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Negative urgency and the dual pathway model of bulimic symptoms: A longitudinal analysis

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Abstract

Objective: This study extends the dual pathway model (DPM) of bulimic symptoms by considering the bidirectional effects amongst symptoms of depression, dietary restraint, and bulimia. We also assessed the influence of negative urgency, a personality construct associated with bulimic symptoms, on the DPM.

Method: Participants were 244 females (M_{age} = 23.77 years) from the general community. Variables pertinent to the DPM as well as negative urgency were assessed at baseline, and symptoms of depression, dietary restraint, and bulimia were reassessed at 1-month follow-up.

Results: Excellent model fit was obtained once modifications were made to the DPM and the extended model that included negative urgency. Cross-sectional paths replicated the DPM as hypothesized, with the exception that time 1 (T1) body mass index failed to predict T1 body dissatisfaction. Although no bidirectional effects were observed, T1 depression predicted dietary restraint at time 2 (T2). Negative urgency was shown to provide incremental predictive utility of T1 pressure to be thin, T1 body dissatisfaction, and T1 and T2 depression.

Conclusion: Findings lend support to the DPM and suggest that depression might be a risk factor for later dietary restraint. The results are also consistent with the notion that negative urgency may be an independent risk factor for symptoms of bulimia. However, short-term longitudinal effects of these putative risk factors require further evaluation.

KEYWORDS

bulimia nervosa, depression, dual pathway model, eating disorders, negative urgency

1 | INTRODUCTION

A number of factors have been identified that confer risk to the development and maintenance of bulimic symptoms, including symptoms of negative mood (e.g., depression, anxiety, and negative affect; Pallister & Waller, 2008; Puccio, Fuller-Tyszkiewicz, Ong, & Krug, 2016), body mass index (BMI), sociocultural pressure to be thin, the internalization of an unrealistic thin ideal, body dissatisfaction (Stice, Gau, Rohde, & Shaw, 2017), and personality traits such as perfectionism, obsessive compulsive disorder traits (Cassin & von Ranson, 2005), and impulsivity (Pearson, Wonderlich, & Smith, 2015; Waxman, 2009). One etiological model that attempts to account for many of these risk factors—and their interrelations—is the dual pathway model of bulimic pathology (DPM; Stice, Nemeroff, & Shaw, 1996).

1.1 | The DPM of bulimic pathology

The DPM (as presented in Figure 1) posits that elevated BMI increases perceived pressure from one's social environment to achieve an ultra-thin body and directly contributes to increased feelings of body dissatisfaction (Stice et al., 1996). Perceived sociocultural pressure to be thin and the internalization of this unrealistic thin ideal also generate the experience of being dissatisfied with one's body, because the thin ideal is largely unattainable (Pidgeon & Harker, 2013). According to the model, increased body dissatisfaction leads to bulimic symptoms via the dual pathways of dietary restraint and negative mood. These mechanisms increase the risk of bulimic symptoms, because binge-eating may compensate for the caloric deprivation associated with restrained eating and also reduce negative mood states. Dietary restraint is also thought to generate negative mood in instances where this pattern of restraint is not upheld and/or via the impact of restricting calories on mood (Stice, 2002). Thus, the DPM proposes specific causal and directional relations amongst the factors implicated in the development of bulimic symptoms. Despite its theoretical appeal in being able to capture the multifaceted aetiology of bulimic symptoms, very few studies have replicated the DPM as proposed by Stice et al. (1996) in its entirety or tested the model longitudinally (Stice, Shaw, & Nemeroff, 1998) to determine its predictive utility.

1.2 | Empirical evidence for the DPM

Support for the DPM has been provided by a number of cross-sectional studies (e.g., Duemm, Adams, & Keating, 2003; Evans, Tovée, Boothroyd, & Drewett, 2013; Girard, Chabrol, & Rodgers, 2018; Hutchinson, Rapee, & Taylor, 2010; Maraldo, Zhou, Dowling, & Vander Wal, 2016; Mason & Lewis, 2015; Ouwens, van Strien, van Leeuwe, & van der Staak, 2009; Ruisoto et al., 2015; Stice et al., 1996; van Strien, Engels, van Leeuwe, & Snoek, 2005; Welsh & King, 2016; Womble et al., 2001), with the model found to account for 9% (van Strien et al., 2005) to 71% (Stice et al., 1996) of the variance in disordered eating symptoms. Possible reasons for such variation in findings may be due to differences in the operationalization of disordered eating symptoms (e.g., overall disordered eating symptoms [Evans et al., 2013] opposed to binge-eating symptoms [van Strien et al., 2005], as well as differences in the variables that have been included/omitted from the models being tested (see below for further discussion).

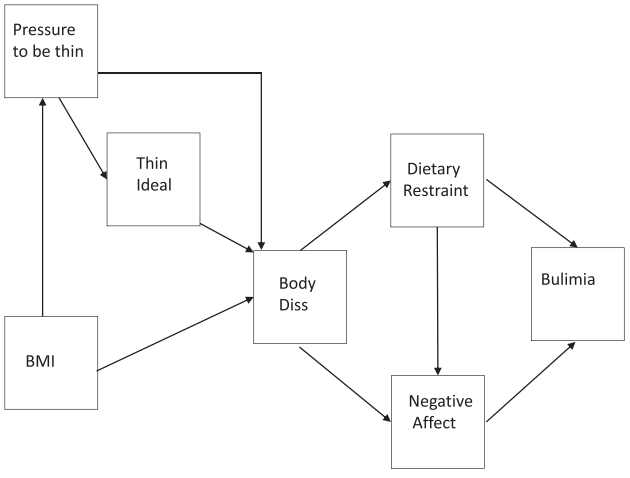

FIGURE 1 Visual conceptualization of the dual pathway model. BMI: body mass index; Thin Ideal: thin ideal internalization; Body Diss: body dissatisfaction; Bulimia: bulimic symptoms

Despite support for the model overall, several specific pathways have been less consistently supported in the literature. For instance, whereas some studies have reported an effect of dietary restraint on bulimic symptoms (Duemm et al., 2003) and overall disordered eating symptoms (Evans et al., 2013), others have failed to find an effect of dietary restraint on bulimic symptoms (Hutchinson et al., 2010; van Strien et al., 2005) or overeating (Ouwens et al., 2009). Moreover, with the exception of one study (Stice et al., 1996), cross-sectional studies have not tested the DPM in its entirety as originally proposed by Stice et al. (1996). Given the interrelatedness of constructs within the DPM, it is possible that bivariate associations that ignore the role of other predictors overestimate these relations in studies that have only assessed some aspects of the DPM. Hence, some pathways may be missing from the models that have been tested and/or may be unnecessary. Modelling the DPM in its entirety would provide surer footing to address these possible limitations.

In addition to the cross-sectional studies above, six longitudinal studies have assessed the model (Allen, Byrne, & McLean, 2012; Dakanalis et al., 2014; Puccio, Kalathas, Fuller-Tyszkiewicz, & Krug, 2016; Salafia & Gondoli, 2011; Sehm & Warschburger, 2017; Stice et al., 1998). These longitudinal studies provided support for the majority of the paths proposed by the DPM; for example, all studies reported a significant effect of dietary restraint on the respective measure of disorder eating symptoms that were utilized. The only study (Stice et al., 1998) that assessed the overall model reported that all hypothesized paths were positive and significant. Stice et al. (1998) reported that their model accounted for 33% of the variance in bulimic symptoms, and hence, the model identified specific factors that might be targeted for interventions designed to prevent the development of bulimic symptoms.

Despite these findings, this group of longitudinal studies omitted the following variables: pressure to be

thin (Allen et al., 2012; Dakanalis et al., 2014; Sehm & Warschburger, 2017) and thin ideal internalization (e.g., Allen et al., 2012; Dakanalis et al., 2014; Salafia & Gondoli, 2011; Sehm & Warschburger, 2017). Further, in two of the studies (Dakanalis et al., 2014; Puccio, Kalathas, et al., 2016), the variable BMI was utilized as a covariate (as opposed to a predictor), such that all variables in the model were regressed onto BMI, hence confounding the relative influence of BMI on the model. Accordingly, further research that examines the entire model as proposed by Stice et al. (1996) is required to determine whether the model is able to be replicated longitudinally.

