A New Integrative Model For The Co-Occurrence of Non-Suicidal Self-Injury Behaviours and Eating Disorder Symptoms

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Abstract

Objective: The high co-occurrence of non-suicidal self-injury (NSSI) behaviours and eating disorder (ED) symptoms suggests these conditions share common aetiological processes. We assessed whether insecure attachment and maladaptive schemas were related to NSSI and ED symptoms through affect dysregulation, impulsivity, self-esteem, and body dissatisfaction.

Method:123 ED patients and 531 individuals from the community completed an online survey, which included measures assessing the variables of interest.

Results: The model was a good fit for the ED group, however only a revised model reached an acceptable fit for the community sample. In the community group, impulsivity was a shared predictor for NSSI and bulimic symptoms, whereas affect dysregulation was a unique predictor for NSSI in both the ED and community groups. No other variables were shared by NSSI and ED symptoms in the two groups. Both attachment and maladaptive schemas were implicated in the pathways leading to ED and NSSI symptoms in the clinical ED and the community sample. The variance explained for NSSI and drive for thinness were highest for the clinical ED sample (29% and 57% respectively).

Conclusion: Common factors may underlie NSSI and ED symptoms, however, these factors may become more specific and less prevalent as a function of disorder severity.

Plain English Summary

We assessed whether insecure attachment and maladaptive schemas (cognitive distortions) were related to non-suicidal self-injury (NSSI) and eating disorder (ED) symptoms through affect (emotion) dysregulation, impulsivity, self-esteem, and body dissatisfaction. A total of 123 ED patients and 531 individuals from the community completed an online survey, which included measures assessing the variables of interest. We found that the proposed model was a good match for the clinical ED, but not the community group. Affect dysregulation was directly associated with NSSI in both the ED and community and impulsivity was related to NSSI and bulimic symptoms only in the community group. Both attachment and maladaptive schemas were indirectly related to ED and NSSI symptoms in both groups. We conclude that common factors may underlie NSSI and ED symptoms, however, these factors may become more specific and less prevalent as a function of disorder severity.

Introduction

Non-suicidal self-injury (NSSI; e.g., cutting, burning, biting) is frequently observed among individuals with eating disorders (EDs), with a meta-analysis reporting the lifetime history of NSSI to be 27.3% amongst ED patients (1). This high comorbidity suggests shared factors for both EDs and NSSI (2). To explain the co-occurrence of these behaviours, Svirko and Hawton (2007) (3) and Claes and Muehlenkamp (2014) (2) proposed a conceptual model of risk, which included adverse childhood events, affect dysregulation, impulsivity, low self-esteem, and dissociation. Although this model has received preliminary empirical

support in the literature (4–6), results have demonstrated that the model explains only a small amount of variance in NSSI and ED symptoms (4); suggesting that the model might benefit from the inclusion of processes previously implicated in both EDs and NSSI. Two factors that are known to influence NSSI and ED, yet to been tested in the context of a comprehensive, integrated model are insecure attachment (7) and early maladaptive schemas (8). The current study is the first to test an extension of the existing conceptual model (2, 3), including these variables.

Current models explaining the relationship between EDs and NSSI

Svirko and Hawton (2007) (3) and Claes and Muehlenkamp (2014) (2) provided a conceptual model of potential factors underlying the association between NSSI and EDs. In this proposed model, key distal risk factors such as major traumatic events, personality, culture and a maladaptive family environment lead to more proximal pathological processes, such as impulsivity, affect dysregulation, dissociation, self-criticising cognitive styles (low self-esteem), need for control, and obsessive-compulsive tendencies. In turn, these factors are thought to lead to the development of NSSI and EDs (2, 3).

To date, only two studies have empirically evaluated this theoretical model in a clinical ED population (5, 6), and one study assessed the model in a university sample (4). Muehlenkamp et al. (2011) (5) tested a simplified version of the model among 422 ED female inpatients. The model assessed whether childhood trauma, low self-esteem, psychopathology (anxiety and depression), dissociation, and body dissatisfaction predicted NSSI. Findings revealed that childhood trauma had an indirect relationship with NSSI, mediated by low self-esteem, psychopathology, body dissatisfaction, and dissociation. The study also found that dissociation and body dissatisfaction were crucial factors in accounting for individual differences in NSSI. Although the model was a good fit to the data, it accounted for only a modest amount of the variance in NSSI (15%).

In the second clinical study, Vieira et al. (2018) (6) assessed a range of risk factors (both precursors and pathological processes) in 245 female ED outpatients with and without NSSI. In line with the conceptual model (2, 3), experiences of physical and sexual abuse acted as distal risk factors for NSSI in EDs, which in turn, were mediated by more proximal risk factors. Specifically, the paths from physical abuse to NSSI and ED were mediated by negative self-evaluation, substance use, and suicide attempts (6).

The third empirical study assessed associations between emotional distress, emotion regulation, avoidance, NSSI, and ED psychopathology in 230 female undergraduates (4). The study found that the relationship between emotional distress and avoidance was mediated by limited access to emotion regulation strategies. Again, although the model provided a good fit to the data, the variance accounted for was only 16% for NSSI and 26% for ED pathology. These studies provide preliminary support for the conceptual model proposed by Svirko and Hawton (2007) (3) and Claes and Muehlenkamp (2014) (2). However, the modest variance in NSSI and ED accounted for by the proposed models suggest that other

important factors related to NSSI and ED may need to be incorporated to better explain the psychological processes which link NSSI and EDs.

While previous theories have focussed predominantly on intrapersonal factors related to NSSI and ED behaviours, considerable evidence suggests that both NSSI and EDs are associated with interpersonal, social and cognitive difficulties which predispose and maintain these behaviours (9, 10). Self-injury and ED symptoms frequently occur in interpersonal contexts and are the result of the cognitive interpretation of these relationships (1). As such, incorporating interpersonal and cognitive factors may add explanatory power and enhance our understanding of the factors linking NSSI and EDs.

Insecure attachment and early maladaptive schemas in EDs and NSSI

Attachment is an interpersonal factor that has been broadly implicated in both NSSI (7) and EDs (10, 11). One possible adverse consequence of attachment difficulties is the development of early maladaptive schemas, defined as implicit negative beliefs or cognitive representations about one's self and one's relationship with the environment that is self-perpetuating and relatively stable over time (12, 13). Maladaptive schemas (i.e. emotional deprivation, insufficient self-control, mistrust/abuse and unrelenting standards), have commonly been implicated in EDs (14–16) and NSSI (17, 18), and have also been found to mediate the relationship between attachment and ED and NSSI symptoms (16, 18). Therefore, the literature has identified insecure attachment and maladaptive schemas as essential precursors of both EDs and NSSI. However, no known study has empirically investigated these interpersonal and cognitive constructs concurrently within an integrated model of ED symptoms and NSSI.

