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CHILD SEXUAL ABUSE PREDICTS POOR OUTCOME

Co-occurrence of avoidant personality disorder and child sexual abuse predicts poor outcome
in longstanding eating disorder

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Abstract

Few consistent predictive factors for eating disorder have been identified across studies. In the current five year prospective study, the objective was to examine whether (1) personality disorder and child sexual abuse predict the course of severity of eating disorder symptoms after inpatient treatment and (2) how the predictors interact. A total of 74 patients with long standing eating disorder and mean age of 30 years were assessed at the beginning and end of inpatient therapy and at one-, two- and five-year follow-up. Mixed model was used to examine the predictors. Avoidant personality disorder and child sexual abuse interacted in predicting high levels of eating disorder over a long-term course. These results suggest that eating disorder, avoidant personality disorder and sequelae after child sexual abuse are potential targets for treatment that need further investigation.

Keywords: Eating disorder, personality disorder, child sexual abuse, mediators, moderators.

Co-occurrence of avoidant personality disorder and child sexual abuse predicts poor outcome in longstanding eating disorder

Eating disorder (ED) rank among the 10 leading causes of disability among young women (Mathers, Vos, Stevenson, & Begg, 2000) and anorexia nervosa (AN) has the highest mortality rate of all mental disorders (Millar et al., 2005; Zipfel, Lowe, Reas, Deter, & Herzog, 2000). Factors predicting treatment outcome and course in ED have been sought mainly in three specific areas; individual, environmental and therapeutic. So far, few predictive factors have been replicated across studies. Several studies have evaluated the effects of personality disorder (PD) on the course and outcome of ED. Obsessive-compulsive PD traits have been found to be a poor prognostic feature among anorexic patients (Lilenfeld, Wonderlich, Riso, Crosby, & Mitchell, 2006; Steinhausen, 2002), whereas histrionic PD indicated a favorable outcome (Steinhausen, 2002). Avoidant PD is one of the most prevalent PD in the ED literature (Rosenvinge, Martinussen, & Ostensen, 2000). The Diagnostic and Statistical Manual of Mental Disorders Fourth Edition (DSM-IV) reports prevalence of avoidant PD between 0.5 and 1.0% in the general population however, across ED diagnoses this increases from 16 to 27% making it one of the most common PD diagnoses in the ED population (Sansone, Levitt, & Sansone, 2005). The longitudinal study on treatment-seeking anorexics and bulimics from Herzog found no evidence that PD predict either longitudinal outcome (Herzog et al., 1999), or ED relapse (Keel, Dorer, Franko, Jackson, & Herzog, 2005). However, a reanalysis on the same sample revealed that avoidant-insecure scores showed consistent associations with poor functioning and outcome, including failure to show ED improvement, poor global functioning after five years, and high treatment utilization after five years (Thompson-Brenner et al., 2008). Despite the high prevalence of avoidant PD, there is still limited understanding of the specific impact of avoidant PD in

patients with ED on the medium to long-term course of ED. In our study we have used both borderline, obsessive-compulsive, and avoidant PD as predictors in the analyses.

Very few studies have examined whether having experienced child sexual abuse (CSA) could predict a poor outcome in ED, despite clinical experience indicating that this may be the case. Rodriguez et al. (Rodriguez, Perez, & Garcia, 2005) found that a history of sexual abuse and exposure to other violent acts is related to poor early response to treatment of ED, as well as to greater dropout and relapse rates. However, as these authors point out, there is a need to evaluate the long term impact of such a history, and whether CSA may predict treatment outcome in ED. There may be a high prevalence of CSA among individuals seeking inpatient treatment for AN (Carter, Bewell, Blackmore, & Woodside, 2006) and a history of CSA can be associated with more severe psychiatric disturbance overall and a higher rate of dropout for patients of the binge-purge subtype (Carter et al., 2006). In summary, there are reasons to hypothesize that CSA among ED patients may predict a poor treatment outcome and a non-favorable course.

