Identification of Psychological Risk Factors for Eating Disorder Symptomatology in Women

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Abstract

Theoretical Background

Eating disorders (ED) are serious psychiatric conditions characterized by severe physical and mental health consequences (Treasure, Claudino, & Zucker, 2010). Individuals suffering from full or partial ED manifestations, experience substantial personal impairment, distress and a loss in quality of life (Mond, Hay, Rodgers, Owen, & Beumont, 2005). In order to understand why individuals become ill, and to develop strategies to prevent illness onset, research about risk factors for the development of an ED, has been intensified over the last two decades. However, to date the empirical evidence about risk factors for ED is still sparse and inconsistent (Jacobi, Hayward, De Zwaan, Kraemer, & Agras, 2004). Additionally, the temporal course of a risk factor prior to the onset of ED symptomatology has hardly been explored. Both issues require prospective longitudinal studies taking into account the specific methodological requirements of risk factor research (Kraemer et al., 1997).

Study Aims

The present research aims to identify risk factors for the onset of ED symptomatology and to explore the temporal course of those factors prior to the onset.

Methods

Based on the requirements of how to identify risk factors (Kraemer et al., 1997), an observational longitudinal study targeting a university student population was conducted. At two sites (University of Minho, Portugal; University of St. Etienne, France) 151 female participants completed monthly Internet-based measurements assessing both ED symptoms and potential risk factors. For the latter, eating, weight and shape related factors, as well as unspecific factors (self-esteem, emotional dysregulation, and perfectionism) were taken into account. The development of

substantial ED symptoms (e.g. low body weight, binge eating, vomiting, misuse of laxatives) were used as outcome criterion (i.e. “partial ED”).

Results

Over a period of twelve months 14.6 percent of the participants met the criterion of partial ED. Using survival analyses, several eating related factors (e.g. dietary restraint, eating concern, affect-regulatory eating), weight/shape related factors (e.g. weight concern, shape concern, body embarrassment) and unspecific factors (self-esteem, emotional dysregulation) were identified to substantially increase the risk for the onset of ED symptomatology. The trajectories of risk factors were explored using a multilevel modeling approach. The results suggest that risk factors can be divided into those which increase over the period of four months (eating and weight/shape related risk factors) and those which show a stable course over time (unspecific risk factors).

Discussion

Several factors (eating, shape, weight related, and unspecific factors) which increase the risk of ED symptomatology were identified. Risk factors such as weight and shape concerns, self-esteem, and emotional dysregulation were found to be already elevated four months prior to the onset of ED symptomatology, and thus, can be classified as “early” risk factors. These factors appear to be especially useful for the early detection of high-risk individuals and for the development of tailored risk-based prevention approaches. These findings are consistent with the results of a recent prospective study examining the onset of partial and full ED in a high-risk sample (Jacobi et al., 2011).

Zusammenfassung

Theoretischer Hintergrund

Essstörungen sind schwerwiegende psychiatrische Erkrankungen, die mit weitreichender körperlicher und psychischer Belastung einhergehen (Treasure et al., 2010). Betroffene, die an einer vollen oder partiellen Essstörung erkrankt sind, leiden unter starken individuellen Beeinträchtigungen und eingeschränkter Lebensqualität (Mond et al., 2005). Trotz vielfältiger Forschungsbemühungen, Risikofaktoren für die Entwicklung von Essstörungen zu identifizieren, um einerseits zu einem besseren Verständnis der Ätiologie beizutragen und andererseits Präventionsansätze zu entwickeln, ist die empirische Befundlage bislang eher bescheiden (Jacobi, Hayward et al., 2004). Nahezu völlig unerforscht blieben zudem bis dato die zeitlichen Verläufe von Risikofaktoren vor Beginn einer Essstörung.

Studienziele

Die Ziele der vorliegenden Forschungsarbeit sind einerseits die Identifizierung von Risikofaktoren für die Entwicklung einer manisfesten Essstörungssymptomatik und andererseits die Exploration der zeitlichen Verläufe dieser Faktoren vor Auftreten der Essstörungssymptomatik.

Methoden

Für die angemessene Identifizierung von Risikofaktoren ist die Erhebung longitudinaler Daten erforderlich (Kraemer et al., 1997). In der vorliegenden Arbeit wurde eine längsschnittliche Beobachtungsstudie an einer Stichprobe von Universitätsstudentinnen durchgeführt. An zwei Studienorten (Universität Minho, Portugal; Universität St. Etienne, Frankreich) füllten 151 Teilnehmerinnen monatliche Online-Befragungen aus. Gemessen wurden sowohl Essstörungssymptome als auch Faktoren bezüglich Essen, Gewicht und Figur, sowie unspezifischere Konstrukte wie Selbstwert, emotionale Dysregulation, und Perfektionismus. Als Outcomemaß (d.h.

„partielle Essstörung“) wurde der Beginn substanzieller Essstörungssymptome (z.B. Untergewicht, Essattacken, Erbrechen, Missbrauch von Abführmitteln) verwendet.

Ergebnisse

Innerhalb von zwölf Monaten erfüllten 14,6 Prozent der Teilnehmerinnen das Kriterium einer partiellen Essstörung. Survival-Analysen ergaben, dass Faktoren bezüglich Essen (z.B. essensbezogene Sorgen, gezügeltes und emotionales Essen), Gewicht und Figur (z.B. körperliche Unzufriedenheit), sowie unspezifische Konstrukte wie Selbstwert und emotionale Dysregulation, das Risiko innerhalb von zwölf Monaten Essstörungssymptome zu entwickeln, wesentlich erhöhten. Die zeitlichen Verläufe der Risikofaktoren ab vier Monate vor Beginn der Essstörungssymptome, wurden anhand von Mehrebenenanalysen untersucht. Die Ergebnisse sprechen für eine Einteilung der Faktoren in solche, die über den Beobachtungszeitraum ansteigen (essens-, gewichts- und figurbezogene Faktoren), und solche die einen stabilen Verlauf zeigen (unspezifische Faktoren).

Diskussion

Es konnte gezeigt werden, dass mehrere der untersuchten Faktoren (essens-, gewichts- und figurbezogene Aspekte sowie unspezifische Konstrukte) das Risiko für die Entwicklung einer substantiellen Essstörungssymptomatik erhöhen. Sorgen bezüglich Gewicht und Figur, niedriger Selbstwert und emotionale Dysregulation waren schon vier Monate vor Beginn der Essstörungssymptomatik erhöht, und wurden daher als „frühe“ Risikofaktoren identifiziert. Diese Faktoren erscheinen besonders sinnvoll um Personen mit hohem Risiko zu ermitteln und entsprechende Präventionsprogramme zu entwickeln. Die vorliegenden Ergebnisse bestätigen die Befunde einer kürzlich publizierten Längsschnittstudie, welche Risikofaktoren für partielle und volle Essstörungssymptomatik in einer Hochrisikogruppe untersuchte (Jacobi et al., 2011).

I. Theoretical background

This theoretical section gives an introduction about eating disorders including diagnostic criteria and clinical characteristics, summarizes the concepts of risk, and risk factors, respectively, and provides an overview of risk factors for eating disorders.

1 Eating disorders

Eating disorders (ED) are serious psychiatric problems faced mainly by women and girls and often characterized by a chronic course and recurrent relapses (Fairburn, Cooper, Doll, Norman, & O'Connor, 2000; Lewinsohn, Striegel-Moore, & Seeley, 2000). The following section overviews the clinical pictures of anorexia nervosa (AN), bulimia nervosa (BN), and eating disorders not otherwise specified (EDNOS), including binge eating disorder (BED), which is labeled as a disorder requiring further study in the DSM-IV (American Psychiatric Association, 2000). Although there is still rigorous epidemiologic data missing, the prevalence rates for AN in the United States and Western Europe are estimated consistently to be approx. 0.3 percent, whereas the mean prevalence of BN is approx. 1.0 percent (Hoek & van Hoeken, 2003). Nevertheless, the prevalence of subthreshold conditions, which are still clinically concerning, is much higher (The McKnight Investigators, 2003; Wittchen, Nelson, & Lachner, 1998). Not only full, but partial manifestations as well, are related with substantial co-morbidity and mortality (Birmingham, Su, Hlynsky, Goldner, & Gao, 2005; Hoek, 2006). Although the financial and social impact of these serious psychiatric disorders on disability and productivity remains largely unknown, the impact on both mental and physical health with severe impairment, distress and a loss in quality of life for the individual (Mond et al., 2005), and for relatives and close others (de la Rie, van Furth, De Koning, Noordenbos, & Donker, 2005) has been proven.

1.1 Anorexia nervosa

1.1.1 Clinical characteristics

Anorexia nervosa (AN) is considered to be a severe psychiatric illness (Klump, Bulik, Kaye, Treasure, & Tyson, 2009), which is characterized by a refusal or inability to maintain a normal, i.e. healthy body weight. Often the body weight is reduced below 85 percent of the ideal range. Individuals who are affected by AN and are still in their growth period often fail to gain the expected increase in body weight (and sometimes in height as well). Individuals suffering from AN are often trapped in a vicious circle: although they increasingly lose body weight, they continue to be obsessed and dissatisfied with their weight and perceived size of their bodies, thus, leading to different unhealthy compensatory behaviors to maintain a constant weight loss (e.g. fasting, dieting, excessive exercise, self-induced vomiting, purging). Great importance is attached by these individuals to shape and weight, making it central for their self-perception, and influencing self-esteem and self-worth.

Although amenorrhea is a diagnostic criterion in the DSM-IV (American Psychiatric Association, 2000), its relevance remains questionable. No meaningful differences were found between anorexic individuals with or without amenorrhea (Gendall et al., 2006). For more details of the future consideration of the diagnostic criteria of amenorrhea, refer to section I.1.4. The peak age of onset reported from epidemiological studies is between 15 and 19 years (Lucas, Beard, O'Fallon, & Kurland, 1991). Nevertheless, single case reports suggest increasing presentations in preadolescent children (Gowers, Crisp, Joughin, & Bhat, 1991) and new illness onsets are reported in mid- and late-life (Beck, Casper, & Andersen, 1996; Inagaki et al., 2002).

Regarding the gender distribution, there is a dominant bias towards female individuals suffering from AN: The gender ratio women to men is approx. 9:1 (American Psychiatric Association, 2000). Typical personality characteristics of individuals with AN include anxiety, perfectionism, low self-esteem, obsessions, and harm avoidance (Lilenfeld, Wonderlich, Riso, Crosby, & Mitchell, 2006). Commonly comorbid psychiatric conditions include anxiety disorders (Kaye, Bulik, Thornton, Barbarich, & Masters, 2004) and major depression (Brewerton et al., 1995; Halmi et al., 1991). Some evidence suggests that the anxiety disorder predates the onset of the ED symptomatology (Bulik, Sullivan, Fear, & Joyce, 1997).

1.1.2 Diagnostic criteria

According to DSM-IV criteria, a diagnosis of AN requires a refusal to maintain a body weight at or above a minimally normal weight for age and height. In addition, an intense fear of gaining weight or becoming fat, even though underweight is required. Although the amenorrhea criteria which is described in the DSM-IV (American Psychiatric Association, 2000) as “the absence of at least three consecutive cycles”, is a discrete diagnostic criteria, the clinical utility is currently questioned (Attia & Roberto, 2009). The DSM-IV distinguishes two subtypes of AN based on an individual’s absence (restricting type) vs. presence (binge-eating/purging type) of binge eating or purging behavior.

The diagnostic criteria for AN are presented in Table VII.1 in detail (see annex, p. 126): Diagnostic and Statistical Manual for Mental Disorders IV-TR (American Psychiatric Association, 2000) and the International Classification of Diseases-Versions 10 (WHO, 1992).

1.2 Bulimia nervosa

1.2.1 Clinical characteristics

Bulimia nervosa (BN) is characterized by recurrent episodes of binge eating behavior followed by dysfunctional and inappropriate compensatory measures. Generally, binge eating is defined as the consumption of an abnormally large amount of food accompanied with feelings of loss of control. Measures of inappropriate compensatory behaviors aiming to prevent weight gain include self-induced vomiting, the misuse of laxatives (or diuretics and other agents to prevent weight gain), fasting, and excessive exercise. Typically, the onset of BN starts in adolescence or early adulthood. Unlike individuals suffering from AN, most of the BN cases maintain a normal body weight (Hoek & van Hoeken, 2003). Epidemiological studies estimate the gender ratio to be approx. 9:1, women to men (Hoek & van Hoeken, 2003), but problematically, the diagnostic criteria themselves appear to be biased regarding gender: Men seem to engage more in non-purging forms of compensatory behavior, i.e. excessive exercise (Lewinsohn, Seeley, Moerk, & Striegel-Moore, 2002), whereas women endorse more frequently in purging behaviors. Comorbidities in individuals with BN are common: approx. 80 percent of individuals diagnosed with BN receive an additional psychiatric diagnose during their life span (Fichter & Quadflieg, 2004) including anxiety, depression, substance abuse, and personality disorders (Braun, Sunday, & Halmi, 1994; Brewerton et al., 1995). Individuals with BN and AN share some personality features including high perfectionism, and harm avoidance, and low self-esteem. Some more specific features to individuals with BN comprise high impulsivity, novelty seeking, and low self-directedness (Fassino, Amianto, Gramaglia, Facchini, & Abbate Daga, 2004).

1.2.2 Diagnostic criteria

According to DSM-IV, a diagnosis of BN requires both binge eating and compensatory behavior at least twice a week over a minimum of three consecutive months. Additionally, individuals report that their self-evaluation and self-esteem is overly influenced by weight and shape, whereas individuals with a diagnosis of AN feature an intense fear of weight gain. Furthermore, a diagnosis of BN is only given if criteria for AN are not met, thus, only if individuals have a body mass index (BMI) greater than 17.5 or the equivalent in children and adolescents. The DSM-IV distinguishes two subtypes of BN: if individuals utilize purging as a compensatory behavior which includes self-induced vomiting and the misuse of laxatives, diuretics, or enemas (purging type) or if they only use restricted eating and excessive exercise to prevent weight gain (non-purging type). It is noteworthy, that the ICD-10 (WHO, 1992) reports only vomiting, the use of purgatives and alternate periods of starvation as compensatory behaviors for BN. The rationale behind this alternative is that vomiting and laxative misuse truly have a stronger societal pathologizing character compared with exercise or restrictive eating. Table VII.2 (annex, p. 127) presents DSM-IV-TR and ICD-10 diagnostic criteria for BN.

1.3 Eating disorders not otherwise specified

1.3.1 Clinical characteristics

Eating disorders not otherwise specified (EDNOS) is a diagnostic residual category including ED that have clinical severity but do not meet diagnostic criteria for either AN or BN. Since the publication of DSM-III (American Psychiatric Association, 1980), the American Psychiatric Association (APA) has included in their manuals either "atypical" (DSM-III; American Psychiatric Association, 1987) or "not otherwise specified" categories (DSM-IV; American Psychiatric Association, 2000). The rationale of this initiative was to address the problem of covering every presentation encountered in clinical practice. These diagnoses intend to "indicate a category within a class of disorders that is residual to the specific categories in that class" (DSM-III; American Psychiatric Association, 1987 p. 23). For an ED, the current version of the DSM (DSM-IV; American Psychiatric Association, 2000) specifies six different examples of presentations of EDNOS:

- all features of AN except amenorrhea;
- all features of AN except remaining in a normal weight range;
- all criteria for BN except the frequency of binge eating or purging twice a week or the duration of 3 months;
- regular inappropriate compensatory behavior after eating small amounts of food;
- chewing and spitting out food; and
- binge eating disorder (BED).

Epidemiological studies and clinical reports estimate that more than 50 percent of ED cases in the community fall into the broad category EDNOS (Fairburn & Cooper, 2007; Turner & Bryant-Waugh, 2004), suggesting that the current nomenclature for ED is imperfect (Walsh & Sysko, 2009).

In Portugal, e.g. EDNOS accounted for over 77 percent of all diagnosed cases of ED in the Portuguese community (Machado, Machado, Gonçalves, & Hoek, 2007).

Additionally, *partial* ED, referring to individuals who report only a subset of the symptoms of a particular disorder (e.g. compensatory behaviors but not binge eating or vice versa), are common in community samples (Stice, Marti, Shaw, & Jaconis, 2009). Furthermore, these symptoms are associated with functional impairment, distress, suicide attempts, medical complications, and increased risk for current and future psychiatric and medical problems (Crow, Stewart Agras, Halmi, Mitchell, & Kraemer, 2002; Garfinkel et al., 1995; Milos, Spindler, Schnyder, & Fairburn, 2005; Mond et al., 2006; Striegel-Moore, Seeley, & Lewinsohn, 2003).

1.3.2 Diagnostic criteria

Binge Eating Disorder

The history of Binge Eating Disorder (BED) becoming a discrete diagnostic category in the psychiatric nosology for ED was slow and controversial (Fairburn, Welch, & Hay, 1993; Walsh, 2007). Currently, BED is labeled as a specific example of EDNOS. Although the symptom of binge eating behavior was already described over 50 years ago (Stunkard, 1959) in a subset of obese individuals, the DSM-IV (American Psychiatric Association, 2000) includes BED as a disorder requiring further study (see annex, Table VII.3, p. 128). Individuals suffering from BED do engage regularly in binge eating behavior, but unlike individuals with BN, they do not regularly engage in compensatory behaviors. Although, the binge eating frequency cut off is considered to be at least twice per week (the same as in BN), this criterion is not well supported by empirical data (Garfinkel et al., 1995).

1.4 Eating disorders in DSM-5

The DSM-5 is scheduled to be published in Autumn 2013, and the DSM-5 Task Force and the Eating Disorders Work Group are revising momentarily modifications of the newest issue. One of the first tasks of the Work Group was to review the existing literature relevant to possible changes in DSM-5. The literature reviews published in issues 7 and 8 of the International Journal of Eating Disorders (2009) addressed the following key points:

- the requirement for amenorrhea for AN,
- the utility and validity of sub-typing AN and BN,
- the appropriateness of the ‘‘twice a week’’ frequency criterion for BN and BED, and
- the utility and validity of sub-typing for BED.

The next section summarizes the literature reviews and forecast possible changes in the DSM-5 edition.

Significant attention is paid to reducing the diagnostic cases falling into the broad residual category of EDNOS. A diagnosis of EDNOS is the most commonly given ED diagnosis in clinical practice, with high prevalence rates (e.g. 50% of individuals with ED; Turner & Bryant-Waugh, 2004). Problematically, this group is highly heterogeneous and therefore difficult to characterize (Mitchell et al., 2007). Furthermore, individuals with EDNOS are rarely included in research studies (Fairburn & Bohn, 2005), which results in a lack of evidence for this diagnostic category. Thus, the illness course, outcome, or treatment possibilities for patients with EDNOS remain largely unknown (Rockert, Kaplan, & Olmsted, 2007). Nevertheless, there is evidence indicating that EDNOS is common, severe, and persistent (Fairburn et al., 2007).

One of the aspects coming under scrutiny is the amenorrhea criterion from the diagnostic scheme of AN. This diagnostic criterion is described in the DSM-IV as “the absence of at least three consecutive menstrual cycles” (American Psychiatric Association, 2000, p. 589). Attia and Roberto (2009) reviewed the existing literature to examine the utility of amenorrhea as a distinguishing diagnostic criterion for AN and as an indicator of illness severity. Based on the findings, the authors give the recommendation that the amenorrhea criteria should be withdrawn as a core diagnostic feature, but should be noted as additional information about clinical severity.