The current study

The current study aimed to assess a novel extension of the conceptual model proposed by Svirko and Hawton (2007) (3) and Claes and Muehlenkamp (2014) (2) by integrating the additional shared interpersonal factor of attachment and the cognitive precursor of schemas for NSSI and EDs. Based on this new socio-cognitive-emotional model of NSSI and EDs (see Fig. 1), we expected that insecure attachment would be related to early maladaptive schemas, which in turn, would be associated with NSSI and ED behaviours through variables proposed in the existing conceptual model (2, 3), including affect dysregulation, impulsivity, self-esteem, and body dissatisfaction. Given that ED symptoms vary on a continuum independent of a clinical diagnosis (19), and non-clinical individuals with NSSI also exhibit a significantly higher level of eating pathology compared to non-NSSI individuals (20), this study also assessed whether the model was invariant across a clinical ED and a community sample.

In the current model, maladaptive schemas were summarized as schemas related to Cluster B (insufficient control, emotional deprivation and mistrust/abuse schemas) and Cluster C (failure to achieve, social undesirability, subjugation and unrelenting standards) schemas, according to the schemas commonly representing these Clusters of personality disorders [Cluster B = borderline, antisocial, histrionic and narcissistic personality disorders and Cluster C = avoidant, dependent and obsessive-

compulsive personality disorders (21)]. These schemas were chosen because previous studies (15) have found significant associations between these two Clusters of schemas and EDs, and to maximise model parsimony. Considering the clinical implications of the comorbidity between NSSI and EDs, there is a need to identify shared factors and potential intermediary mechanisms underlying these behaviours so that effective prevention and early intervention efforts can be established

Methods

Participants

A total of 123 female participants with a lifetime ED diagnosis [54 Anorexia Nervosa (AN)-Restrictive, 18 AN-Binge Purge, 18 Bulimia Nervosa, 11 Binge Eating Disorder, 17 Other Specified Eating and Feeding Disorder, and 5 Unspecified Feeding or Eating Disorders] were recruited from two clinical ED units in Melbourne and other ED associations across Australia. Formal ED diagnosis was determined by psychiatrist report in clinical settings according to the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) (21), and by self-report in other settings. The average age of onset of an ED diagnosis among this group was 15 years [Standard Deviation (SD) = 4.25], and 32% indicated that they were currently recovered from their ED. The community sample comprised 531 females from Australia. The mean age for the total sample was 22.48 years (*SD* = 8.13), and most of the participants were single, Caucasian, and currently studying at a University in Australia. The inclusion criteria for both samples included being female and at least 18 years of age.

Measures

Sociodemographics. Information on participant age, height, weight, ethnicity, employment status, marital status, highest completed education, lifetime ED status, and age of ED onset were obtained. Body Mass Index (BMI) was calculated as the ratio of weight (kg) to height squared (m^2).

Attachment style. Attachment style was measured using the Revised Experiences in Close Relationships scale [ECR-R; (22)]. The ECR-R is a 36-item self-report measure that assesses individual differences in *attachment anxiety* (18 items) and *attachment avoidance* (18 items). Items were scored on a 7-point scale from 1 (strongly disagree) to 7 (strongly agree). Items are scored on a 7-point scale from 1 (strongly disagree) to 7 (strongly agree). The ECR-R is a reliable measure of adult attachment (23).

Schemas. Early maladaptive schemas were assessed using seven of 15 subscales of the Young Schema Questionnaire – Short Form [YSQ-SF; (24)]: (1) insufficient self-control; (2) emotional deprivation; (3) mistrust/abuse; (4) failure to achieve; (5) social undesirability; (6) subjugation; and (7) unrelenting standards. All schema scales include 5 items, which are scored on a 6-point scale ranging from 1 (completely untrue of me) to 6 (describes me perfectly). A higher score is indicative of a more maladaptive belief. The seven subscales were collapsed into two schema Clusters (15): (1) maladaptive Cluster B schemas (emotional deprivation and mistrust/abuse) and (2) maladaptive Cluster C schemas (failure to achieve, social undesirability subjugation and unrelenting standards).

Affect dysregulation, self-esteem, impulsivity and NSSI symptoms. Affect dysregulation, self-esteem, impulsivity and NSSI symptoms were assessed using the Borderline Personality Questionnaire [BPQ; (25, 26)]. The BPQ is an 80-item self-report measure with items are scored as true or false. Four of the nine subscales were used in the current study: *impulsivity* (9 items), *affective instability* (to measure affect dysregulation, 10 items), *self-image* (to measure self-esteem, 9 items) and *suicide/self-mutilation* (to evaluate NSSI behaviours, 7 items). One item from the suicide/self-mutilation subscale was excluded ("I have made a suicide attempt in the past") as it assessed suicide attempts and not self-injury behaviours, leaving this scale with a total of 6 items.

Eating disorder symptoms. Three subscales [*body dissatisfaction* (10 items), *bulimia* (8 items), and *drive for thinness* (7 items)] of the Eating Disorder Inventory 3 (EDI-3; (27)) were used to assess ED symptoms. Items are rated on a 6-point scale. The EDI-3 has demonstrated good psychometric properties (28) in previous studies.

Procedure

Consenting adults were provided with a link to the online questionnaire via Qualtrics Online Survey Software, which included the measures outlined above. Participants were asked to fill in the survey at their convenience. For the two participating clinics consenting, individuals were provided with this link during the intake interview. All data was collected in accordance with the Declaration of Helsinki and was approved by a University in Melbourne and two Hospitals in Melbourne. Participants were allowed to withdraw from the study at any time without affecting their treatment.

Statistical Analyses

Descriptive statistics and univariate group difference analyses were conducted using IBM SPSS version 25 (SPSS, Chicago, IL, USA). T-tests for continuous and chi-square tests for categorical variables were used to assess whether ED patients and the community sample differed on sociodemographic data or variables intended for the path analysis. Pearson correlations were undertaken to evaluate associations between variables before conducting the path analysis. Strength of the correlation was determined by Pearson's r, with $|.10| < r < |.30|$ indicating weak correlations, $|.30| < r < |.50|$ medium correlations, and $r > |.50|$ indicating a strong relationship (29).

Path analysis using MPlus software was used to test the hypothesised model in Fig. 1. The model was run separately for the ED and the community sample to ascertain goodness of fit for each group. Following recommended criteria, non-significant chi-square ($p > .05$), Comparative Fit Index (CFI) values above .95, Root Mean Square Error of Approximation (RMSEA) below .10, and Standardised Root Mean Residual (SRMR) below .08 were used to indicate acceptable model fit (30). In the event of poor model fit, modification indices were consulted, and recommended paths were added if theoretically or logically justifiable.

