Chapter

Risk and Maintenance Factors for Eating Disorders: An Exploration of Multivariate Models on Clinical and Non-Clinical Populations

Stefania Cella, Mara Iannaccone, Annarosa Cipriano and Paolo Cotrufo

Abstract

The recognition of factors involved in the development and maintenance of eating disorders (EDs) may support the choice of therapeutic strategies and improve the prevention/treatment of eating pathologies and their outcomes. Based on this consideration, the overall purpose of the chapter is to investigate how some psychological characteristics link to EDs. It is organized as follows. First, the epidemiological aspects, risk, and maintaining factors for ED are outlined. Next, we present the findings from our two studies. The purpose of the first study was to identify predictors associated with the severity of eating symptomatology. Then, the objective of the second study was to provide an understanding of the relationship among perceived parental bonding, self-esteem, perfectionism, body shame, body mass index, and ED risk and mainly to test a predictive ED risk model in a non-clinical sample. In conclusion, the major findings and practical implications are discussed.

Keywords: perceived parental bonding, self-esteem, perfectionism, body shame, body mass index, eating disorders, risk factors

1. Eating disorder risk and maintaining factors: an overview

Eating disorders (EDs) are highly prevalent psychological conditions characterized by abnormal eating behaviors that may lead to serious health problems and even cause death [1]. The existing diagnostic classifications of EDs include anorexia nervosa (AN), bulimia nervosa (BN), eating disorders not otherwise specified (EDNOS), avoidant/restrictive food intake disorder (ARFID), pica and rumination disorder. Additionally, the Diagnostic and Statistical Manual of Mental Disorders-5 (DSM-5) [2] supports binge eating disorder (BED) as a correct diagnosis on par with AN and BN.

In the framework of the European Study of the Epidemiology of Mental Disorders (ESEMeD) project, a lifetime prevalence rate of 0.93% for AN, 0.88% for BN, and 1.92% for BED have been found for females [3]. In a large population-based survey in the United States, Hudson and colleagues [4] have reported a lifetime prevalence of 0.9, 1.5, and 3.5% for AN, BN, and BED, respectively. More recently, a national survey has found a lifetime prevalence of DSM-5 defined AN, BN, and

BED of 0.80, 0.28, and 0.85%, respectively. Individuals with lifetime BED were found to have a later age of onset of ED and longer ED episodes duration [5].

Even though eating pathologies have been traditionally associated with females [6], males are also at risk for developing EDs [7, 8]. It was estimated that approximately 14% of AN [9], 10–15% of BN [10], and 40% of BED cases [11] were men. Prevalence rates of 0.3, 0.5, and 2.0% were found for AN, BN, and BED among men, who also accounted for approximately 25% of all EDs cases [4]. However, several studies have pointed out an upward trend in EDs prevalence rates among males [12–13]. Furthermore, empirical research shows that, in males, homosexual orientation is associated with higher body dissatisfaction and abnormal eating behaviors [14].

Adolescence to young adulthood is the peak risk period of onset for EDs symptomatology [15]. A recent longitudinal study, for instance, showed increases in weight preoccupation, body dissatisfaction, and bulimic behaviors from 11 to 25 years [16]. The Growing Up Today Study found that binge eating increased with age and peaked in late adolescence [17]. More generally, several authors have reported the presence of body dissatisfaction and drive for thinness even in children aged between 5 and 11 [18, 19] and have demonstrated that weight concerns, body dissatisfaction, and weight status increase with age [20, 21].

Some studies have found that the levels of EDs are highest in younger individuals [22]. Women with EDs with later age of onset (>25 years) might report less severe eating symptomatology compared with women with the typical age of onset (<25 years) [23].

In terms of the ED occurrence, several variables have been suggested as possible predisposing factors for these pathologies. In this section, we review some of the known risks and maintaining factors for the development of eating disturbances.

1.1 Parental bonding

It has long been recognized that family factors are essential features in the development, maintenance, and therapeutic outcome of EDs.

Selvini Palazzoli [24] was one of the first authors who observed some typical patterns in ED families functioning, such as an overprotective relationship with mother and a distant relationship with father.

Similarly, psychosomatic family model [25] suggested that a family environment characterized by enmeshment, overprotectiveness, and rigidity plays a key role in the etiology of AN. On the other hand, insecure attachment patterns were found to be prevalent in ED patients [26].

Overall, empirical evidence on parental bonding—generally assessed by the Parental Bonding Instrument [27]—highlights the importance of low paternal care and high maternal overprotection in the occurrence of ED symptomology in both clinical and non-clinical samples [28, 29]. Parental care refers to a continuum of behaviors ranging from affection and warmth to coldness and rejection. In contrast, parental protectiveness exists along a continuum that ranges from behaviors indicating encouragement of autonomy/independence as opposed to strict control with regulations and intrusiveness [27].