The current issue of the DSM (DSM-IV; American Psychiatric Association, 2000) provides two subtypes for AN and BN, respectively: for AN the restricting (AN-R) and the binge-eating/purging (AN-BP) subtypes, and for BN the purging (BN-P) and nonpurging (BN-NP) subtypes. For the upcoming edition of the DSM the validity and utility of the sub-typing schema is questioned. A literature review of the AN subtypes (Peat, Mitchell, Hoek, & Wonderlich, 2009) summarized that dividing individuals with AN into restrictors versus binge/purge subtypes lacks predictive validity. The revision of the BN sub-typing system yielded a gradual difference in severity from BN-P (most severe) through BN-NP to BED (least severe). However, there was no convincing evidence for the validity or utility of the BN-NP diagnosis (Hoeken, Veling, Sinke, Mitchell, & Hoek, 2009).

The frequency criterion in DSM-IV (American Psychiatric Association, 2000) for the diagnosis of BN is “at least twice a week, on average, for 3 months” (criterion C). A review of the literature summarized that evidence on the prognostic utility of the binge eating frequency criterion is largely lacking (Wilson & Sysko, 2009). However, the number of studies available is limited, and, thus, the optimal diagnostic threshold

for binge eating remains to be determined. The authors consider the options to relax the frequency criterion to an once a week frequency threshold or to label “recurrent” binge eating over the past 3 months with mild (twice a month to once a week), moderate (once to twice a week) or severe (daily).

Binge eating disorder (BED) was included in the DSM-IV (American Psychiatric Association, 2000) as a provisional ED diagnosis and as a disorder requiring further study. The reviewed literature showed reasonable evidence that BED can be differentiated from other existing ED and is associated with significant impairment and clinical levels of ED psychopathology (Wonderlich, Gordon, Mitchell, Crosby, & Engel, 2009). Therefore, the authors consider including BED into DSM-5 as a discrete diagnostic category.

Summary chapter I.1.4

The DSM-5 will include three discrete diagnostic categories, namely anorexia nervosa (AN), bulimia nervosa (BN), and binge eating disorder (BED), and the diagnostic residual category of eating disorders not otherwise specified (EDNOS). A recent position paper of the Academy for Eating Disorders (AED) claims that these potential fatal disorders “are biologically based, serious mental illnesses” (Klump et al., 2009; p. 1). Individuals who report only partial ED symptoms have high health care utilization, too (Stice et al., 2009), due to experienced distress and impairment (Crow et al., 2002; Garfinkel et al., 1995; Milos et al., 2005; Mond et al., 2006; Striegel-Moore et al., 2003).

2 Risk factors

In the last two decades, an endeavor has been made to identify risks and risk factors. In order to achieve a better understanding of the etiology of psychiatric disorders, and to reduce the risk for adverse outcomes, strategies to identify and cope with risk factors have been implemented. A crucial problem in investigating risk factors has been the inconsistent use of terminology (Kraemer et al., 1997), not only between different fields (psychology, epidemiology, medicine) but even within the different fields.

2.1 Definition of risk factors

Although the field of risk factor research in psychology has grown rapidly during the last years and the term *risk factor* has been often used, precise definitions for risk factor terminology were only proposed a bit more then a decade ago (Kazdin, Kraemer, Kessler, Kupfer, & Offord, 1997; Kraemer et al., 1997). Kraemer et al. (1997) suggested the following definition for risk:

> *A risk factor is defined as a characteristic, experience or event that, if present, is associated with an increase in the probability (risk) of a particular outcome over the base rate of the outcome in the general (unexposed) population (p. 379).*

Summarizing the terminology introduced by Kazdin et al. (1997) and Kraemer et al. (1997), a risk factor is regarded as any measurable characteristic of a subject in a specified population which (1) precedes the outcome of interest and (2) which can dichotomize the population into two subpopulations: the high- and the low-risk group. Thus, the probability of the outcome in the high-risk group must be shown to be greater than in the low-risk group.

Clarifying the key words in the given definition, the term *outcome* refers to a particular event/characteristic which wants to be prevented (risk factor) or promoted (protective factor). Therefore, the term *risk* refers to an adverse antecedent condition increasing the probability of the incidence of an adverse, or undesirable outcome. The term *protective factor* displays the other end of the continuum, describing characteristics which are related to a decrease in the risk likelihood.

For risk factor research a critical feature is that outcomes are probabilistic. Thus, risk factors influence the likelihood of an outcome rather than determine the outcome. In a high-risk group, many individuals may not show the outcome, while other individuals who do not seem to be at risk may show the outcome (Kraemer et al., 1999).

2.2 Key features of the risk factor concept

The risk factor concept can be described using three key interrelated features (Kraemer et al., 1997): (1) *relational*, (2) *conditional*, and (3) *process*:

(1) The *relational* properties of the risk factor concept describe that a characteristic, event, or experience relates to a particular outcome. (2) The *conditional* aspects indicate that this risk factor-outcome relation depends on a number of conditions, including characteristics of the risk factor itself, the population, other variables with which that factor may be associated, and characteristics of the outcome. (3) Finally, the risk factor concept is a *process* per se because it refers to a characteristic, event, or experience that in some way influences, initiates, or alters critical processes within a person’s life or the systems within which he or she functions.

2.3 Taxonomy of risk factors

Kraemer et al. (2001) developed a methodological basis for investigating how risk factors work together. Assuming that in a (defined) population, an association between a potential risk factor and the outcome can be shown, the factor is called a *correlate.* However, any characterization measured concomitantly with or after the outcome may potentially be a concomitant phenomenon or consequence of the outcome (i.e. a symptom). Only if a correlate can be demonstrated to precede the outcome, the term *risk factor* is justified. The temporal precedence is a crucial criterion in establishing a factor's status as a risk factor. Furthermore, a risk factor that can be shown to change spontaneously within a subject (e.g. age, weight) or to be changed by an intervention (e.g. medication or psychotherapy) is called a *variable risk factor.* A risk factor that cannot be demonstrated to change or be changed (e.g. race, gender, year of birth) is called a *fixed marker.*

In general, the label that is assigned for a given factor always reflects the current state of scientific knowledge about the factor. Thus, accumulating more knowledge about a factor can change its status. In addition, the status of some factors (e.g. pubertal timing) may also change depending on the period of assessments. Thus, a variable risk factor for which it can be shown that manipulation changes the risk of the outcome (e.g. onset of disorder) is called a *causal risk factor.* If that cannot be shown, it is called a *variable marker.* A factor may be variable prior to a certain event (e.g. developmental phase, infection), and therefore be classified as a variable risk factor or marker but become immutable after the event and consequently be classified as a fixed marker. According to this taxonomy, the clinical significance of a risk factor (beyond a statistically significant association between risk factor and outcome) should be indicated by the magnitude of the association – the so called *potency* of the factor.

2.4 Study designs to investigate risk factors

Taking the considerations of the abovementioned concept into account, the decision for a study design (i.e. cross-sectional vs. longitudinal) already determines which factors can be studied. Table I.1 summarizes the risk factor taxonomy and which study design is required to investigate particular factors.

Table I.1 Risk factor typology and identification methods

Term	*Definition*	*Study Design*
Noncorrelate	No significant association between factor and outcome	Cross-sectional & longitudinal studies
Correlate	Statistically significant association between factor & outcome	Cross-sectional studies (epidemiological, case-control, family or family history studies)
Risk factor	Significant statistical & clinical association between factor & outcome; precedence	Longitudinal studies
Fixed marker	Risk factor that cannot be changed or change spontaneously	Cross-sectional studies (medical records or birth registers) Longitudinal studies (including twin & genetic studies)
Variable risk factor	Risk factor that can be changed or can change spontaneously	Longitudinal studies
Variable marker	Variable risk factor, manipulation does not change the risk of outcome	Randomized clinical trials (preventive or intervention study)
Causal risk factor	Variable risk factor, manipulation changes the risk of outcome	Randomized clinical trials (preventive or intervention study)

2.5 Study requirements to investigate risk factors

To investigate risks, risk factors and the relation to outcome, Kraemer et al. (1997, p. 342) set forth the following requirements:

- define the outcome clearly and completely and measure it validly;
- define the population and sample it properly;
- define the risk factor, establish that it defines a characteristic that occurs before the outcome, and measure it validly and reliably;
- use analytic procedures that lead to definitions of high- and low-risk groups and establish that a statistically significant difference exists in the risks of these groups;
- use analytic procedures that lead to meaningful demonstrations of potency.

Summary chapter I.2

This paragraph addressed the concept of risk and risk factors by introducing a theoretical framework (Kazdin et al., 1997; Kraemer et al., 1997). This approach overcames the inconsistent use of terminology by proposing a taxonomy which clearly defines the term *risk* and *risk factor*, respectively. Beyond the correlational aspect of a factor with an outcome, the term *risk factor* is only justified if the potential risk factor as a correlate precedes the outcome temporally. This temporal precedence is a crucial criterion to establish a factor's status as a risk factor.

Furthermore, the paragraph discussed what study design is required to investigate a particular risk factor, concluding that the investigation of a variable risk factor requires a longitudinal study design.

3 Risk factors for eating disorders

The following section aims to summarize putative risk factors for the development of an ED. For a better overview, the risk factors will be divided into three major and broad categories: biological, social, and psychological risk factors.

3.1 Biological risk factors

A position paper of the Academy for Eating Disorders (Klump et al., 2009, p. 1) outlined the evidence supporting that ED are “biologically-based forms of severe mental illnesses”. Research on biological risk factors for ED has focused predominantly on genetic factors and neurobiological disturbances. Therefore, this section gives an overview about the evidence currently available on the importance of genetic factors in the liability to ED and on possible neurobiological vulnerabilities.

3.1.1 Genetics

It is beyond controversy that ED arise from an interaction between environmental events and biological and developmental features of the individual (Treasure et al., 2010). Therefore, genes, environment, and neurobiology are inseparable. Many of the psychological risk factors for ED might ultimately be a reflection of genetic predisposition and gene-environment interactions (Fairburn, Cowen, & Harrison, 1999).

Evidence from twin and family studies suggest that AN, BN, and BED are complex genetic diseases, and for each disorder, the estimated heritability ranges between 50 percent and 83 percent (Bulik et al., 2003; Bulik & Tozzi, 2004; Javaras et al., 2008). About a third of genetic risk for ED and depression (Wade, Bulik, Neale, & Kendler, 2000), anxiety disorders (Keel, Klump, Miller, McGue, & Iacono, 2005), and addictive disorders (Baker, Mazzeo, & Kendler, 2007) is shared.

To date, chromosomal regions and genes that may contribute to the genetic diathesis have been identified in molecular genetic studies: Especially, areas on chromosomes 1, 4, and 10 have been discussed as contributing to the risk for AN (Bacanu et al., 2005; Devlin et al., 2002; Grice et al., 2002); less consistent molecular genetic findings are available for BN and EDNOS. It is noteworthy that so far most of the genetic studies are limited due to a lack of power – a serious obstacle to genetic research in general. However, since 2007, the Genetic Consortium for Anorexia Nervosa (GCAN), which is led by the University of North Carolina Eating Disorders Program, is conducting a genomewide association study on over 4000 DNA samples from individuals with AN (for more information see the GCAN project homepage). This huge study will allow to replicate preliminary findings and detect new associations on a large-scale sample.

3.1.2 Neurobiology

Growing evidence suggests that neurobiological vulnerabilities contribute substantially to the etiology and maintenance of AN and BN (Kaye, 2008). It could be demonstrated that an altered brain serotonin (5-HT) function is involved both in the dysregulation of appetite, satiety, and impulse control (Kaye, Strober, & Jimerson, 2004). Alterations of brain structure (Mühlau et al., 2007), metabolism (Katzman, 2005) and neurochemistry (Kaye, Strober et al., 2004) can be detected, if malnourished individuals with AN. Similar alterations are found in individuals with BN (Kaye, Strober et al., 2004). In addition, profound disturbances of brain serotonin (Kaye, Bailer, Frank, Wagner, & Henry, 2005), within the neuropeptide systems (Kaye, Strober et al., 2004) and brain neurocircuitry (Mühlau et al., 2007) can be identified both in individuals with AN and BN. These disturbances and alterations are associated with regions which are related to the regulations of appetite, mood,

cognitive function, impulse control, energy metabolism, and autonomic and hormonal systems (Kaye, Strober et al., 2004). After recovery from the disorders these alterations often continue.

Summary chapter I.3.1

Over the last decade, the knowledge of biological risk factors, especially the heritability of ED, has been extended. However, the understanding of which genes, or rather gene sequences, are associated with the development of ED, and how these genes are expressed in a gene-environment interaction (epigenetics) is still in its infancy. The launch of Genome-Wide Association Studies (GWAS) is a promising endeavor to overcome problems of sample size and lack of positive results in replication studies. Although, GWAS have identified single-nucleotide polymorphisms (SNPs) implicating hundreds of robustly replicated loci (i.e. specific genomic locations) for common traits (Need & Goldstein, 2010), the genetically based risk prediction is that currently known variants explain too little about the risk of disease occurrence to be of clinically useful predictive value (Manolio, 2010).

3.2 Social risk factors

Social risk factors such as gender and age, acculturation and adverse life events, respectively, play an important role in the development of ED (Striegel-Moore & Bulik, 1997). In the following, the abovementioned factors are discussed.

3.2.1 Gender

In the U.S., the National Comorbidity Survey Replication (Hudson, Hiripi, Pope, & Kessler, 2007) found lifetime prevalence rates of 0.3 percent in men and 0.9 percent in women for AN and 0.5 percent in men and 1.5 percent in women for BN, respectively. It is one of the most consistent findings in the ED literature that females with an ED outnumber males by a sizeable margin in every study (Hoek, 2006). The female to male ratio from population-based studies is estimated to be in the range of 9:1 for both AN and BN (American Psychiatric Association, 2000). Thus, female gender is considered to be a fixed marker for the development of ED (Jacobi, Hayward et al., 2004).

3.2.2 Age

A consistent finding from both clinic- and population-based samples is the peak in incidence of ED during adolescence and early adulthood (Hoek, 2006; Hoek & van Hoeken, 2003). Epidemiological studies have pointed out that the highest incidence rates for AN are among females aged 15 to 19 years, and for BN among females aged 20 to 24 years (Hoek, van Hoeken, & Katzmann, 2003; Keski-Rahkonen, Raevuori, & Hoek, 2008). Although the onset of AN in individuals older than 24 years is considered to be very rare, a meta-analysis found evidence for ED in elderly patients (Lapid et al., 2010). More then 80 percent of the detected cases were female

and experienced a late onset for AN. The typical onset for BN occurs during young adulthood with few cases starting during adolescence (Fairburn & Harrison, 2003).

3.2.3 Adverse life events and sexual abuse

Adverse or stressful life events and their relationship to psychopathology in general have already been discussed thirty years ago (e.g. Brown & Harris, 1978) and since then studied extensively. However, only few studies have investigated the association between stressful life events and the onset of ED. Furthermore, the evidence as to whether individuals with ED have experienced more stressful life events than normal controls remains inconsistent (Horesh et al., 1996; Horesh et al., 1995; Troop & Treasure, 1997; Welch, Doll, & Fairburn, 1997). Among stressful life events, the role of sexual abuse, especially childhood sexual abuse, as a risk factor for ED has been studied the most (for a review see Wonderlich, Brewerton, Jocic, Dansky, & Abbott, 1997). Results indicate that childhood sexual abuse appears to be a consistent risk factor for ED, especially for the bulimic spectrum. A meta-analysis (Smolak & Murnen, 2002) found a relationship between childhood sexual abuse and ED: childhood sexual abuse was associated with an increased likelihood of ED symptomatology. It is most likely that sexual abuse temporally preceded ED onset. Thus, childhood sexual abuse is considered to be a non-specific risk factor for the development of an ED (Jacobi, Hayward et al., 2004).

Taken together, there is some evidence that patients with AN and BN experience more severe life events before the onset of their ED than healthy control subjects. However, this relationship does not seem to be specific for patients with ED, but for psychiatric patients in general.

3.2.4 Acculturation

The so-called sociocultural model of ED emphasizes the female beauty ideal of pronounced thinness which is dominant in “Western” culture. This objectification of the female body is assumed to lead to the social pressure of being thin, which in turn fosters an internalization of this thin ideal and body dissatisfaction. Individuals, that pursue the thin ideal and rate their physical appearance accordingly, are at high-risk for dieting, negative affect, and eating pathology (Cafri, Yamamiya, Brannick, & Thompson, 2005). Although cultural theories of ED have an excellent face validity, the mechanism(s) in which way cultural factor of the thin ideal contribute to ED risk, is largely unknown.

Summary chapter I.3.2

Strong evidence suggests that female gender and the period of adolescence and young adulthood increase the risk of developing an ED. The experience of stressful and adverse life events, in particular sexual abuse, increases the vulnerability for the development of psychiatric disorders in general. Why some individuals are able to cope with such adverse life events, while others develop a particular psychiatric disorder, is largely unknown. Furthermore, the pathways from an adverse life event to the development of an ED remain widely unclear. The so-called sociocultural model aims to explain why ED are mainly prevalent in cultures that value extreme female thinness. Thin ideal internalization seems to be associated with body dissatisfaction, which in turn could lead to the development of ED related symptoms. However, these pathways remain hypothetical.

3.3 Psychological risk factors

Psychological risk factors such as weight concern or body dissatisfaction play an important role in the development and/or maintenance of ED. In the following, the most prominent psychological factors which are under discussion to increase the probability of the development of ED, are discussed including co-morbidities with axis I and axis II psychiatric disorders.

3.3.1 Body dissatisfaction

Body dissatisfaction or negative body image, hypothetically enhances dieting and negative affect, which in turn increases the risk for eating pathology (Bruch, 1962). In this framework, body dissatisfaction putatively leads to dieting behavior because individuals who are dissatisfied with their body might believe that this is an effective way of controlling body weight. Moreover, body dissatisfaction may also foster negative affect because physical appearance may have a central evaluative dimension. Finally, body dissatisfaction may directly promote compensatory behaviors (e.g. vomiting) that characterize e.g. the bulimic spectrum of ED. In fact, body dissatisfaction predicted increased dieting (Stice, 2001; Wertheim, Koerner, & Paxton, 2001) and negative affect (Stice & Bearman, 2001). Body dissatisfaction also predicted bulimic symptom onset (Killen et al., 1996; Killen et al., 1994), an increase in bulimic pathology (Stice, 2001) and eating pathology in general (Wertheim et al., 2001). It is noteworthy that other studies did not find significant relations between body dissatisfaction and increase in bulimic or ED symptoms (Gardner, Stark, Friedman, & Jackson, 2000).

3.3.2 Negative affect

The so-called affect regulation model postulates binge eating as a method to deal with negative emotion. Individuals are assumed to use binge eating behavior to provide comfort and distraction from adverse emotions. The use of extreme compensatory measures (e.g. vomiting) to reduce concern and anxiety about impending weight gain, a consequence of binge eating, might reflect a similar attempt to overcome negative affect. The empirical evidence supporting the theoretical background of these associations is mixed (for a summary see Stice, 2002): while some studies found that increased ED symptoms were predicted by negative affect (Wertheim et al., 2001; Wichstrøm, 2000), other studies found non significant relations of negative affect to the increase of eating pathology (Gardner et al., 2000; Keel, Fulkerson, & Leon, 1997; Leon, Fulkerson, Perry, Keel, & Klump, 1999). Therefore, the question whether negative affect as a variable risk factor or only a correlate remains unanswered so far.