Given the marked differences in the initial model fit and proposed revisions for the ED and the community separately, a common model was not deemed suitable. Instead, final models are reported separately for

each group. Model-implied mediation pathways were tested for significance using bias-corrected bootstrapping with 5,000 bootstraps (31). In the final models age was included as a co-variate for all dependent variables. In the following results section only the significant paths are being outlined. Model parameters for non-significant paths can be provided on request from the corresponding author.

Results

Sociodemographic characteristics

Significant group differences were observed for age, ethnicity, and marital and employment status and BMI, with the ED group being significantly more likely to be older, Caucasian, married, unemployed and having a lower average BMI than the community group. Conversely, the community sample contained significantly more Asian, single, and student participants than the ED group. There were no significant group differences on the highest level of education attained (Table 1).

Group differences in study variables

Table 2 presents differences between the ED and community sample in the study variables included in the proposed socio-emotional model of NSSI and ED. ED patients scored significantly higher than community individuals on all variables. Table 2 also presents the Cronbach values for all measures, which provided satisfactory (α = .60 for impulsivity for the community sample) to excellent values (α = .97 for failure to achieve schema for the ED sample).

Path Analyses

Correlational analyses (Supplementary Table 1) were run separately for the ED and the community samples to examine bivariate relationships, which formed the basis for the path analyses. Significant correlations were found between all the variables included in the model for both the ED and the community groups, with mainly moderate to large effect sizes.

ED group. The proposed model had good fit overall; chi square$_{(df=26)}$ = 25.22, p = .51, CFI = 1.00, RMSEA = .000, SRMR = .032. The predictor variables accounted for over one-quarter of the variance in NSSI, with significant unique contributions from affect dysregulation and age. Bulimic symptoms were uniquely predicted by body dissatisfaction, impulsivity, and age. In total, the predictors accounted for 33% of the variance in bulimic symptoms. The model accounted for 57% of the variance in drive for thinness, with significant unique contributions from self-esteem and body dissatisfaction.

The predictors combined accounted for fifty-two percent of the variance in body dissatisfaction, with significant contributions from impulsivity, affect dysregulation, and self-esteem. Impulsivity was significantly predicted by maladaptive Cluster B schemas. In total, 20% of the variance in impulsivity was accounted for in this model. The model accounted for 56% of the variance in self-esteem; with maladaptive Cluster C schemas being the only significant predictor.

Affect dysregulation was significantly predicted by age, maladaptive Cluster B schemas and Cluster C schemas, with 47% of the variance in affect dysregulation accounted for overall.

Maladaptive Cluster B and C schemas had 39% and 45%, respectively, of their variance explained. Both maladaptive schema Clusters B and C were predicted by anxious attachment, and avoidant attachment. Maladaptive Cluster B and Cluster C schemas were significantly, positively related.

The relationships between NSSI and bulimia and drive for thinness were non-significant after controlling for predictors in the model. Bulimia and drive for thinness were significantly related.

Community group. The proposed model was a poor fit overall for the community group; chi square$_{(df=26)}$ = 144.98, p < .001, CFI = 0.949, RMSEA = .093, SRMR = .054. Inspection of the modification indices identified four plausible paths to add to the model to improve fit: (1) a covariance term between affect dysregulation and self-esteem, (2) anxious attachment predicting affect dysregulation, (3) anxious attachment predicting self-esteem, and (4) maladaptive Cluster C schemas directly predicting NSSI. With these revisions, the model had acceptable fit; chi square$_{(df=22)}$ = 51.82, p < .001, CFI = 0.987, RMSEA = .051, SRMR = .033. Relationships among modelled variables are reported below for this refined model.

Almost one-quarter (24%) of the variance in NSSI was accounted for, with significant unique contributions from affect dysregulation, impulsivity, and maladaptive Cluster C schemas. Bulimic symptoms were uniquely predicted by body dissatisfaction, impulsivity, and self-esteem. In total, the predictors accounted for 39% of the variance in bulimic symptoms. Over half the variance (51%) in drive for thinness was accounted for by the model, with body dissatisfaction demonstrated a significant unique contribution.

Twenty-seven percent of the variance in body dissatisfaction was accounted for by its predictors combined, with significant contributions from impulsivity and self-esteem. Impulsivity was significantly predicted by maladaptive Cluster B schemas and age. In total, 16% of the variance in impulsivity was accounted for in this model. The model accounted for 53% of the variance in self-esteem; maladaptive Cluster C schemas and anxious attachment were the only significant predictors.

Affect dysregulation was significantly predicted by maladaptive Cluster B and Cluster C schemas, and anxious attachment, with and 36% of the variance in affect dysregulation accounted for. Maladaptive Cluster B and Cluster C schemas had 31% and 29% of their variance explained, respectively. Anxious and avoidant attachment were significant predictors of maladaptive Cluster B schemas and maladaptive Cluster C schemas. Age was also a significant predictor of maladaptive Cluster C schemas.

Maladaptive Cluster B and Cluster C schemas were significantly and positively related, as was impulsivity with affect dysregulation. The relationships between NSSI and bulimia and drive for thinness were non-significant after controlling for predictors in the model. Bulimia and drive for thinness remained significantly related.

Indirect effects

ED group. The total indirect effect from the attachment variables to ED outcomes showed that anxious attachment on bulimia (*ß* = .14, 99% CIs: .05, .26), drive for thinness (*ß* = .24, 99% CIs: .12, .39), and NSSI (*ß* = .13, 99% CIs: .03, .26) were significant for the ED group. As shown in Table 3, the relationship between a.) anxious attachment and bulimia and b.) anxious attachment and drive for thinness was mediated by Cluster B and C, self-esteem, impulsivity, affect dysregulation, and body dissatisfaction. The relationship between anxious attachment and NSSI was mediated by Clusters B and C, affect dysregulation and self-esteem.

Results showed a significant total indirect effect of avoidant attachment to bulimia (*ß* = .09, 99% CIs: .02, .20), with drive for thinness (*ß* = .17, 99% CIs: .04, .31), and NSSI (*ß* = .08, 99% CIs: .01, .19) showing significant indirect effects. These relationships were the same as for anxious attachment apart that for NSSI self -esteem was a significant mediator.