CSA and PD may interact in several ways to affect ED outcome and course. First, CSA could moderate the effects of PD on the ED course. For instance, avoidant PD may interfere more with improvement in ED symptoms during and after treatment in the presence of the post-traumatic sequelae of child sexual abuse such as post-traumatic intrusions associated with eating. Specifically among avoidant persons, such intrusions may evoke the shame and fear of rejection easily felt by these persons (Andrews, 1998), leading to more avoidance and less engagement in the therapeutic process. Second, PD could mediate the relationship between CSA and ED (mediator model). For instance, CSA may lead to PD, and PD, in turn, may affect ED course and outcome. Third, CSA and PD may independently contribute to predict course and outcome of ED. Each model has distinct clinical implications. If the mediator model is correct, then we can hypothesize that the best target for

changing ED would be to modify the PD, because this would remove the link between trauma-related symptoms (CSA) and ED. By contrast, if the moderator model is correct, then the target would be to modify either the trauma-related symptoms (CSA) or PD, or both in order to facilitate a positive outcome, because both would contribute in maintaining or aggravating the ED. This would only make sense if trauma-related symptoms were found to mediate the association between CSA and ED course/outcome.

Prior publications have reported results from the sample studied here (Ro, Martinsen, Hoffart, Sexton, & Rosenvinge, 2005b; Ro, Martinsen, Hoffart, & Rosenvinge, 2005a; Ro, Martinsen, Hoffart, Sexton, & Rosenvinge, 2005a; Vrabel, Ro, Martinsen, Hoffart, & Rosenvinge, 2009; Vrabel, Rosenvinge, Hoffart, Martinsen, & Ro, 2008). In the present paper we investigated different models for the influence of CSA and PD on the course of ED after intensive inpatient treatment. More specifically, our research questions were:

1. Do PD and CSA separately predict the course of severity of ED symptoms measured by global Eating Disorder Examination after inpatient treatment?
2. How do CSA and PD interact to affect the course of ED after inpatient treatment? Does PD mediate, does CSA moderate, or do they independently influence the course of ED?

Method

Participants

From August 1998 to June 2001, 92 patients were admitted consecutively to a specialized ED unit at a psychiatric hospital. The admission criteria were (1) symptoms of AN, bulimia nervosa (BN), and eating disorder not otherwise specified (EDNOS) that impaired daily functioning, (2) inadequate responses to previous treatment, (3) and age above 18 years. Finally, (4) those with severe medical complications and body mass index < 14 were excluded from inpatient treatment. Six patients discharged themselves during the first two weeks and

86 patients, one male and 85 female, entered the study. All were Caucasian. Participants were assessed at five points in time. A total of 77 patients (90 %) were available for the five year follow-up, and for 74 of these complete SCID-II data were available from admission, constituting the sample of the present study (Figure 1). The nine patients who dropped out of the study did not differ significantly from those retained. Structured Clinical Interview for DSM-IV Axis I diagnoses (SCID-I) was used to determine if a patient had axis I psychiatric disorders at the five-year follow-up. A total of 11 patients in the whole series met the criteria for post-traumatic stress disorder PTSD at the five-year follow-up. All belonged to the CSA-group. No other SCID-I diagnosis is reported. The participants were informed about the study and gave written consent to participate. The study was conducted in compliance with the requirements specified for this project by the official regional committee for medical and health related research.

#INSERT FIGURE 1 HERE#

Hospital treatment

Patients were allocated to separate treatment programs for AN and BN. The programs lasted 22–23 weeks for AN and sub-threshold AN with normal weight (i.e. EDNOS) and 15 weeks for BN and sub-threshold BN (i.e. EDNOS). The treatment was a multicomponent inpatient program focusing on ED symptoms based on cognitive-behavioural therapy. The goals were to identify and change dysfunctional thinking, feelings and behaviour regarding food, body image and interpersonal relationships. Procedures of exposure (to forbidden food), and response prevention (for vomiting and purging) were used. The components included a combination of group sessions focusing on ED symptoms, individual treatment and milieu therapy.

Dependent measures

The measures of interest were the global Eating Disorder Examination (EDE) score at all time points, admission, discharge, one-, two- and five-year follow-up.

