

## Invited Article

# Eating Disorders and Chronic Post Traumatic Stress Disorder: Issues of Psychopathology and Comorbidity

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*This article reviews in both directions the recent literature on the relationship between Eating Disorders (ED) and chronic Post Traumatic Stress Disorder (PTSD) to shed some light on an area of comorbidity for too long overlooked. PTSD may be the commonest anxiety disorder in ED but it seems to be independent from ED. Clinicians should be aware that PTSD might represent more than just an occasional occurrence in ED clinical work and should know how to approach it. Copyright © 2002 John Wiley & Sons, Ltd and Eating Disorders Association.*

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## BACKGROUND AND INTRODUCTION

Modern nosology (DSM III, APA, 1980; DSM III -R, APA, 1987; DSM IV, APA, 1994;) has clarified the diagnoses of Eating Disorders (Anorexia Nervosa, Bulimia Nervosa, Binge Eating Disorder, Eating Disorders Not Otherwise Specified; ED, AN, BN, BED, EDNOS) and of Post Traumatic Stress Disorder (PTSD). Over the last two decades all these diagnostic labels have bloomed in the number of scientific papers published each year. Both are now known to be common in the general population (Hoeck, 1993; Kessler *et al.*, 1995).

Furthermore, they have been 'secularized' by the media. In the public eye, they epitomize some aspects of the Western culture: concern for

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body weight and shape on one hand and victimization, emotional damage and reimbursement on the other.

ED and PTSD share a somewhat similar history: perceived as somatic conditions or mere spiritual afflictions according to different cultures and ages. They have often been missed or misdiagnosed by clinicians who perceived these syndromes as the 'expression' of something else (e.g. ascetism, Simmonds disease, monosymptomatic psychosis for ED; cowardice, shell shock, post-traumatic neurosis and compensation neurosis for PTSD). In fact, both ED and PTSD result from the evolution of the concept of hysteria to which they owe their first descriptions in psychiatry (Silvermann, 1995; Van der Kolk *et al.*, 1996).

Both researchers and clinicians in the field of ED and those into PTSD have always been aware of the complex and proteiform nature of these disorders. Both need integrative multidisciplinary approaches to be assessed and treated effectively and in both cases the body is secondarily affected by the psychological state which is in turn worsened by the ensuing physical condition.

However, in ED treatment, the clinician deals with the relation body–mind from a somehow opposite perspective than that of his counterpart who treats PTSD. In ED, there is a justified concern, for somatic complications and medical experts are so often involved in the cure of ED. In contrast, an almost total lack of concern for the psychosomatic dimensions of traumatization is the rule in PTSD clinical treatments (Foa *et al.*, 2000). The main symptoms targeted are intrusive thoughts of the trauma, avoidance of it and dysfunctional autonomic activation.

Both ED experts and 'psychotraumatologists' are aware of the difficulties of their positions: the former deal with the somatically oriented mental state of patients who need to break through to the psychological in order to proceed with therapy. Finding meaning in trauma is alluring and may bring more material to the therapeutic sessions. On the other hand, PTSD clinicians are aware of their patients' discomfort with the body but they find hard it to reach the 'body' (Van der Kolk, 1996). Knowing that trauma is etched in the non-verbal parts of the brain they would find it helpful to have access to trauma at more somatic levels. The notion that an ED might be associated with traumatic stress, with PTSD or even be a somatic 'equivalent' of PTSD could open the way to new stimulating thoughts and, possibly, to innovative clinical developments (Torem and Curdue, 1988).

This article was not intended as a review of the meaning of trauma in ED aetiology. Instead, it will straddle both the field of eating disorders and that of post traumatic stress and will exam the relationships between them. Only ED literature in which a complete PTSD diagnosis was formulated has been taken into account.

## THE DIAGNOSES AND CLINICAL FEATURES

PTSD is currently conceptualized as a complex anxiety disorder with an oscillation between reexperiencing the trauma in memories and dreams, on one hand, and generalized avoidance of stimuli reminiscent of the event on the other. Autonomic activation is present in phasic states, especially when trauma is reexperienced. The course of PTSD can be chronic and therapy-resistant (Kessler *et al.*, 1995) and its impact on the individual's quality of life can be devastating.