Community group. Inspection of the indirect effects from the attachment variables to ED outcomes showed that the total indirect effects of anxious attachment on bulimia (*ß* = .22, 99% CIs: .16, .29), drive for thinness (*ß* = .20, 99% CIs: .14, .26), and NSSI (*ß* = .24, 99% CIs: .17, .30) were significant in the community group. As shown in Table 3, the relationship between a.) anxious attachment and bulimia and b.) anxious attachment and drive for thinness were mediated by Cluster B and C schemas, self-esteem, impulsivity, and body dissatisfaction. The relationship between anxious attachment and NSSI was mediated by affect dysregulation, self-esteem, and Cluster B and C schemas.

The total indirect effects of avoidant attachment on bulimia symptoms (*ß* = .06, 99% CIs: .03, .10), drive for thinness (*ß* = .05, 99% CIs: .02, .08), and NSSI (*ß* = .07, 99% CIs: .02, .12) were also significant. These relationships were the same as for anxious attachment apart that for NSSI, impulsivity and not self-esteem was a significant mediator.

Discussion

Informed by the conceptual model of risk proposed by Svirko and Hawton (2007) (3) and Claes and Muehlencamp (2014) (2), this study examined a novel socio-cognitive-emotional model of NSSI and ED symptoms in a clinical ED and community group. Our model provided a good fit to the data in the clinical ED sample, but a poor fit in the community sample, suggesting the model does not generalise across groups, or the spectrum of severity for EDs and NSSI. For the community sample only a revised model, which included several additional pathways (e.g., anxious attachment to affect dysregulation and self-esteem, and maladaptive Cluster C schemas directly to NSSI), achieved an acceptable fit. Therefore, the initial proposed model required an increased complexity to account for the correlations between ED and NSSI symptoms in a community sample. The significance of predictors differed between the clinical ED group and the community sample, with affect dysregulation being a unique contributor for NSSI in both the ED and community groups. The only shared factor for NSSI and bulimic symptoms found for the community, but not the ED sample, was impulsivity.

Unique and shared predictors for NSSI, bulimia, and drive for thinness

We found that affect dysregulation was a unique predictor for NSSI, but not bulimia or drive for thinness in both the ED and the community groups. Several studies have also revealed positive associations between NSSI and anxiety and depression, often regarded as proxies for affect dysregulation, in ED patients (5, 32). Claes et al. (2010) (5) for instance reported that affect dysregulation was the primary motivation for all types of NSSI in EDs except bruising. Regarding community samples, a study using path analysis found that emotion dysregulation predicted positive and negative affect after engaging in NSSI. However, positive, not negative affect was responsible for more subsequent lifetime NSSI behaviours (33). These findings indicate that future studies would benefit from assessing the distinctive roles of both negative and positive affect in predicting NSSI. Finally, in terms of the previous models that assessed parts of our proposed model, Muehlenkamp et al. (2012) (34) also revealed that NSSI was related to depression through dissociation, whereas disordered eating was motivated by body dissatisfaction.

Contradicting previous studies, which have suggested that disordered eating may also function to regulate emotions (35), in the current study affect dysregulation was not related to any of the ED symptoms. This finding might be because the measurement of affect dysregulation in the current study was more general and not specific to regulating body image dissatisfaction, which may explain why it was not a significant predictor in our path-analyses. Overall, our findings indicate that a more general emotional risk factor (i.e., affect dysregulation) may be related explicitly to NSSI in EDs, but that this factor may not be sufficiently body focused to influence ED behaviours.

Surprisingly, impulsivity was a significant unique predictor of NSSI, and it was also a shared factor for bulimic symptoms and NSSI in the community, but not in the ED group. While this finding partially supports the findings of previous non-clinical ED population studies, which revealed positive correlations between NSSI, impulsiveness, and eating pathology (36), it contradicts other studies that have reported shared contributing factors for impulsivity and NSSI in clinical ED patients, especially in binge/purging ED subtypes (1). The non-significant finding for impulsivity for the ED group might be attributable to the mainly restrictive symptomatology (i.e. AN-Restrictive) present in this group (37). It is also worth noting that apart from impulsivity being shared between NSSI and bulimic symptoms in the community group, no other shared factors between NSSI and ED symtpoms were observed.

In the community group schemas relating to Cluster C personality disorders, were directly associated with NSSI. This finding is consistent with the findings by Anderson et al. (2018) (4), which revealed that experiential avoidance, a proxy for Cluster C personality disorders, was the most influential contributing factor to NSSI in their community sample. Insecure attachment may lead individuals to be socially avoidant, and steer away from close involvement with others to protect themselves against anticipated rejection, which then contributes to the adoption of maladaptive coping strategies, such as NSSI, to manage these difficulties (38).

As regards to the ED related outcome variables, our findings indicated a direct relationship between impulsiveness and body dissatisfaction for bulimic symptoms, for the ED and community groups. Furthermore, for the community sample, self-esteem was also a significant predictor for bulimic symptoms. This finding is in accordance with other studies that have shown impulsiveness, low self-esteem and body dissatisfaction to be important triggering and maintaining factors for bulimic symptoms in both clinical (14) and community (39, 40) samples.

Finally, we observed that low self-esteem and body dissatisfaction were related to drive for thinness in the ED group, which is in line with previous research (41). However, for the community group, the only significant direct contributor to ED symptoms was body dissatisfaction. Overall body dissatisfaction appears to be the most significant factor for ED symptoms in both the clinical ED and community sample, a finding that has been supported by a previous meta-analysis of risk factors for EDs (40).

Variance accounted for in NSSI, bulimia and drive for thinness.

Results showed that our model explained a higher amount of variance for NSSI, bulimia and drive for thinness than previous studies (4, 34). This was the case for the models assessing the ED and the community samples. The variance explained for NSSI in our path-analyses was 29% for the ED group and 24% for the community group. For the ED outcome variables, the variance ranged from 33% for bulimic symptoms and 57% for drive for thinness for the ED group. For the community group, these values ranged from 39% for bulimic symptoms to 51% for drive for thinness. Our percentages are almost double the amount of variance explained for by NSSI and ED symptoms in other studies (4). Therefore, including insecure attachment and early maladaptive schemas in our model appears to enhance our understanding of the processes underlying ED symptoms and NSSI.

Despite our model accounting for significantly more variance in our primary outcome variables than other studies, a large amount of variance was still unexplained. Future studies should examine important predisposing factors including aversive childhood experiences, family characteristics (e.g., parenting styles) as well as more immediate emotional (e.g., dissociation) and cognitive (e.g., need for control) variables that have been outlined in the original theoretical models of the co-occurrence of ED symptoms and NSSI (2, 3). However, increased complexity in our theoretical understanding of these processes comes with increased statistical complexity, which makes model fitting extremely difficult.