Global EDE score. The EDE interview (Fairburn & Cooper, 1993) was used to assess global EDE score and generate ED diagnoses. This is a semi-structured interview that assesses ED psychopathology. It focuses on the previous 28 days, except for diagnostic items, that are rated for duration similar to the DSM IV. It is generally considered the best established instrument for assessing ED. A mean global EDE value is calculated on a 0-6 point scale, and this global EDE value is the dependent measure in this study. The Cronbach's alpha for global EDE ranged from .82 – .93 during all assessment points. At the five-year follow-up the first author conducted all interviews, except one. All interviews at the five-year follow-up were videotaped, and 20 tapes were randomly selected for blind rating by an experienced clinician. Inter-rater reliability was high, with intraclass coefficients (ICC (1,1)) of .99 for the global EDE at five-year follow-up. For all 20 interviews there was

complete agreement between the two raters about absence or presence of objective binge episodes.

Predictor measures

CSA. Consistent with the conceptualization of Sanders and Becker-Lausen (Sanders & Becker-Lausen, 1995), CSA was defined as any involuntary, repetitive sexual experiences with an adult (not necessarily a parent or relative) that occurred before the age of 16. Data on these measures were collected retrospectively based upon official medical charts. Details regarding the abuse (e.g. was the abuse penetrative, did it involve physical contact, was it intra-familial) were not recorded in a sufficiently systematic way to allow them to be included in the statistical analyses. The first author categorized the cases based on each patient's record and personal interviews at each follow-up point. Thereafter, an experienced psychiatrist, who knew the series well through both clinical practice and research, categorized cases blind to the first author (Ro, Martinsen, Hoffart, & Rosenvinge, 2005; Ro, Martinsen, Hoffart, Sexton, & Rosenvinge, 2005), concluding with 19 cases. However, the first author had one additional case based on spontaneous report of CSA at the five-year follow-up with which the third author was not familiar, so in the end there were 20 cases.

PD. The Structured Clinical Interview for DSM-IV Axis II diagnoses, (SCID-II) (First, Spitzer, Gibbon, & Williams, 1995) was conducted during inpatient treatment. To reduce the risk of false positive PD diagnoses due to ED symptoms such as binges or malnutrition, the SCID-II interviews were conducted during the last part of the hospital stay. Patients were interviewed regarding stable personality traits prior to admission.

The patients' individual therapists conducted the SCID-II interviews during hospital treatment. Twenty random SCID-II interviews were videotaped and evaluated by a blind rater. The inter-rater reliability of diagnosis was good ($K = .88$) (Ro et al., 2005).

Statistical Analysis

Data were analyzed by SPSS version 16.0. Analysis of variance (ANOVA) and chi-square analysis were used to compare baseline characteristics.

Our research question concerns upper level mediation in multilevel data (Krull & MacKinnon, 1999). That is, we relate constant features (e.g. CSA, PD) of upper level units, patients, to lower level units, the repeated measurements of the ED symptoms. Therefore, we used linear mixed effects models which can take account of, and adjust for, the interdependence of the repeated observations within individuals that is typical in multilevel longitudinal data. This dependency is accounted for by introducing individual-specific random effects and by modeling the covariance structure of the residuals. In this study, Akaike's Information Criterion was used to compare the fit of different models. Global EDE was the dependent variable and CSA, obsessive-compulsive, borderline and avoidant PD were the predictors. Time was computed in half-years (0, 1, 2, 4, 10). A first order autoregressive covariance structure of the residuals AR (1,1), introducing subject-specific random effects for initial level, fitted our data best. Mixed model analyses including time and predictor as covariates in a first step, and adding predictor X time in a second step, were conducted separately for each predictor. Mediation was tested using Baron and Kenny's recommendations (Baron & Kenny, 1986).

Inter-rater reliability at five-year follow-up was calculated as interclass correlation coefficients ICC (1,1) (Shrout & Fleiss, 1979).

The present study is theory-driven and exploratory in nature, leading us to use significance level of .05 (Kraemer, Wilson, Fairburn, & Agras, 2002). Effects sizes were computed as Cohen's *d*.

Results

Baseline Characteristics

Baseline characteristics are presented in Table 1. The mean age at admission was 29.0 years ($SD = 7.3$). Age of onset of ED was 15.8 years ($SD = 4.8$) and illness duration was 13.1 years ($SD = 7.3$).