1.3 | Bidirectional linkages between constructs

Recent research into the links between depression and eating pathology indicate that these constructs might be bidirectionally related, and thus, are risk factors for each other (Puccio, Fuller-Tyszkiewicz, et al., 2016). This raises the possibility that the DPM might be better conceptualized as having bidirectional feedback loops between specific paths (e.g., between the depression-bulimic symptoms). In line with this view, two studies (Bradford & Petrie, 2008; Sehm & Warschburger, 2017) have tested for longitudinal bidirectional associations between constructs in the DPM. Bradford and Petrie (2008) tested for bidirectional links between internalization of the thin ideal and body dissatisfaction, depressive affect and body dissatisfaction, and depressive affect and disordered eating over a 6-month time period. They found that internalization of the thin ideal and body dissatisfaction were bidirectionally related, as were depressive affect and disordered eating. By contrast, Sehm and Warschburger (2017) found no evidence to support bidirectional relationships over a 20-month time lag between body dissatisfaction, restrained eating, negative affect, and binge-eating symptoms. Indeed, unlike other studies into the DPM (Dakanalis et al., 2014), Sehm and Warschburger (2017) found no evidence to support any paths that predicted binge-eating symptoms. Sehm and Warschburger (2017) speculated that past longitudinal studies might have found significant effects for paths that predicted their disordered eating construct because such studies assessed shorter (e.g., a time lag of less than approximately 2 years), opposed to longer, time lags. These two studies provide an important starting point in assessing whether any linkages in the DPM might be better operationalized as bidirectional. As neither of these studies examined the DPM in its entirety, and because the depression-eating pathology relationship might be bidirectional, future research is needed to test a revised version of the DPM that includes feedback loops between the depression-bulimic symptoms pathways.

1.4 | Negative urgency and the dual pathway model

Impulsivity is a personality factor that is considered etiological to the development of eating pathology, especially binge-purging symptomatology (Pearson et al., 2015; Stice, 2002; Waxman, 2009). Impulsivity is conceptualized as a multidimensional construct comprising four facets: negative urgency, lack of premeditation, lack of perseverance, and sensation seeking (Whiteside & Lynam, 2001). Of these facets, negative urgency (i.e., the disposition to act in rash or regrettable ways as the result of intense negative affect; Whiteside & Lynam, 2001) has been shown to be a transdiagnostic risk factor for the development of bulimic symptoms (Fischer, Smith, & Cyders, 2008; Pearson et al., 2015) and depressive symptoms (Smith, Guller, & Zapolski, 2013). Negative urgency likely leads to bulimic symptoms by limiting individuals' focus on consequences. Within the context of the DPM the link between binge-eating symptoms and negative urgency may also arise via the negative mood pathway, as negative urgency, depression and binge-eating symptoms might be generated by the motivation to reduce or avoid negative internal states (Heatherton & Baumeister, 1991; Quigley, Wen, & Dobson, 2017; Whiteside & Lynam, 2001).

To date, trait impulsivity and negative urgency have received little attention in relation to the DPM. Cross-sectional studies have examined the influence of trait impulsivity (Mason & Lewis, 2015) and negative urgency (Racine & Martin, 2016; Wenzel, Weinstock, Vander Wal, & Weaver, 2014) on paths within the DPM. Wenzel et al. (2014) found that negative urgency was related to bulimic symptoms after controlling for BMI, depression, and dietary restraint. This study did not, however, assess the direct influence of negative urgency on other variables in the model. Thus, it remains unclear how negative urgency might affect these other variables. Mason and Lewis (2015) reported that trait impulsivity did not lead to binge-eating symptoms, but that it did exert an effect on body shame, food-related cognitions, and negative affect. Finally, Racine and Martin (2016) found that interactions amongst negative urgency and other variables implicated in the DPM, including pressure to be thin, thin ideal internalization, body dissatisfaction, and dietary restraint, were significantly related to bulimic symptoms. By contrast, the researchers found that these interactions did not link to other types of psychopathology including depressive symptoms or problematic alcohol use. Racine and Martin

(2016) speculated that disordered eating-specific risk factors likely influence individuals with negative urgency to experience disordered eating as opposed to other forms of psychopathology. Hence, there is evidence to suggest that negative urgency might be an important addition to the DPM.

1.5 | Limitations of past literature

In light of the above, important questions remain unanswered in the literature. Only two (Puccio, Kalathas, et al., 2016; Stice et al., 1998) of the six longitudinal studies that have assessed the DPM utilized a 6-month or less time lag between points of assessment. It is possible that the effects between paths might be better assessed across shorter time lags (e.g., Bradford & Petrie, 2008), and thus, further research is required to test this idea. Second, depression and eating pathology have been shown to be bidirectionally related (Puccio, Fuller-Tyszkiewicz, et al., 2016); however, no studies have assessed the potential longitudinal bidirectional effects amongst symptoms of depression and disordered eating in the context of the entire DPM. Prior studies have typically been unable to control for other variables known to predict depression and disordered eating (i.e., body dissatisfaction) because such variables have not been included in bivariate associations (Puccio, Fuller-Tyszkiewicz, et al., 2016). The DPM provides candidates for possible shared risk factors between depression and disordered eating, and hence, enables one to test the possibility that the depression–disordered eating relationship might be reciprocal whilst controlling for these shared risk factors.

1.6 | The current study

This study aimed to assess two models. Model 1 assessed the entire DPM longitudinally and sought to examine whether feedback loops exist amongst symptoms of depression and disordered eating (dietary restraint and bulimia) by measuring these symptoms at baseline and then again at 1-month follow-up. Model 2 examined the personality trait negative urgency as an exploratory predictor of all other variables to determine if negative urgency added predictive power to the DPM. We opted to operationalize negative affect using the construct of depression because there seems to be strong evidence for a bidirectional relationship between depression and bulimic symptoms (Puccio, Fuller-Tyszkiewicz, et al., 2016); however, to our knowledge, no such meta-analysis exists for the potential bidirectional relationship between "negative affect" and bulimic symptoms.

Further, we operationalized "negative affect" using a measure of depressive symptoms because different facets of "negative affect" have been shown to relate differentially to disordered eating variables (Puccio, Fuller-Tyszkiewicz, et al., 2017). Hence, because the mechanisms which link different facets of negative affect to bulimic symptoms might differ as a function of negative mood type, grouping together different facets of "negative affect" into a single variable might confound the relationships being tested, and thus, might limit the interpretation of results.

It was hypothesized that:

1 Model 1: All paths for the variables included in the DPM would be positive and significant, and the pathways between symptoms of depression, bulimia, and dietary restraint at both time 1 (T1) and time 2 (T2) would be significant and bidirectional (see Figure 2).
2 Model 2: Negative urgency would positively and significantly predict all variables in the DPM.

2 | METHOD

2.1 | Participants

As part of a larger study (Puccio. Kathalas, et al., 2016), participants were a convenience sample of 245 females aged 18 to 54 years of age (M_{age} = 23.90 years, SD = 7.19). The decision to include only females in this study was made because eating disturbances, including bulimia nervosa and dietary restraint, primarily affect women (Hoek, 2006; Streigel-Moore & Bulik, 2007; Wittchen & Jacobi, 2005), and there is also a paucity of research on males regarding the variables of interest. Thus, we aimed to test the model in a female sample before seeing whether the model can generalize to a male sample. Of this sample, 127 participants (M_{age} = 19.37 years, SD = 2.49) were undergraduate students from a Melbourne university, 58 (M_{age} = 27.78 years, SD = 0.80) were recruited from the community in Australia, and 60 (M_{age} = 29.13 years, SD = 5.75) were recruited from the United States via the online labour system Clickworker. In exchange for participation in the study, the university students received course credit, the Australian community sample were entered into a draw to win an iPad mini, and the Clickworker participants were reimbursed €8 (equivalent to approximately AUD $12). Eligibility criteria for the study were that participants were female and over the age of 18 years. All participants provided informed consent prior to completing the study. Ethics approval was obtained from the lead author's university ethics board.