3.3.3 Self-esteem

The assumption that low self-esteem or a negative self-concept plays an important role in many clinically derived theories of ED was already mentioned by Bruch (1962). She described disturbances in the self-concept as a “paralyzing sense of ineffectiveness” (p. 191), and suggested that these might be more basic than the disturbances in body image. In the latest version of the DSM-IV (American Psychiatric Association, 2000) it is explicitly addressed, that the self-evaluation of individuals with AN and BN is “highly” or “unduly” influenced by their body weight and shape (criterion C for AN, criterion D for BN). Although the concepts of low self-esteem, and negative self-evaluation have been studied in numerous cross-sectional studies, most of these investigations have major limitations (Jacobi, Hayward et al., 2004).

Disregarding the cross-sectional character of the studies and the impossibility of conclusion about the temporal order of low self-esteem and the onset of the ED, other shortcomings are: the absence of psychiatric control groups, and a lack of control for depressive symptomatology, although depression and low self-esteem are highly correlated (Neiss, Stevenson, Legrand, Iacono, & Sedikides, 2009), and ED and depressive disorders are highly comorbid (Godart et al., 2007). Nevertheless, some longitudinal based studies confirmed the presence of a negative self-concept or low self-esteem, prior to the onset of an ED (Ghaderi & Scott, 2001; Leon, Fulkerson, Perry, & Early-Zald, 1995), the specificity as a variable risk factor remains unclear. Therefore, it seems reasonable to assume that they are not highly specific for ED.

3.3.4 Perfectionism

Perfectionism has long been linked to ED: Already, Bruch (1978) characterized young AN patients as fulfilling “every parent's and teacher's idea of perfection” and demonstrating “pleasing superperfection” (p. 59). While the link with AN is longstanding and broadly accepted, the relation of perfectionism to BN is less clear.

The broad body of evidence regarding the relationship between perfectionism and ED is summarized in three extensive reviews: Jacobi, et al. (2004) consider perfectionism as a “specific correlate” (p. 44), whereas Stice (2002) in his meta-analytic review, suggests that perfectionism may “be a risk factor for bulimic symptoms and a maintenance factor for more general eating pathology, and that perfectionism may interact with other risk factors” (pp. 838). Lilenfeld et al. (2006) concluded in their review of personality and ED, that the limited prospective research suggests that perfectionism may be a “predisposing personality trait” (p. 316), preceding and increasing the risk for the development of an ED.

3.3.5 Weight concern

Weight concern has been a widely assessed and studied factor. Two studies from Killen et al. (1996; 1994) reported the course of weight concerns in relation to ED onset over a period of several years. In these studies weight concern comprises fear of weight gain, negative body image, dieting behavior, and specific ED attitudes. Having high scores of weight concern was a reliable predictor for the onset of eating pathology. Therefore, weight concern seems to be a well-supported factor which is classified as a variable risk factor (Jacobi, Hayward et al., 2004).

3.3.6 Comorbidity

Epidemiological studies from industrialized countries estimate that over 50 percent of cases of the common psychiatric disorders meet the criteria of more than one psychiatric diagnosis (Andrews, Slade, & Issakidis, 2002; Jacobi, Wittchen et al., 2004). However, it is not well studied whether comorbid conditions arise before the onset of an ED or display secondary complications or maintenance factors. In individuals suffering from an ED psychiatric comorbidities occur often, including axis I disorders such as depression, bipolar disorder, anxiety disorders (obsessive-compulsive disorder, panic disorder, social anxiety disorder and other phobias, and post-traumatic stress disorder) and substance abuse as well as axis II disorders such as borderline personality disorder.

Mood disorders

A critical review of the literature estimates that the lifetime prevalence of at least one mood disorder is 40 percent in AN-R and 60 percent in AN-BP, respectively, whereas the prevalence ranges from 50 to 90 percent in BN (Godart et al., 2007). A family study reports that the familial coaggregation of ED with mood disorders is highly significant, suggesting that ED and mood disorders have common familial causal pathways

(Mangweth et al., 2003). In both AN and BN, major depression is the most commonly diagnosed comorbid psychiatric disorder (Herzog, Keller, Sacks, Yeh, & Lavori, 1992) with a lifetime prevalence of 68 percent for AN (Halmi et al., 1991) and 63 percent for BN (Brewerton et al., 1995). There are less studies investigating comorbidity with bipolar disorder: McElroy, et al. (2005) reported in a review of clinical studies, a weighted mean lifetime prevalence of bipolar disorder of more then 7 percent in ED samples. Bipolar disorder was mainly associated with BN (Herzog et al., 1992).

Few studies have looked at the comorbidity of mood disorders and ED focusing on the relative onset chronology of either mood disorders or ED. At least one mood disorder preceded the onset of AN and BN in respectively 25 percent and 36 to 61 percent of the cases (Brewerton et al., 1995; Schwalberg, Barlow, Alger, & Howard, 1992; Smith, Feldman, Nasserbakht, & Steiner, 1993). Major depression preceded BN in 32 to 71 percent of the cases (Hudson, Pope, Jonas, & Yurgelun-Todd, 1983; Kendler et al., 1991). Although, the rates of a mood disorder preceding an ED are highly variable depending on the particular study, it is evident that comorbid mood disorders may play an important role in the development of ED, but especially in the maintenance and severity of ED.

Anxiety disorders

Clinical and epidemiological studies have consistently shown that the majority of individuals with AN or BN experience one or more anxiety disorders (Godart, Flament, Perdereau, & Jeammet, 2002; Kaye, Bulik et al., 2004). Therefore, it has been suggested that anxiety is central in both the etiology and maintenance of AN and BN. Swinbourne and Touyz (2007) reported in a review that the lifetime prevalence rates of at least one anxiety disorder vary from 25 (Keck et al., 1990) to 75 percent in BN (Schwalberg et al., 1992), and from 23 (Laessle, Kittl, Fichter,

Wittchen, & Pirke, 1987) to 75 percent in AN (Deep, Nagy, Weltzin, Rao, & Kaye, 1995). Furthermore, studies have shown that, in most cases, the onset of the anxiety disorder precedes the onset of the ED (Bulik, 2003).

Obsessive-compulsive disorder (OCD) has been found to have the highest comorbidity rates with ED compared with other comorbid anxiety disorders (e.g. Kaye, Bulik et al., 2004). Milos et al. (2002) reported that individuals with OCD comorbidity have a longer history of an ED and are likely to have developed the ED at an earlier age, whereas, in contrast, Thornton and Russell (1997) found that OCD usually predates the onset of an ED, suggesting that OCD may be a risk factor for developing an ED.

Studies investigating comorbid post traumatic stress disorders (PTSD) among individuals with ED have reported comorbidity rates ranging from 11 to 52 percent (Gleaves, Eberenz, & May, 1998; Kaye, Bulik et al., 2004; Turnbull et al., 1997). Most prominent in this context is a history of trauma, particularly childhood sexual abuse, which is considered to be a non-specific risk factor for the development of an ED (see section I.3.2.3, p. 32).

Studies investigating ED and comorbid agoraphobia have led to mixed results: while in some studies significantly higher lifetime prevalence rates of agoraphobia have been reported in the AN group compared to controls (Halmi et al., 1991), another study by Råstam et al. (1995) reported no differences. The lifetime prevalence of agoraphobia in BN is estimated from zero (Schwalberg et al., 1992) to over 17 percent, depending on the investigation (Laessle, Wittchen, Fichter, & Pirke, 1989).

A number of studies have demonstrated associations between social phobia and ED: Halmi, et al. (1991) found significantly higher lifetime rates of social phobia in their ED group (33.9%) compared to controls (3.2%). However, not all studies have

reported this pattern of results. A study by Bulik, et al. (1997), found a significantly higher rate of social phobia amongst women with BN (30.2%) compared to normal controls (6.1%), but did not find an increased rate amongst women with AN (5.9%). Social phobia most often preceded the development of an ED (Godart et al., 2002; Kaye, Bulik et al., 2004).

Considerably less research has focused on the relationship between generalized anxiety disorder and ED. One study identified generalized anxiety disorder comorbidity of 13 percent for AN and 7 percent for BN (Kaye, Bulik et al., 2004), whereas another study reported rates of 46 percent for AN and 31 percent for BN (Godart et al., 2003). The age of onset for generalized anxiety disorder was reported to be prior to or concurrent with the onset of BN in 75 percent of the cases, indicating that anxiety may be a predisposing factor rather than a consequence of BN (Bulik, Wade, & Kendler, 2001).

Personality disorder

Research suggests that personality disorders are frequently diagnosed among clinical and community samples with ED (for a review see Bornstein, 2001). Among individuals with personality disorders, ED are the most common comorbid axis I diagnoses (Grilo et al., 2007). A meta-analysis (Cassin & von Ranson, 2005) suggested that in particular, avoidant personality disorder is most prominent in all individuals with ED, supporting the hypothesis that ED affected individuals tend to be overly concerned with acceptance and approval, and fear criticism and rejection (Narduzzi & Jackson, 2000). Obsessive-compulsive personality disorder is reported to be frequently seen among individuals with AN-R and also quite common among those with BN, reflecting that these individuals are dispositional perfectionistic and have high personal standards (Hewitt, Flett, & Ediger, 1995). Dependent personality

disorder, characterized by an excessive need to be taken care of, is similarly common among individuals with AN-R and BN. Borderline personality disorder, which is marked by impulsivity and instability, is commonly seen among individuals who engage in binge eating, supporting previous findings that impulsivity, sensation seeking, and novelty seeking are typical of binge eating syndromes (Bulik, Sullivan, & Joyce, 1999). A study investigating the association of personality disorders with the subsequent development of eating problems found that individuals with personality disorders by age 22 were at an elevated risk for an ED at a mean age of 33 years (Johnson, Cohen, Kasen, & Brook, 2006).

Summary chapter I.3.3

Although ED comorbidities such as mood and anxiety personality disorders are common, there is mixed evidence that these comorbid conditions precede the onset of an ED. Therefore, studies examining the temporal onset of particular symptoms are required to establish comorbities as a risk factor in a narrow sense.

Among psychological risk factor, weight concerns, body dissatisfaction, negative affect, low self-esteem, and perfectionism are well studied factors in the association with ED. However, the findings are inconsistent and further well planned and conducted risk factor studies are needed.

II. Study aims

This section combines the general question of how to identify risk factors adequately and the aims of the present study. Theoretical requirements – especially formulated by Kraemer et al. (1997) – for the investigation of risk factors in general, are translated into the aim of identifying risk factors for the onset of ED symptomatology. Furthermore, the need to explore the temporal course of those factors prior to the onset of ED symptoms – especially for prevention programs – is explained, and a second research aim is conveyed.

1 Identifying risk factors for eating disorder symptomatology and measuring their potency

In general, the examination of risk factors has become a widespread field in research. Particularly over the past two decades risk factor research has contributed to both a broader understanding of the etiology of psychiatric disorders and enhanced applications of preventive approaches.

Conceptually, risk factor research refers both to the study of antecedent conditions and subsequent outcomes, and the many ways in which both are related. The processes involved in the emergence of the phenomena of interest – i.e. the onset of ED – are analyzed. Importantly, a better understanding of how a risk factor contributes to an outcome, allows the establishment of e.g. screening tools to identify high-risk individuals, and preventive approaches tailored to address potent risk factors.

Generally, theoretical features of risk factors and how to investigate them and their relationship to outcome adequately (see section I.2.5, p. 27), are set out in detail by Kraemer et al. (1997). However, the translation from theoretical requirements for identifying risk factors into feasible risk factor studies includes some difficulties

regarding study design and analytic procedures. Beyond a clear a priori definition of outcome, risk factors and study population, the temporal precedence of the risk factor has to be established. Only if the potential risk factor as a correlate precedes the outcome temporally, the term *risk factor* should be used (see Kraemer et al., 1997; p. 338). Thus, prospective study designs are essential in establishing whether a factor antecedes the outcome of interest (if retrospective data are not available or considered as non reliable).

An important application from risk factor research is the development of reliable screening tools to detect high-risk individuals. Generally, analytic procedures have to be developed, which lead to definitions of high- and low-risk groups, and a significant difference between these groups regarding the probability of outcome. The groups need to be determined empirically, which in return requires a dichotomization of the total population into two subpopulations: a “high-risk” vs. a “low-risk” group, such that the risk of the outcome is shown to be greater in the high-risk than in the low-risk subpopulation (Kraemer & Gibbons, 2009). However, establishing cut points for an optimal dichotomization of a population, presents a challenging endeavor.

As an additional requirement for the examination of risk factors – introduced by Kraemer et al. (1997) – a measure of strength for the influence of the risk factor needs to be established. Therefore, clinical significance of the risk factor has to be considered, beyond a mere statistically significant association between risk factor and outcome. Such a measure, the *potency* of a risk factor, refers to a special type of effect size that describes the extent to which the high- and low-risk subpopulations defined by this particular risk factor differ in their risk of experiencing the outcome (Kraemer et al., 1999). Several indicators to measure the potency of a risk factor are available (e.g. the odds ratio).

Summarizing these requirements, the identification of a risk factor presents a sequential research procedure, where each step needs to be conducted with caution. To date, well planned and conducted risk factor studies in the field of ED are lacking: e.g. in a comprehensive meta-analysis about psychosocial and biological risk factors for the onset of ED (Jacobi, Hayward et al., 2004), the authors included 320 studies and reviews. However, only 28 longitudinal studies were found from which only 15 were included for classification of risk factors. For the large number of cross-sectional studies, the temporal precedence of the risk factors could only be established via retrospective measures, while longitudinal studies allowed for testing the temporal precedence criterion.

One identified risk factor from the meta-analysis is illustrated exemplarily: The factor weight concern only was assessed directly in two of the included longitudinal studies (Killen et al., 1996; Killen et al., 1994). Both studies allowed for the calculation of a potency measure indicating medium to strong potency. Thus, the authors concluded that weight concern, as assessed in longitudinal research, seems to be a potent, well-supported factor classified as a variable risk factor in the Kraemer typology (Kraemer et al., 1997). Although the number of studies investigating weight concern as a risk factor is far from satisfactory, these two studies by Killen et al. (1996; 1994) demonstrate the adequate sequential procedure of identifying a risk factor.

The present risk factor study was designed in regard to the lack of well planned and conducted studies according to the state of the art in risk factor research (Kraemer et al., 1997). Furthermore, many factors for ED symptomatology are discussed, but less are identified as risk factors in prospective studies. In the present study numerous factors (e.g. weight concerns, body dissatisfaction, emotional dysregulation, self-esteem, and perfectionism) are tested whether they increase the risk for ED symptomatology.

2 Temporal occurrence and course of risk factors for eating disorder symptomatology

The need to identify risk factors for an outcome arises both from the aim to extend the empirical knowledge of understanding how individuals become ill, and the objective to establish measures to prevent high-risk individuals from becoming ill. For such prevention programs, it is essential to know when a risk factor is elevated in reference to the outcome.

Assuming the etiology of a psychiatric disorder follows a continuum where the same variables that differentiate controls from subthreshold individuals should also differentiate subthreshold individuals from individuals with a full-blown clinical picture (e.g. for ED Stice, Killen, Hayward, & Taylor, 1998), at some threshold (defined through diagnostic criteria) a categorical diagnosis is given (American Psychiatric Association, 2000). Along this continuum – from healthy, via subthreshold, to full clinical conditions – a risk factor could be manifested at an early vs. late state in reference to the time of categorical onset of the disorder. Theoretically, one risk factor might be expressed (i.e. elevated) chronologically close to the onset of the disorder (“late” risk factor), whereas others might already be manifested a long time before the actual start of the disorder (“early” risk factor). Such an “early” risk factor provides valuable information for early detection and prevention approaches: those factors can be used to screen for high-risk subjects well in advance and offer them risk-tailored prevention programs at an early stage.

Figure II.1 illustrates exemplarily “early” and “late” risk factors along the continuum of developing a full-blown ED.

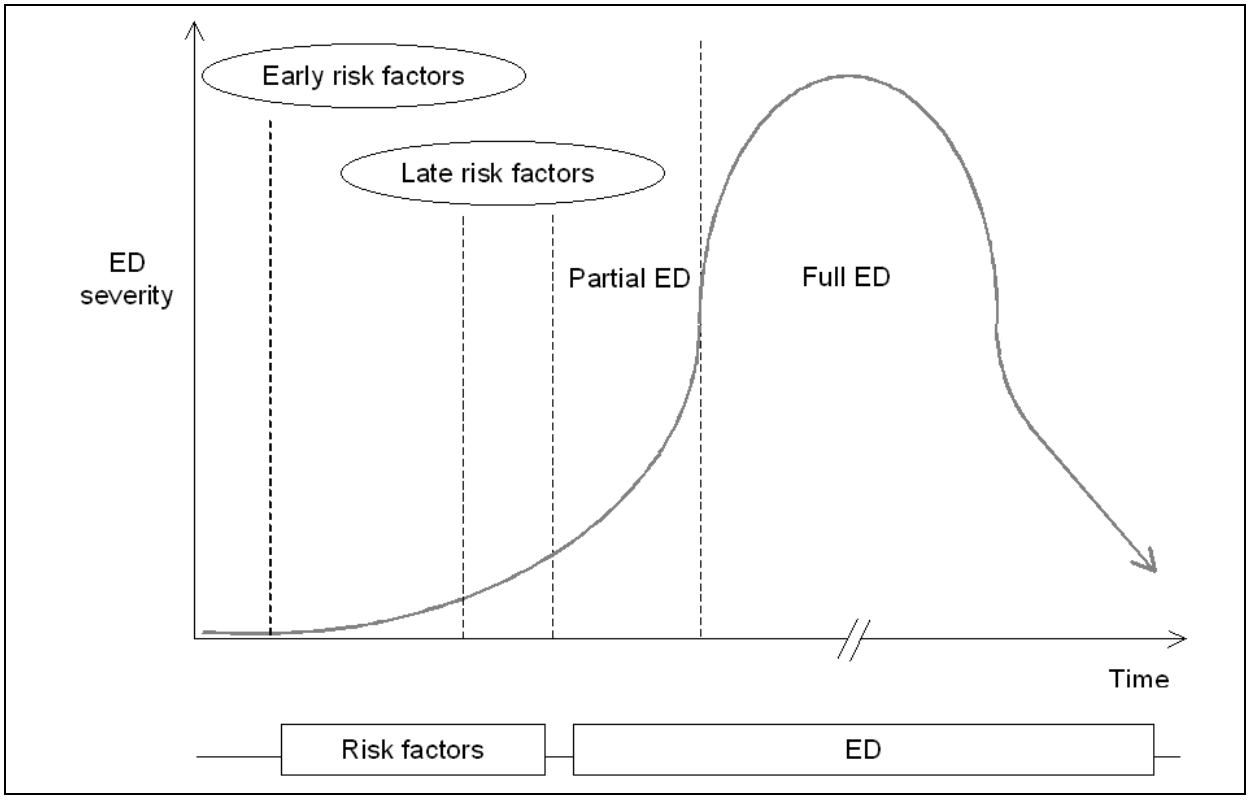

Figure II.1 Early & late risk factors

In this exemplary schema, the pathway to a full diagnosed ED develops from a healthy state via discrete steps of partial or subthreshold conditions to the full clinical picture of the disorder. Accordingly, an “early” risk factor is already manifested well in advance of the onset of the full clinical condition, whereas a “late” risk factor is only manifested shortly before e.g. the partial or full ED starts.