Despite their apparent distance in presentation and clinical picture between PTSD and ED, there are similarities. Firstly, the two disorders share the presence of ruminations. They tend—annoyingly—to be called 'obsessive' by many in ED and 'intrusive' in PTSD. Indeed, intrusions may be the hallmark of PTSD. When compared with typical obsessions, both the intrusions of PTSD and obsessive thoughts of ED have a stronger link with objective 'external reality' (trauma; food).

In AN, the phobic and obsessive ruminations about food are followed by restraint and avoidance, with a worsening effects of obsessive thoughts. In BN, the response to persistent thoughts about food tends to be fear of situations where food is involved with avoidance until a spell of craving explodes in the form of a binge attack, reinforcing the bulimic cycle (Fairburn, 1995). In PTSD the mind tries not to think of the trauma but this inevitably, triggered by internal as well as external minor cues, exerts an almost magnetic attraction to these thoughts. Painful memories and the distressing accompanying emotions are brought back to the present with immediate psychological and physical discomfort. Further generalized avoidance for situations and relations potentially reminiscent of the trauma follows. In both ED and PTSD, the resulting aggravated symptoms may worsen self-esteem and depressive states and the interruption of this vicious circle is vital to improvement. Treatments rely on lessening the power of the bingeing/purging cycle and of the intrusion/avoidance oscillation (Horowitz *et al.*, 1979).

Favaro *et al.* (2000) in a group of binge-eating Holocaust survivors with PTSD noticed that the victims continued until the present to have 'persistent and specific thoughts about food and eating'. Interestingly, central serotonin imbalance is postulated both for ED and PTSD. This is supported by the therapeutic effect of serotonergic agents such as fluoxetine for ED (APA, 1993) and sertraline for PTSD (Gottlieb, 1999).

A second psychopathological symptom commonly found both in ED and in PTSD is dissociation.

Even though the presence of dissociative states is not an essential feature among DSM IV symptoms with diagnostic value, its importance both in ED and PTSD is well known and widely recognized. Dissociative

symptoms can occur both in the dynamic of the trauma (Marmar *et al.*, 1994) and during the course of chronic PTSD. Amnesia for the traumatic episode, flashbacks in which the trauma is re-lived or re-enacted with feelings of estrangement from the traumatic memories are all forms of dissociation seen in PTSD. Dissociative reactions are thought to occur in the face of overwhelming traumatic stress in the form of a freezing/numbing state. ED patients may present high levels of dissociative symptoms. Comparing 103 ED patients with an age- and sex-matched healthy control group Dalle Grave *et al.* (R. Dalle Grave *et al.*, presentation at the Fifth Annual Spring Conference of the International Society for the Study of Dissociation, Amsterdam, 1995) found dissociative symptoms in 20 per cent of patients, a figure significantly higher than that of controls. The bulimic symptomatology showed most association with dissociative states.

Bingeing often occurs in a blurred and 'disconnected' mental state (see Vanderlinden and Vandereyken, 1997a) and might be a way to obtain an analgesic response against pervasive anxiety and desperation. According to Nijenhuis and Vanderlinden (1995) a very young child has little alternative to a paralysis response in the face of extreme stress except for refusal and rapid ingestion of food as seen in stressed animals. Age-related immaturity of the major information processing organizer of the brain—especially the hippocampal system—would explain the lack of more sophisticated coping strategies. Such reactions could result in permanent change in eating patterns, creating the base for an analgesic defensive binge mechanism in front of stress that might be resistant to change.

## CO-MORBIDITY

The concept of co-morbidity refers to coexistent psychiatric diagnoses with no necessary inference about their interdependence. Both ED and PTSD are characterized by high rates of co-morbidity, especially for alcohol and substance abuse/dependency, depressive and anxiety disorders.

From the perspective of ED, the practical advantages of detecting co-morbid PTSD over merely looking for histories of traumatization are several. Firstly, detection of PTSD allows for the study of more objective clinical phenomena with increased opportunity for new research. Secondly, no inference by the researcher about causation is warranted and no expectancy is fostered of a cathartic effect on ED symptoms after the 'resolution' of trauma. Thirdly, and most importantly, the ED patient might benefit from having disturbing PTSD symptoms detected and treated on the basis of 'state of the art' therapeutic guidelines.

There is increasing research interest in the issue of co-morbidity. Two of the most celebrated 'psychotraumatologists' of our time have published scientific papers on ED (McFarlane *et al.*, 1988; Van der Kolk *et al.*, 1991). Some authors have studied this comorbidity in non-clinical populations (Dansky, 1997, 2000; Favaro and Santonastaso, 2000), others have studied already diagnosed subjects (Turnbull *et al.*, 1997) or severely affected inpatients (Gleaves and Eberenz, 1994; Gleaves *et al.*, 1998; Lipshitz *et al.*, 1999; Striegel Moore *et al.*, 1999).