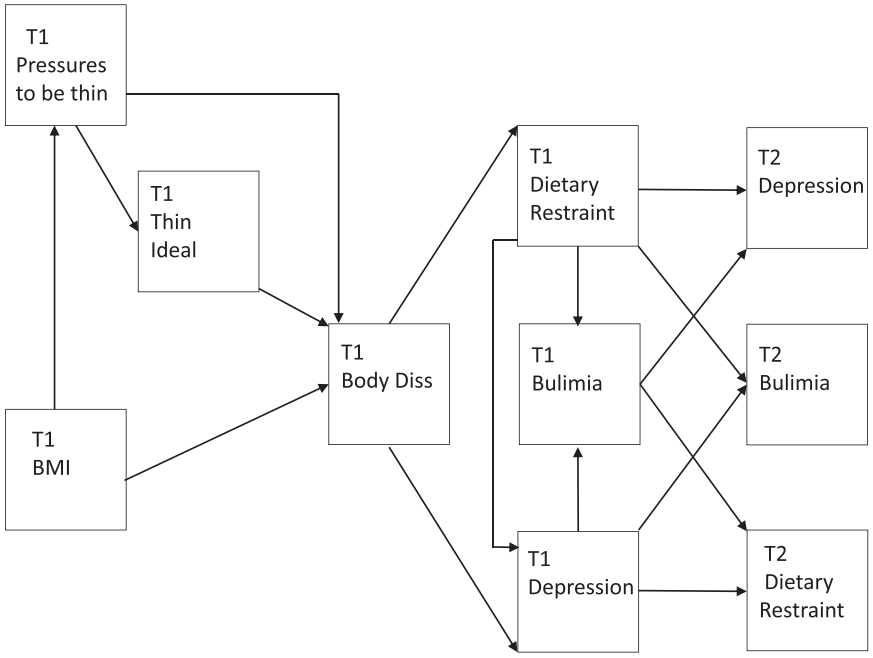

FIGURE 2 Visual conceptualization of the dual pathway model including potential bidirectional effects amongst depression, bulimic symptoms, and dietary restraint. BMI: body mass index; Thin Ideal: thin ideal internalization; Body Diss: body dissatisfaction; Bulimia: bulimic symptoms; Depression: depressive symptoms; T1: time 1; T2: time 2

2.2 | Materials

2.2.1 | Demographics

Participants provided information on their age, ethnicity, country of birth, first language, education, employment status, marital status, and current height and weight. Self-reported weight and height were used to calculate BMI.

2.2.2 | Thin ideal internalization

Thin ideal internalization was assessed at T1 using the Ideal Body Stereotype Scale-Revised (Stice, Fisher, & Martinez, 2004). The scale consists of six items that assess the acceptance of socially sanctioned standards of thinness. Items were rated using a 5-point Likert scale ranging from 1 (*strongly disagree*) to 5 (*strongly agree*) from which an average total score was calculated. The reliability coefficient for thin ideal internalization indicated good internal reliability at T1 (Cronbach's $\alpha = 0.78$).

2.2.3 | Pressure to be thin

Perceived pressure from the media, family, and friends to have a thin body was assessed at T1 using the Perceived Socio-cultural Pressure Scale (Stice et al., 1996). Participants rated 10 items using a 5-point rating scale ranging from 1 *(none*) to 5 (*a lot*) from which an average total score was calculated. Internal consistency for the scale was high at T1 (Cronbach's $\alpha = 0.86$).

2.2.4 | Body dissatisfaction

Body dissatisfaction was assessed at T1 using the Body Parts Satisfaction Scale-Revised (Petrie, Tripp, & Harvey, 2002). The measure consists of 15 items addressing an individual's satisfaction with various body parts, muscle tone, and overall body satisfaction. Participants rated each item using a 6-point rating scale ranging from 1 (*extremely dissatisfied*) to 6 (*extremely satisfied*) from which an average total score was calculated and then reverse scored to provide an index of body dissatisfaction. The internal consistency of this scale at T1 was high (Cronbach's $\alpha = 0.88$).

2.2.5 | Negative urgency

Negative urgency was assessed at T1 using the negative urgency subscale from the Urgency, Premeditation, Perseverance, and Sensation Seeking Scale (Whiteside & Lynam, 2001). The 12-item negative urgency subscale measures the tendency to experience strong impulses, frequently under conditions of negative affect. Items were rated using a 4-point rating scale ranging from 1 (*strongly disagree*) to 4 (*strongly agree*) from which an average total score was calculated. The internal consistency of the negative urgency scale at T1 was high (Cronbach's $\alpha = 0.90$).

2.2.6 | Depressive symptoms

Depressive symptoms were assessed at T1 and T2 using the 10-item short form of the Centre for Epidemiologic Studies Depression Scale (CES-D; Radloff, 1977).

Participants rated each item in terms of the frequency that each mood or event occurred during the past week on a 4-point rating scale ranging from 0 (*rarely or none of the time, less than one day*) to 3 (*all of the time, five-seven days*), from which an average total score was calculated. The internal consistency of the CES-D was good at T1 (Cronbach's $\alpha = 0.75$) and at T2 (Cronbach's $\alpha = 0.88$).

2.2.7 | Dietary restraint

Dietary restraint was assessed at T1 and 1-month follow-up assessment (T2) using the dieting subscale from the Eating Attitudes Test-26 (EAT-26; Garner, Olmsted, Bohr, & Garfinkel, 1982). The subscale consists of 13 items from which an average total score was calculated. Using a 6-point rating scale ranging from 1 (*never)* to 6 (*always*), participants rated the extent to which 13 dieting statements (e.g., *I am aware of the calorie content of the food that I eat*) described their behavior. Higher scores indicated greater levels of dietary restraint. The internal consistency of this subscale was good at T1 (Cronbach's $\alpha = 0.82$) and good at T2 (Cronbach's $\alpha = 0.85$).

2.2.8 | Bulimic symptoms

Bulimic symptoms were assessed at T1 and T2 using the bulimia and food preoccupation subscale from the EAT-26 (Garner et al., 1982). The bulimia and food preoccupation subscale consists of six items that measure participants' bulimic behaviors such as bingeing and purging (e.g., *I have gone on binge eating episodes where I feel I may not be able to stop*). Items were rated on a 6-point rating scale from 1 (*never*) to 6 (*always*) from which an average total score was calculated. Higher scores indicated greater levels of bulimic symptoms. Internal consistencies for bulimic symptoms were low at T1 (Cronbach's $\alpha = 0.58$) and good at T2 (Cronbach's $\alpha = 0.79$).

2.3 | Procedure

At T1, a questionnaire assessing participant demographics, thin ideal internalization, pressure to be thin, body dissatisfaction, negative urgency, depressive symptoms, dietary restraint, and bulimic symptoms was administered online to participants via the software programme Qualtrics (Qualtrics, 2015). Approximately 1 month later (T2), participants completed a follow-up questionnaire, also administered online via Qualtrics, which assessed depressive symptoms, dietary restraint, and bulimic symptoms.

2.4 | Statistical analyses

All analyses were performed using SPSS version 23 and Mplus version 8 (Muthén, Muthén, & Asparouhov, 2017). Path analysis was used to test all specified models. Age was included as a covariate for all variables, given prior evidence of age-related differences in body image and disordered eating (Allen et al., 2013; Bucchianeri, Arikian, Hannan, Eisenberg, & Neumark-Sztainer, 2013). T2 variables (depression, dietary restraint, and bulimic symptoms) were regressed onto DPM-implied predictors as well as T1 versions of themselves (e.g., T1 depression predicting T2 depression) to facilitate prediction of change in these dependent variables over time. Modifications were completed if theoretically justifiable improvements to model fit could be obtained (Kline, 2015).

Adequacy of model fit was assessed using chi-square, the comparative fit index (CFI), and the root mean square error of approximation (RMSEA). The following cut-offs for standard fit indices were used: non-significant chi-square for excellent fit, CFI > 0.95 for excellent fit, and >0.90 for adequate fit; RMSEA < 0.06 for good fit, and >0.10 for poor fit (Marsh, Hau, & Wen, 2004). Significant standardized beta weights indicated whether the hypothesized paths in the models were supported. R-squared estimates are reported for variables that are predicted by others in the model.