Reviewing the existing studies about risk factors for ED, the use of such an approach is sparse: e.g. a large prospective study which aims to identify risk factors for adolescent girls (The McKnight Investigators, 2003), found that thin body preoccupation and social pressure predicted the onset of ED during a three year follow-up. Throughout the course of the study, both risk factors showed stable but elevated trajectories over time.

Although the study lacks a detailed exploration of the temporal course of these two factors, the effects of both thin body preoccupation and social pressure on the later onset of ED, were already expressed three years prior the actual start of the disorder. The early elevations of the risk factors and the stable course throughout the three years, argues for an “early” risk factor.

In the present study, the temporal occurrence and course of risk factors prior to the onset of partial ED are examined in order to establish “early” vs. “late” risk factors for the onset of ED symptomatology. “Early” risk factors could provide useful information to first detect high-risk individuals, and second refer them earlier to prevention programs which may counteract illness onset.

Summary chapter II

The identification of a risk factor requires a sequential procedure: the association between factor and outcome has to be proven statically significant, temporal precedence of the factors has to be established, and beyond the mere statistical significance, a measure of potency is needed. Such planned prospective studies are lacking in the field of ED. In order to identify risk factors for the onset of ED symptoms, the present study uses a prospective observational design. Different factors (eating behavior disturbances, affective/cognitive impairment, perfectionism, anxiety and depression, etc.) are tested in order to examine whether they increase the risk for ED symptomatology. Additionally, the potency of each factor is analyzed.

The detailed exploration of temporal occurrence and course of risk factors prior to the outcome yields additional information of the factor and the association to outcome.

Especially for detecting high-risk individuals and planning prevention programs, it is crucial to know which risk factors are already elevated well in advance of the onset of ED symptoms (“early” risk factor). In order to test for “early” and ”late” risk factors, the present study explores the occurrence and trajectories of risk factors prior to the onset of ED symptoms. In detail, factors will be classified into those which are already elevated months prior to the onset of ED symptoms (“early” risk factors), and those which show a steep increase shortly before the onset of the partial ED (“late” risk factors).

Overall, the present study addresses two aims:

1. Aim: Identification of variables which increase the risk of developing ED symptoms, and determination of the potency for these variables.
2. Aim: Exploration of trajectories of variables prior to the partial ED onset to establish “early” and “late” risk factors.

III. Methods

This section summarizes the procedural, methodological and analytical issues of the present research: The network within the study was conducted, the study design and procedure, as well as the measurements and data analysis procedures.

1 The INTACT network

The present study was conducted within the research cooperation named INTACT.[1] The acronym INTACT stands for "Individually Tailored Stepped Care for Women with Eating Disorders", and is a Research and Training Network (MRTN-CT-2006-035988) funded by the European Commission in the Marie Curie Program (European Commission, 2007). It consists of nine European partners and aims to address the challenge of the development of new strategies for the prevention and treatment of ED in a multi-disciplinary and inter-sectorial way. The centers represent institutions with research interest in the field of ED and a high academic reputation in this area:

- University Hospital Heidelberg (Germany)
- University of Saint Etienne (France)
- Charles University Prague (Czech Republic)
- King's College London (UK)
- Semmelweis University Budapest (Hungary)
- University of Minho (Portugal)
- Rudolf Magnus Institute of Neuroscience (The Netherlands)
- Geneva University Hospital (Switzerland)
- NetUnion Sarl (Switzerland)

For further and detailed information on the network nodes, please refer to the official project homepage www.intact-rtn.eu.

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[1] *Acknowledgment*: The present research was supported by the European Commission, grant MRTN-CT-2006-035988

The European Commission stated that the Marie Curie Research and Training Networks should first undertake a comprehensive research project, and second provide a structured training program (European Commission, 2006, p. 10):

> *These [Networks] provide the means for research teams of recognised international stature to link up, in the context of a well-defined collaborative research project, in order to formulate and implement a structured training programme for researchers in a particular field of research.*

The overall scientific objective of the INTACT network is to combine empirical and conceptual research and technical innovations to optimize care provision for women with ED. Combining the scientific expertise in the field of ED, the nine European partners aim to expand the empirical knowledge by addressing the following key aspects:

- determining the role of genetic, psychological and sociocultural factors and their interaction for the development of an ED,
- identify mechanisms and influences determining the process of getting ill, getting well and staying well, and
- developing novel e-health tools for the optimization of health provision.

Based on the facts that not all women at risk for the development of an ED actually become ill and not all of those who develop the disorder benefit from the same type, duration, and intensity of care, the innovative contribution of INTACT can be seen in the development of individually tailored treatment and stepped care interventions.

The present study on psychological risk factors for ED symptomatology is one out of 14 projects that build the INTACT research program.

2 Data collection

The data collection started in May 2008 and ended in July 2010. The recruitment took place at two sites in Braga, Portugal and St. Etienne, France.[2] At both study sites, ethical approval was obtained from the Internal Review Boards (IRB) at the University of Minho and at the University of St. Etienne. The study was conducted in accordance with the Good Clinical Practice guidelines (European Commission, 2002), international, and state laws, as well as the declaration of Helsinki (World Medical Association, 1996). All participants had to give their informed consent before registering for participation.

2.1 Recruitment

The recruiting process in Portugal mainly took place at the University of Minho via class visits, by dissemination of flyers and posters and university emailing. Furthermore, more open strategies were chosen to increase the sample size, e.g. via Facebook. The University of Minho (Universidade do Minho; www.uminho.pt) has a high reputation as an academic institution in Portugal and is located in the Minho region of Northern Portugal. The university has a student population of 16.000, out of which 1.900 are postgraduate students.

In St. Etienne students were also informed about the study by dissemination of flyers and posters. In addition recruitment took place at medical appointments, which are offered from the University of St Etienne free of charge to all freshmen entering the university in the first year of their studies. Furthermore, recruitment via Facebook was carried out as well. The University of St. Etienne (Université Jean Monnet, St. Etienne;

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[2] *Acknowledgment*: The data collection at the French site was accomplished by Linda Szabo.

http://portail.univ-st-etienne.fr) is a multidisciplinary academic institution in the heart of France with about 14.000 students. Participants were recruited mainly from the University of St. Etienne, but other universities from the Rhone-Alpes region participated in the recruitment process, too (universities of Lyon, Grenoble, Chambéry, Roanne, and St. Chamond).

2.2 Internet-based data collection

Data were collected via the Internet using the software Web-AKQUASI (Percevic, Gallas, Arikan, Mößner, & Kordy, 2006). Web-AKQUASI was developed by the Center for Psychotherapy Research at the University Hospital Heidelberg for the purpose of online data collection and data management. Various measures (such as 128-Bit SSL encoded data transfer and storage, specific access control etc.) have been implemented to ensure safe transfer and storage of data. Web-AKQUASI has been used for quality assurance and outcome monitoring in numerous psychosomatic and psychiatric hospitals, and is the core data collection tool for several projects monitoring the course of symptoms. Within the INTACT network the data of nine studies are collected via Web-AKQUASI. The program has proven to be technically reliable and psychometrically valid (Percevic et al., 2006).

While participants filled in questionnaires, plausibility and completeness were checked automatically. Data were stored at the center in Heidelberg. The study centers in Braga, Portugal and St. Etienne, France could independently check the status of the data collection. No personal identifying information (e.g. names etc.) was stored, except email addresses in order to remind the participants to complete the assessments. Due to data security issues, the email addresses were stored on a separate server and deleted with completion of the study.

3 Procedure

The following paragraph describes the study procedure for participants exemplary. Individuals came to know about the study via class visits, emailing, poster, and flyers. Study homepages (Portugal: http://gepa.cipsi.uminho.pt/eva; France: www.lisa-st-etienne.eu, see annex, Figure VII.1, p. 129) provided information including terms, conditions and rights. In case of willingness to participate, individuals registered by entering a valid email address on the study homepage. Participants were fully informed about the conditions of the study and had to actively agree on an (electronic) informed consent (IC). After registration participants received a welcome email with a personalized hyperlink directing them to their first assessment (see annex, Figure VII.2, p. 130). Upon activating the hyperlink, the personal assessment was opened displaying the first questionnaire (see annex, Figure VII.2, p. 130). After completion of the assessment participants received an email thanking them for their contribution.

The data collection software was programmed to send emails every 30 days to remind participants about their monthly assessment. In case participants did not respond to the questionnaires, two additional emails were sent. An assessment was rated as completed if participants had answered 80 percent of the questionnaires. In case participants did not complete three consecutive assessments the reminding procedure was stopped.

Participants were informed that they could contact the investigators (by email or a contact form on the study homepage). Thus, participants could withdraw from the study at anytime without giving a reason and demand deletion of all their data. Only four participants (Portuguese site = 3, French site = 1) asked for elimination from the study, therefore no data from these participants were used for any analyses.

4 Design

An observational longitudinal study was conducted to identify risk factors for the development of ED symptoms. In order to investigate the temporal course of risk factors prior to the onset of partial ED, repeated measures have to be carried out (Jacobi, Hayward et al., 2004). In the following, the frequency of the assessments and the employed measures are described.

4.1 Assessments

The participants completed monthly Internet-based assessments. Table III.1 gives an overview of the frequencies of six first monthly assessments; participants continued to complete assessments until they did not respond to three consecutive assessments or asked to stop their participation.

Table III.1 Assessments

	Months						
	1	*2*	*3*	*4*	*5*	*6*	*n*
Socio-demographic questions	●						
Eating Disorder Examination Questionnaire (EDE-Q)	●	●	●	●	●	●	
Clinical & Research Inventory for Eating Disorders (CR-EAT)	●	●	●	●	●	●	
Short Evaluation of Eating Disorders (SEED)	●	●	●	●	●	●	
Depression Anxiety Stress Scale (DASS, 21 item version)							
Portugal	●	●		●		●	
France	●					●	

Note: Portugal = Portuguese study site, France = French study site, n = monthly assessments continued until participants did not complete three consecutive assessments or asked to stop their participation

The core battery of questionnaires – which were administered monthly – consisted of the Eating Disorder Examination Questionnaire (EDE-Q), the Clinical and Research Inventory for Eating Disorders (CR-EAT), and the Short Evaluation of Eating Disorders (SEED). Additionally, the 21-item version of the Depression Anxiety Stress Scale (DASS-21) was administered at the Portuguese site every other month, whereas at the French site only at the initial and sixth assessment. After the sixth monthly assessment, the measurements continued with the core battery of questionnaires, and the DASS-21 at the Portuguese site. The SEED was used to measure the outcome criteria “partial ED” (see section III.4.3), the other questionnaires to establish putative risk factors (e.g. restraint, controlled, and affect-regulatory eating, weight and shape concern, body embarrassment, perfectionism, anxiety, and depression, emotional dysregulation, etc.) for the onset of partial ED.

4.2 Measures

4.2.1 Outcome measure

Short Evaluation of Eating Disorders

The Short Evaluation of Eating Disorders (SEED; Bauer, Winn, Schmidt, & Kordy, 2005) is a short screening instrument for ED symptomatology. With six items, the questionnaire assesses core characteristics of ED and total severity indices for AN and BN are provided. Moderate construct validity and good criterion validity, as well as a good sensitivity to change have been demonstrated (Bauer et al., 2005).

4.2.2 Risk factor measures

Eating Disorder Examination Questionnaire

The Eating Disorder Examination Questionnaire (EDE-Q; Fairburn & Beglin, 1994) is a self-report 36-item questionnaire derived from the interview version of the Eating

Disorder Examination (EDE; Fairburn & Cooper, 1993). The instrument contains a total score and four subscales (dietary restraint, eating concern, shape concern, and weight concern). Identical to the interview, the questionnaire spans a 28-day time frame scored on a 7-point Likert scale. The EDE-Q is widely regarded as the “gold-standard” for the assessment and diagnosis of DSM-IV ED (Garner, 2002), and has been used with increasing frequency in clinical and community investigations of ED symptoms (Anderson & Williamson, 2002). Similar to the EDE, it provides a comprehensive assessment of specific psychopathology of eating disordered behavior in a relatively brief self-report format. Studies of the validity of the EDE-Q have demonstrated a high level of agreement between the EDE-Q and EDE in assessing the core attitudinal features of ED psychopathology (Binford, Le Grange, & Jellar, 2005; Black & Wilson, 1996). Regarding the psychometric properties, the EDE-Q demonstrated acceptable levels of internal consistency (Peterson et al., 2007 [total score: $\alpha = .90$; subscales: $\alpha = .70 – .83$]), good concurrent validity, and acceptable criterion validity (Mond, Hay, Rodgers, Owen, & Beumont, 2004).

Clinical and Research Inventory for Eating Disorders

The Clinical and Research Inventory for Eating Disorders (CR-EAT; Fassnacht, 2007) contains 70 items on a 6-point Likert scale assessing psychological and behavioral characteristics on the basis of the assumption that disordered eating is multidimensional in its nature. The measurement was designed especially for Internet-based data collection (Moessner, Fassnacht, & Bauer, under review). Using a factor analytic approach, 12 subscales have been developed measuring weight preoccupation (WP), control over eating (CE), emotional dysregulation (ED), affect-regulatory eating (AE), self-esteem (SE), concerns about negative evaluation (CN), body embarrassment (BE), restrained eating behavior (RE), societal expectations of

weight and shape (SO), harmful weight regulation (HW), and perfectionism (personal [PP] and familial expectation [PF]). Three global factors addressing Eating Behavior Disturbance (EBD: subscales WP CE, BE, RE, SO, HW), Affective/Cognitive Impairment (ACI: subscales ED, AE, SE, CN) and Perfectionism Total (PER: subscales PP, PF) are provided. The questionnaire showed moderate to good internal consistencies (total score: α = .96; sub scales: α = .62 - .95) and test-retest reliabilities (total score: r = .93; subscales: r = .71 - 91), and good construct validity (Moessner et al., under review).

Depression Anxiety Stress Scale

The Depression Anxiety Stress Scale (DASS; Lovibond & Lovibond, 1995), a 42 item self-report questionnaire, measures the negative emotional states of depression, anxiety and stress. On a 4-point Likert scale, the severity of each state over the past week is assessed. In addition to the basic 42-item questionnaire, a short 21-version, the DASS-21, is available, and studies demonstrated similar factor structure and psychometric features of the short version (Antony, Bieling, Cox, Enns, & Swinson, 1998; Henry & Crawford, 2005): The internal consistency (depression : α = .88 – .94; anxiety: α = .82 – .87; stress: α = .90 – .91) and concurrent validity of the DASS-21 were in acceptable to excellent ranges.

4.3 Outcome criterion

Given the rather small prevalence rates of ED (see section I.1, p. 13), less stringent conditions than the full DSM-IV diagnostic criteria were used as outcome criterion. To establish the definition of a *partial* ED referring to individuals who report only a subset of symptoms of a particular disorder (e.g. only compensatory behaviors but no binge eating or vice versa), the following criteria were used:

- at least one binge per week and / or,
- at least one vomit per week and / or,
- at least one misuse of laxatives per week and / or,
- BMI less than 18 and / or,
- start of treatment for ED.

Participants who met one of these criteria at least at one time in their assessments were rated as “partial ED” cases.[3] The outcome criteria were assessed using the SEED questionnaire (see section III.4.2, p. 55).

[3] In the following, the expressions onset of partial ED, onset of ED symptomatology, and onset of ED symptoms are used synonymously.

5 Statistical analysis

The following section overviews the statistical analyses both for the identification of risk factors, and the exploration of the temporal course of these factors prior to the onset of partial ED.

5.1 Identification of risk factors for the onset of eating disorder symptomatology

To identify risk factors for the later onset of ED symptomatology, survival analyses are used. Survival analysis typically focuses on time to event data by using follow-up time from a defined starting point to the occurrence of a given event, e.g. the time from a diagnosis of a disease to death. The present study uses the Kaplan-Meier method with a possible survival time from the start of participation to the onset of partial ED. Right censoring of the data occurs commonly due to the small incidence rates of ED symptoms. Consequently, no mean survival times are calculated. As the metric unit of the variable time, the completed monthly assessments are used. An interval of 12 months is chosen to be analyzed.

In order to examine which factors increase the risk for the onset of partial ED, the continuous variables (total and subscale scores of the EDE-Q, CR-EAT and DASS-21) are dichotomized with a median split that, for every variable, establishes a low- vs. high-risk group. Statistical differences of the survival distributions of the two groups (low- vs. high-risk) are tested by using the log-rank test.

In order to examine the potency of a particular risk factor, the odds ratio is used. The odds ratio (OR) is defined as the odds of an outcome among exposed individuals divided by the odds of an outcome among unexposed, where p is the estimated

probability of an event occurring in a exposed group, and *q* is the estimated probability of the same event occurring in a unexposed group:

$$OR = \frac{p(1-q)}{q(1-p)}$$

An OR of 1 indicates that the outcome is equally likely to occur in both groups, whereas an OR greater than 1 indicates that the condition or event is more likely to occur in the exposed group.

5.2 Temporal course of factors prior to the onset of eating disorder symptomatology

To explore the temporal course of factors prior to partial ED onset, trajectories of variables over time are analyzed. Therefore, data from participants prior to the onset of ED symptoms are used in a multilevel model approach. The framework of multilevel modeling offers powerful and flexible ways to analyze longitudinal data sets. Basically, it provides a method to characterize response patterns or trajectories of growth on an individual or group level over time (Raudenbush, 2001). Furthermore, it enables a distinction between person- or group-level characteristics and their association to variations in the growth patterns (Raudenbush & Bryk, 2002). A major advantage of multilevel modeling is that these approaches can overcome many of the limitations with which traditional approaches are confronted while analyzing longitudinal data.

Especially relevant for the present study, multilevel models can deal with unbalanced data, missing observations and varying measurement occasions across individuals. Sometimes multilevel models are referred to as hierarchical linear models, too. The notion *hierarchical* refers to the fact that data sets have a hierarchical (*nested*) data structure. In the present longitudinal data set, observations taken over time are

nested within subjects, giving a two-level hierarchical structure. The variation of responses within subjects over time is at the lowest level (level one) and the variation of the underlying mean responses between subjects is at level two. The response, as a linear function of time (*t)* measured at more then one point of time, is expressed by the following equation (Goldstein, 2005),

$$\gamma_{ij} = \beta_{0j} + \beta_{1j} t_{ij} + \varepsilon_{ij} \quad (1)$$

where the first subscript (i) refers to the measurement point, and the second to the individual (j). The response is γ_{ij} and t_{ij} the time for the j-th individual at the i-th time point. The intercept coefficient β_{0j} and the slope coefficient β_{1j} – as the average growth rate – allows for variations across individuals in the present analyses, where data from participants who developed ED symptoms are used. The measurement point where participants reported at least one case criterion is used as centering point. A four month time interval is chosen to be examined (due to available data). In order to allow a chronologically detailed exploration of factors, the variable time (monthly assessments) is refined into days, giving a time frame of 120 days prior to the onset of the partial ED. In the following, the centering point – the onset of the criterion “partial ED” – is marked as day zero; chronologically earlier days receive negative values (e.g. the assessment 30 days prior the onset of the partial ED, is marked with –30, etc.).