Other papers, mainly case reports, describe difficult multi-personality patients who also had very complicated and atypical ED and PTSD diagnosis decades after childhood abuse (Levin *et al.*, 1992; Tobin *et al.*, 1995; Vanderlinden and Vandereyken, 1997b).

However, there are major differences between the ED and the PTSD literature. For instance, the typical participant in any ED clinical study is a young female while PTSD studies tend to consider ageing male war veterans. Moreover, PTSD therapeutic response might be different in civil subjects and, particularly, in women (Gottlieb, 1999). New lines of PTSD research tend to consider samples drawn from the general population.

## PTSD IN THE COURSE OF ED

Only recently, after considerable time spent in debate and research over the presence and meaning of trauma, especially sexual abuse, in the premorbid history of ED patients (Palmer, 1995), has the focus been shifted from the traumatic history to the presence of current PTSD symptoms in ED patients (Gleaves and Eberenz, 1994).

Looking at a non-clinical population (3006 female subjects) Dansky *et al.* (1997) screened for victimized and traumatized subjects in the bulimic responders. They found lifetime and current PTSD respectively in 36.9 and 21.4 per cent of BN patients, compared with 21 and 7.1 per cent of BED patients and 11.8 and 4.2 per cent of the non-ED population. When controlling for victimization the prevalence of BN among the victimized and PTSD positive subjects (7.9 per cent) was significantly higher than that of victimized subjects without PTSD (2.7 per cent) and the non-victim group (1.7 per cent). ED and PTSD prevalences were 2.5 and 12.5 per cent, while that for major depression was 15.2 per cent. PTSD subjects were considered to be at 3.36 times the risk of developing BN, while direct victimization alone increased the same risk by 1.86 times. A second analysis of the same population revealed that the clinical significance of PTSD in ED might extend to the role of a mediator between BN and other psychiatric disorders such as alcohol abuse (AA)

and dependence (Dansky *et al.*, 2000). PTSD prevalence was higher than that of Major Depression both in BN with AA and in the BN without AA groups. These data contrast with those of Lilenfeld (Lilenfeld *et al.*, 1997) who found PTSD more frequently in the BN without AA group. Dansky *et al.* concluded that comorbid PTSD could be one of the reasons why AA is frequent in BN.

Turnbull *et al.* (1997) diagnosed lifetime PTSD in 11 per cent (18/164) of a mixed AN–BN ED female British patient population. PTSD did not seem to be associated with a specific ED diagnosis. So far this was the only ED–PTSD comorbidity study performed in the UK and its results have to be considered in the context of the lack of epidemiological data on PTSD in the UK. High parental control was the most commonly found childhood adversity theme reported by PTSD patients, exceeding in frequency both child sexual and physical abuse (44.4, 23.1 and 23.5 per cent of PTSD patients, respectively). Of PTSD-positive subjects 44.0 per cent reported having had a controlling environment versus 6.0 per cent of PTSD-negative subjects.

The authors speculate that an ED patient growing up in a controlling environment might be less skilled and therefore at greater risk of developing PTSD when confronted with life stress. The atmosphere of upbringing may undermine the sense of personal security and predispose to PTSD in later life. The central feature of the traumatic experience, at least in ED childhood histories, might be the emotional rather than the physical or sexual abuse as suggested by Kent who carried out a study on 236 female volunteers (Kent *et al.*, 1999).

Knowing the type and nature of the trauma may not be essential when looking for the presence of PTSD symptoms (Gleaves and Eberenz, 1994; Gleaves *et al.*, 1998).

In their first study on ED treatment resistance Gleaves and Eberenz (1994) found that highly victimized patients were much at risk for treatment failure. They hypothesized that such failure could have been explained by the presence of post traumatic symptoms and recommended treatment of those symptoms before starting ED therapy. In their study on a population of 294 hospitalized women with ED (AN, BN and EDNOS), the same authors found high rates of current PTSD self-rated symptomatology at admission to the ED unit. In fact, 74 per cent of subjects reported at least one traumatic event and 54 per cent of the sample could be diagnosed as suffering from lifetime PTSD regardless of the severity and quality of their ED. However, ED and PTSD symptomatology seemed independent and the latter was associated with significant depression, anxiety and dissociation. Since no healthy or psychiatric comparison groups were employed, caution should be used in the interpretation of such high PTSD rates.