The absolute skew value for T2 bulimic symptoms exceeded the value of 2, suggesting that the assumption for univariate normality was not satisfied (Kline, 2015). Positive skew for these variables was to be expected given the non-clinical sample (Tabachnick & Fidell, 2007). To address the issue of skewed variables, we used the MLR estimator in Mplus, which is a maximum likelihood estimator with robust standard errors to account for non-normal data (Muthén et al., 2017).

Finally, Bayesian Information Criterion (BIC; Schwarz, 1978) values were used to compare model fit for the models with and without negative urgency included. The model with lower BIC values is deemed a better fit. Following Raftery's (1995) guidelines, BIC differences between models of 0–2 indicate weak support, BIC differences of 2–4 indicate positive support, >6 reflect strong support, and >10 indicate very strong support for the model with the smaller BIC value.

3 | RESULTS

3.1 | Descriptive analyses

Table 1 presents summary statistics for demographic variables for the overall sample and the three participant subgroups (university students, Australian community

TABLE 1 Sociodemographics and results from chi-square difference tests on participant sociodemographics as a function of sample type

	Frequency	Percent (%)	UNI	USA	AU	χ^2	*df*	*p*
First language is English						10.110	2	0.006
Yes	215	88.10	104	55	56			
No	29	11.90	23	4	2			
Ethnic background						69.565	12	<0.001
African	11	4.50	0	11	0			
Caucasian or White	140	57.60	53	43	46			
Eastern Asian	21	8.60	18	1	2			
Southern Asian	31	12.70	26	1	4			
Hispanic or Latin American	4	1.60	3	1	0			
Middle Eastern	6	2.50	5	0	1			
Other	26	12.50	21	3	5			
Highest education						123.958	12	<0.001
Primary school	1	0.40	0	1	0			
Some secondary education	1	0.40	0	1	0			
Completed year 12	111	45.50	90	11	10			
Some tertiary education	45	18.44	25	16	4			
University degree or equivalent	86	35.24	12	30	44			
Employment status						216.625	10	<0.001
Full-time	44	18.10	0	20	24			
Part time	15	6.10	0	11	4			
Casual	11	4.50	0	3	8			
Unemployed	15	6.10	0	14	1			
Student	150	61.50	127	3	20			
Other	9	3.70	0	8	1			
Marital status								
Single, never married	172	70.50	118	22	31	95.669	12	<0.001
Married without children	11	4.50	1	7	3			
Married with children	23	9.43	0	17	6			
Divorced or separated	7	2.87	0	5	1			
Living with partner	31	12.70	7	8	16			

Note. AU: Australian community sample; UNI: Melbourne University student sample; USA: American community sample; χ^2: chi-square value; *df*: degrees of freedom; *p*: probability value.

sample, and the U.S. community sample). Approximately half of the participants were Caucasian, had completed some tertiary education, and were single. The majority of participants were Australian, employed full-time, and spoke English as their first language.

Table 2 presents descriptive statistics for all key variables of interest for the total sample and by participant subgroup. Chi-square difference tests (see Table 1) and one-way analyses of variance and post hoc Scheffe tests for continuous data (see Table 2) showed significant mean differences amongst the three subgroups on all sociodemographic variables and on a number of variables implicated in the DPM. Two categorical dummy variables were created to control for these differences. Each dummy variable was used as a covariate in the models tested such that all DPM variables were regressed onto these dummy variables. Results showed that 20.10% and 12.30% of participants scored at/or above the cut-score of 20 for the EAT-26 at T1 and T2, respectively, and that 17.20% and 17.60% of participants scored at/or above the cut-score of 16 for the CES-D at T1 and T2 respectively.

Paired sample *t* tests were utilized to examine if symptoms of depression and disordered eating changed significantly from T1 to T2. Results showed no significant

TABLE 2 Descriptive statistics and results from one-way analyses of variance and post hoc Scheffe tests as a function of sample type of variables utilized in the study

Variable	Range	*M*	*SD*	Skew	Kurtosis	UNI *n* = 127	USA *n* = 59	AU *n* = 58	*F*	*df*	*p*	Comparison group	Post hoc *p* value
Age (years)	18–54	23.90	7.19	-	-	19.71 (3.81)	29.15 (5.64)	27.59 (8.59)	70.19	2	<0.001	UNI vs. USA UNI vs. AU USA vs. AU	<0.001 <0.001 <0.001
Body mass index	14.27–62.14	24.34	7.66	-	-	21.89 (5.05)	27.11 (5.43)	21.34 (4.33)	24.48	2	<0.001	UNI vs. USA UNI vs. AU USA vs. AU	<0.001 0.715 <0.001
Negative urgency	1–4	2.38	0.61	0.08	−0.45	2.42 (.58)	2.34 (.66)	2.33 (.41)	0.56	2	0.574	-	-
T1 Pressures to be thin	1–6	2.90	1.11	0.75	−0.06	3.16 (1.16)	2.24 (.51)	2.98 (1.20)	17.96	2	<0.001	UNI vs. USA UNI vs. AU USA vs. AU	<0.001 0.486 0.001
T1 Thin ideal internalization	1–5	3.41	0.79	−0.69	0.52	3.67 (.65)	2.68 (.71)	3.51 (.68)	47.13	2	<0.001	UNI vs. USA UNI vs. AU USA vs. AU	<0.001 0.232 <0.001
T1 Body dissatisfaction	1.07–4.80	2.91	0.64	0.26	0.29	2.98 (.57)	2.93 (.75)	2.74 (.62)	3.01	2	0.051	-	-
T1 Depression	0–3.5	1.04	0.62	0.87	0.59	0.98 (.56)	1.46 (.65)	.73 (.49)	25.70	2	<0.001	UNI vs. USA UNI vs. AU	<0.001 0.023
												USA vs. AU	<0.001
T2 Depression	0–2.80	1.02	0.63	0.63	−0.30	1.09 (.63)	1.13 (.66)	0.76 (.51)	7.17	2	0.001	UNI vs. USA UNI vs. AU USA vs. AU	0.923 0.003 0.005
T1 Dietary restraint	0–2.38	0.57	0.58	1.46	2.50	0.58 (.59)	0.68 (.59)	0.44 (.52)	2.367	2	0.096	-	-
T2 Dietary restraint	0–2.23	0.46	0.50	1.52	1.79	0.52 (.54)	0.42 (.44)	0.38 (.45)	1.658	2	0.193	-	-
T1 Bulimic symptoms	1–2.50	0.49	0.61	1.26	0.70	0.39 (.57)	0.92 (.64)	0.29 (.41)	23.613	2	<0.001	UNI vs. USA UNI vs. AU USA vs. AU	<0.001 0.578 <0.001
T2 Bulimic symptoms	1–2.50	0.27	0.46	2.20	5.18	0.34 (.51)	0.21 (.43)	0.19 (.37)	2.616	2	0.075	-	-

Note. T1: time 1; T2: time 2; *N*: sample size; *M*: mean; *SD*: standard deviation; UNI: Melbourne University student sample; USA: American community sample; AU: Australian community sample; *df*: degrees of freedom; *F*: *F* value from one-way analysis of variance; *p*: probability value; ^: mean (standard deviation) of total scale-score values; post hoc *p* value: *p* value derived from Scheffe test; −: not relevant.

difference between the mean level of T1 and T2 symptoms of depression: $t(1) = 0.175$, $p = 0.676$, Cohen's $d = 0.033$. By contrast, the mean level of T2 dietary restraint was significantly lower than at T1: $t(1) = 20.514$, $p < 0.001$, Cohen's $d = 0.288$, and the mean level of bulimic symptoms at T2 was significantly lower than at T1: $t(1) = 42.535$, $p < 0.001$, Cohen's $d = 0.440$.