Yet, up till now, few studies have investigated whether parental bonding might be correlated with the severity of disordered eating symptoms. Among ED patients, high parental overprotection is associated with suicidal behavior [30]. Body image disturbances, considered as one of the major clinical features of eating pathology [31], resulted in being predicted by low parental care and high parental overprotection [29, 32].

In the study by Canetti and colleagues [33], anorexic participants reported perceiving both parents as less caring and fathers as more controlling than control

group participants; moreover, maternal control and paternal care were associated with higher symptom severity. A recent cross-sectional study has shown that the quality of the father-daughter relationship (i.e., overprotective and avoidant) plays a critical role in understanding the ED onset and maintenance [29].

Similar findings were reported by Rienecke and colleagues [34], the presence of paternal criticism—and not maternal—showed a significant predictive power for less psychological improvement in ED psychopathology at the end of family-based treatment for adolescents AN.

Overall, the overprotective behavior of the parents might be a result of the ED and often starts as a consequence of the disorder [35]. Researchers suggest that eating pathology may influence family dynamics and environment, which in turn may unconsciously affect ED symptoms maintenance and evolution [36].

In terms of the psychological dynamics underlying the association between parental bonding and eating pathologies, several researchers have suggested the existence of potential mediating mechanisms involved in such association. Turner and colleagues [37], for instance, reported that paternal care and maternal overprotection had an indirect effect on ED symptoms through the mediating effect of maladaptive core beliefs (i.e., schemas related to defectiveness/shame and dependence/incompetence).

Likewise, maladaptive perfectionism was found to mediate the pathway from parental psychological control and ED patterns [38]. In our previous study [28], on a large sample of adolescents, the link between the parental bonding pattern typified by low paternal care/maternal overprotection and dysfunctional eating attitudes were found to be mediated by self-concept. Our data were consistent with the study of Perry and colleagues [39] in which a parental bonding pattern characterized by low care and over-protection affected self-concept formation, which, in turn, affected eating psychopathologies in adulthood.

1.2 Self-esteem

The literature on EDs shows that a patient's self-concept is fundamentally characterized by low self-esteem, which is considered a critical vulnerability factor in the development of these diseases [40]. In a review focusing on causes of EDs, low self-esteem is one of the prominent features strongly implicated in the onset of the pathology [41].

In a series of interesting papers exploring the self-esteem dimensions, Geller and colleagues found that shape- and weight-based self-esteem and intimate relationship-based self-esteem were related to higher ED symptoms [42, 43].

It seems that white women are most at risk for having low self-esteem and difficulty with eating problems [44]. Still, several studies have shown that an impoverished sense of self is an essential contributor to ED symptomatology [45], and it is correlated with a negative treatment outcome [46]. Similarly, in a study carried out by Brechan and Kvalem [47], the effect of body dissatisfaction on restrained eating, binge eating, and compensatory behavior was completely mediated by self-esteem.

However, some studies do not support the existence of a direct relationship between low self-esteem and eating pathology. In some studies, self-esteem did not emerge as a significant predictor of disordered eating [48, 49]. Moreover, some researchers suggest that future studies should focus on a more informative multifaceted construct of self-esteem [50] and investigate interactive effects with other predisposing factors for a better understanding of the link between self-esteem and eating problems [45]. In this regard, self-esteem has been suggested to predict disordered eating via body shame [51], also among obese youth [52]. A study by Goossens and colleagues [53] provided evidence for the hypothesis that an insecure

relationship with parents may act as a mediating variable in the pathway from self-esteem and dysfunctional eating patterns.

1.3 Perfectionsim

Perfectionism is a personality trait that is characterized by setting excessively high personal standards of performance [54]. Frost and colleagues [54] viewed perfectionism as being constituted of adaptive (healthy) and maladaptive (dysfunctional) perfectionism.

In a cross-sectional research designed to explore perfectionism across different stages of EDs recovery, ED patients scored significantly higher than healthy controls on the maladaptive perfectionism factor [55]. Further studies have reported that elevated levels of both adaptive and maladaptive perfectionism are strongly associated with body dissatisfaction [56] and ED psychopathology [57–59], including BN [60]. A percentage of about 40% of AN patients (Mage = 15.3 years) have a very high score on self-oriented perfectionism and perfectionistic self-presentation. The authors concluded that in this subgroup of patients, it would be necessary to address these psychological characteristics to achieve a good outcome [61]. Several studies have highlighted that a higher level of perfectionism might be detrimental for disease duration and prognoses [62, 63], also among children and adolescents with EDs [64, 65]. Previous studies have shown that perfectionism predicts ED onset and maintaining [66]. Similarly, an experimental study has suggested that perfectionism represents a causal risk factor for ED pathology [67]. On the other hand, these findings are not systematically replicated in other studies: perfectionism—adaptive and maladaptive types—does not emerge as a risk factor for eating disturbances [68, 69], and the specific mechanism by which perfectionism uses its influence on eating psychopathology has, up till now, to be recognized.