The statistical packages SPSS 17.0 and S-Plus 8.0 are used for data analyses.

5.3 Sampling

For longitudinal data analyses, repeated measurements from each individual are required. Thus, in the present study data from participants with only one completed assessment are excluded.

According to Andersen (1995), women and men with ED differ mainly before and after their acute phase of the disorder. Given the scope of the INTACT research (Individually Tailored Stepped Care for Women with Eating Disorders) and the mentioned differences in the etiology of ED between the genders, male participants are excluded from further analyses.

For survival analyses, data prior to the occurrence of the event of interest are required. If the event already occurs at the first measurement point of a subject, these data are missing. Thus, participants who fulfill the partial ED criteria already at their first measurement are excluded from the data used for survival analyses.

Hierarchical linear modeling approaches are able to manage unbalanced or missing data (see section III.5.2, p. 60). However, including data of participants who fulfill the partial ED criteria already at their first measurement, the results would be biased due to more severe ED symptomatology of these participants. To test for differences between the two groups (ED case at first measurement vs. ED case at a later measurement), total mean scores of the EDE-Q, CR-EAT and DASS-21 are tested at the time point where participants report partial ED (Table III.2, p. 63).

Table III.2 Comparison: Case at first assessment vs. case at a later assessment I

	Case at first assessment			Case at > first assessment			Statistic	
	n	*M*	*SD*	*n*	*M*	*SD*	*t-value*	*p-value*
EDE-Q	58	2.68	1.43	25	1.90	1.21	2.36	.021
CR-EAT	62	3.49	0.91	24	3.18	0.86	1.40	.165
DASS-21	22	61.27	29.57	11	43.09	22.37	1.79	.083

Note: n = number of participants, M = mean, SD = standard deviation, EDE-Q = Eating Disorder Examination Questionnaire total score, CR-EAT = Clinical and Research Inventory for Eating Disorders total score, DASS-21 = Depression Anxiety Stress Scale total score

The results yield significant differences in the EDE-Q total score; $t(81) = 2.36$, $p < 0.05$: participants who already fulfill the partial ED criteria at the initial measurement point show higher EDE-Q total scores.[4]

As a result, the hierarchal linear modeling, yields higher slopes for all EDE-Q, CR-EAT and DASS-21 scales in the models which include all cases compared to models which include only the cases reporting the partial ED criteria at a later assessment (see annex, Table VII.6, p. 133).

Taken together, results suggest that individuals who fulfill the partial ED criteria at the initial assessment, show more severe ED symptomatology then individuals who develop the partial ED criteria during the observation time of the study. To avoid a bias in the hierarchal linear modeling, cases meeting ED criteria at their initial assessment are excluded.

[4] *Note*: For a more detailed comparison, EDE-Q subscales, CR-EAT global scales and DASS-21 subscales are presented in annex, Table VII.4, p. 131

IV. Results

1 Sample description

As described in the methods section (see section III.5.3, p. 62), data from the following participants are excluded:

- participants who complete only one assessment,
- male participants,
- participants who report partial ED at the first assessment.

After excluding participants, two subsamples are obtained: subsample 1 (n = 151) is used for survival analyses – to identify risk factors; subsample 2 (n = 25) is used for hierarchal linear modeling – to explore the temporal course of factors prior to partial ED onset. Partial ED cases are established by using the criteria described in section III.4.3 (p. 58).

The flow chart (Figure IV.1) gives an overview about the sampling process, and excluded and included participants.

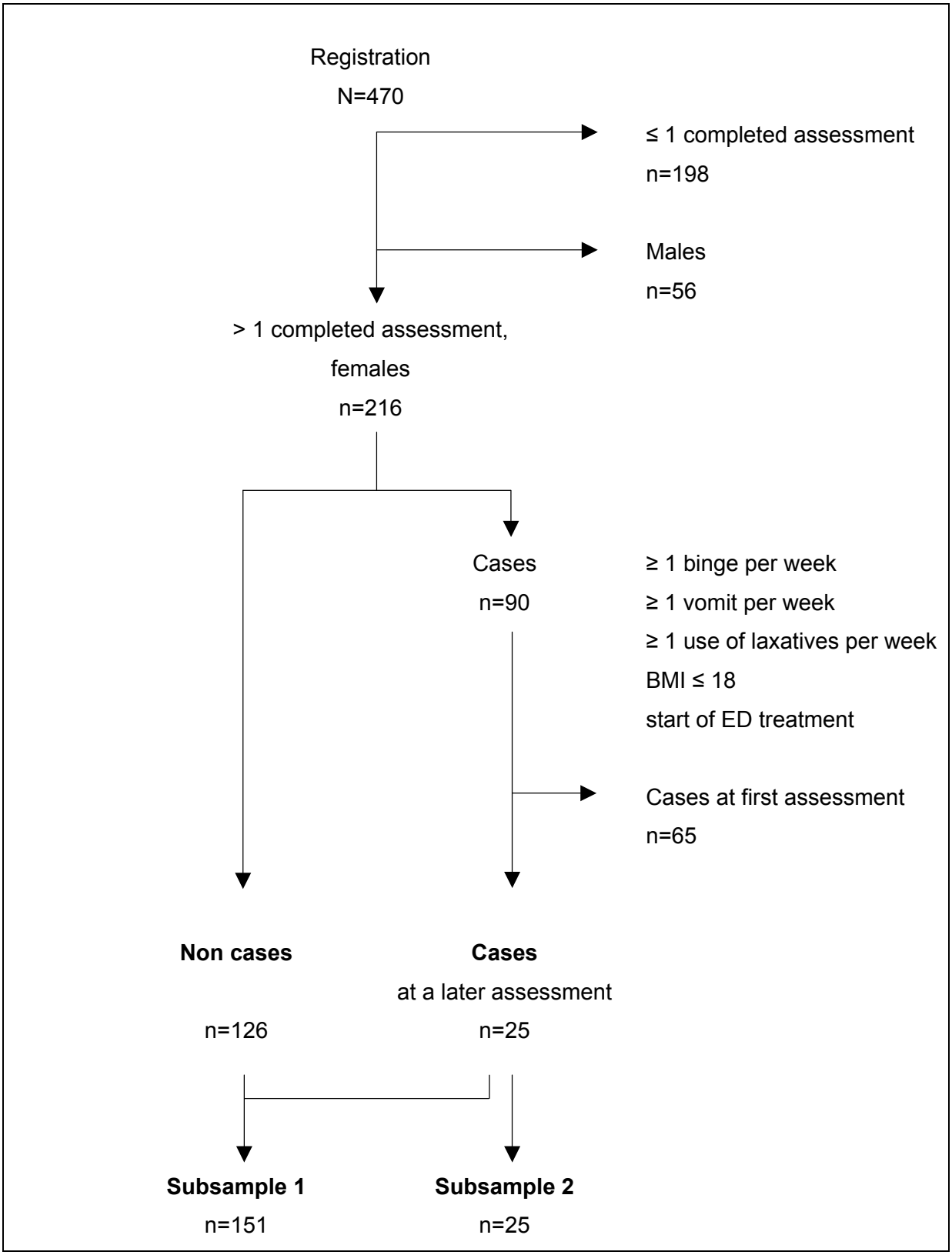


Figure IV.1 Sampling

From the 470 registered participants, 198 individuals (42.13%) are excluded due insufficient data (≤ 1 completed assessment), and 56 individuals (11.91%) due to male gender. From the remaining sample of 216 participants, 90 individuals (41.67%) fulfill at least one partial ED criterion during the total observation time (“Cases”), whereas 126 individuals (58.33%) never meet criteria for partial ED (“Non cases”). From the 90 partial ED cases, 65 individuals (72.22%) are excluded due to fulfilling the partial ED criteria at their first measurement (“Cases at first assessment”). The remaining sample of cases (“Subsample 2”), consists of 25 individuals. Those and the 126 non cases, compose the “Subsample 1” (n = 151).

1.1 Subsample 1

Subsample 1 is used to address the first aim – the identification of risk factors for the onset of ED symptoms. The subsample consists of partial ED cases (n = 25) and non cases (n = 126). Table IV.1 briefly describes subsample 1. On average participants are 22.29 years old (range = 18-50), and completed 7.44 numbers of assessments (median = 5).

Table IV.1 Description subsample 1

	PT		F		all	
Site (%)	94	(62.3)	57	(37.7)	151	(100.0)
Age (SD)	22.88	(5.16)	21.33	(2.25)	22.29	(4.34)
Assessment (SD)	7.78	(6.32)	6.88	(4.93)	7.44	(5.83)

Note: PT = Portugal, F = France, SD = standard deviation

There are no significant differences between the partial ED cases and the non cases regarding the following variables: percentage of participants from the Portuguese vs. French site, χ^2 (1, *N* =151) = 0.065, *p* = .799; average age, *t*(148) = 0.156, *p* = 0.876; and average number of completed assessments, *t*(150) = 0.04, *p* = 0.968 (for more details see annex, Table VII.7, p. 134).

1.2 Subsample 2

Subsample 2 is used to address the second aim – the temporal course of factors prior to partial ED onset. The subsample includes only partial ED cases (n = 25). Table IV.2 briefly describes subsample 2. Two third of the partial ED cases are from the Portuguese site; on average participants are 22.42 years old (range = 18-30), and complete 7.48 numbers of assessments (median = 5).

Table IV.2 Description subsample 2

	PT		F		all	
Site (%)	15	(60.0)	10	(40.0)	25	(100.0)
Age (SD)	23.17	(3.11)	21.30	(2.58)	22.42	(3.00)
Assessment (SD)	8.93	(7.27)	5.30	(3.23)	7.48	(6.17)

Note: PT = Portugal, F = France, SD = standard deviation

The composite outcome criteria “partial ED” includes binge eating behavior, vomiting, misuse of laxatives, a BMI less than 18, and the start of ED treatment. Table IV.3 shows the partial ED criteria at the time when the 25 subjects first reported ED symptoms.[5]

Table IV.3 Case according to partial ED criteria

	PT	F	all		χ^2	*p*-value
Binge (%)	12	9	21	(84.0)	4.46	.504
Vomit (%)	2	0	2	(8.0)	1.45	.229
Misuse of laxatives (%)	5	0	5	(20.0)	4.17	.041
Start of ED treatment (%)	1	1	2	(8.0)	0.09	.763

Note: PT = Portugal, F = France, SD = standard deviation, χ^2 = χ^2 test statistic

From the 25 partial ED cases, 21 subjects (84%) fulfill the criterion of binge eating behavior at least once per week, 5 subjects (20%) report the misuse of laxatives as means of controlling shape or weight, 3 subjects (8%) report either vomiting or start of ED treatment; none of the participants meet the criterion of a BMI less than 18. From the 21 binge eaters, 5 subjects (23.8%) report at least one other partial ED criterion simultaneously. Participants report only the misuse of laxatives significantly more at the Portuguese site, χ^2 (1, *N* =25) = 4.17, *p* = .041.

[5] *Note*: n = 5 participants reported more the one partial ED criteria simultaneously.

2 *Identification of risk factors for the onset of eating disorder symptomatology*

In order to identify risk factors for the later onset of ED symptoms, survival analyses are conducted. First the cumulative probability of staying well over a period of twelve months is presented (Figure IV.2).

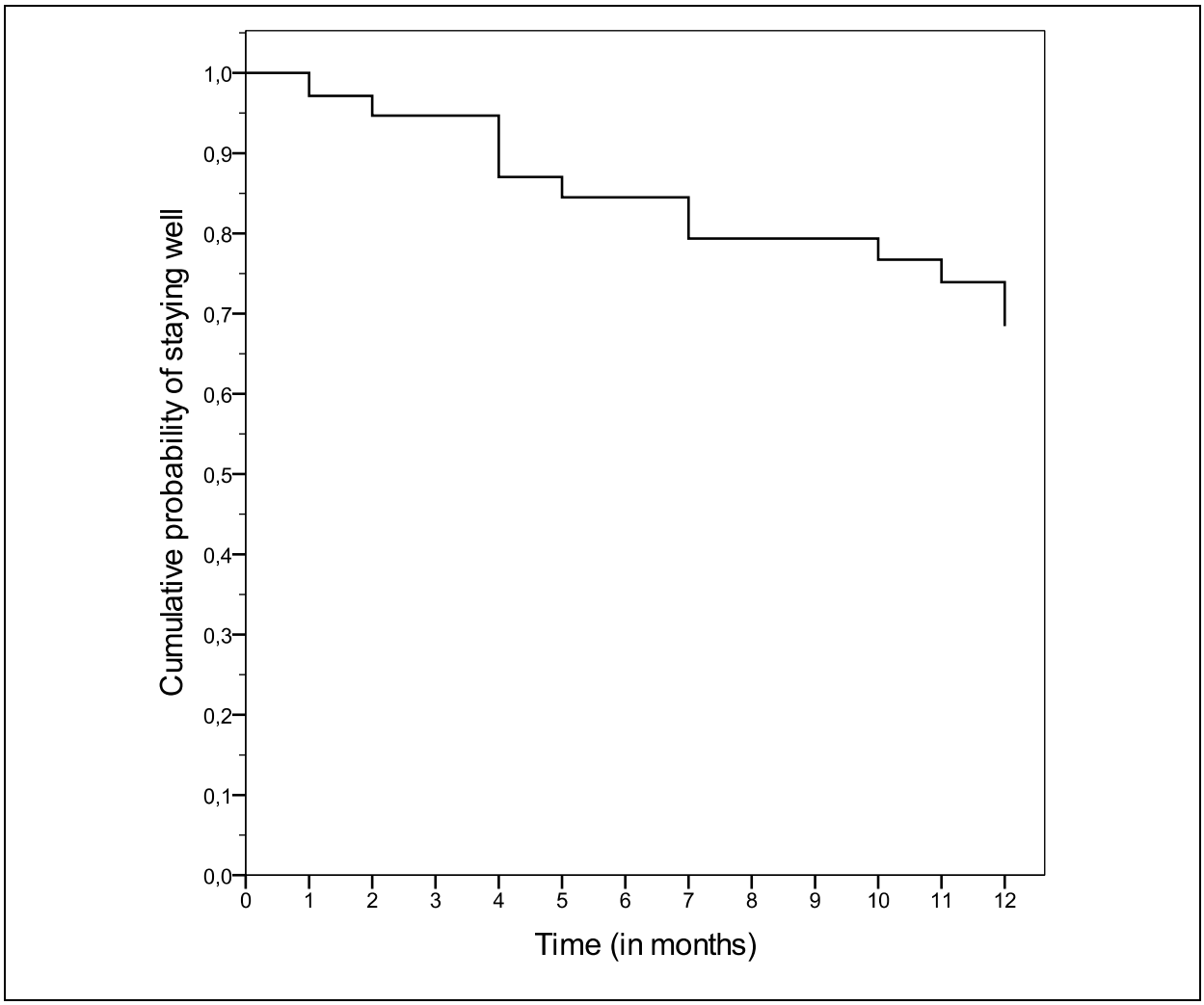


Figure IV.2 Probability of staying well

The survival curve displays the percentage of participants who experience at least one ED symptom vs. participants who stay well over the period of twelve months (for more information about the observation time, see section III.5.1, p. 59).

The life table shows the numbers of participants who stay well, who terminate (due to loss to follow-up), who are exposed to the risk for experiencing partial ED onset, the number of partial ED onsets, and the hazard rate (Table IV.4).

Table IV.4 Probability of staying well: Life table

Time in months	N staying well	N terminated	N exposed to risk	N events	% staying well	Cumulative % staying well	Hazard rate
0	151	0	151.0	0	1.00	1.00	.00
1	151	22	140.0	4	.97	.97	.03
2	125	13	118.5	3	.97	.95	.03
3	109	14	102.0	0	1.00	.95	.00
4	95	17	86.5	7	.92	.87	.08
5	71	5	68.5	2	.97	.84	.03
6	64	11	58.5	0	1.00	.84	.00
7	53	7	49.5	3	.94	.79	.06
8	43	4	41.0	0	1.00	.79	.00
9	39	8	35.0	0	1.00	.79	.00
10	31	2	30.0	1	.97	.77	.03
11	28	1	27.5	1	.96	.74	.04
12	26	25	13.5	1	.93	.68	.08

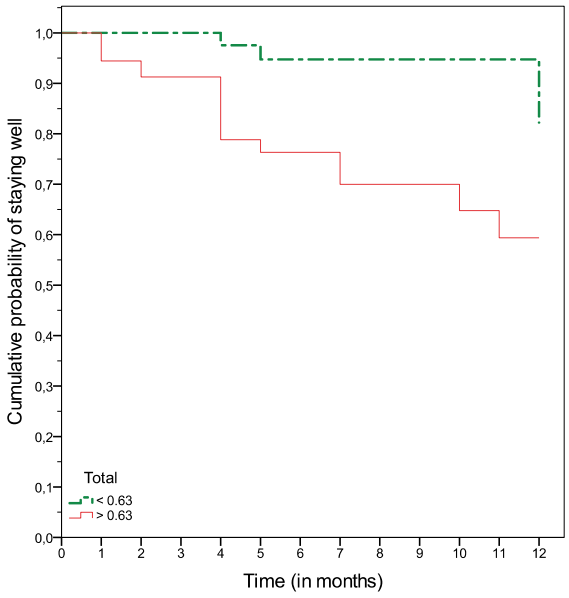
Note: N staying well = number of participants who stay well, N terminated = number of participants who terminate the study, N exposed to risk = number of participants who are exposed to the risk of experiencing partial ED onset, N events = number of partial ED onsets, % staying well = percentage of participants who stay well, Cumulative % staying well = cumulative percentage of participants who stay well

Out of the 151 participants, 126 individuals (83.44%) never report partial ED criteria, whereas 25 (16.56%) fulfill at least one criterion during the total observation time: 22 individuals meet the partial ED criteria during a period of twelve months (14.57% of the 151 participants), while 3 individuals fulfill the criterion later in time. The cumulative probability of staying well yields 68 percent over a period of twelve months.

In order to test risk factors for the onset of partial ED, low- vs. high-risk groups – established with a median split in the total, global, and subscales of the EDE-Q, CR-EAT, and DASS-21 – are compared.

EDE-Q total and subscales

First, the EDE-Q total and subscales (dietary restraint, eating concern, weight concern, shape concern) are tested as risk factors for the onset of partial ED. Figure IV.3 displays the survival curves of the low- and high-risk groups for the EDE-Q total scale over a period of twelve months.



Total

Figure IV.3 Probability of staying well: EDE-Q total scale

Figure IV.4 displays the survival curves of the low- and high-risk groups for the EDE-Q subscales (dietary restraint, eating concern, weight concern, shape concern).