3.2 | Path analyses

3.2.1 | Model 1: The DPM with bidirectional links between symptoms of bulimia, depression, and dietary restraint

Results indicated that the model was a good fit for the data, $\chi^2(27) = 31.039$, $p = 0.269$, CFI = 0.996, RMSEA = 0.025. A specification search was undertaken to improve the fit of the model. Based on this specification search, the path from T1 body dissatisfaction ➔ T2 depression was added. This path (presented in Figure 3) was chosen based on previous findings that have observed a direct relationship between these variables (e.g., Salafia & Gondoli, 2011). Proposed effects of the covariate age on T1 and T2 dietary restraint, T1 depression, and T2 bulimic symptoms were found to be non-significant; hence, these non-significant paths were trimmed from the model. Together, these revisions to the proposed model (both model trimming and addition) resulted in an excellent fit of the data, $\chi^2(30) = 27.505$, $p = 0.5967$, CFI = 1.000, RMSEA = 0.000, BIC = 6,358.544.

All cross-sectional paths of the DPM as proposed by Stice et al. (1996) were significant, with the exception of the path from T1 BMI ➔ T1 body dissatisfaction. T1 depression was significantly predicted by T1 body dissatisfaction ($b = 0.190$, $p = 0.004$) and T1 dietary restraint ($b = 0.250$, $p < 0.001$), which in combination explained 33% of the variance in T1 depression. T1 dietary restraint was significantly predicted by T1 body dissatisfaction ($b = 0.507$, $p < 0.001$), explaining 28% of the variance in T1 dietary restraint. T1 bulimic symptoms was significantly predicted by T1 depression ($b = 0.168$, $p = 0.001$) and T1 dietary restraint ($b = 0.608$, $p < 0.001$) which together explained 59% of the variance in T1 bulimic symptoms. Regarding T2 symptoms, T2 depression was significantly predicted by T1 body dissatisfaction ($b = 0.343$, $p < 0.001$) and T2 dietary restraint ($b = 0.229$, $p = 0.001$), which in combination explained 43% of the variance in T2 depression. T2 dietary restraint was significantly predicted by T1 depression ($b = 0.123$, $p = 0.019$), which explained 58% of the variance in T2 dietary restraint. Finally, T2 bulimic symptoms was significantly predicted by T2 depression ($b = 0.136$, $p = 0.025$) and T2 dietary restraint ($b = 0.357$, $p < 0.001$), which in combination explained 48% of the variance in T2 bulimic symptoms.

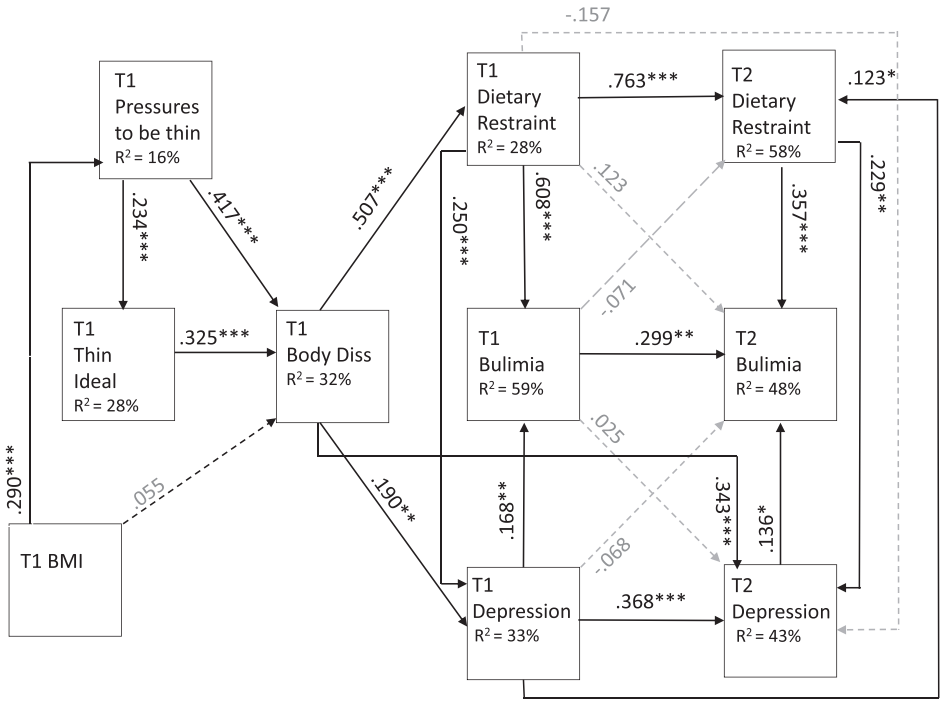

FIGURE 3 Standardized beta weights from the path analysis of Model 1. BMI: body mass index; Thin Ideal: thin ideal internalization; Body Diss: body dissatisfaction; Bulimia: bulimic symptoms; Depression: depressive symptoms; T1: time 1; T2: time 2; $^{*}p < 0.05$; $^{**}p < 0.01$; $^{***}p < 0.001$. Bold lines denote significant paths and broken lines denote non-significant paths

3.2.2 | Model 2: A revised DPM to include negative urgency

Building upon Model 1, negative urgency was included as a predictor of each variable in Model 2. This model was an excellent fit for the data, $\chi^2(32) = 28.483$, $p = 0.645$, CFI = 1.000, RMSEA = 0.000, BIC = 6,581.520. Results showed that negative urgency exerted a significant effect on all variables in the model with the exception of the following: T1 negative urgency ➔ T1 thin ideal internalization, T1 bulimic symptoms, T1 dietary restraint, and T2 dietary restraint. A new model (see Figure 4) was therefore specified that trimmed these non-significant paths. These revisions resulted in an excellent fit of the data, $\chi^2(37) = 34.550$, $p = 0.585$, CFI = 1.000, RMSEA = 0.000, BIC = 6,287.050.

With negative urgency included as a predictor, the following paths, which were significant in Model 1, reduced to non-significance: T1 body dissatisfaction ➔ T1 depression, and T2 depression ➔ T2 bulimic symptoms. Inclusion of negative urgency to the model increased the R-squared value in the relevant dependent variables by 1–11%, which is small yet non-trivial (Cohen, 1992). However, the inclusion of negative urgency to the model resulted in no change in the R-squared value to T1 bulimic symptoms. Comparison of BIC values for the model including negative urgency (BIC = 6287.050) versus not including negative urgency (BIC = 6358.544) showed very strong evidence that inclusion of the negative urgency variable provided better model fit.

4 | DISCUSSION

This study assessed the DPM as proposed by Stice et al. (1996) and included a longitudinal extension of the model that examined possible bidirectional effects amongst symptoms of depression, dietary restraint, and bulimia over a 1-month follow-up. This study also investigated whether the inclusion of negative urgency increased the predictive utility of the DPM beyond the current factors within the model. Results from Model 1 showed that all hypothesized cross-sectional paths of the DPM were significant and positive, with the exception of the path T1 BMI ➔ T1 body dissatisfaction. Indeed, analyses indicated that with a few suggestions for refinement, the model as proposed by Stice et al. (1996) was well replicated. The cross-sectional results of our study lend support to the proposition that pressure to be thin and thin ideal internalization are associated with body dissatisfaction and that a dual-pathway process links body dissatisfaction to bulimic symptoms via negative mood and restrained eating. However, results showed no evidence for bidirectional effects amongst symptoms of depression and disordered eating over a 1-month time interval, but it was found that T1 depression predicted T2 dietary