Indirect effects of insecure attachment and maladaptive schemas

In the current study, the mediation analyses revealed that both anxious and avoidant attachment were related to both NSSI and ED symptoms through maladaptive Cluster C and B schemas as well as the emotional variables (e.g., impulsivity, affect instability, self-esteem). This finding indicates that early in illness progression, there may be a range of general risk factors that relate to both NSSI and ED symptoms. However, as the illness progresses, associations may become narrower and more specific, which can be seen in the fact that apart from impulsivity in the community sample, no other immediate

factors were shared between NSSI and ED symptoms. This notion is in line with staging models for mental illnesses such as psychosis (42) and more recently also EDs (43).

NSSI and ED symptoms frequently occur in interpersonal contexts (44, 45). Caregiver neglect and traumatic events during childhood are influential risk factors for NSSI behaviours in ED populations (12, 20, 46). These difficult early experiences can lead to the development of insecure attachment styles, which manifest as either avoidant or anxious forms of attachment (47). Attachment difficulties may predispose an individual to fear negative social evaluation (9), including schemas related to Cluster B (e.g., emotional deprivation) and Cluster C (e.g., failure) personality disorders (48). In adulthood, these core beliefs remain dormant until they are activated by situations or life events that are relevant to that specific schema (13). These factors may then become central in triggering both ED and NSSI symptoms through other emotional and body image-related variables, and inevitably serve to maintain both problem sets.

Limitations

Our findings may be considered in the context of the study's limitations. First, as the data is cross-sectional, future research may benefit from the use of longitudinal designs, which may include constructs such as attachment and schemas earlier in life. It is also important to highlight that our model tested for independent associations for NSSI and ED symptoms. If a variable accounted for some common, shared variance between NSSI and ED symptoms, this was unfortunately not captured by our current model. It is for instance possible that ED symptoms lead to NSSI, as individuals try to find an alternative coping style to ED behaviours or struggle with associated shame and distress. The idea that NSSI and ED symptoms may be more causally linked in this way, could not be considered in the current study due to the cross-sectional data.

Second, the ED sample differed in terms of illness status, with 35% of the sample already recovered from their ED, which may have affected our results. A mixed sample between current and recovered ED samples is common in ED populations that have not exclusively been ascertained from a tertiary setting. These studies have commonly referred to lifetime, rather than current ED statuses (49). It is also worth acknowledging that some of the current traits (i.e., attachment. impulsivity) are considered to be endophenotpyes, that have been found to be elevated, even after the recovery from an ED (50).

Third, due to the small sample size for the different ED subdiagnoses, we were not able to assess different models for ED subtypes. Future research may consider the use of a more balanced ED sample with equal distribution of participants across all ED subtypes, to determine any existing differences in the variables of interest between ED subtypes.

Fourth, the model is limited to females living in Australia, and we, therefore, do not know whether our findings are generalisable to males and individuals from other countries.

Finally, there are limitations in the conceptualisation of the model such that the current variables may not fully explain the variance in NSSI and ED symptoms indicating other contributing factors. Future research

may, therefore, continue to assess variables related to both problem behaviours, including other variables that have recently been implicated in both ED and NSSI, such as rejection sensitivity (51), social rank (52), and alexithymia (53).

Withstanding these limitations, the proposed conceptual model is the first model assessing a wide range of interpersonal, cognitive, and emotional factors known to be associated with both NSSI and ED, in an ED as well as a community sample.

Clinical implications

The current findings highlight the importance of screening for NSSI within clinical ED populations, and for clinicians to formulate the overlapping and distinct processes which contribute to both problem sets. Knowledge of the shared contributing factors between EDs and NSSI as well as the functional role self-harm and disordered eating may serve for individuals, may contribute to improved clinical decision-making regarding treatment and support. Specifically, those presenting with comorbidity may benefit from treatments that target both ED and NSSI symptoms such as dialectical behavioural therapy (54), schema therapy (24), interpersonal therapy (55), and emotion regulation training programs (56)]. Finally, there is the need to address the role of insecure attachment, both through the prevention of insecure attachment and maladaptive schemas using early intervention parenting programs (57), as well as fostering secure attachment for those already engaged in therapy. The results also highlight the importance of identifying those risk factors more pertinent for individuals with less severe disordered eating such as those participants within the community, to identify where to target prevention strategies.

Conclusions

In conclusion, the current study expanded upon previous conceptual models of NSSI in EDs. Using an attachment framework, we examined the shared contributions of interpersonal, cognitive, and emotional difficulties which may lead to ED symptoms and NSSI in an ED and a community sample. The results from our path-analyses found that our model provided a good fit for the ED, but not the community sample, indicating that the model was not directly generalisable to a community sample. Except for impulsivity, we did not find other direct shared factors for NSSI, and ED symptoms and this finding was only observed in the community, but not the ED sample. Affect dysregulation was a unique predictor for NSSI, but not ED symptoms, in both samples, indicating that additional and ED specific factors may be involved in the development of EDs. Future research may consider extending the proposed models to gain further understanding of interpersonal, cognitive, and emotional difficulties associated with EDs and NSSI. Testing these models longitudinally may help to differentiate the proximal versus distal risk factors included in our proposed model.

Abbreviations

NSSI
Non-suicidal self-injury

ED
Eating Disorder
AN
Anorexia Nervosa
DSM-5
Diagnostic and Statistical Manual of Mental Disorders
SD
Standard Deviation
BMI
Body Mass Index
ECR-R
Revised Experiences in Close Relationships scale
YSQ-SF
Young Schema Questionnaire – Short Form
BPQ
Borderline Personality Questionnaire
EDI-3
Eating Disorder Inventory 3
CFI
Comparative Fit Index
RMSEA
Root Mean Square Error of Approximation
SRMR
Standardised Root Mean Residual

Declarations

Ethics Approval and Consent

All data was collected in accordance with the Declaration of Helsinki and was approved by a University in Melbourne [Human Ethics Sub-Committee (HESC)] and two Hospitals in Melbourne (Melbourne Health and the Melbourne Clinic). This information has been anonymized in the paper.

Data availability statement:

The datasets during and/or analysed during the current study available from the corresponding author on reasonable request.

Competing interests

The authors declare that they have no competing interests

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Authors' contributions

I.K. and M.D.A developed the study concept. All authors contributed to the study design. Testing and data collection were performed by T.P. M.F. performed the data analysis and interpretation of the data. I.K., M.D.A and K.B. drafted the paper, and S.G., L.K., A.B.D. and J.T. provided critical revisions. All authors approved the final version of the paper for submission.