Interestingly, this study did not consider the possibility that trauma-negative ED patients might have PTSD symptoms. This is not a contradiction in terms, since traumata not meeting the requirements for DSM-based operational criteria for traumatic events might be enough to break the lowered threshold to trauma of the ED patient. Moreover, the possibility that ED patients might have PTSD without an acknowledged defined precipitant trauma, or with no memory of it, should be explored, especially when knowing that PTSD symptomatology can be highest in subjects who 'can't tell' their trauma (Vrana and Lauterbach, 1994).

## ED IN THE COURSE OF PTSD

In spite of the lack of psychosomatic implications in the DSM criteria for PTSD, we know that a significant burden is posed by traumatized people on primary medical care and on general hospitals (Van der Kolk, 1996). However, we know very little of the body of the traumatized patient and about his/her eating behaviour. Not a single paper has been published on the eating patterns of known, clinically active PTSD cases. Perhaps this is not surprising given the predominantly male gender-based nature of typical PTSD studies. However, in a group of 120 Bosnian women with post traumatic symptoms, lack of appetite and body weight, more than vomiting, were highly correlated with the status of refugee and were deemed to be reactions to the trauma of displacement (Ljubotina and Libby, 1998). In the same sample the frequency of 'menses problems' changed from 25.5% before the war to 42.6% after it. The mediating effect of depression was very likely in this sample. Prisoners of war (POWs) are a population in which PTSD may reach 80 per cent in frequency (Sutker *et al.*, 1993). Many are known to be starved in captivity and may remain underweight for decades after release (Wolfe and Kimerling, 1996). Suicide and severe psychopathology are common in these subjects. AN might be a syndrome chronically present in some cases (Der Loos, 1990; Tobin *et al.*, 1995).

Bingeing is a behaviour associated with traumatization and PTSD in POWs (Polivy *et al.*, 1994) and in Holocaust survivors (Favaro *et al.*, 2000). Polivy *et al.* found binge eating in 198 POWs but not in 67 World War II combat veterans.

Favaro screened 51 Italian Nazi concentration camp survivors for Binge Eating (BE) and PTSD. Lifetime BE was found in 33 per cent of cases and current BE in 14 per cent. Lifetime PTSD rates were 35.3 per cent and current 25.5 per cent. Even if the association between the two disorders did not reach statistical significance (39 per cent in those with PTSD and 30 per cent in those without PTSD), BE and PTSD were still

clinically relevant some 50 years after internment. Furthermore, in a study of the offspring of Holocaust survivors (100 subjects, 71 females) Yehuda *et al.* (1998) found two current ED cases and 15 PTSD. No ED cases and only one PTSD were found in the 44 age-matched subjects (21 females) in the comparison group. An explorative pilot study (Y. Reibman *et al.*, poster at the IIIrd World Conference for the International Society for Traumatic Stress Studies, Melbourne, March 2000) found elevated EDI -2 and EAT 26 mean scores in a group of 10 civil patients attending a PTSD clinic in Jerusalem after a variety of traumatic events (seven car accidents, two war, one terrorist attack).

All subscales were elevated except Body Dissatisfaction in EDI-2. 'Dieting' scores stood out among EAT-26 subscales. However, no complete ED diagnostic screening was performed on the subjects and the reliability of the use of specific ED screening tools in such a context is questionable. In a small cohort of civil victims of kidnapping (24 subjects, 19 males), A. Favaro *et al.* (2000) found that after captivity at least one had developed BN. Occasional case reports have been published on AN following physical and psychological trauma such as a severe car accident. Damlouji and Ferguson (1985) reported that distortion in body image and self perception following trauma was particularly linked to the disfigurement perceived, or misperceived, after the injury. Marked depression was also present.

A study on motor vehicle accident (MVA) survivors found 8.1 per cent current and lifetime ED in the 62 subjects with full PTSD out of 158 participants versus 4.4 per cent in those with subsyndromal PTSD, 3.9 per cent in the MVA survivors without PTSD and 2.2 per cent in matched controls not exposed to MVA (Blanchard *et al.*, 1995). Most of the study participants were female (68.4 per cent) and were recruited through advertisements offering psychotherapy. It is therefore likely that the rates of ED reflect a biased sample and perhaps the prevalence of a pre-existing condition. In contrast, no evidence of change in eating patterns was acknowledged by the 105 subjects of a study on 'whiplash' who were asked about changes in biological rhythms after in the first 3 months after the crash (Mantero *et al.*, 2001).