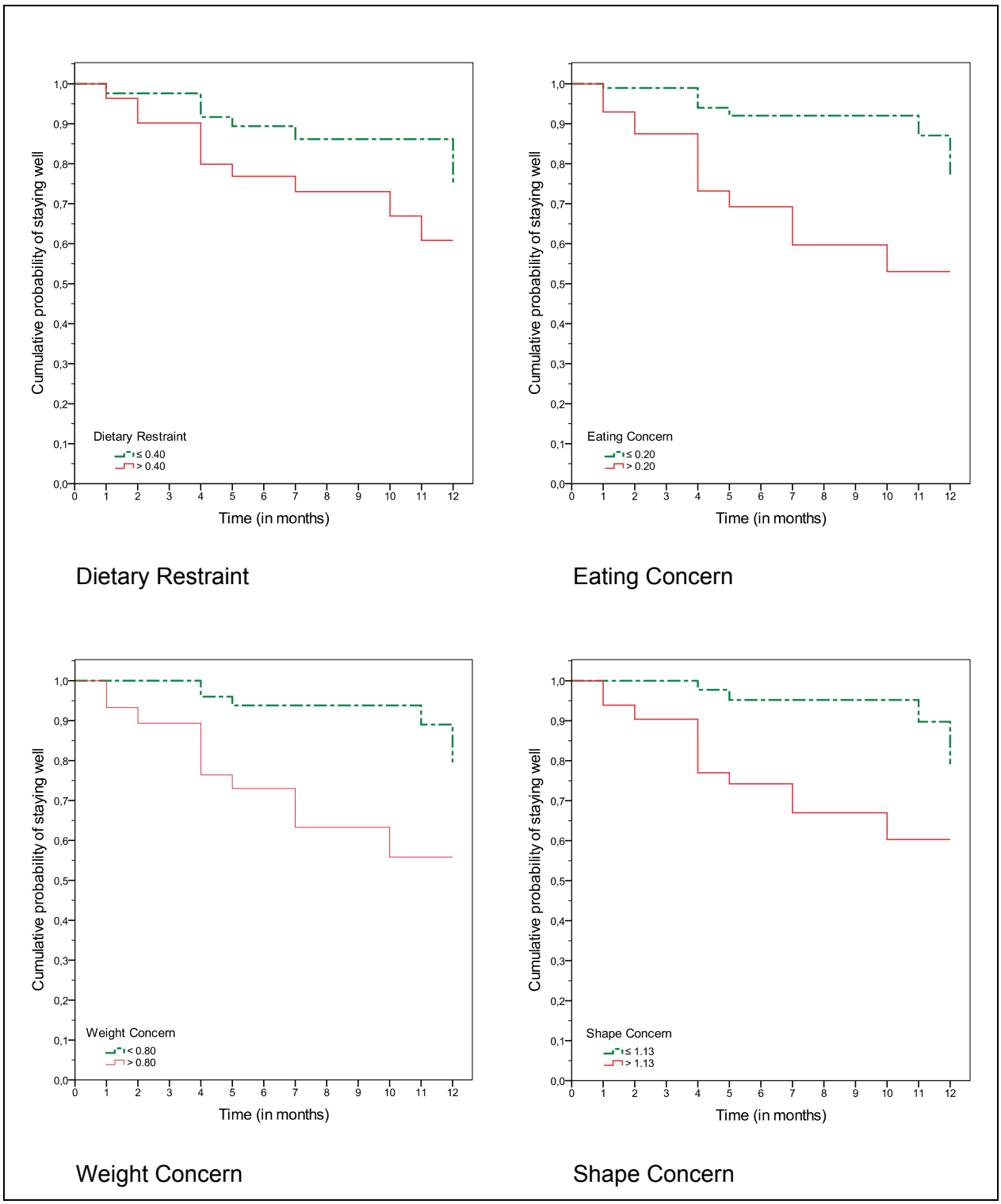


Figure IV.4 Probability of staying well: EDE-Q subscales

In order to identify risk factors for the onset of ED symptoms, differences in the survival curves of low- vs. high-risk individuals are tested for all EDE-Q scales.[6] Table IV.5 shows the cumulative probabilities of staying well for the low- vs. high-risk groups, and the log-rank test statistics.

Table IV.5 Difference in the cumulative probability of staying well: EDE-Q scales

	Cut off	Cumulative probability of staying well		χ^2	*p*-value
		low risk	*high risk*		
TO	> 0.63	0.82	0.59	9.71	.002
DR	> 0.40	0.75	0.61	4.22	.040
EC	> 0.20	0.77	0.53	12.69	.000
WC	> 0.80	0.80	0.56	11.59	.001
SC	> 1.13	0.79	0.60	10.34	.001

Note: Cut off = cut off scores for high-risk groups, χ^2 = log-rank test statistic, TO = Total Score, DR = Dietary Restraint, EC = Eating Concern, WC = Weight Concern, SC = Shape Concern

Survival curves of low- vs. high-risk individuals differ significantly for all EDE-Q scales: high-risk individuals in dietary restraint, χ^2 (1, *N*=149) = 4.22, $p < .05$; eating concern, χ^2 (1, *N*=147) = 12.69, $p < .01$; weight concern, χ^2 (1, *N*=147) = 11.59, $p < .01$; and shape concern, χ^2 (1, *N*=147) = 10.34, $p < .01$, are more likely to experience the onset of ED symptoms during a period of twelve months.

[6] *Note*: Life tables for the EDE-Q scales are presented in annex, Table VII.10 – Table VII.14, pp. 140.

In order to examine the potencies of the EDE-Q scales, odds ratios are calculated. Table.IV.6 presents the number of events (onset of partial ED) according to the risk groups, and the odds ratios with 95% confidence intervals.

Table.IV.6 Risk potencies: EDE-Q scales

	n	N of events		OR	95% CI
		low risk	*high risk*		
TO	147	3 / 69	17 / 78	5.01	1.41 – 17.84
DR	149	8 / 91	13 / 58	2.55	1.00 – 6.53
EC	147	7 / 100	13 / 47	3.95	1.48 – 10.55
WC	147	5 / 82	15 / 65	3.78	1.31 – 10.96
SC	147	4 / 76	16 / 71	4.28	1.37 – 13.42

Note: n = number of participants, N of events = number of participants experiencing partial ED onset, OR = odds ratio, 95% CI = 95% confidence interval of odds ratio, TO = Total Score, DR = Dietary Restraint, EC = Eating Concern, WC = Weight Concern, SC = Shape Concern

The odds ratios for the EDE-Q subscales range between 2.55 and 4.28 (confidence interval lower bounds > 1).[7] The odds ratio of the EDE-Q total scale suggests that the odds of experiencing a partial ED onset for high-risk individuals are 5.01 times greater than the odds for low-risk individuals.

CR-EAT total and global scales

Second, the CR-EAT total and global scales (eating behavior disturbance, affective/cognitive impairment, and perfectionism total) are tested as risk factors for the onset of ED symptoms.

[7] An exception is the lower bound of the 95% confidence interval for the EDE-Q subscale Dietary Restraint: confidence interval lower bound = 1.

Figure IV.5 displays the survival curves of the low- and high-risk groups for the CR-EAT total and global scales over a period of twelve months.[8]

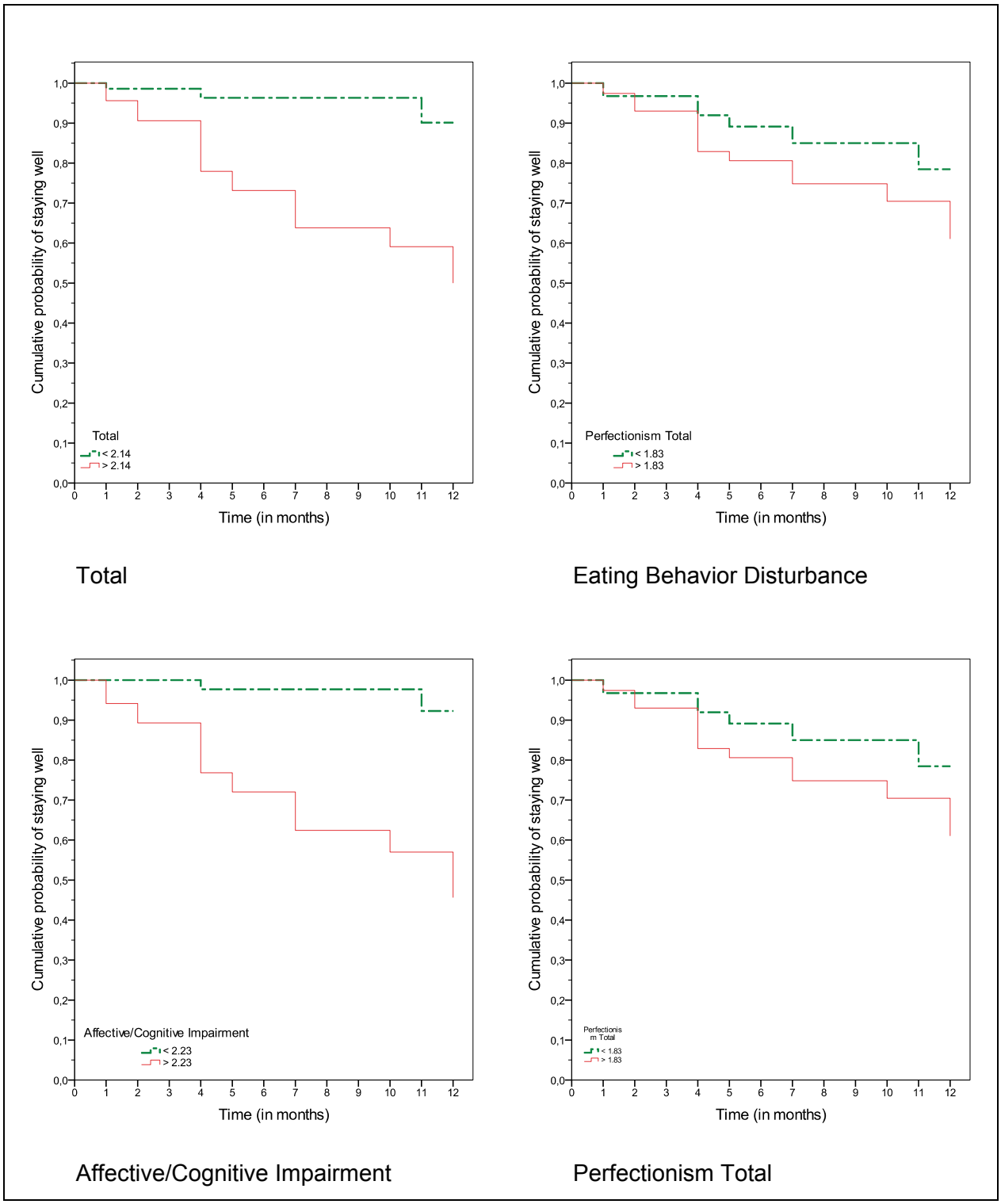


Figure IV.5 Probability of staying well: CR-EAT total & global scales

[8] *Note*: Survival curves of the CR-EAT subscales are displayed in annex, Figure VII.3 – Figure VII.5, pp. 135.

Differences in the survival curves of low- vs. high-risk individuals are tested for the CR-EAT total and global scales.[9] Table IV.7 shows the cumulative probabilities of staying well for the low- vs. high-risk groups, and the log-rank test statistics.[10]

Table IV.7 Difference in the cumulative probability of staying well: CR-EAT scales

	Cut off	Cumulative probability of staying well		χ^2	p-value
		low risk	*high risk*		
TO	> 2.14	0.90	0.50	13.56	.000
EBD	> 1.76	0.76	0.59	8.50	.004
ACI	> 2.23	0.92	0.46	18.17	.000
PER	> 1.83	0.78	0.61	1.71	.191

Note: Cut off = cut off scores for high-risk groups, χ^2 = log-rank test statistic, TO = Total Score, EBD = Eating Behavior Disturbance, ACI = Affective/Cognitive Impairment, PER = Perfectionism Total

Survival curves of low- vs. high-risk individuals differ significantly for the CR-EAT total scale χ^2 (1, *N*=150) = 13.56, $p < .01$; and the global scales eating behavior disturbance, χ^2 (1, *N*=150) = 8.50, $p < .01$; and affective/cognitive impairment, χ^2 (1, *N*=150) = 18.17, $p < .01$. No differences are found for the survival curves of the global scale perfectionism, χ^2 = (1, *N*=150) = 1.71, $p = .191$.

[9] *Note*: Life tables of the CR-EAT scales are presented in annex, Table VII.15 – Table VII.30, pp.145.
[10] *Note*: Cumulative probabilities of staying well, and log-rank test statistics of the CR-EAT subscales are presented in annex, Table VII.8, p. 138.

In order to examine the potencies of the CR-EAT total and global scales, odds ratios are calculated.[11] Table.IV.8 presents the number of events (onset of partial ED) according to the risk groups, and the odds ratios with 95% confidence intervals.

Table.IV.8 Risk potencies: CR-EAT scales

	n	N of events		OR	95% CI
		low risk	*high risk*		
TO	150	3 / 75	19 / 75	6.33	1.80 – 22.31
EBD	150	5 / 75	17 / 75	3.40	1.19 – 9.68
ACI	150	2 / 76	20 / 74	10.27	2.32 – 45.50
PER	150	7 / 66	15 / 84	1.68	0.65 – 4.37

Note: n = number of participants, N of events = number of participants experiencing partial ED onset, OR = odds ratio, 95% CI = 95% confidence interval of odds ratio, TO = Total Score, EBD = Eating Behavior Disturbance, ACI = Affective/Cognitive Impairment, PER = Perfectionism Total

The odds ratios for the CR-EAT total and the global scales eating behavior disturbance and affective/cognitive impairment range between 3.40 and 10.27 indicating significant greater odds for the high-risk individuals to experience the event of partial ED onset. The perfectionisms global scale yields a non significant odds ratio (95% confidence interval lower bound < 1).

DASS-21 total and subscales

Finally, the DASS-21 total and subscales (depression, anxiety, stress) are tested as risk factors for the onset of partial ED.

[11] *Note*: Odds ratios for the CR-EAT subscales are presented in annex, Table.VII.9, p. 139.

Figure IV.6 displays the survival curves of the low- and high-risk groups for the DASS-21 total and subscales over a period of twelve months.

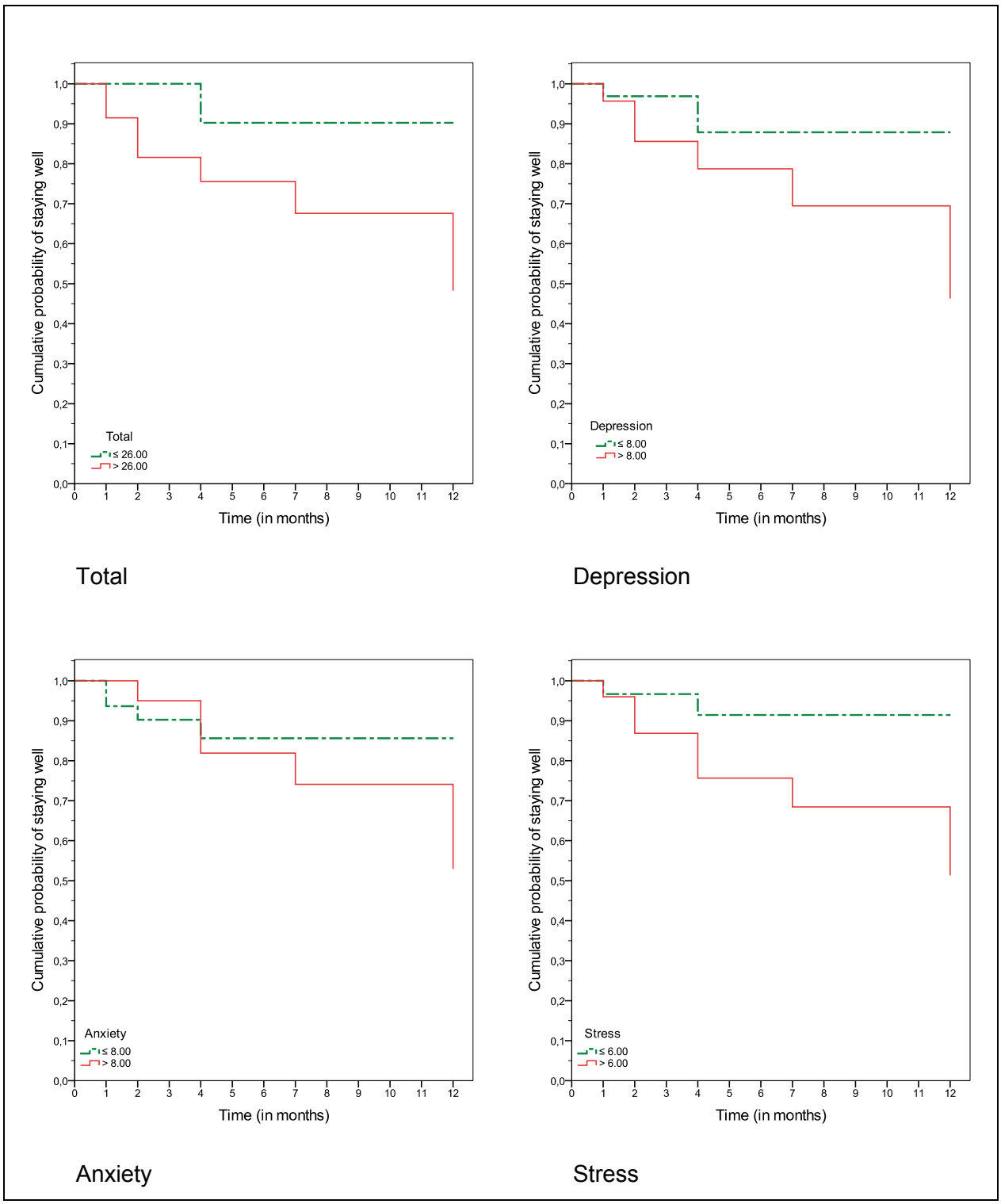


Figure IV.6 Probability of staying well: DASS-21 total & subscales

In order to identify risk factors for the onset of ED symptoms, differences in the survival curves of low- vs. high-risk individuals are tested for all DASS-21 scales.[12] Table IV.9 shows the cumulative probabilities of staying well for the low- vs. high-risk groups, and the log-rank test statistics.

Table IV.9 Difference in the cumulative probability of staying well: DASS-21 scales

	Cut off	Cumulative probability of staying well		χ^2	p-value
		low risk	*high risk*		
TO	> 26.00	0.90	0.48	4.66	.031
DE	> 8.00	0.88	0.46	2.63	.105
AN	> 8.00	0.86	0.53	0.41	.522
ST	> 6.00	0.78	0.61	3.39	.066

Note: Cut off = cut off scores for high-risk groups, χ^2 = log-rank test statistic, TO = Total Score, DE = Depression, AN = Anxiety, ST = Stress

Only the survival curves of the DASS-21 total scale differ significantly χ^2 (1, N=59) = 4.66, $p < .05$. No differences are found between low- and high-risk individuals in depression, χ^2 (1, N=59) = 2.63, p = .105; anxiety, χ^2 (1, N=59) = 0.41, p = .522; and stress, χ^2 (1, N=59) = 3.39, p = .066, regarding the probability of experiencing the onset of ED symptoms during a period of twelve months.

[12] *Note*: Life tables of the DASS-21 scales are presented in annex, Table VII.31 – Table VII.34, pp. 161.

In order to examine the potencies of the DASS-21 total and subscales, odds ratios are calculated. Table.IV.10 presents the number of events (onset of partial ED) according to the risk groups, and the odds ratios with 95% confidence intervals.