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Consent for publication

All authors have approved the manuscript for submission to the Journal of Eating Disorders

References

1. Cucchi A, Ryan D, Konstantakopoulos G, Stroumpa S, Kacar AS, Renshaw S, et al. Lifetime prevalence of non-suicidal self-injury in patients with eating disorders: a systematic review and meta-analysis. Psychol Med. 2016;46(7):1345–58.
2. Claes L, Muehlenkamp JJ. Non-suicidal self-injury in eating disorders. Heidelberg: Germany Springer; 2014.
3. Svirko E, Hawton K. Self-injurious behavior and eating disorders: the extent and nature of the association. Suicide Life Threat Behav. 2007;37(4):409–21.
4. Anderson NL, Smith KE, Mason TB, Crowther JH. Testing an Integrative Model of Affect Regulation and Avoidance in Non-Suicidal Self-Injury and Disordered Eating. Arch Suicide Res. 2018;22(2):295–310.
5. Muehlenkamp JJ, Claes L, Smits D, Peat CM, Vandereycken W. Non-suicidal self-injury in eating disordered patients: a test of a conceptual model. Psychiatry Res. 2011;188(1):102–8.
6. Vieira A, Machado B, Moreira C, Machado P, Brandão I, Roma-Torres A, et al. Eating disorders and non-suicidal self-injury: Structural equation modelling of a conceptual model. Eur Eat Disord Rev. 2018;26(5):431–7.
7. Glazebrook K, Townsend E, Sayal K. The role of attachment style in predicting repetition of adolescent self-harm: A longitudinal study. Suicide Life-Threat Behav. 2015;45(6):664–78.
8. Waller G. Schema-level cognitions in patients with binge eating disorder: a case control study. Int J Eat Disord. 2003;33(4):458–64.

9. Arcelus J, Haslam M, Farrow C, Meyer C. The role of interpersonal functioning in the maintenance of eating psychopathology: A systematic review and testable model. Clin Psychol Rev. 2013;33(1):156–67.
10. Caglar-Nazali HP, Corfield F, Cardi V, Ambwani S, Leppanen J, Olabintan O, et al. A systematic review and meta-analysis of 'Systems for Social Processes' in eating disorders. Neurosci Biobehav Rev. 2014;42:55–92.
11. Tasca GA. Attachment and eating disorders: a research update. Curr Opin Psychol. 2019;25:59–64.
12. Voderholzer U, Schwartz C, Thiel N, Kuelz AK, Hartmann A, Scheidt CE, et al. A comparison of schemas, schema modes and childhood traumas in obsessive-compulsive disorder, chronic pain disorder and eating disorders. Psychopathology. 2014;47(1):24–31.
13. Young JE, Klosko JS, Weishaar ME. Schema therapy: a practitioner's guide. New York: Guilford Press; 2003.
14. Dingemans A, Spinhoven P, Van Furth E. Maladaptive core beliefs and eating disorder symptoms. Eat Behav. 2006;7(3):258–65.
15. Pauwels E, Dierckx E, Schoevaerts K, Claes L. Early Maladaptive Schemas in Eating Disordered Patients With or Without Non-Suicidal Self-Injury. Eur Eat Disord Rev. 2016;24(5):399–405.
16. De Paoli T, Fuller-Tyszkiewicz M, Krug I. Insecure attachment and maladaptive schema in disordered eating: The mediating role of rejection sensitivity. Clin Psychol Psychother. 2017;24(6):1273–84.
17. Castille K, Prout M, Marczyk G, Shmidheiser M. The early maladaptive schemas of self-mutilators: Implications for therapy. J Cogn Psychother. 2007;21(1):58.
18. Langhinrichsen-Rohling J, Thompson K, Selwyn C, Finnegan H, Misra T. Maladaptive schemas mediate poor parental attachment and suicidality in college students. Death Stud. 2017;41(6):337–44.
19. Peck LD, Lightsey OR Jr. The eating disorders continuum, self-esteem, and perfectionism. J Couns Dev. 2008;86(2):184–92.
20. Kostro K, Lerman JB, Attia E. The current status of suicide and self-injury in eating disorders: a narrative review. J Eat Disord. 2014;2:19.
21. APA. American Psychiatric Association. Diagnostic and statistical manual of mental disorders (DSM-5®). American Psychiatric Pub.; 2013.
22. Fraley RC, Waller NG, Brennan KA. An item response theory analysis of self-report measures of adult attachment. J Pers Soc Psychol. 2000;78(2):350.
23. Sibley CG, Fischer R, Liu JH. Reliability and validity of the revised experiences in close relationships (ECR-R) self-report measure of adult romantic attachment. Pers Soc Psychol Bull. 2005;31(11):1524–36.
24. Young JE. Cognitive Therapy for Personality Disorders: A Schema-Focused Approach. 3 ed. Sarasota: Professional Resource Exchange; 1999.

25. Fonseca-Pedrero E, Paino M, Lemos-Giraldez S, Sierra-Baigrie S, Gonzalez MP, Bobes J, et al. Borderline personality traits in nonclinical young adults. J Pers Disord. 2011;25(4):542–56.
26. Poreh AM, Rawlings D, Claridge G, Freeman JL, Faulkner C, Shelton C. The BPQ: a scale for the assessment of borderline personality based on DSM-IV criteria. J Pers Disord. 2006;20(3):247–60.
27. Garner DM. Eating disorder inventory-3 (EDI-3). Professional manual. Odessa: Psychological Assessment Resources; 2004.
28. Clausen L, Rosenvinge JH, Friborg O, Rokkedal K. Validating the Eating Disorder Inventory-3 (EDI-3): A Comparison Between 561 Female Eating Disorders Patients and 878 Females from the General Population. J Psychopathol Behav Assess. 2011;33(1):101–10.
29. Cohen J. Statistical power analysis for the behavioral sciences. 2nd ed. Hillsdale: Erlbaum; 1988.
30. Schermelleh-Engel K, Moosbrugger H, Müller H. Evaluating the fit of structural equation models: Tests of significance and descriptive goodness-of-fit measures. Psychol Methods. 2003;8(2):23–74.
31. Shrout PE, Bolger N. Mediation in experimental and nonexperimental studies: New procedures and recommendations. Psychol Methods. 2002;7(4):422.
32. Favaro A, Santonastaso P. Self-injurious behavior in anorexia nervosa. J Nerv Ment Dis. 2000.;188(8):537–42.
33. Jenkins AL, Schmitz MF. The roles of affect dysregulation and positive affect in non-suicidal self-injury. Arch Suicide Res. 2012;16(3):212–25.
34. Muehlenkamp JJ, Peat CM, Claes L, Smits D. Self-injury and disordered eating: expressing emotion dysregulation through the body. Suicide Life Threat Behav. 2012;42(4):416–25.
35. Ambwani S, Morey LC. Food consumption as affect modulation in borderline personality. J Pers Disord. 2015;29(2):261–74.
36. Black EB, Mildred H. A cross-sectional examination of non-suicidal self-injury, disordered eating, impulsivity, and compulsivity in a sample of adult women. Eat Behav. 2014;15(4):578–81.
37. Wagner AF, Vitousek KM. Personality Variables and Eating Pathology. Psychiatr Clin North Am. 2019;42(1):105–19.
38. Bartholomew K, Horowitz LM. Attachment styles among young adults: A test of a four-category model. J Pers Soc Psychol. 1991;61(2):226.
39. Hovrud L, Simons R, Simons J, Korkow J. Non-suicidal self-injury and bulimia: the role of emotion dysregulation and body dissatisfaction. Eat Weight Disord. 2019:1–9.
40. Stice E. Risk and maintenance factors for eating pathology: A meta-analytic review. Psychol Bull. 2002;128(5):825.
41. Shea ME, Pritchard ME. Is self-esteem the primary predictor of disordered eating? Pers Individ Differ. 2007;42(8):1527–37.
42. McGorry PD, Nelson B, Goldstone S, Yung AR. Clinical staging: a heuristic and practical strategy for new research and better health and social outcomes for psychotic and related mood disorders. Can J Psychiatry. 2010;55(8):486–97.