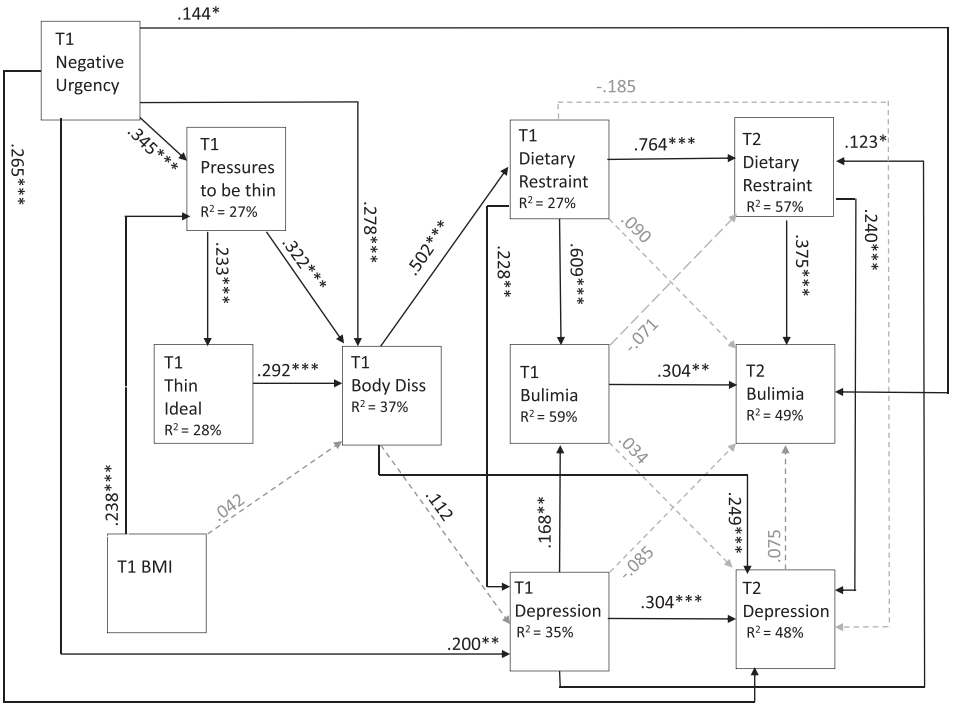

FIGURE 4 Standardized beta weights from the path analysis of Model 2. BMI: body mass index; Thin Ideal: thin ideal internalization; Body Diss: body dissatisfaction; Bulimia: bulimic symptoms; Depression: depressive symptoms; T1: time 1; T2: time 2; $^{*}p < 0.05$; $^{**}p < 0.01$; $^{***}p < 0.001$. Bold lines denote significant paths and broken lines denote non-significant paths

restraint. Results from Model 2 showed that the inclusion of negative urgency improved the explanatory power of the model.

4.1 | Model 1: Replication of the DPM

Model 1 provided support for the DPM, with several minor modifications. Contrary to expectations, T1 BMI was not associated with T1 body dissatisfaction. BMI is thought to lead to body dissatisfaction because cultural norms to achieve and maintain a thin body shape require individuals to have an unrealistically low BMI (e.g., Stice, 2002), and thus, females with an "elevated" BMI are thought to feel dissatisfied with their body shape and size, because in such cases, individuals fall short of the culturally defined thin ideal. This null effect of T1 BMI on T1 body dissatisfaction is consistent with one study (Allen et al., 2012) but inconsistent with other studies which reported a significant positive effect (Evans et al., 2013; Mason & Lewis, 2015; Salafia & Gondoli, 2011; Stice et al., 1996; Stice et al., 1998). It is currently unclear why the present study found that T1 BMI was not associated with T1 body dissatisfaction. This is particularly surprising given that BMI was significantly and positively associated with other body image constructs as predicted, notably pressures to be thin. The mean level of BMI in this study was somewhat higher than the mean level reported in past studies (Stice et al., 1996), and thus, this difference might go some way towards explaining the null effect of T1 BMI on T1 body dissatisfaction. Yet, according to the DPM, it would be expected that higher levels of BMI would exert a stronger effect on body dissatisfaction, and thus, it remains unclear why BMI showed no relationship to body dissatisfaction. To date, only a paucity of studies (Allen et al., 2012; Evans et al., 2013; Mason & Lewis, 2015; Salafia et al., 2010; Stice et al., 1998; Stice et al., 1996) have examined the influence of BMI on specific paths within the DPM. Therefore, it is difficult to draw firm conclusions regarding the influence of BMI to the DPM, and thus, future studies that examine the effect of this variable on the model are required.

Results from Model 1 suggest that the variable body dissatisfaction may further add to prediction within the model. T1 body dissatisfaction was shown to predict T2 depression, a finding that is consistent with past research (Paxton, Neumark-Sztainer, Hannan, & Eisenberg, 2006). Researchers (e.g., Paxton et al., 2006; Stice & Bearman, 2001) have contended that puberty, and the associated changes to females' body weight, shape, and size, increases the discrepancy between one's actual body shape and the thin ideal. They have argued that appearance is a critical evaluative factor for females in Western culture, and therefore, greater levels of body dissatisfaction cause increased feelings of negative affect. Results from Model 1 support this proposition and highlight the importance of including this variable in the DPM.

4.2 | Evidence for bidirectional links amongst symptoms of depression and disordered eating

The prediction that symptoms of depression assessed at T1 and T2 would be bidirectionally related was not supported in Model 1. However, results showed that T1 depression predicted T2 dietary restraint. The finding is inconsistent with a past meta-analysis (e.g., Puccio, Fuller-Tyszkiewicz, et al., 2016), which showed that across 30 studies, depression and disordered eating were bidirectionally related. However, this meta-analysis included studies of varying time intervals (ranging from 7 days to 7 years) and only one study (Mackinnon et al., 2011) that utilized a time interval of 1 month or less. In their study, Mackinnon et al. (2011) found that binge-eating symptoms at T1 predicted T2 depression at 1-week follow-up and that T2 depression predicted T3 binge-eating symptoms at an additional 1-week follow-up.

The paucity and inconsistency of findings at these shorter time intervals warrants further research attention to determine whether meaningful change occurs across short time horizons. The breadth of null findings from the present study may indicate that the proposed relationships do not influence each other reciprocally over these shorter time periods. This seems to be supported further by the small magnitude of change in several of the modelled variables in the present study (Cohen's d range = 0.033 to 0.440), suggesting that change processes involving these variables may take longer than 1-month to materialize. Even so, the finding that at least some paths may exert longitudinal effects over a 1-month interval and that variables such as dietary restraint and bulimic symptoms changed across this timeline suggests an alternative possibility; namely, that the rate of change in these constructs differs and that some processes for some of the variables in the model occur over much shorter time intervals than others. Further research with multiple and varied follow-up time-points may help to clarify the role of time in observing hypothesized effects of body image on disordered eating, as per the DPM.

Another explanation for this non-significant bidirectional finding is that the effects amongst symptoms of depression, dietary restraint, and bulimia might be stronger in one direction than another, thus accounting for the non-significant bidirectional links observed but equally explaining why T1 depression was found to predict T2

dietary restraint. If the hypothesis that the symptoms in question are bidirectionally related is true, then our data suggest that the effects of depression on dietary restraint might materialize first and that any possible reciprocal effects amongst symptoms of depression, dietary restraint, and bulimia might take greater time to develop. Thus, these effects might not be constant, but rather, the transmission of effects might occur at a specific stage in the progression of the illness. For example, it is possible that the effects of depression on dietary restraint is exacerbated during symptom onset of depression and that feedback loops amongst all three constructs might become activated once symptom levels have reached a threshold. It is also possible that respective symptoms have limited effect on each other across time once symptoms are well developed. Results indicated that approximately 20% and 12% of the sample at T1 and T2, respectively, were at, or above, the total cut-score of 20 for the EAT-26, our measure of disordered eating. This suggests that a majority of participants were drawn from a largely non-clinical population, and accordingly, our results possibly indicate that such feedback loops might not hold in non-clinical samples. Thus, although our findings do not support the view that a bidirectional relationship exists amongst these symptoms in question, confounding factors described above (e.g., differences in time intervals for assessing symptoms) indicate that further research is required to test this proposition in clinical and non-clinical samples.

4.3 | Model 2: The DPM and negative urgency

Results showed that, relative to Model 1, Model 2 which included negative urgency (Figure 4) was a better fit for the data and that the variable added small but non-trivial predictive power to the model (R-squared rage = 1–11%). Findings illustrated that negative urgency was positively and significantly associated with T1 pressure to be thin, T1 body dissatisfaction, and T1 depression. Negative urgency also positively and significantly predicted T2 depression and T2 bulimic symptoms. The finding that T1 negative urgency was associated with T1 pressure to be thin is consistent with one past study (Racine & Martin, 2016) that reported a significant correlation between the two constructs. It is possible that individuals who are less likely to focus on consequences are also less able to adhere to the thin ideal, which in turn, likely elevates such individuals' BMI, and hence, increases susceptibility to internalize pressures to be thin.