INSERT TABLE 1

Mixed model analysis of the global EDE score over time

Mean global EDE score was 4.18 at admission, 3.10 at discharge, 3.20 at one-year follow-up, 2.96 at two-year follow-up and 2.48 at five-year follow-up. Mixed model analyses including time and predictor as covariates in a first step, and adding predictor X time in a second step, were conducted separately for each predictor. As Table 2 shows, both avoidant PD $t(72) = 4.60, p < .0001$ and CSA $t(71.94) = 5.18, p < .0001$ predicted the level of global EDE and in addition, CSA predicted the rate of improvement over time $t(146.18) = 3.33, p < .001$ (Table 2). Borderline and obsessive-compulsive PD did not predict either the level of global EDE, or the rate of improvement.

INSERT TABLE 2#

Investigation of the influence of PD and CSA on ED

Because we were interested in the possible additive and interactive effects of PD and CSA, each of the PD was analyzed together with CSA.

Mediation Model. Following the recommendations of Baron and Kenny for testing mediation (Baron et al., 1986), we first examined whether avoidant, borderline, and obsessive-compulsive PD mediated the relationship between CSA and global EDE. Using mixed model, CSA was found to be a significant predictor of global EDE score in interaction with time (see Table 2). Next, CSA was found not to predict avoidant ($r = .15, n.s.$), borderline ($r = .12$) and obsessive-compulsive ($r = -.13$) PDs. An association between the putative mediator and the predictor is a condition for mediation, however we found no evidence that

avoidant, borderline and obsessive-compulsive PDs play a mediating role in the association of CSA and ED.

Moderator model. The results for avoidant PD and CSA are presented in Table 3. The three-way interaction between CSA, avoidant PD and time was significant. Follow-up analysis showed that there was only significant avoidant X time effect in the CSA group, $t(20.74) = 3.42, p < .01$ and not in the non-CSA group, $t(73.88) = 0.02, ns$. This interaction effect is depicted in Figure 2.

#INSERT TABLE 3#

#INSERT FIGURE 2 HERE#

The three-way interactions between CSA, obsessive-compulsive PD and time $t(83.62) = -1.42$ and CSA, borderline PD and time $t(85.32) = -0.01$, were both non-significant.

Discussion

Using mixed models, avoidant PD and CSA separately predicted the course of ED severity using global EDE as a measure. When they were included in the analysis together, thus controlling for each other, only CSA predicted the course of ED. Finally, avoidant PD and CSA had an interactive effect on the course of ED. Follow-up analyses showed that avoidant PD predicted the course of ED only among patients experiencing CSA. Thus it seems that the presence of CSA moderates the effect of avoidant PD on the course of ED. CSA and the PD were not significantly correlated, indicating that the PD did not mediate the effect of CSA on the course of ED.

ED and avoidant PD is often unnoticed by clinicians (Hinrichsen & Waller, 2006). To our knowledge there are currently no available studies describing treatment effectiveness with patients experiencing both ED and avoidant PD (Hinrichsen et al., 2006). This is noteworthy as research shows that avoidant PD is a common problem among ED patients (Cassin & von Ranson, 2005; Sansone et al., 2005), and that patients with an avoidant PD may be less likely

to benefit from standard cognitive-behavioural therapy for ED (Ghaderi, 2006). However, our study shows that avoidant PD alone does not predict ED course after inpatient treatment, but CSA, moderates the avoidant PD – ED course relationship. The clinical implication of this may be that avoidant persons experience i.e. post-traumatic intrusions associated with eating as a sequelae of CSA. These intrusions may evoke shame and fear easily felt by these persons, leading to more avoidance. The ED symptoms may function as an adaption to counter for both the social emptiness and post-traumatic intrusions.

Limitations

A major strength of this study is that ED has been prospectively assessed five times by structured interviews. One important question is whether the findings may be generalized to ED patients in general, as they have been recruited from a specialist treatment unit. However, similar clinical pictures regarding symptom severity and comorbidity are commonly seen in ordinary clinical settings as well (Haas & Clopton, 2003; Palmer, 2006). However, our findings are limited in generalizability with respect to gender and ethnicity. We also have to acknowledge that this is a relatively old sample of ED patients. Thus, future replication studies are due from a younger sample and countries with a larger population size, and cultural diversity.