In this regard, Bardone-Cone and colleagues [58] have reported that the investigation of mediating pathways from perfectionism to ED pathology was mostly absent from the literature. How perfectionism related to the EDs risk factors and potential mediating variables affecting the relationship between perfectionism and disordered eating remain mostly unknown.

Several studies support the association between low self-esteem, perfectionism, and varying degrees of ED patterns [70], "Indeed, the combination of low self-esteem and perfectionism is not unusual among those who binge, and especially those with BN, AN, or an atypical ED, and it may well contribute to the development of the problem" ([71], p. 65). Still, "as he was nearing the end of his life, Michelangelo began working on what many people believe to be his most important work, the Florentine Pietà. After working intensely for almost a decade, he entered his studio one day and took a sledgehammer to the sculpture. He broke away the hands and legs and nearly shattered the work before his assistants dragged him away. Why did Michelangelo attempt to destroy one of his greatest creations, a statue that has been described as among the finest works of the Renaissance? Disillusioned and isolated in the last decades of his life, Michelangelo had a heightened sense of perfectionism that was exarcebed by his failure to live up to the exceptions of his father, who viewed being a sculptor as akin to begin a manual laborer. Michelangelo, it seems, had self-esteem issues" ([72], p. 158).

Literature supports the hypothesis that a combination of low perception of control and low self-esteem moderates the effects of perfectionism on drive for thinness, BN, and body [73].

In conclusion, as previously suggested [74], the role of perfectionism in the etiology and maintenance of EDs remains unclear.

1.4 Body shame

Eating pathologies have been described as "disorders of shame" [75]. A positive relationship between shame and eating pathology has been found [76, 77].

However, several studies have concentrated upon shame explicitly associated with the body rather than to general shame.

Body shame is the shame one feels about one's body or any part of it [78]. Moreover, body shame can also relate to how one's body functions [79], and it represents a better concept than "body dissatisfaction" in work with EDs [80].

Body shame is a strong predictor of disordered eating [81, 82].

In a longitudinal study designed to explore the role of body shame and general shame in predicting increases in eating symptoms over 2.5 years in a sample of women with a past or current ED, body shame exclusively predicted an increase in AN symptoms [83]. Dorian and colleagues [84] have found that body shame is uniquely predictive of eating disturbance in a female clinical sample and in a male non-clinical sample. However, both body and characterological shame predicted eating psychopathology in a non-clinical female sample. In sum, body shame would seem to have a causal role in the ED onset.

In addition, the severity of ED symptomatology has been linked to feelings of bodily shame in the eating context [81, 85]. Troop and colleagues [83], for instance, found that shame was uniquely associated with the severity of both AN and BN symptoms. Goss and Gilbert [79] proposed a model based on the functional role of eating disordered beliefs and behaviors in the management of shame. The authors offered a model process based on risk factors (i.e., genetic factors, personality, early attachment history, abuse or rejection experiences, and cultural factors) that might predispose people to develop both shame proneness and ED proneness. These factors cause shame, and to defend themselves against adverse social outcomes, individuals may attempt to change their body shape and weight. Then, they may feel around in their ability to manage their weight, but when they are not able to do so, they feel further shame. This leads to a shame-pride circle that maintains the pathology.

Overall, shame can be described both as cause and consequence of symptoms in eating pathology [80]. Unfortunately, there are few findings about body shame, and much is unknown about how it operates in ED development. Literature supports the suggestion that body shame may act as a mediator in the relationship between self-esteem and disordered eating [51].

Regarding the determinants of body shame, some research has provided evidence associating poor perceived parenting and subsequent shame [86]. The perception of low parental care and high parental protectiveness in childhood was found to be related to shame in young adulthood [87]. Murray and colleagues [88] suggested that dysfunctional parenting practices may lead to individuals' feelings of inadequacy and worthlessness, so it might be clinically essential to examine the psychological consequences of such a family experience, such as shame. Specifically, the authors found that paternal overprotection was related to bulimic symptoms through the mediation effect of shame. On the other hand, it has been shown that parenting practices failed to predict the vulnerability to body image shame, directly or indirectly. In a study examining the determinants of body shame, Markham and colleagues [89] found that body-image esteem, global self-worth, appearance comparison, and internalization of the thin ideal accounted for 62% of the variance in body-image shame.

1.5 Body mass index

It would seem that the body mass index (BMI) plays a more critical role in promoting the risk factors for EDs than indirectly maintaining eating pathology [90].