The finding that negative urgency was linked to bulimic symptoms is consistent with one DPM study (Wenzel et al., 2014) but inconsistent with another (Mason & Lewis, 2015) that showed a non-significant effect of trait impulsivity on binge-eating symptoms. Mason and Lewis (2015) operationalized impulsivity by assessing it as a higher order trait, whereas both this study and Wenzel et al. (2014) assessed impulsivity by examining the lower order facet of negative urgency. This difference in result likely reflects the idea that impulsivity is a multifactored construct, and thus, one aspect of the construct—negative urgency—is perhaps more strongly linked to bulimic symptoms versus other aspects of the construct, such as sensation seeking or a lack of premeditation. Indeed, our results lend support to the belief that negative urgency directly generates symptoms of bulimia (Pearson et al., 2015), arguably because both constructs are mechanisms that are utilized to reduce, avoid, and/or escape negative internal states. Results also showed a significant effect of negative urgency on depression. This is consistent with one previous study that showed an effect of trait impulsivity on depression in the context of the DPM (Mason & Lewis, 2015). Our finding is not surprising given that negative urgency reflects a tendency to avoidance and/or escape negative states and that depression is associated with similar "avoidant/escape" mechanisms (Quigley et al., 2017). The current results therefore support the idea that negative urgency is a transdiagnostic risk factor for both bulimic symptoms (Pearson et al., 2015) and depression (Smith et al., 2013) and that its inclusion to the DPM is warranted.

It is reasonable to speculate that negative urgency generates bulimic symptoms as the result of a dysfunctional belief whereby one perceives a lack of volitional control over one's thoughts/behaviors whilst distressed. Individuals with this low locus of self-control might experience greater inability to achieve and/or maintain the thin ideal (i.e., fail to adhere to strict dietary restriction regimes due to engaging in binge-eating episodes), which in turn, might generate body dissatisfaction and reinforce avoidant/escape mechanisms that perpetuate bulimic symptoms and negative mood.

4.4 | Strengths and limitations of this study

The present study has numerous strengths, including that this is only the second longitudinal study to assess the DPM in its entirety (Stice et al., 1998). Further, this is the first study to our knowledge to examine the relative influence of negative urgency to the entire DPM. Despite these strengths, this study does have limitations to consider. First, the self-report methodology of this study might have led to participants' overestimating/underestimating their level of symptomatology. Thus, future designs would benefit from utilizing structured

clinical interviews. Second, the T1 reliability of the bulimia subscale was low which likely reduced the correlations amongst T1 bulimia with other factors in the model. Although all hypothesized variables at T1 showed a significant effect on T1 bulimic symptoms, the results of this study should be interpreted with caution. Finally, this study assessed three subgroups that varied on a range of characteristics (e.g., symptomatology levels). Dummy variables were created to control for these differences, thus minimizing the impact that these differences might have had on results.

4.5 | Clinical implications

Results of this study highlight that early intervention aimed at helping individuals manage negative internal states may reduce the chance of developing disordered eating/an eating disorder. Interventions that help individuals adaptively regulate emotions (Mallorquí-Bagué et al., 2018) and interoceptive deficits (see Preyde, Watson, Remers, & Stuart, 2016 for an example) might be efficacious in reducing one's likelihood of engaging in disordered eating as they reduce the likelihood that one might act rashly (i.e., binge/purge) during times of distress.

5 | CONCLUSION

This study is only the second longitudinal investigation to assess the entire DPM (Stice et al., 1998). Results showed support for the model as all hypothesized paths were confirmed, with the exception that T1 BMI failed to link to T1 body dissatisfaction. Contrary to expectations, results showed no evidence for any bidirectional feedback loops amongst symptoms of depression and disordered eating at T1 and T2, but it was found that T1 depression predicted T2 dietary restraint. Finally, it was found that the inclusion of negative urgency to the model provided significantly better model fit relative to the model that did not include negative urgency hence validating the inclusion of this variable to the DPM. Further testing is required to confirm the replicability of present longitudinal findings in clinical and non-clinical samples, to determine if any feedback loops exist within the DPM, and to further understand the influence of personality factors on the development of bulimic symptoms.

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REFERENCES

Allen, K. L., Byrne, S. M., & McLean, N. J. (2012). The dual-pathway and cognitive-behavioural models of binge eating: Prospective evaluation and comparison. *European Child & Adolescent Psychiatry*, *21*(1), 51–62.

Bradford, J. W., & Petrie, T. A. (2008). Sociocultural factors and the development of disordered eating: A longitudinal analysis of competing hypotheses. *Journal of Counseling Psychology*, *55*(2), 246–262.

Bucchianeri, M. M., Arikian, A. J., Hannan, P. J., Eisenberg, M. E., & Neumark-Sztainer, D. (2013). Body dissatisfaction from adolescence to young adulthood: Findings from a 10-year longitudinal study. *Body Image*, *10*(1), 1–7.

Cassin, S. E., & von Ranson, K. M. (2005). Personality and eating disorders: A decade in review. *Clinical Psychology Review*, *25*(7), 895–916. https://doi.org/10.1016/j.cpr.2005.04.012

Cohen, J. (1992). A power primer. *Psychological Bulletin*, *112*(1), 155–159.

Dakanalis, A., Timko, C. A., Carrà, G., Clerici, M., Zanetti, M. A., Riva, G., & Caccialanza, R. (2014). Testing the original and the extended dual-pathway model of lack of control over eating in adolescent girls. A two-year longitudinal study. *Appetite*, *82*, 180–193.

Duemm, I., Adams, G. R., & Keating, L. (2003). The addition of sociotropy to the dual pathway model of bulimia. *Canadian Journal of Behavioural Science/Revue Canadienne Des Sciences du Comportement*, *35*(4), 281–291.

Evans, E. H., Tovée, M. J., Boothroyd, L. G., & Drewett, R. F. (2013). Body dissatisfaction and disordered eating attitudes in 7-to 11-year-old girls: Testing a sociocultural model. *Body Image*, *10*(1), 8–15.

Fischer, S., Smith, G. T., & Cyders, M. A. (2008). Another look at impulsivity: A meta-analytic review comparing specific dispositions to rash action in their relationship to bulimic symptoms. *Clinical Psychology Review*, *28*(8), 1413–1425.

Garner, D. M., Olmsted, M. P., Bohr, Y., & Garfinkel, P. E. (1982). The eating attitudes test: Psychometric features and clinical correlates. *Psychological Medicine*, *12*(4), 871–878.

Girard, M., Chabrol, H., & Rodgers, R. F. (2018). Support for a modified tripartite dual pathway model of body image concerns and risky body change behaviors in French young men. *Sex Roles*, *78*(11–12), 799–809.

Heatherton, T. F., & Baumeister, R. F. (1991). Binge eating as escape from self- awareness. *Psychological Bulletin*, *110*(1), 86–108.

Hoek, H. W. (2006). Incidence, prevalence and mortality of anorexia nervosa and other eating disorders. *Current Opinion in Psychiatry*, *19*(4), 389–394.

Hutchinson, D. M., Rapee, R. M., & Taylor, A. (2010). Body dissatisfaction and eating disturbances in early adolescence: A structural modeling investigation examining negative affect and peer factors. *The Journal of Early Adolescence*, *30*(4), 489–517.

Kline, P. (2015). *A handbook of test construction (psychology revivals): Introduction to psychometric design: Routledge*.

Mackinnon, S. P., Sherry, S. B., Graham, A. R., Stewart, S. H., Sherry, D. L., Allen, S. L., & McGrath, D. S. (2011). Reformulating and testing the perfectionism model of binge eating among undergraduate women: A short-term, three-wave longitudinal study. *Journal of Counseling Psychology*, *58*(4), 630–646.

Mallorquí-Bagué, N., Vintró-Alcaraz, C., Sánchez, I., Riesco, N., Agüera, Z., Granero, R., & Fernández-Aranda, F. (2018). Emotion regulation as a transdiagnostic feature among eating disorders: Cross-sectional and longitudinal approach. *European Eating Disorders Review*, *26*(1), 53–61.

Maraldo, T. M., Zhou, W., Dowling, J., & Vander Wal, J. S. (2016). Replication and extension of the dual pathway model of disordered eating: The role of fear of negative evaluation, suggestibility, rumination, and self-compassion. *Eating Behaviors*, *23*, 187–194.