## SEX- AND AGE-RELATED ISSUES

Age and gender affect both vulnerability as well as resilience in the face of trauma. It should be noticed as an example that Child Sexual Abuse (CSA) does not fulfil strictly applied criterion A for the diagnosis of PTSD. Nevertheless, its devastating effects are now beginning to be known and



fully acknowledged. Young age, a risk factor for PTSD, contributes to the resulting damage, described as 'complex PTSD' (Herman, 1992).

Looking for diagnoses of PTSD in 74 adolescent psychiatric inpatients Lipschitz *et al.* (1999) found 24 (32.4 per cent) subjects meeting criteria for current PTSD and CSA was the commonest trauma (69 per cent). Six (25 per cent) of those with PTSD had a concomitant ED, versus 3 per cent in those without. Rates of Somatization Disorder was also very high (34 per cent) in PTSD adolescents. Interestingly, males with PTSD were more likely to have a comorbid ED than females. Dissociative symptoms as well as depression and suicidality were very high in both genders.

Examination of large numbers of discharge notes of Veteran Administration psychiatric populations led Striegel Moore *et al.* (1999) to find that 0.30 per cent of female veterans (mean age 51.4 years) and 0.02 per cent male veterans (mean age 60.2 years) had an ED (Striegel Moore *et al.*, 1999). Mood disorder, anxiety disorder and personality disorder were the commonest diagnoses of this subsample. PTSD was the commonest anxiety disorder in women with ED. One-quarter of females with ED had a co-morbid PTSD as opposed to 8 per cent in the non-ED clinical population. Overlap between PTSD and Borderline Personality Disorder was modest, whilst the sample in general showed high rates of co-morbid psychopathology.

As for men, ED was registered more often as a secondary diagnosis and high co-morbidity rates for mood disorder, substance abuse or dependence disorder, mood disorder, schizophrenia or other diagnoses were found. No relevant association with PTSD (8 per cent in ED versus 12 per cent in the general clinical population) was found in men.

Reactivation of remote traumata explains many cases of late onset PTSD, sometimes in the second half of life or even old age when vulnerability to trauma is on the increase. Interestingly, cases have been reported in which common physical traumata were followed by the insurgence of ED and PTSD in well-functioning subjects with a history of CSA (Tobin *et al.*, 1995). A hypomanic and hyperphagic lifestyle was predominant before the clinical onset of ED, precipitated by a bone fracture or a surgical trauma. This clinical evolution was considered by the authors as the depressive collapse of a defensive strategy against severe CSA post traumatic symptoms in the face of a reactivating situation.

## DISCUSSION AND FUTURE DEVELOPMENTS

The present evidence appears to be too scanty to draw any definite conclusions on the subject. There are still more questions than answers.

However this article may offer food for further thoughts on a clinically relevant issue. The possibility that ED and PTSD might co-exist in the same patient is there and should be taken into account. Perhaps some provisional conclusions can be attempted.

### *ED onset after trauma and in the course of chronic PTSD*

The debate over the post traumatic aetiology of ED has a long history (McFarlane *et al.*, 1988). Results from the National Women's Study showed traumatic events preceding BN onset were detectable in 84 per cent of cases (Brewerton *et al.*, 1999). In fact, the incidence of many psychiatric disorders, beside PTSD, is elevated after trauma (McFarlane and Papay, 1992). It seems probable that ED might be among them. Systematic studies are lacking.

However, since the perception of trauma is highly subjective it is also probable that a number of different stressors might be involved, including environmental and educational styles of upbringing. The pathogenic relevance of sexual abuse should not overshadow the role played by other childhood traumatic experiences that might have major emotional impact. It is also possible that early traumatic events are reactivated by common life events (e.g. losses, surgical operations, car accidents) in situations of increased vulnerability (e.g. retirement, relocation) with precipitation of an ED among other psychopathological symptoms (Tobin *et al.*, 1995). ED can be precipitated by major life stressors in which attachment bonds are threatened as in sexual abuse or in which severe and prolonged starvation is involved as in captivity. In both cases concomitant PTSD symptoms are likely to be present.