Obese individuals are at higher risk for developing an eating pathology [91]. Indeed, a low BMI represents a protective factor against the development of disturbed eating in adolescent girls [92, 93]. Moreover, BMI in childhood is a significant predictor of restrained eating in early adolescence [94].

BMI at admission can be considered as a significant predictor of outcome in AN [95]. The link between BMI and mortality in BN has also been investigated. Severe BN patients may be at higher risk of death, especially if suicide has been attempted previously or in case of a low minimum BMI at admission [96].

In terms of the underlying mechanisms that linked BMI and eating psychopathology, in a study by Fan and colleagues [97], BMI was not found to have a direct influence on ED symptoms, and the authors concluded that weight control concerns and behaviors could mediate this relationship. To answer the question of what causes a high BMI, risk factors for obesity included parental fatness—although only a few longitudinal studies have investigated the parent-child fatness association—social factors, birth weight, timing or rate of maturation, physical activity, dietary factors, and other behavioral and psychological factors [98, 99].

Parental overweight is one of the main predictors for the development of childhood overweight and obesity [100], but parents can influence child body weight through specific feeding behaviors and practices, such as restriction, pressure to eat, and monitoring [101, 102], or more broadly through their general parental attitudes and style of interacting with children (for a review, see [103]). In a study about the influence of parental care in childhood on the risk of obesity in young adulthood, parental neglect was found to significantly affect the risk of adult obesity, independent of age and body mass index in childhood, sex, and social background. Instead, receiving overprotective parental support did not affect [104].

In terms of the psychological dynamics underlying parental-child relationship, possible mediating factors are considered. Overall, a growing body of research has focused on maternal sensitivity and emotion regulation. A poor quality of the early maternal-child relationship, characterized by low levels of maternal sensitivity, may be linked to childhood overweight and obesity through the development of potential difficulties in children's ability to regulate emotions [105]. In this regard, emotion dysregulation in early childhood is implicated in the development of obesity in early adolescence [106]. Similarly, empirical research suggested that authoritarian parenting (high control and low levels of emotionally responsiveness) may influence children's self-regulation skills [107] and, in turn, to be positively associated with child weight status [108, 109]. A longitudinal study showed that more inadequate maternal emotional regulation abilities during pregnancy were able to predict, at 7 months of age of the baby, the quality of the early mother-child feeding patterns [110], and the body mass index of the child at three years of age [111].

1.6 Multivariate etiologic models

Most of the studies about the ED onset have focused on the risk factors unconnectedly, precluding understanding about interactive effects.

In one of the few research in this area studies, Bardone-Cone and colleagues [112] found that perfectionism, body dissatisfaction, and self-esteem interact to predict bulimic symptoms. Specifically, women who perceived themselves to be overweight and who had elevated levels of perfectionism and lower levels of self-esteem were most at risk for bulimic symptoms. However, this interactive model has received mixed support [113–115], for example, it has been considered valid concerning maintenance and exacerbation, but not with the onset of bulimic symptoms [116]. Expanding the model to EDs patients under psychotherapy treatment, Watson and colleagues [74] found that binge eating and purging were not

significantly predicted by the three-way interaction term neither in concurrent nor in prospective analyses (i.e., examined as a moderator of treatment outcome). The authors concluded raising concerns about the robustness of the three-way model. Otherwise, it could have been affected by an inadequate conceptualization of the perfectionism construct.

A study by Ghaderi [117] suggested that a combination of low self-esteem, high body concern, low perceived support from the family, and more relative use of escape avoidance coping constitute a risk profile that later would lead to the development of ED. However, the author did not investigate if and how the predictor variables interact with each other in explaining ED onset.

In terms of the mechanism that perpetuates EDs, according to the cognitive-behavioral theory of the maintenance of BN [118], a dysfunctional system for evaluating self-worth is central to the continuation of the pathology. People with eating disturbances judge themselves principally based on their eating behaviors, shape, or weight and their ability to control them. Most of the other clinical features can be considered as stemming directly from this overvaluation of eating, shape, and weight that represents the "core of psychopathology". This original theory of continuance on BN could embrace four additional maintaining mechanisms, which concern the influence of clinical perfectionism, low core self-esteem, mood intolerance, and interpersonal difficulties. However, a common mechanism is involved in the persistence of BN, AN, and the atypical EDs resulting in the transdiagnostic theory of the maintenance of the full range of eating disturbances [119]. Moving from this theory, Lampard and colleagues [120] concluded that a mixture of factors (i.e., transdiagnostic and disorder-specific) might be involved in the maintaining mechanism of ED disorder symptomatology. The transdiagnostic model of EDs might be applied to improve our understanding of muscle dysmorphia, additionally to eating psychopathology [121].