Despite attempts to counter it a limitation of the study is the potential recall bias due to the retrospective recollection of CSA experiences. Moreover, a conservative definition of CSA was chosen to reduce the risk of false positive cases (i.e. affirmative reports of repetitive CSA experiences). Also the risk of CSA in the non-CSA group was reduced by a careful interview-based screening for possible abuse at all five measure points. However our strict definition may limit our findings to individuals with repetitive child abuse experiences. Further studies would benefit from using standardized and validated measures of CSA.

Another limitation in our study is that it is impossible to control for confounding factors during admission (e.g. admission length, medication use, treatment adherence) that may contributed to patients' outcome. In addition, following admission the patients had received other kind of treatments that we have no reliable data on. In other words, the findings related to CSA and Avoidant PD as predictors of poor outcome on ED may be influenced by such unmeasured confounds.

Since this was an exploratory study with a p-value of .05, there is a danger of type-II error. The sample size is relatively small, thus deflating statistical power for many of the statistical tests. On one hand, this small sample size means there is little risk of type I error. On the other hand other predictors may have been significant in a larger sample.

Conclusion

The present findings identify CSA and avoidant PD as predictors of the course of ED. The findings support the clinical notion that patients with CSA experiences are more difficult to treat. However, the prediction of CSA is complex as it appears to moderate the effect of avoidant PD on ED course. These results suggest that ED, avoidant PD and sequelae after CSA are potential targets for treatment that need further investigation.

Table 1. Socio-demographic variables at admission for all patients ($N = 74$) and the series divided into diagnostic groups

Variables	AN ($n = 13$)	BN ($n = 37$)	EDNOS ($n = 24$)	Total ($N = 74$)	Chi-Square	F-value	Post-Hoc test, LSD	df
Age, years ($M \pm SD$)	24.4 (5.0)	29.2 (7.0)	31.3 (7.8)	29.0 (7.3)		4.04*	AN<BN, EDNOS	71
Duration of illness, years ($M \pm SD$)	7.2 (2.8)	13.3 (7.1)	15.9 (7.7)	13.1 (7.3)		6.90**	AN<BN, EDNOS	71
Duration of treatment, years ($M \pm SD$)	2.7 (2.0)	2.8 (2.1)	3.1 (2.4)	2.9 (2.1)		0.07		
Previous inpatient treatment n (%)	9 (69)	19 (51)	15 (63)	43 (58)	NS			
Self-mutilation n (%)	6 (46)	23 (62)	10 (42)	39 (53)	NS			
Eating disorder symptoms								
BMI ($M \pm SD$)	15.0 (1.3)	21.1 (3.9)	21.6 (5.3)	20.2 (4.8)		12.44***	AN<BN, EDNOS	71
Binging n (%)	3 (23)	36 (97)	8 (33)	47 (64)	NS			
Vomiting n (%)	5 (38)	35 (95)	11 (46)	51 (69)	NS			
Use of laxatives n (%)	6 (46)	13 (35)	5 (21)	25 (34)	NS			
PD								

Any PD (%)	8 (62)	27 (73)	18 (75)	53 (72)	<i>NS</i>
Avoidant PD (%)	8 (62)	18 (49)	13 (54)	39 (53)	<i>NS</i>
Obs-comp PD (%)	6 (46)	7 (19)	5 (21)	18 (24)	<i>NS</i>
Borderline PD (%)	2 (15)	11 (30)	3 (13)	16 (22)	<i>NS</i>

Note. AN = anorexia nervosa; BN = bulimia nervosa; EDNOS = eating disorder not otherwise specified; BMI = body mass index; PD = personality disorder; Obs-comp = obsessive-compulsive PD.

* $p < .01$. ** $p < .001$. *** $p < .0001$.

Table 2. Predictive relationship between PD, CSA and global EDE ($N = 74$)

Predictor	<i>B</i>	<i>SE</i>	<i>df</i>	<i>t</i>	<i>d</i>
Avoidant PD	1.18.	0.26	72.00	4.60**	0.54
Avoidant PD x Time ^a	0.59	0.36	151.45	1.61	0.13
Obs-comp PD	0.07	0.34	71.99	0.19	0.02
Obs-comp PD x Time ^a	0.00	0.04	147.34	0.09	0.01
Borderline PD	0.44	0.35	71.99	1.27	0.15
Borderline PD x Time ^a	0.02	0.04	147.66	0.36	0.03
CSA	1.46	0.28	71.94	5.18**	0.61
CSA x Time ^a	0.13	0.04	146.18	3.33*	0.28

Note. Time was both significant alone at a .001-level (not shown in the table). Each predictor was first entered alone, and then the interaction term was added. PD = personality disorder; obs-comp = obsessive-compulsive; CSA = child sexual abuse.