43. Treasure J, Cardi V, Leppanen J, Turton R. New treatment approaches for severe and enduring eating disorders. Physiol Behav. 2015;152:456–65.
44. Brodsky BS, Groves SA, Oquendo MA, Mann JJ, Stanley B. Interpersonal precipitants and suicide attempts in borderline personality disorder. Suicide Life Threat Behav. 2006;36(3):313–22.
45. Brown MZ, Comtois KA, Linehan MM. Reasons for suicide attempts and nonsuicidal self-injury in women with borderline personality disorder. J Abnorm Psychol. 2002;111(1):198.
46. Vieira A, Ramalho S, Brandao I, Saraiva J, Goncalves S. Adversity, emotion regulation, and non-suicidal self-injury in eating disorders. Eat Disord. 2016;24(5):440–52.
47. Bowlby J. Attachment and loss. Separation: Anxiety and anger. II. New York: Basic Books; 1973.
48. Simard V, Moss E, Pascuzzo K. Early maladaptive schemas and child and adult attachment: A 15-year longitudinal study. Psychol Psychother. 2011;84(4):349–66.
49. Root TL, Pisetsky EM, Thornton L, Lichtenstein P, Pedersen NL, Bulik CM. Patterns of co-morbidity of eating disorders and substance use in Swedish females. Psychol Med. 2010;40(1):105–15.
50. Bardone-Cone AM, Butler RM, Balk MR, Koller KA. Dimensions of impulsivity in relation to eating disorder recovery. Int J Eat Disord. 2016;49(11):1027–31.
51. Bondü R, Bilgin A, Warschburger P. Justice sensitivity and rejection sensitivity as predictors and outcomes of eating disorder pathology: A 5-year longitudinal study. Int J Eat Disord. 2020.
52. Pinto-Gouveia J, Ferreira C, Duarte C. Thinness in the pursuit for social safeness: An integrative model of social rank mentality to explain eating psychopathology. Clin Psychol Psychother. 2014;21(2):154–65.
53. Greene D, Boyes M, Hasking P. The associations between alexithymia and both non-suicidal self-injury and risky drinking: A systematic review and meta-analysis. J Affect Disord. 2020.;260:140 – 66.
54. Linehan MM. Dialectical behavior therapy for borderline personality disorder: Theory and method. Bull Menninger Clinic. 1987;51(3):261.
55. Rieger E, Van Buren DJ, Bishop M, Tanofsky-Kraff M, Welch R, Wilfley DE. An eating disorder-specific model of interpersonal psychotherapy (IPT-ED): Causal pathways and treatment implications. Clin Psychol Rev. 2010;30(4):400–10.
56. Berking M, Ebert D, Cuijpers P, Hofmann SG. Emotion regulation skills training enhances the efficacy of inpatient cognitive behavioral therapy for major depressive disorder: a randomized controlled trial. Psychother Psychosom. 2013;82(4):234–45.
57. Lock J, le Grange D. Family-based treatment of eating disorders. Int J Eat Disord. 2005;37(S1):64-S7.

Tables

Due to technical limitations, table 1,3 is only available as a download in the Supplemental Files section.

Table 2: Descriptive statistics and group difference tests

Figures

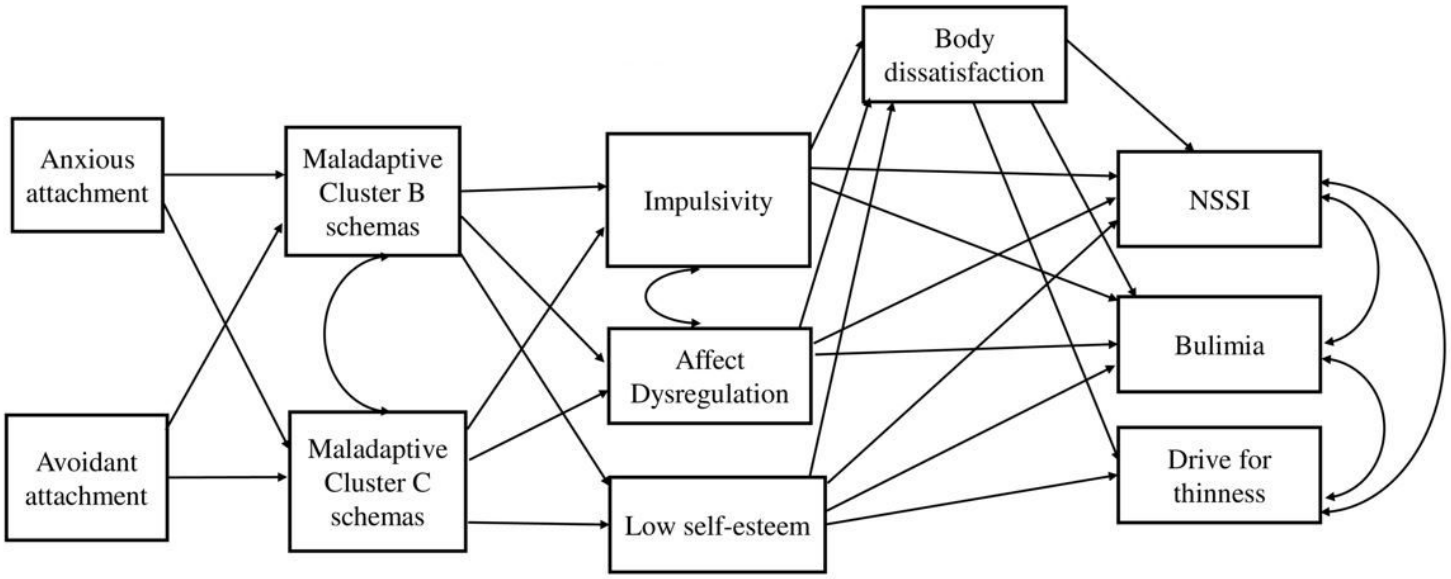

Figure 1

Proposed path model for NSSI and ED symptoms Age is included as a covariate for all DVs in the model (but omitted from figure for simplicity).