2. Identifying predictors associated with the severity of eating concerns in females with eating disorders

Investigating factors that contribute to the onset and development of EDs has been the focus of previous studies. Several variables have been suggested as possible predisposing and perpetuating factors for EDs pathologies: perceived parental bonding, self-esteem, perfectionism, and body shame are among the factors that have been investigated separately. However, studies explicitly evaluating different predictors associated with the risk and severity of eating symptoms are limited in the literature [122, 123].

Based on this consideration, we have conducted a study [124] to identify predictors associated with the severity of disordered eating symptomatology. Identifying which of the individual variables (self-esteem, perceived parental care and protectiveness, body shame, and perfectionism) significantly predicted the severity of eating symptomatology for ED patients was the main research question that has driven our work.

The study was approved by the ethics committee of the Faculty of Psychology (University of Campania "Luigi Vanvitelli").

We gathered data from inpatients and outpatients referred to specialized residential ED treatment units in Northern, Central, and Southern Italy. At intake, a clinical interview was administered by ED clinicians for the assessment of diagnosis. All participants had a primary ED diagnosis DSM-IV [125]. Participants were tested at early stages—in order to avoid strong treatment effects—and at variables points during the treatment.

We screened 80 female eating disordered patients aged 13–40 years old through the self-report measures of parental behavior, self-esteem, perfectionism, body shame, and ED risk.

It is worth noting that the comparisons between AN, BN, and BED patients on the study variables highlighted only a few statistically significant differences. Based on these findings, patients who fall below the different diagnostic categories for eating disturbances seem to share several psychological characteristics. However, results indicate that greater severity of the eating symptomatology could be related to the diagnosis of BN. In our opinion, these results seem to further support the hypothesis of a shared psychopathological core of EDs, and BN could be regarded as a "failed" AN [126, 127]. Future treatment research should broaden the clinical understanding of this suggestion.

In line with empirical research, maladaptive perfectionism was found to be strongly linked to eating concerns, followed by body shame and low self-esteem. On the other hand, differently from previous studies, parental care and protectiveness were not related to the level of eating symptomatology. Linear regression analysis, as displayed in **Table 1**, demonstrated that maladaptive perfectionism ($p < 0.001$), body shame ($p < 0.05$), and self-esteem ($p < 0.05$), significantly predicted ED symptom severity, and explained a significant proportion of variance in ED symptomatology (adjusted $R^2 = 0.450$).

While both adaptive and maladaptive perfectionism were found to be correlated to EDs symptomatology, only maladaptive perfectionism was significantly and positively associated with eating concerns. These findings seem to support recent studies pointing out the role of maladaptive perfectionism—but not adaptive—in the prediction of eating symptomatology [57]. However, this datum could be due to an inadequate assessment of the adaptive perfectionism. Future studies should look at perfectionism as a multidimensional construct and further investigate the specificity of both functional and dysfunctional perfectionism contribution and their interplay with ED maintenance.

In line with previous findings [84], shame about the body emerged as a significant predictor of the level of eating concerns. However, research is needed because few studies have investigated the role of shame in ED maintenance, and further investigation might examine other forms of shame (specific and generalized).

Interestingly, these results seem to contradict the findings of previous research [49], and suggest that low self-esteem is a strong predictor of ED symptomatology. In this sense, low self-esteem consistently emerges as one of the core features of ED pathology.

Model	B	Standard error	β	*T*	P	R^2	Adjusted R^2	*F*
1. Maladaptive Perfectionism (constant)	4.608 .615	9.884 .109	.538	4.666 5.642	.642 .000	.290	.281	31.838
2. Maladaptive Perfectionism (constant)	−19.382 .491	11.081 .106	.430	−1.749 4.651	.084 .000	.403	.387	25.961
Bodily Shame	2.629	.689	.353	3.815	.000			
3. Maladaptive Perfectionism (constant)	9.059 .389	13.892 .105	.341	.652 3.696	.516 .000	.471	.450	22.537
Bodily Shame	2.173	.669		3.248	.002			
Self-Esteem	-1.106	.354		-3.126	.003			

Table 1.
Stepwise regression model and statistics for dependent variable [124].

Unexpectedly, perceived parental care and protectiveness were not found being associated with the level of eating concerns. In contrast with ED literature, these findings might be due to cultural differences. Furthermore, it is possible that perceived parental bonding might participate in promoting the risk factors for eating pathology rather than indirectly maintaining the disturbance. It would be interesting to clarify this question.

In our opinion, it is helpful to recognize psychological variables significant for considerations in the treatment of these patients. Identifying potential predisposing and maintaining factors may enhance our understanding of ED symptomatology and support the choice of targeted therapeutic strategies to improve ED treatments and outcomes. Specific attention should be paid to helping ED patients to improve overall self-esteem. In addition, the treatment of maladaptive perfectionism and body shame might help in reducing ED symptomatology.