Table.IV.10 Risk potencies: DASS-21 scales

	n	N of events		OR	95% CI
		low risk	*high risk*		
TO	59*	2 / 33	7 / 26	4.44	0.85 – 23.21
DE	59*	3 / 34	6 / 25	2.72	0.62 – 11.94
AN	59*	4 / 33	5 / 26	1.59	0.39 – 6.51
ST	59*	2 / 31	7 / 28	3.88	0.74 – 20.23

Note: n = number of participants, N of events = number of participants experiencing partial ED onset, OR = odds ratio, 95% CI = 95% confidence interval of odds ratio, TO = Total Score, DE = Depression, AN = Anxiety, ST = Stress, * = the DASS-21 was only administered (at best) at every other monthly assessment

Non significant odds ratios are obtained for the DASS-21 total and the global scales (95% confidence interval lower bound < 1).

Summary chapter IV.2

Individuals with high scores in dietary restraint, eating concern, weight concern, shape concern, eating behavior disturbance, and affective/cognitive impairment, are more likely to experience the onset of ED symptoms during a twelve months period. Strong potencies are obtained especially for eating concern, weight concern, shape concern, eating behavior disturbance, and affective/cognitive impairment.

3 Temporal course of factors prior to the onset of eating disorder symptomatology

In order to explore the temporal course of factors prior to partial ED onset, trajectories of these factors are explored. Intercepts and slopes are estimated using a hierarchical linear modeling approach (see section III.5.2, p. 60).

EDE-Q total and subscales

First, the trajectory of the EDE-Q total scale is analyzed. Figure IV.7 shows the course over a period of 120 days prior to the onset of partial ED.

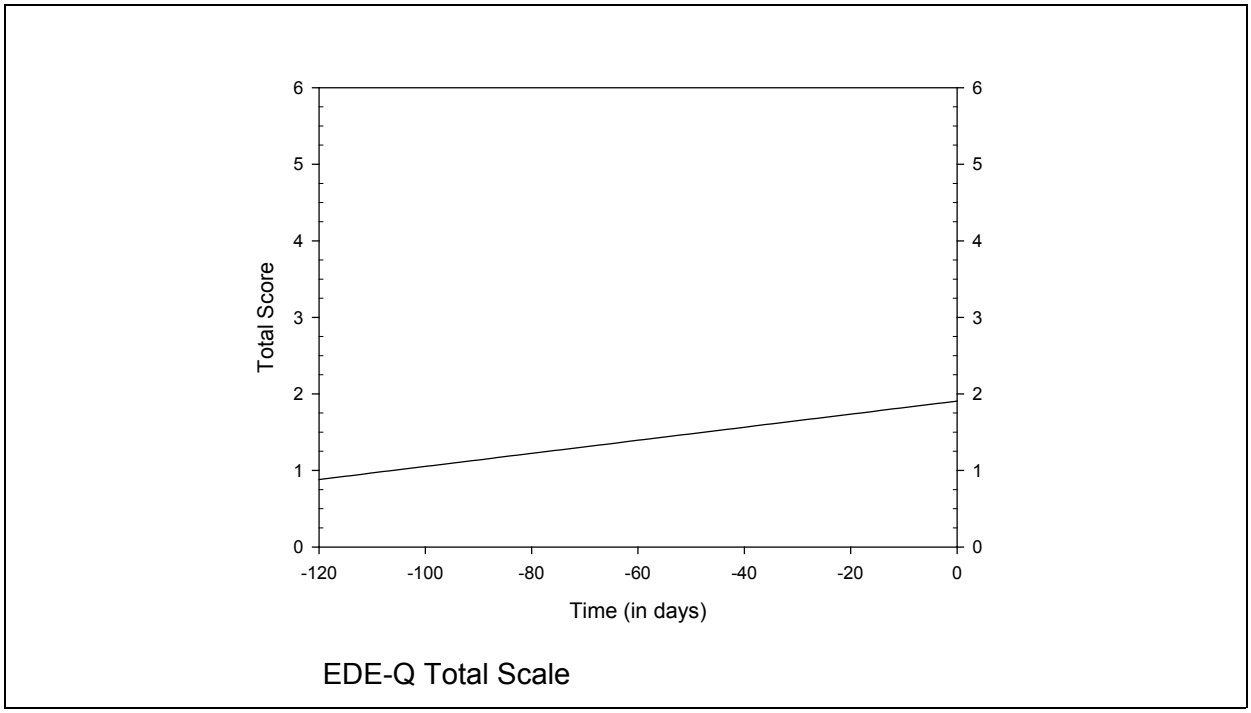


Figure IV.7 Temporal course of the EDE-Q total scale

The trend of the EDE-Q total scale increases over time (120 days prior to partial ED onset): On average the partial ED cases report increasing EDE-Q scores at the monthly measurements.

Figure IV.8 shows the trajectories of the EDE-Q subscales (dietary restraint, eating concern, weight concern, and shape concern).

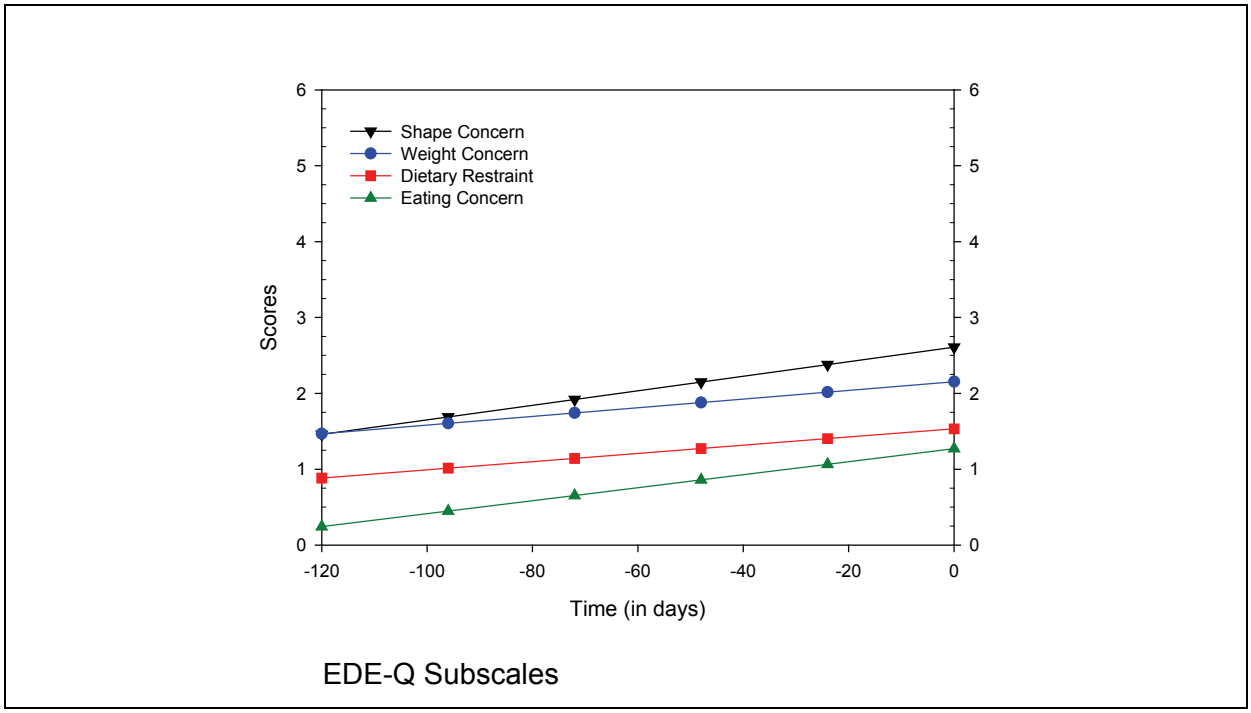


Figure IV.8 Temporal courses of the EDE-Q subscales

All EDE-Q subscales are increasing over the period of four months prior to the onset of ED symptoms. Table IV.11 reports the intercepts[13] and slopes for the EDE-Q scales.

Table IV.11 Temporal course of the EDE-Q scales: Intercept & slope

	Intercept	t-value	p-value	Slope	t-value	p-value
TO	1.91	8.72	.000	0.008538	4.71	.000
DR	1.53	5.72	.000	0.005404	2.71	.009
EC	1.27	4.94	.000	0.008566	3.49	.001
WC	2.15	8.57	.000	0.005714	3.50	.001
SC	2.61	10.27	.000	0.009570	4.37	.000

Note: TO = Total Score, DR = Dietary Restraint, EC = Eating Concern, WC = Weight Concern, SC = Shape Concern

[13] *Note*: Time was centered at the onset of partial ED symptoms.

Over the period of 120 days, both the trajectories of the EDE-Q total scale, and EDE-Q subscales increase significantly. Every month participants report on average an increase of 0.26 points on the EDE-Q total scale, dietary restraint increases by 0.16, eating concern by 0.26, weight concern by 0.17, and shape concern by 0.29 points, respectively. Hundred and twenty days prior to the partial ED onset, the projected average scores of participants are 1.47 points for weight concern, 1.46 points for shape concern, 0.88 points for dietary restraint, and 0.24 points for eating concern, respectively.

CR-EAT total and global scales

Next, trajectories of the CR-EAT total and global scales (eating behavior disturbance, affective/cognitive impairment, and perfectionism total) are analyzed over the period of 120 days prior to partial ED onset (Figure IV.9).

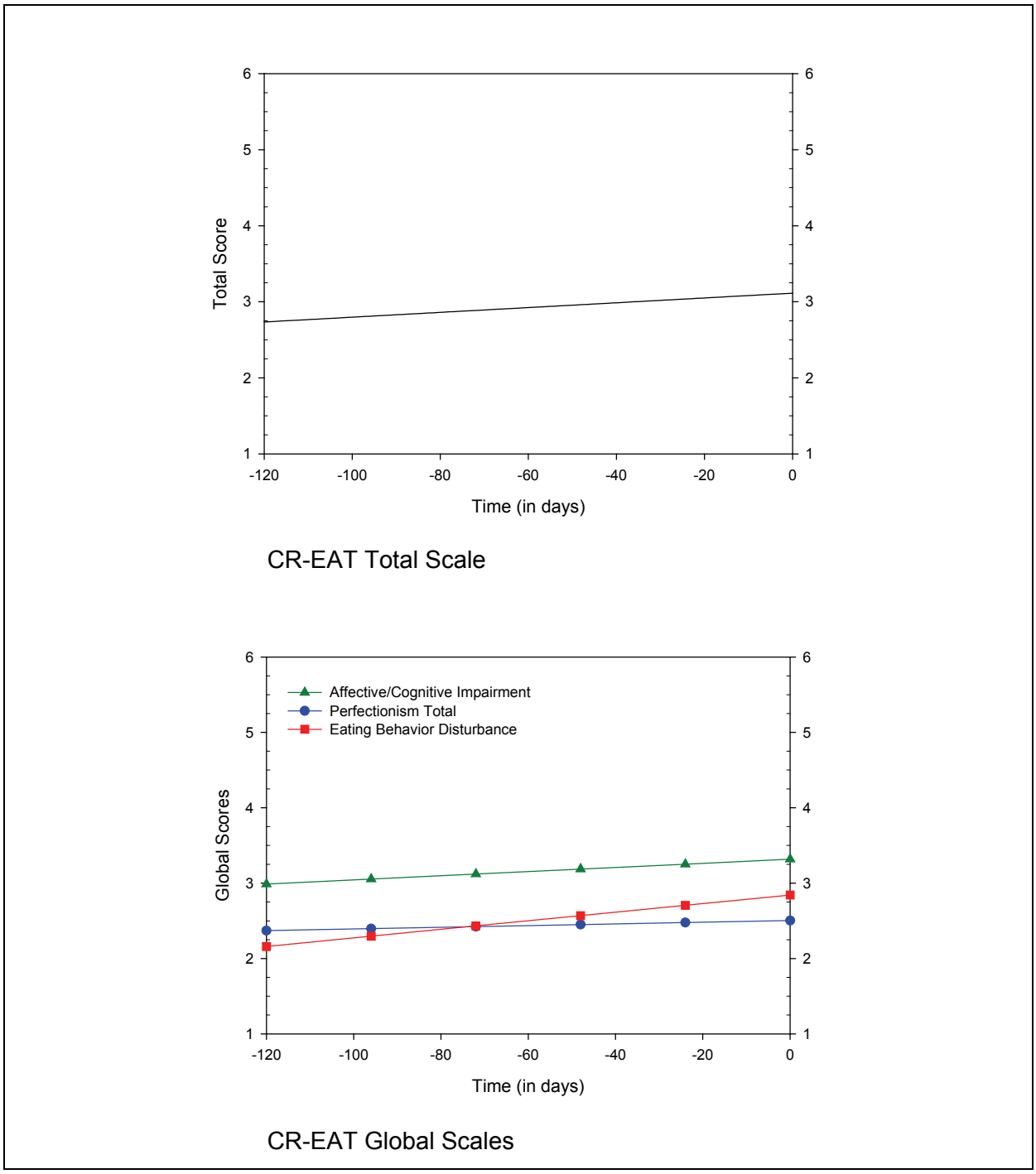


Figure IV.9 Temporal courses of the CR-EAT total & global scales

In Table IV.12 the intercepts and slopes of the CR-EAT total and global scales are presented.

Table IV.12 Temporal course of the CR-EAT total & global scales: Intercept & slope

	Intercept	t-value	p-value	Slope	t-value	p-value
TO	3.11	20.00	.000	0.003158	3.54	.000
EBD	2.84	15.99	.000	0.005696	5.12	.000
ACI	3.32	17.33	.000	0.002755	2.52	.015
PER	2.50	16.51	.000	0.001107	0.10	.324

Note: TO = Total Score, EBD = Eating Behavior Disturbance, ACI = Affective/Cognitive Impairment, PER = Perfectionism Total

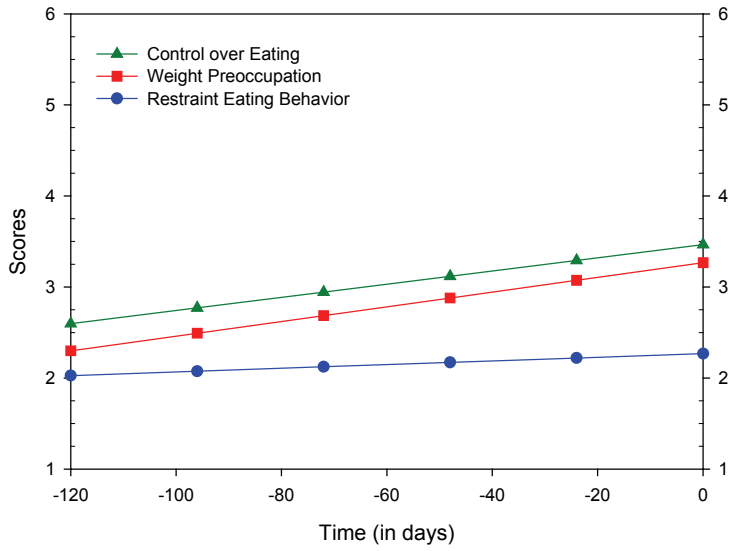
Both the CR-EAT total scale, and the two global scales eating behavior disturbance and affective/cognitive impairment show a significant increase. However, no significant increase is obtained in the global scale perfectionism. On average participants report monthly an increase of 0.17 points in eating behavior disturbance, and 0.08 points in affective/cognitive impairment. Projected average scores 120 days prior to the partial ED onset, are 2.99 points for affective/cognitive impairment, 2.37 points for perfectionism and 2.16 points for eating behavior disturbance.

In the following, trajectories of the CR-EAT subscales are analyzed. For a better overview the subscales are presented based on the three global scales: eating behavior disturbance, affective/cognitive impairment, and perfectionism total.

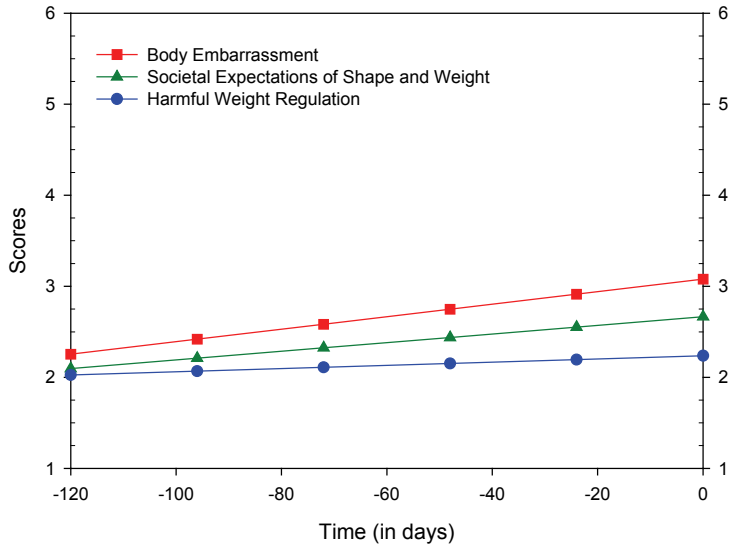
CR-EAT subscales of the global scale eating behavior disturbance

The global scale eating behavior disturbance includes the CR-EAT subscales weight preoccupation, control over eating, body embarrassment, restraint eating behavior, societal expectations of shape and weight, and harmful weight regulation.

Figure IV.10 displays the trajectories of the 6 CR-EAT subscales which compile the global scale eating behavior disturbance.



CR-EAT Eating Behavior Disturbance Subscales I



CR-EAT Eating Behavior Disturbance Subscales II

Figure IV.10 Temporal courses of the CR-EAT subscales I

Table IV.13 gives an overview about the intercepts and slopes of these 6 CR-EAT subscales.

Table IV.13 Temporal course of the CR-EAT subscales I: Intercept & slope

	Intercept	t-value	p-value	Slope	t-value	p-value
WP	3.27	14.41	.000	0.008070	5.40	.000
CE	3.47	13.56	.000	0.007236	3.05	.004
BE	3.08	12.18	.000	0.006868	3.41	.001
RE	2.27	11.53	.000	0.002004	1.08	.284
SO	2.67	12.79	.000	0.004746	3.65	.000
HW	2.24	10.31	.000	0.001763	1.46	.150

Note: WP = Weight Preoccupation, CE = Control over Eating, BE = Body Embarrassment, RE = Restraint Eating Behavior, SO = Societal Expectations of Shape and Weight, HW = Harmful Weight Regulation

The subscales weight preoccupation, control over eating, body embarrassment, and societal expectations of shape and weight increase all significantly over the period of 120 days prior to the onset of partial ED. On average participants report every month 0.24 points higher weight preoccupation, 0.22 points higher control over eating, 0.21 points higher body embarrassment, and 0.14 points higher societal expectations of shape and weight. Projected average scores 120 days prior to the partial ED onset, are 2.60 points for control over eating, 2.30 points for weight preoccupation, 2.25 points for body embarrassment, 2.10 points for societal expectations of shape and weight.

Non significant slopes are found for the subscales restraint eating behavior and harmful weight regulation. Projected average scores 120 days prior to partial ED onset, are for both scales 2.03 points.

CR-EAT subscales of the global scales affective/cognitive impairment and perfectionism total

Figure IV.11 shows the trajectories of the CR-EAT subscales emotional dysregulation, affect-regulatory eating, self-esteem, concern about negative evaluation (global scale affective/cognitive impairment), familial expectations, and personal expectations (global scale perfectionism total).