	ED (n=123)	Cronbach alpha	Community (n=531)	Cronbach alpha	*t*	*p*	*d*
Mean (SD)							
Attachment anxiety	3.93 (1.13)	.93	3.51 (1.20)	.94	3.49	.001	.36
Attachment avoidance	4.05 (1.25)	.95	3.30 (1.13)	.94	6.45	< .001	.63
Schema B Cluster					2.41	.017	.25
Insufficient self-control schema	16.09 (6.69)	.88	14.51 (5.84)	.88			
Emotional deprivation schema	14.00 (7.14)	.92	12.07 (6.32)	.90	2.96	.003	.29
Distrust /abuse schema	15.06 (6.96)	.91	12.83 (5.88)	.89	3.30	.001	.35
Schema C Cluster					8.18	< .001	.87
Failure to achieve schema	19.08 (8.12)	.97	12.64 (6.62)	.95			
Social undesirability schema	19.76 (7.32)	.94	12.86 (6.53)	.94	9.61	< .001	.99
Subjugation schema	16.73 (7.15)	.91	11.68 (5.64)	.88	7.33	< .001	.73
Unrelenting standards schema	23.05 (5.89)	.88	18.46 (5.77)	.85	7.92	< .001	.79
Affect dysregulation	5.98 (2.92)	.84	4.08 (2.95)	.83	6.45	< .001	.65
Low self-esteem	6.18 (2.90)	.87	3.49 (2.69)	.81	9.84	< .001	.96
Impulsivity	2.50 (2.04)	.69	1.43 (1.53)	.60	5.44	<.001	.59
NSSI	3.17 (2.03)	.84	1.19 (1.63)	.81	10.09	< .001	1.08
Drive for thinness	17.94 (8.54)	.91	9.38 (7.26)	.87	10.28	< .001	1.21
Body dissatisfaction	30.09 (12.28)	.93	17.68 (10.55)	.90	10.36	< .001	1.08
Bulimia	11.52 (9.45)	.91	5.85 (6.27)	.87	6.33	< .001	.71

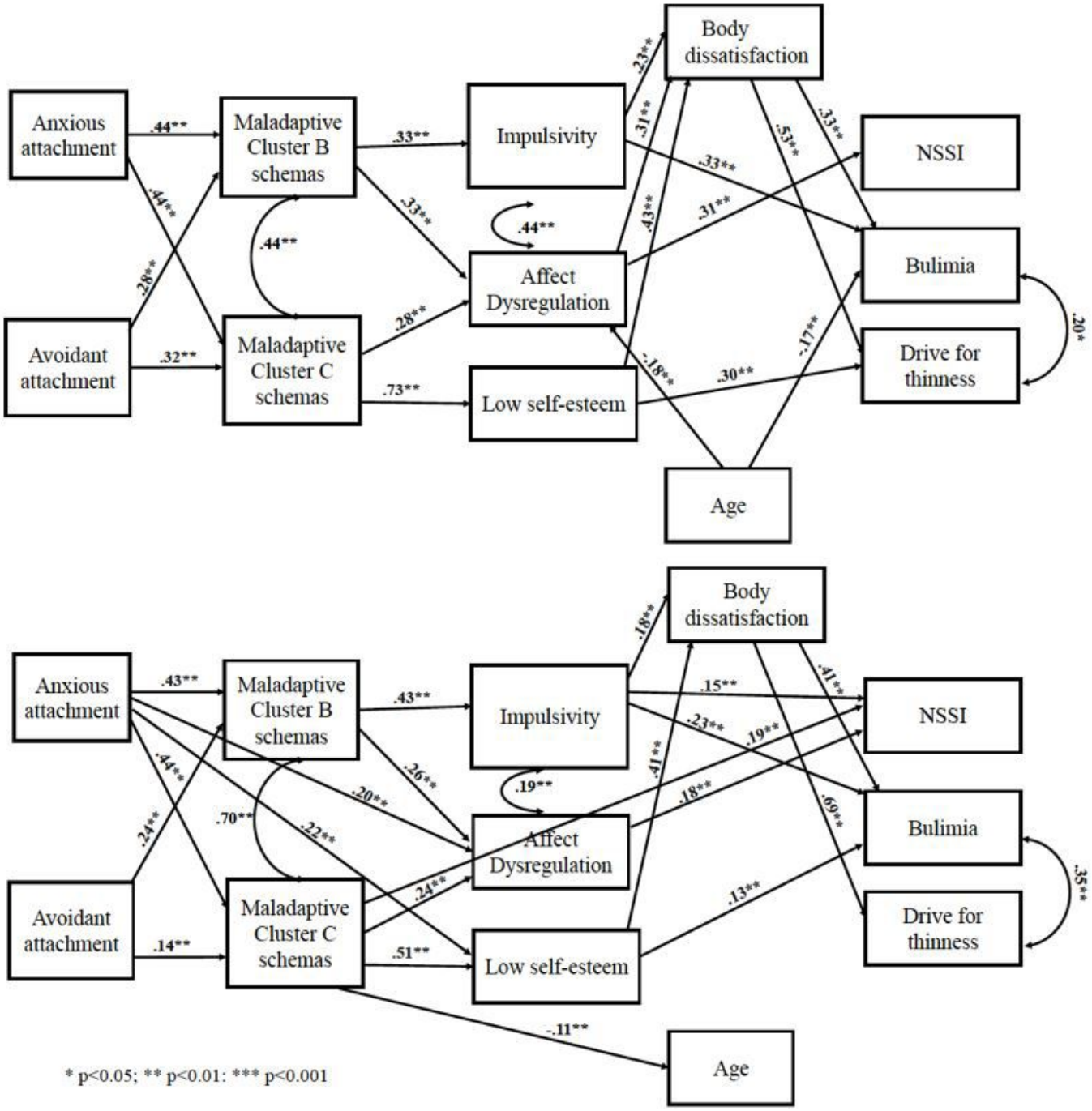

Figure 2

a. Proposed model results for the ED group b. Proposed model results for the community group

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- Table1.jpg
- Table3.jpg

- Table3a.jpg
- SupplementaryTable1NSSIEDJOFEDs.docx