In conclusion, our findings stress the need to investigate these factors further as they might represent negative prognostic factors.

3. Structural equation modeling of possible risk factors for eating disorder onset in female and male adolescents

Given the limited number of studies evaluating multivariate models explaining ED risk, research intended to fill this empirical gap was undertaken. Specifically, we have conducted a study [128] to assess the relationships among perceived parental bonding, self-esteem, maladaptive and adaptive perfectionism, body shame, body mass index, and ED risk with structural equation modeling. Several predictions were advanced concerning these potentially contributing factors. We hypothesized that perceived parental bonding, self-esteem, perfectionism, and BMI do not have a direct effect on ED risk. In our opinion, one potential mean by which these variables are related to ED vulnerability is through their effects on body shame.

Obtaining a clearer picture of how ED relates to these variables could result in an enhanced understanding of the mechanism through which such factors may lead to eating pathology.

This research was conducted on a sample of 1156 high school students—males and females—who ranged in age from 13 to 20 years. Participants were screened through self-report measures of parental behavior, self-esteem, perfectionism, body shame, and ED risk. The height and weight of each individual were measured. This age group was chosen as this cohort is at the most significant risk for eating disturbances, with ED incidences peaking during adolescence to early adulthood [129].

The study received the institutional review board approval.

The results only partially supported the hypothesized model (**Figure 1**), and several interesting findings emerged.

Several studies have documented an association between a bonding behavior pattern characterized by low care and high protectiveness and eating symptomatology [130, 131]. In line with previous empirical research [36], the model tested showed that poor parental care does not have a direct effect on ED risk, but it has a significant indirect effect through low self-esteem. Parental care has also an indirect effect on ED risk through the mediational effect of the BMI. Thus, perceiving neglectful parents may put adolescents at risk of developing obesity and eating problems.

Bearing in mind this suggestion, it seems reasonable that a high BMI is predicted by low parental care, but not parental protectiveness.

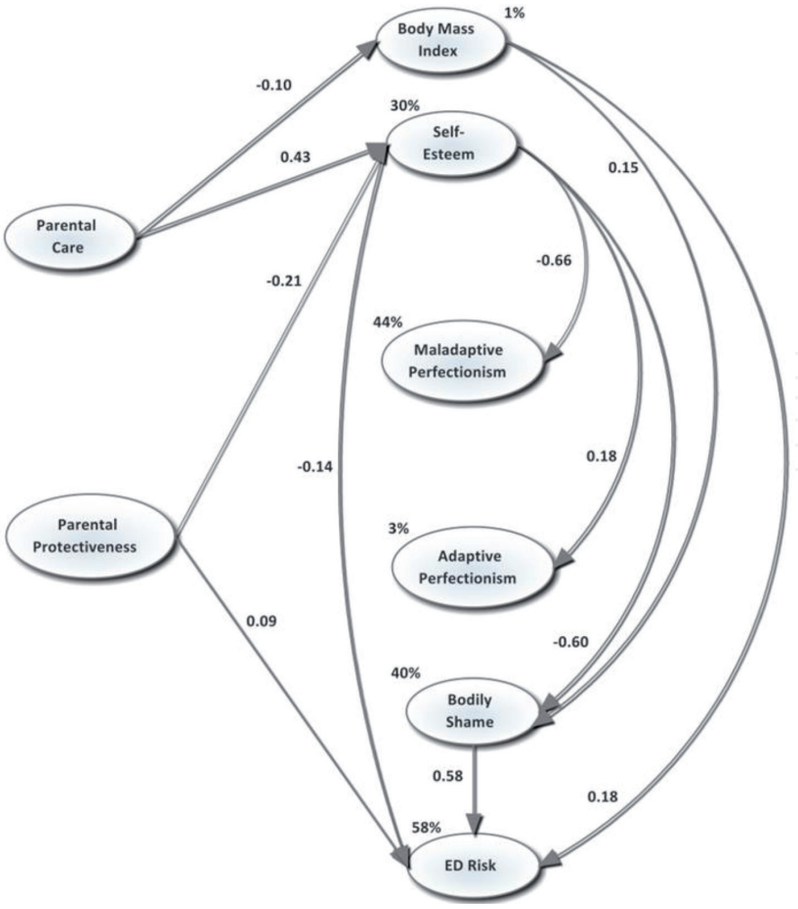

Figure 1.
Illustration of the final model with standardized path coefficients and percentage of variance explained [128].

Speculatively, it could be hypothesized that the relationship between parental overprotectiveness and ED risk might be mediated by the existence of other intervening variables, such as interoceptive awareness.

In this regard, parental protectiveness could not allow individuals to develop a sense of personal needs, altering their interoceptive awareness and, consequently, their eating behavior. Future empirical studies should investigate this suggestion.