Data on POWs and on Holocaust survivors reveal that reduction in body weight and altered food intake can have persisting effects after captivity. Favaro *et al.*, 2000 in a group of PTSD-positive binge eating Holocaust survivors noticed in a recent study that the victims have, to this day '...persistent and specific thoughts about food and eating'. High rates of PTSD are typical of these subjects raising the possibility that post traumatic symptoms can be thematically related to food and eating restrictions.

Binge eating could be a way of relieving tension in the traumatized. They might develop an ED as a non-verbal method of regulating affect and reinforcing a sense of self by adhering to the general demand for thinness (Rorty and Yager, 1996). It is likely that alteration of food intake, when present, follows the phasic time course of chronic PTSD and is subject to spontaneous aggravations and remittance. The role played by age and gender in increasing the vulnerability to an ED-PTSD double diagnosis is unclear. However, previous severe trauma such as

CSA might be reactivated by adult life events leading to complex psychopathological presentations.

### *PTSD onset in the course of chronic ED*

It is probable that the presence of an ED increases vulnerability to traumatic events resulting in minor traumatic events reaching the threshold of PTSD. It would seem very likely that concomitant depression would also be present, at least in adults. Available data do not allow speculation as to which type of ED might particularly increase vulnerability to trauma. Unfortunately, none of the studies examined has considered the presence of subsyndromal or partial PTSD (Schnyder *et al.*, 2001), where only intrusion plus either avoidance or hypervigilance DSM IV criteria are met. The assessment of subclinical forms of PTSD might have helped to assess the clinical importance of post-traumatic symptoms and maybe explain discrepancies in current lifetime PTSD rates across studies. It is also possible that ED and PTSD symptoms are part of a complex chronic clinical picture, are intertwined in presentation (Vanderlinden and Vandereyken, 1997a) and seem to have developed together. Other relevant comorbid psychopathological conditions would often be present: dissociative disorders, depression, anxiety disorders or substance abuse. Impulsivity might be the predominant behavioural style and dysfunctional personality traits would also be present. A history of child neglect and abuse is also likely to be traced.

At a theoretical level, very little is understood about the effect of severe food restriction on the mind. Is dietary restriction a 'traumatic' stressor? Should it be included in PTSD criterion A DSM IV definition of trauma? Might the enduring pain of food restraint and severe dieting parallel Type 2 trauma, a prolonged traumatic stressor (Terr, 1991)? Might it precipitate disordered eating patterns that become chronic and resistant such as the alterations in food intake seen in POWs, in Holocaust survivors or in the typical ED patient? Severe dieting is a self-inflicted sufferance and can be a deadly threat to health. Its consequences on the human mind might encompass, especially in the vulnerable individual, alterations of thoughts, craving and avoidance as well as hypersensitization to external reality with enhanced reactivity and depression, making the analogy between ED and PTSD clinically sound. This possibility is supported by claims that Eye Movement Desensitization and Reprocessing (EMDR), a controversial yet interesting method to access traumatic memories (Shapiro, 1989), along with other techniques like hypnosis (Vanderlinden and Vandereyken, 1997b) might be of help in overcoming the past traumata of the ED patient.

However, evidence of the success of these methods in the treatment of ED remains at the moment only anecdotal (Hudson *et al.*, 1998).

## CONCLUDING REMARKS

Overall this article aims at re-evaluating the importance of PTSD in routine clinical work with ED patients. It should be remembered that PTSD is probably the most devastating anxiety disorder, it is particularly common in women (Kessler *et al.*, 1995) and it is often hidden by the patient, due to lack of insight and awareness of the pathological quality of its symptoms. Nevertheless, it is very disturbing and it is a risk factor for suicidal depression and substance abuse. Hence, attention should be given to traumatic histories in ED and, even more crucially, to the clinical indicators of traumatic stress such as PTSD nuclear symptoms (intrusions, avoidance and hyperarousal).

Given the pandemic proportions reached by body shape and weight concerns in the Western world, it is possible that subjects with all kinds of complaints seek treatment for a supposed ED while having other severe psychological problems which are difficult to verbalize. As an example, binge eating disorder (BED) might be the presenting complaint of an abused woman with undiagnosed PTSD. Evaluation and treatment of PTSD before the ED might be indicated in order to avoid unnecessary ED treatment failures (Gleaves and Eberenz, 1994; Dansky *et al.*, 1997). Concealed PTSD presenting with an ED problem is an occurrence that deserves attention. The clinician needs a certain degree of familiarity with PTSD if treatment is to be successful. In practice, screening for PTSD should be part of the routine assessment in ED services.

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