^a Time = assessment number 0,1,2,4,10.

* $p < .001$. ** $p < .0001$.

Table 3. Predictive model between CSA x avoidant PD x time and global EDE ($N = 74$)

Predicted variable	<i>B</i>	<i>SE</i>	<i>df</i>	<i>t</i>	<i>d</i>
CSA	1.28	0.25	70.98	5.09**	0.60
Avoidant PD	1.00	0.22	70.98	4.51**	0.54
Time	-0.12	0.02	152.06	-6.77**	0.55
CSA x Time ^a	0.13	0.04	148.60	3.13*	0.26
Avoidant PD x Time ^a	0.04	0.04	148.60	1.18	0.10
CSA x Avoidant PD	0.29	0.51	69.94	0.56	0.10
Time ^a x CSA x Avoidant PD	0.22	0.08	148.59	2.87*	0.24

Note. CSA = child sexual abuse; PD = personality disorder.

^a Time = assessment number 0,1,2,4,10.

* $p < .01$. ** $p < .0001$.

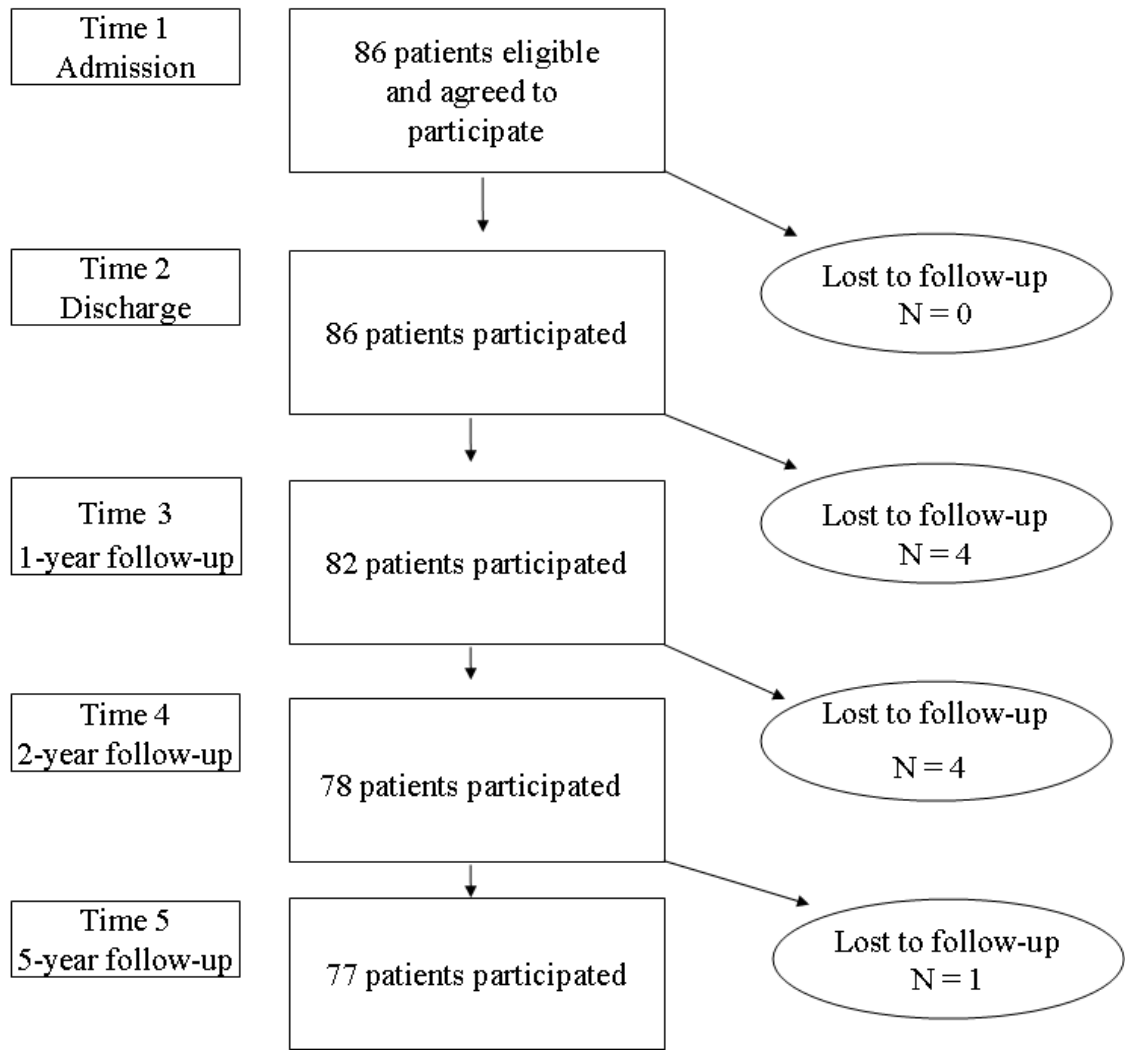


Figure 1. Participation rates

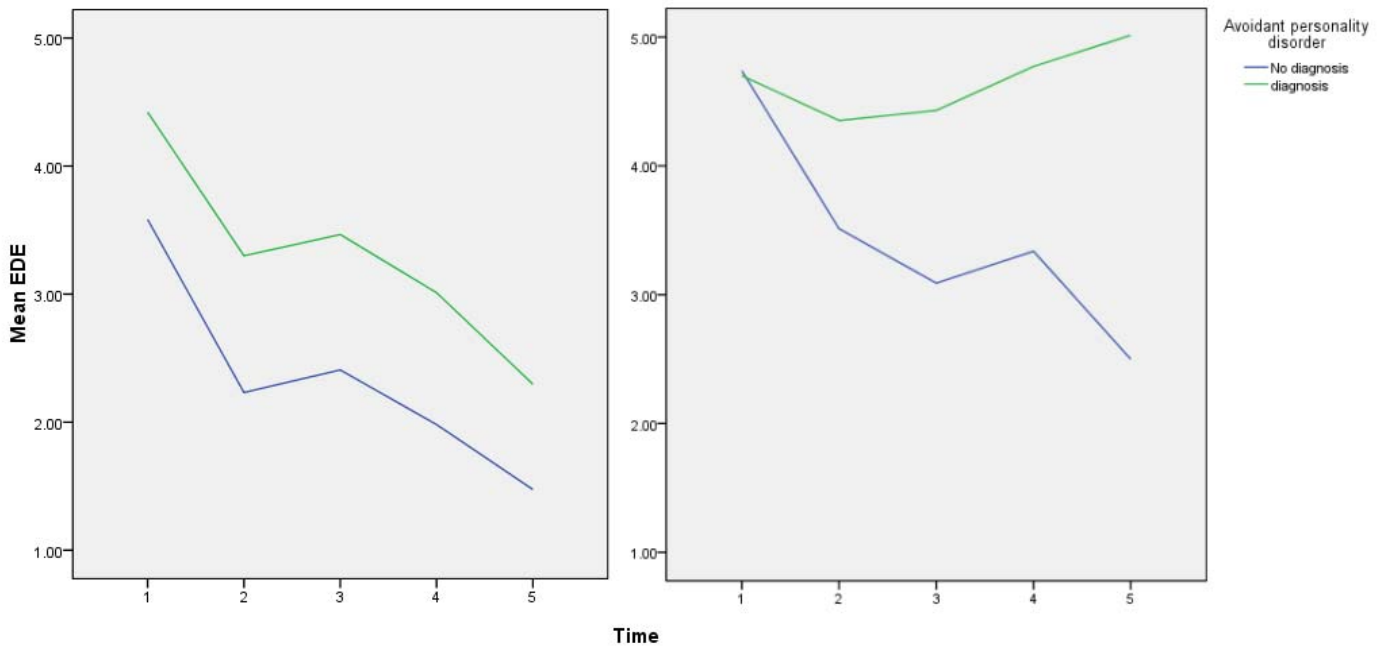


Figure 2. Interaction of avoidant PD x CSA x time. EDE = Eating Disorder

Examination. Left: patients without a history of CSA (n = 54). Right: Patients with a history of CSA (n = 20).

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