Consistent with our hypothesis, parental protectiveness did have both direct and indirect effects—via low self-esteem—on ED risk, as previously suggested [39]. These findings are consistent with research that converges on the role of psychological control (i.e., parental protectiveness) in ED proneness. As such, this finding suggests that overprotectiveness may be more directly relevant to eating disturbance development. Research should further explore this datum and examine the unique contribution of each parent in the etiology of eating pathologies.

Results showed that self-esteem has a direct effect on both maladaptive and adaptive perfectionism. Specifically, the linkage between low self-esteem and maladaptive perfectionism was particularly strong. These findings offer further support to previous studies suggesting that striving to appear perfect is an attempt to compensate for low self-esteem [132]. Individuals with an ED may interpret their mistakes as evidence of personal deficiencies [133]. In this perspective, to be flawless represents an obligation.

Moreover, even if indirectly via self-esteem, a link between parental bonding and perfectionist orientation emerged. This datum partially supports the findings of Soenens and colleagues [38] who have evidenced a relationship between parental psychological control and maladaptive perfectionism.

Contrary to the initial hypothesis, parental care and protectiveness were not found as significant predictors of body shame. This agrees with findings from other

researchers [89]. One potential explanation may be that parental bonding exerts an indirect influence on body shame via self-esteem and BMI. Indeed, as predicted, both low self-esteem and BMI emerged as significant predictors of increased vulnerability to body shame. In this regard, low self-esteem may be regarded as a source of body shame. People who perceive themselves as inadequate or unworthy may be at the most significant risk of experiencing body shame [89].

Model results showed that perfectionism did not predict body shame, contrary to our initial hypothesis. However, the non-significant pathway from both maladaptive and adaptive perfectionism to ED risk offers further support, alongside other studies [68], that these variables may not be vulnerability factors for eating disturbances.

Due to its association with self-esteem, perfectionism could be regarded as a psychological characteristic typically associated with eating disturbances, rather than a risk factor for EDs. However, future research is needed to investigate variables that may intervene in the pathway from perfectionism to ED risk.

In line with previous studies [51], body shame emerged as a mediator in the relationship between self-esteem and ED vulnerability—explaining 71% of the variance. Notwithstanding, self-esteem has also a direct influence on ED. These results further corroborate previous studies recognizing low self-esteem as a critical predictor of ED vulnerability [40, 41].

Although body shame partially mediated the relationship between BMI and eating disturbance vulnerability, BMI had also a positive effect on ED risk. Undeniably, a high BMI has long been considered as a strong ED risk factor [91]. Our results support findings of previous studies [82, 83] suggesting that the experience of shame related to one's body exerts a strong influence on eating disturbances vulnerability. Yet our results suggest that in addition to a direct effect, body shame also serves as a mediator between other risk factors and eating disturbance risk. In this regard, body shame may be a key variable in the pathway to eating disturbances risk and a core diagnostic feature of all eating disturbances. Notwithstanding, only a few studies have investigated the role of body shame in ED onset. Further studies on this construct would be beneficial.

Finally, the evaluation of measurement invariance allowed to conclude that the final model was invariant across gender: no gender differences emerged in the hypothesized pathway to ED risk. Therefore, our study suggests that adopting the same prevention and treatment programs for both males and females may be appropriate.

In conclusion, we extend the work of others who have separately examined the role of perceived parental bonding, self-esteem, body mass index, perfectionism, and shame in EDs, identifying the possible mechanisms through which these variables increase the likelihood of eating problem development.

This is the first study that conceptualizes how several risk factors may work together to create a pathway to eating pathologies. Collectively, perceived parental care and protectiveness, self-esteem, maladaptive and adaptive perfectionism, body shame, and BMI account for 58% of the variance in ED risk. Therefore, these initial findings suggest a promising model.

Provided that the present findings can be replicated longitudinally, they have noteworthy implications for EDs prevention and treatment. Identifying which interpersonal characteristics and personal factors are most relevant in the etiology of EDs may help mental health professionals designing targeted prevention and/or intervention programs during adolescence. Clinicians should consider the routine assessment and treatment of these factors. First and foremost, intervention programs ought to be addressed to reduce subjective feelings of ineffectiveness and shame. Particular attention should be paid to obese youth.

4. Conclusions

Among psychiatric illnesses, EDs have the highest rate of mortality [1], and early detection of cases is essential. Several researchers have investigated the factors that lead to these pathologies in the hope that this information would help in the design of more efficient programs of prevention and treatment. However, few studies have been devoted to understanding how these risk factors work in concert to promote eating disturbance development. A psychological model of risk factors for developing eating pathologies in female and male adolescents was validated in our study [128]. From this model, we can conclude that several ED risk factors are linked among them and occur together to cause the eating disturbance opening up the possibility of translating these findings into a form of intervention.