Marsh, H. W., Hau, K.-T., & Wen, Z. (2004). In search of golden rules: Comment on hypothesis-testing approaches to setting cutoff values for fit indexes and dangers in overgeneralizing Hu and Bentler's (1999) findings. *Structural Equation Modeling*, *11*(3), 320–341.

Mason, T. B., & Lewis, R. J. (2015). Assessing the roles of impulsivity, food-related cognitions, BMI, and demographics in the dual pathway model of binge eating among men and women. *Eating Behaviors*, *18*, 151–155.

Muthén, B., Muthén, L., & Asparouhov, T. (2017). *Regression and mediation analysis using Mplus*. Los Angeles, CA: Muthén & Muthén.

Ouwens, M. A., Van Strien, T., Van Leeuwe, J., & Van der Staak, C. (2009). The dual pathway model of overeating. Replication and extension with actual food consumption. *Appetite*, *52*(1), 234–237.

Pallister, E., & Waller, G. (2008). Anxiety in the eating disorders: Understanding the overlap. *Clinical Psychology Review*, *28*(3), 366–386. https://doi.org/10.1016/j.cpr.2007.07.001

Paxton, S. J., Neumark-Sztainer, D., Hannan, P. J., & Eisenberg, M. E. (2006). Body dissatisfaction prospectively predicts depressive mood and low self-esteem in adolescent girls and boys. *Journal of Clinical Child and Adolescent Psychology*, *35*(4), 539–549.

Pearson, C. M., Wonderlich, S. A., & Smith, G. T. (2015). A Risk and Maintenance Model for Bulimia Nervosa: From Impulsive Action to Compulsive Behavior. *Psychological Review*, *122*(3), 516–535. https://doi.org/10.1037/a0039268

Petrie, T. A., Tripp, M. M., & Harvey, P. (2002). Factorial and construct validity of the body parts satisfaction scale-revised: An examination of minority and nonminority women. *Psychology of Women Quarterly*, *26*(3), 213–221.

Pidgeon, A., & Harker, R. A. (2013). Body-focused anxiety in women: Associations with internalization of the thin-ideal, dieting frequency, body mass index and media effects. *Open Journal of Medical Psychology*, *2*(04), 17–24.

Preyde, M., Watson, J., Remers, S., & Stuart, R. (2016). Emotional dysregulation, interoceptive deficits, and treatment outcomes in patients with eating disorders. *Social Work in Mental Health*, *14*(3), 227–244.

Puccio, F., Fuller-Tyszkiewicz, M., Ong, D., & Krug, I. (2016). A systematic review and meta-analysis on the longitudinal relationship between eating pathology and depression. *International Journal of Eating Disorders*, *49*(5), 439–454. https://doi.org/10.1002/eat.22506

Puccio, F., Fuller-Tyszkiewicz, M., Youssef, G., Mitchell, S., Byrne, M., Allen, N., & Krug, I. (2017). Longitudinal Bi-directional Effects of Disordered Eating, Depression and Anxiety. *European Eating Disorders Review*, *25*(5), 351–358.

Puccio, F., Kalathas, F., Fuller-Tyszkiewicz, M., & Krug, I. (2016). A revised examination of the dual pathway model for bulimic symptoms: The importance of social comparisons made on Facebook and sociotropy. *Computers in Human Behavior*, *65*, 142–150.

Quigley, L., Wen, A., & Dobson, K. S. (2017). Avoidance and depression vulnerability: An examination of avoidance in remitted and currently depressed individuals. *Behaviour Research and Therapy*, *97*, 183–188.

Racine, S. E., & Martin, S. J. (2016). Exploring divergent trajectories: Disorder-specific moderators of the association between negative urgency and dysregulated eating. *Appetite*, *103*, 45–53.

Radloff, L. S. (1977). The CES-D scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement*, *1*(3), 385–401.

Raftery, A. E. (1995). Bayesian model selection in social research. *Sociological Methodology*, *25*, 111–163.

Ruisoto, P., Cacho, R., López-Goñi, J. J., Deus, E. R., Vaca, S., & Mayoral, P. (2015). Gender differences in risk factors for Stice's bulimia in a non-clinical sample. *The Spanish Journal of Psychology*, *18*, 1–10.

Salafia, E. H., & Gondoli, D. M. (2011). A 4-year longitudinal investigation of the processes by which parents and peers influence the development of early adolescent girls' bulimic symptoms. *The Journal of Early Adolescence*, *31*(3), 390–414.

Schwarz, G. (1978). Estimating the dimension of a model. *The Annals of Statistics*, *6*(2), 461–464.

Sehm, M., & Warschburger, P. (2017). The dual-pathway model of binge eating: Is there a need for modification? *Appetite*, *114*, 137–145.

Smith, G. T., Guller, L., & Zapolski, T. C. (2013). A comparison of two models of urgency: Urgency predicts both rash action and depression in youth. *Clinical Psychological Science*, *1*(3), 266–275.

Stice, E. (2002). Risk and maintenance factors for eating pathology: A meta-analytic review. *Psychological Bulletin*, *128*(5), 825–848.

Stice, E., & Bearman, S. K. (2001). Body-image and eating disturbances prospectively predict increases in depressive symptoms in adolescent girls: A growth curve analysis. *Developmental Psychology*, *37*(5), 597–607. https://doi.org/10.1037//0012-1649.37.5.597

Stice, E., Fisher, M., & Martinez, E. (2004). Eating disorder diagnostic scale: Additional evidence of reliability and validity. *Psychological Assessment*, *16*(1), 60–71.

Stice, E., Gau, J. M., Rohde, P., & Shaw, H. (2017). Risk factors that predict future onset of each DSM-5 eating disorder: Predictive

specificity in high-risk adolescent females. *Journal of Abnormal Psychology*, *126*(1), 38–51. https://doi.org/10.1037/abn0000219

Stice, E., Nemeroff, C., & Shaw, H. E. (1996). Test of the dual pathway model of bulimia nervosa: Evidence for dietary restraint and affect regulation mechanisms. *Journal of Social and Clinical Psychology*, *15*(3), 340–363. https://doi.org/10.1521/jscp.1996.15.3.340

Stice, E., Shaw, H., & Nemeroff, C. (1998). Dual pathway model of bulimia nervosa: Longitudinal support for dietary restraint and affect-regulation mechanisms. *Journal of Social and Clinical Psychology*, *17*(2), 129–149.

Streigel-Moore, R. H., & Bulik, C. M. (2007). Risk factors for eating disorders. *American Psychologist*, *62*(3), 181–198.

Tabachnick, B. G., & Fidell, L. S. (2007). *Using multivariate statistics: Allyn & Bacon/Pearson education*.

Van Strien, T., Engels, R. C., Van Leeuwe, J., & Snoek, H. M. (2005). The Stice model of overeating: Tests in clinical and non-clinical samples. *Appetite*, *45*(3), 205–213.

Waxman, S. E. (2009). A systematic review of impulsivity in eating disorders. *European Eating Disorders Review*, *17*(6), 408–425. https://doi.org/10.1002/erv.952

Welsh, D. M., & King, R. M. (2016). Applicability of the dual pathway model in normal and overweight binge eaters. *Body Image*, *18*, 162–167.

Wenzel, K. R., Weinstock, J., Vander Wal, J. S., & Weaver, T. L. (2014). Examining the role of negative urgency in a predictive model of bulimic symptoms. *Eating Behaviors*, *15*(3), 343–349.

Whiteside, S. P., & Lynam, D. R. (2001). The five factor model and impulsivity: Using a structural model of personality to understand impulsivity. *Personality and Individual Differences*, *30*(4), 669–689.

Wittchen, H. U., & Jacobi, F. (2005). Size and burden of mental disorders in Europe—A critical review and appraisal of 27 studies. *European Neuropsychopharmacology*, *15*(4), 357–376.

Womble, L. G., Williamson, D. A., Martin, C. K., Zucker, N. L., Thaw, J. M., Netemeyer, R., & Greenway, F. L. (2001). Psychosocial variables associated with binge eating in obese males and females. *International Journal of Eating Disorders*, *30*(2), 217–221.

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