Notably, a potential mechanism for eating problem onset has been identified.

Overprotective parents often anticipate the physical needs of their children and compromise, albeit unconsciously, their ability to recognize their own need and their autonomy. The perception of parental hyper-involvement and lack of sufficient caring may produce a feeling of ineffectiveness and an impoverished self, which, in our opinion, maybe the root of eating psychopathologies. It would seem that the sense of ineffectiveness that these individuals feel toward themselves has been moved in their bodies through a defense mechanism that works by substituting the object of a painful or dangerous feeling with an acceptable object. Such a mechanism would produce body shame: "I am not inadequate, but my body is". In Freudian psychology, this unconscious mechanism of defense is called displacement [134]. From that perspective, EDs could be an attempt to modify and/or to punish the ashamed body, which has been considered responsible for an individual's ineffectiveness. The body becomes the stage of the illness. This potential mechanism of action (parental overprotectiveness/low care → low self-esteem → body shame → eating pathology) might work in both obese and normal-weight adolescents, but among obese people, this would occur more often because it comes more natural to consider a fat body responsible for the sense of ineffectiveness [52]. In that respect, a person with a high BMI would be at greater risk of developing an ED than others.

This investigation presents a unique contribution to the literature by illustrating a promise predictive ED risk model. However, it is not possible to use the present data to argue for specific casual links, and more research is needed to validate these hopeful results.

Understanding the potential causes of eating problems would permit us to formulate aids in planning prevention/treatment. This etiological model for eating pathology onset could be transformed into a model for early and preventive interventions. Prevention programs that specifically target risk factors may be of benefit. However, the practical utility and clinical significance of this model ought to be examined in prevention studies.

As well, understanding possible psychopathological maintenance mechanism present enormous potential for eating disturbance treatment. Another goal was to determine the relationship of well knows ED risk factors with the severity of eating symptomatology. More specifically, we carry out a study aimed at examining whether BMI, perceived parental care and protectiveness, self-esteem, body shame, and perfectionism (adaptive and maladaptive) provide more information about the level of eating concerns among ED patients [124]. The small size of the sample limited this study: consequently, the structural equation model test could not be used for the clinical population. Overall, our results suggest that maladaptive perfectionism, body shame, and low self-esteem may represent an obstruction for successful treatment, and consequently, it ought to be targeted in psychotherapy. Particular attention should be paid to people with high BMI because they may present more severe eating

symptomatology. To sum up, the current results propose the necessity to consider these potential maintaining factors when designing treatment for this population.

The non-significant pathway from perfectionism to ED risk among non-clinical people seems to suggest that maladaptive perfectionism might represent a perpetuating factor in the ED maintenance exclusively. Perfectionism may correspond to a psychological correlate of the low self-esteem resulted from the need to compensate for a feeling of inadequacy as well as to a factor that makes the individuals more tenacious in achieving their own goal such as diet. In this perspective, perfectionism may be more substantially associated with AN.

On the other hand, parental bonding may play a key role in promoting the predisposing factors for eating pathologies rather than indirectly maintaining the pathology. As previously stated, parental bonding may create a vulnerability to eating disturbances principally through the development of a poor self-concept. The association between low self-esteem and eating disturbance development has been confirmed in our studies. By the potential mechanism of action described above, low self-esteem may represent the root of eating psychopathology and have a fundamental role in the onset as well as in the maintenance of the disorder.

The possible role of BMI in the beginning of EDs has also been established. It is known that obese people are more at risk of developing an ED. In our opinion, this might happen because obese individuals are more likely to move in their bodies the sense of ineffectiveness that they feel toward themselves, and this potential mechanism could also fuel the continuance of the pathology.

Finally, one of the strengths of these two studies includes the unique nature in investigating the role of body shame in EDs, a previously under-researched construct. Our data highlight the potential role of body shame as a critical variable in both ED onset and maintenance and as a core diagnostic feature of all eating disturbances. Further research is needed, but the preliminary results prove promising for application in a clinical setting.

Even though the current results contribute to valuable novel insights into EDs risk factors model, a large body of research proposes a biological model suggesting that genetic, immunological and metabolic aspects contribute to the development of EDs as well (for a review, see [135, 136]).

In conclusion, we focused on psychological risk factors that are important for the development and the maintenance of EDs. To differentiate between those predisposing, precipitating and perpetuating factors might help to develop more successful strategies for the prevention and treatment of these disorders.

Author details

Stefania Cella*, Mara Iannaccone, Annarosa Cipriano and Paolo Cotrufo
Observatory on Eating Disorders, Department of Psychology, University of Campania "Luigi Vanvitelli", Caserta, Italy

*Address all correspondence to: stefania.cella@unicampania.it

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