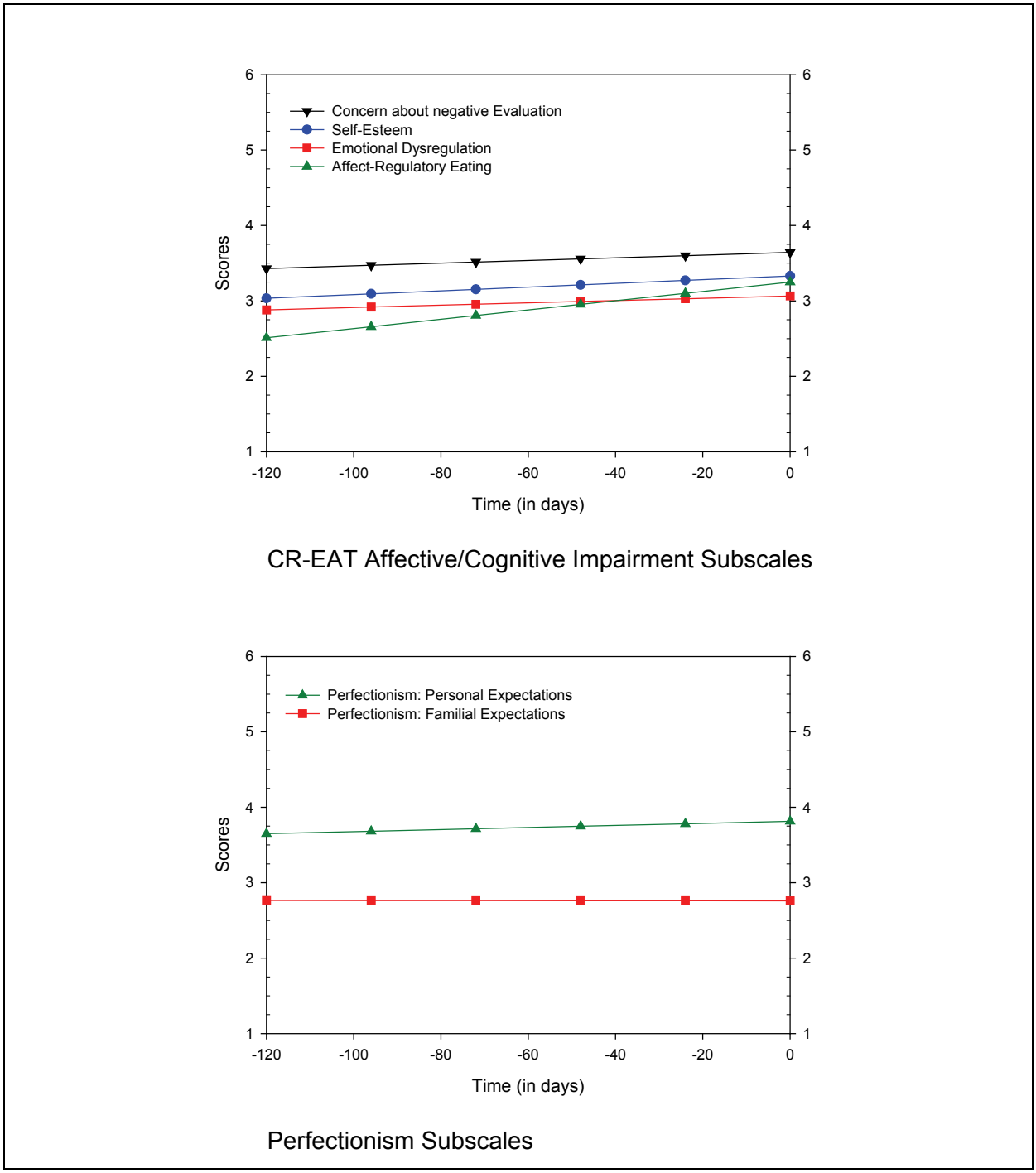


Figure IV.11 Temporal courses of the CR-EAT subscales II

Table IV.14. gives an overview about the intercepts and slopes of the CR-EAT subscales that form the global scales affective/cognitive impairment, and perfectionism total.

Table IV.14 Temporal course of the CR-EAT subscales II: Intercept & slope

	Intercept	t-value	p-value	Slope	t-value	p-value
ED	3.07	12.45	.000	0.001536	0.98	.333
AE	3.25	11.07	.000	0.006154	3.98	.000
SE	3.33	20.72	.000	0.002467	1.59	.118
CN	3.64	15.89	.000	0.001783	1.07	.291
PF	2.76	10.81	.000	-0.000028	-0.01	.986
PP	3.81	18.08	.000	0.001371	0.98	.331

Note: ED = Emotional Dysregulation, AE = Affect-Regulatory Eating, SE = Self-Esteem, CN = Concern about Negative Evaluation, PF = Perfectionism: Familial Expectations, PP = Perfectionism: Personal Expectations

Only affect-regulatory eating increases significantly over the period of 120 days: participants report a monthly increase of 0.18 points starting at 2.51 points 120 days prior the partial ED onset (projected average scores). The subscales emotional dysregulation, self-esteem, and concern about negative evaluation show non significant slopes. All these scales yield elevated scores 120 days prior to the partial ED onset: concern about negative evaluation (3.43), self-esteem (3.03), and emotional dysregulation (2.88). Both perfectionism subscales do not increase over time: participants score on average 2.76 points on the familial expectations scale, and 3.65 on the personal expectations scale, respectively.

DASS-21 total and subscales

In the following the temporal course of the DASS-21 total and subscales (depression, anxiety, stress) are explored.

Figure IV.12 shows the trajectories of the DASS-21 total and subscales over a period of 120 days.

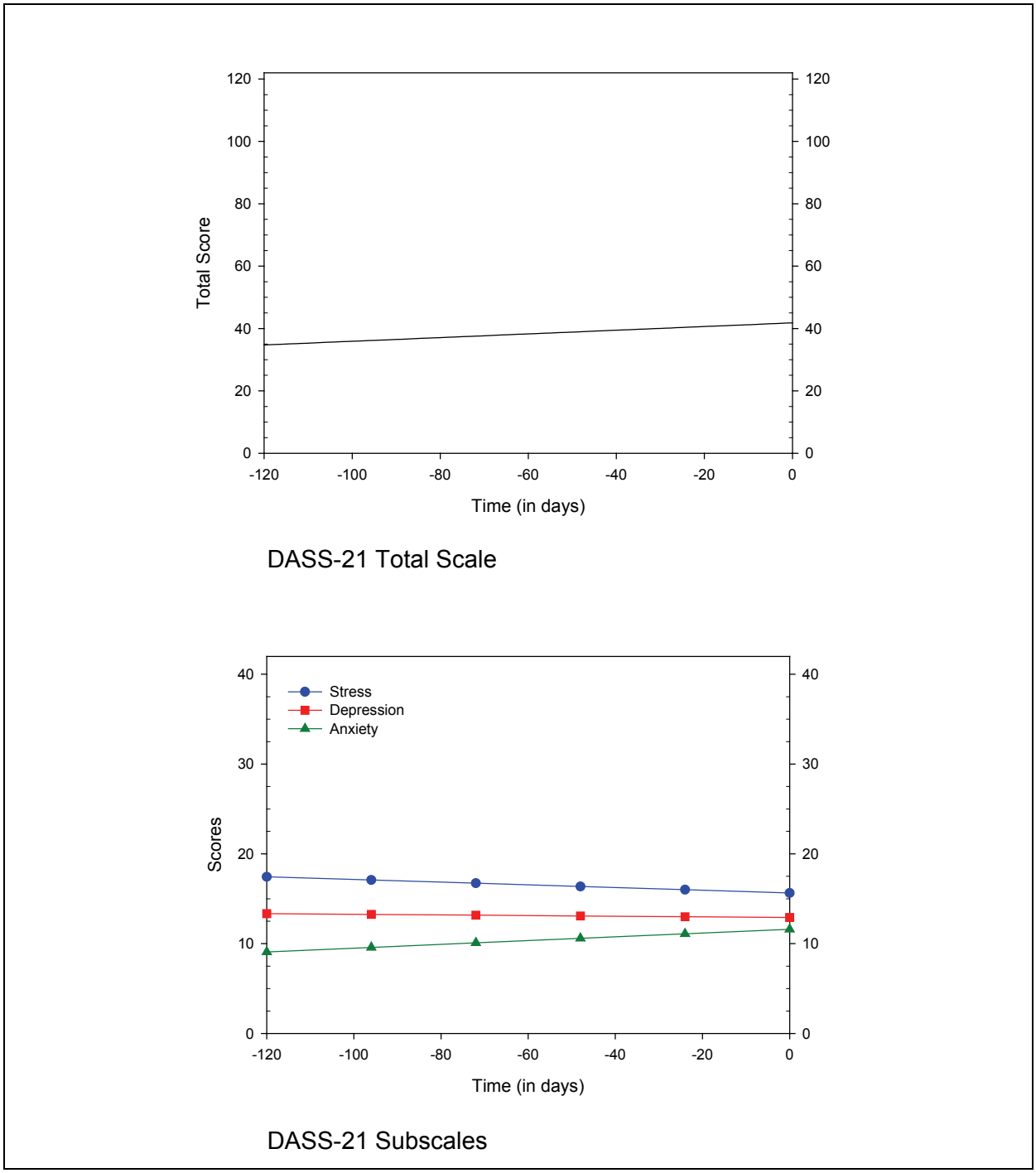


Figure IV.12 Temporal courses of the DASS-21 scales

Intercepts and slopes of the DASS-21 total and subscales are shown in Table IV.15.

Table IV.15 Temporal course of the DASS-21 scales: Intercept & slope

	Intercept	t-value	p-value	Slope	t-value	p-value
TO	41.81	7.50	.000	0.05910	1.06	.325
DE	12.92	5.72	.000	-0.00353	-0.33	.750
AN	11.60	5.22	.000	0.02102	0.71	.501
ST	15.65	7.23	.000	-0.01502	-0.91	.395

Note: TO = Total Score, DE = Depression, AN = Anxiety, ST = Stress

The slopes of all DASS-21 scales are non significant indicating no increase over the period of 120 days.

Summary chapter IV.3

Summarizing the results, the factors can be divided into those which show an increase over time, and those which do not increase. Participants report on average an increase in dietary restraint, eating concern, weight concern, shape concern, and almost in all subscales of the global factor eating behavior disturbance (weight preoccupation, control over eating, body embarrassment, societal expectations of shape and weight). None of the subscales of the global factor affective/cognitive impairment (emotional dysregulation, self-esteem, concern about negative evaluation) – except affect-regulatory eating – show an increase over time. Also, the perfectionism and DASS-21 scales are not increasing over time. Four months prior to ED symptoms high scores are obtained especially for weight concern and shape concern, and the affective/cognitive impairment global scale: Although not increasing over time, emotional dysregulation, and self-esteem, are particularly elevated four months prior the onset of ED symptoms.

V. Discussion

The discussion section is divided into four parts: first, discussion of the results, second, methodological issues (outcome criterion, statistical analyses etc.), third, limitations, and strengths of the present study, and fourth, conclusion and outlook.

1 Discussion of the results

The first part of the study resulted in the identification of several risk factors for ED symptomatology. From the 151 participants 22 individuals met the partial ED criteria during a twelve months period (14.57%). High-risk individuals in the factors dietary restraint, restraint eating behavior, eating concern, control over eating, affect-regulatory eating, weight concern, weight preoccupation, shape concern, body embarrassment, societal expectations of shape and weight, harmful weight regulations, emotional dysregulation, and self-esteem are more likely to experience the onset of partial ED. Summarizing the results, three distinctive groups of risk factors emerged: eating related, weight/shape related, and non-specific risk factors. In the following, these three risk factor groups are discussed in detail.

Eating related risk factors

Eating related behaviors – such as dietary restraint, restraint eating behavior, eating concern, control over eating, and affect-regulatory eating – are identified as risk factors with medium to strong potencies (odds ratio: 2.6 – 6.5). Especially potent factors for increasing the risk for partial ED are affect-regulatory eating, which describes eating due to emotional distress, and eating concern, which describes preoccupation with food, fear of losing control, social eating and feelings of guilt about eating. Controlling food intake or being on a diet presents a well supported precursory factor, if not causal, for ED, especially in BN (Jacobi, Hayward et al.,

2004). However, these dysfunctional behaviors towards food and eating are temporally closely related to ED symptoms such as binge eating, vomiting and misuse of laxatives.

Weight/shape related risk factors

Among the weight/shape related constructs weight concern, weight preoccupation, shape concern, body embarrassment, societal expectations of shape and weight, harmful weight regulations are found to be risk factors with medium potencies (odds ratio: 3.4 – 4.3). Weight preoccupation describing concerns about body fat, and shape concern describing e.g. dissatisfaction with body shape, proved to be especially potent factors for increasing the risk for partial ED.

Some of the weight/shape related constructs such as weight concern, or body embarrassment reflect underlying schemas about body weight and dissatisfaction with physical appearance. Supporting evidence classifies e.g. body dissatisfaction as one of the most consistent and robust risk and maintenance factors for eating pathology in general (Stice, 2002; The McKnight Investigators, 2003). Other factors such as shape concern and harmful weight regulation reflect underlying constructs of over-evaluating body shape and attitudes towards dysfunctional weight regulation.

Non-specific risk factors

High emotional dysregulation, and low self-esteem are identified as non-specific risk factors with strong potencies (odds ratio: 4.9 – 7.1). Emotional dysregulation or negative affect is a well studied and supported risk factor for the development of ED in general (Jacobi, Hayward et al., 2004; Stice, 2002). Although only addressed in very few prospective studies, there is evidence that low self-esteem is a risk factor for ED symptomatology (Ghaderi & Scott, 2001). The specificity of both factors for ED

symptoms particularly remains unclear, although it seems reasonable to assume that both factors are not highly specific for ED.

Non identified risk factors

Perfectionism (operationalized in the present study through the CR-EAT familial and personal expectations scales) are not proven to be risk factors for partial ED. From a clinical point of view, it is well recognized that especially patients with AN often act highly perfectionistic, but empirical findings particularly about premorbid perfectionism are inconsistent (Jacobi, Hayward et al., 2004; Stice, 2002).

In the present study depression, anxiety, and stress are not associated with an increased risk for partial ED. There is evidence about ED symptoms and comorbid depressive or anxiety syndromes (see section I.3.3.6, p. 37). However, there is much less evidence about depressive or anxiety conditions prior to illness onset (Godart et al., 2002; Godart et al., 2007). The non significant influence of depressive and anxiety symptoms for the onset of partial ED in the present study, should be regarded cautiously: due to the frequency of the assessments, depression, anxiety and stress (operationalized through the DASS-21 scales) were only assessed at every other measurement point, producing fewer reports regarding those factors.

The second part of the results, explored the temporal course of risk factors over a period of four months prior to the onset of ED symptoms. Three different patterns of risk factor trajectories were identified: a stable but elevated course over time, an increasing course starting at a low level, and an increasing course starting at an already elevated level.

In order to determine whether the level of a particular risk factor can be considered as normal or already elevated 120 days prior the partial ED onset, a reference group

as comparison is required. To put the levels of the EDE-Q scales that were found in the present study into context, reference scores from a latent class analysis of a large-scale sample of Portuguese females are used (Pinheiro, Bulik, Sullivan, & Machado, 2008) for the purpose of this discussion. The authors report a 4-class solution with a healthy, binge eating, purging, and classic BN class, where both the EDE-Q total and the subscales differed across all latent classes. Similarly, for the CR-EAT scales, reference scores are used from a female clinical subsample which was used for the construction procedure of the questionnaire. As expected the clinical subsample scores higher than women from the normal population on all global and subscales (Moessner et al., under review).

"Late" risk factors

A characteristic for "late" risk factors is an increase over time, while four months prior to ED onset the levels of these factors are comparable to the levels which are obtained in a healthy population (e.g. Moessner et al., under review; Pinheiro et al., 2008).[14] Per definition, "late" risk factors are only of little use for an early detection of individuals who will become ill. These factors can rather be regarded as precursory factors immediately prior to the onset of an illness.

In the present study "late" risk factors include all the eating related and several of the weight/shape related factors. Eating related behaviors such as restraint, controlled or affect-regulatory eating are classified as "late" risk factors due to the non elevated levels prior to and the temporal increase shortly before partial ED onset. Starting at low levels four months prior to the partial ED onset, these factors increase over time:[15]

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[14] *Note*: Reference scores for the EDE-Q and CR-EAT see annex, Table VII.35, p. 165; Trajectories of the EDE-Q and CR-EAT total, global and subscales with reference scores see annex, Figure VII.6 – Figure VII.14, pp. 169.

[15] *Note*: An exception is restraint eating behavior which did not increase.

e.g. at the time when the ED symptoms start, dietary restraint approximates the level of Pinheiro et al.'s (2008) binge eating class; affect-regulatory eating the level of Moessner et al.'s (under review) clinical sample.

Some of the weight/shape related constructs such as weight concern, or body embarrassment are classified as "late" risk factors, too. These factors increase over the period of four months,[16] while the levels of e.g. weight concern, weight preoccupation, body embarrassment, and societal expectations of shape and weight are comparable to the levels that Pinheiro et al. (2008) and Moessner et al. (under review), respectively, reported from a healthy population.

"Early" risk factors

Specific for "early" risk factors, are the elevated levels already identifiable four months prior to the onset of ED symptoms. The identification of an "early" risk factor is an important endeavor for developing screening procedures and prevention approaches in order to detect high-risk individuals and to prevent these at-risk individuals from becoming ill. For such prevention programs, it is essential to know when a risk factor is elevated in reference to the outcome.

In the present study, the non-specific constructs – emotional dysregulation and self-esteem – and shape concern and harmful weight regulation (weight/shape related factors) are classified as "early" risk factors. All of these factors yield already elevated scores four months prior to the onset of ED symptoms: emotional dysregulation exceeds the reference scores of a healthy population reported by Moessner et al. (under review) by 0.68 standard deviations and self-esteem by 0.99, respectively; the shape concern score is comparable to the one of a binge eating class (Pinheiro et al., 2008), the harmful weight regulation score is elevated by 0.66 standard deviations

[16] *Note*: An exception is harmful weight regulation which did not increase.

from the mean of a healthy sample (Moessner et al., under review). The temporal courses of these “early” risk factors over the four month period is stable for the non-specific factors (emotional dysregulation, self-esteem), and for harmful weight regulation, whereas shape concern is increasing.

Summarizing the results, topics such as body shape concerns and dysfunctional weight regulation attitudes appear to be useful ED related constructs in order to screen for individuals who have an increased risk to develop ED. Additionally these issues could be addressed in preventive approaches, in order to change dysfunctional attitudes and behaviors towards food and shape.

Although, it seems reasonable to assume that the risk factors emotional dysregulation, and self-esteem are not highly specific in increasing the risk for ED, prevention programs should address these factors, too: highlighting that someone’s self-concept should not rely only on the evaluation of shape and weight, and alternative ways of dealing with associated distress should be demonstrated.

Integration of the results

Beyond the better understanding of the etiology of ED, risk factor research provides necessary information for the detection of high-risk populations and the creation of ED prevention programs. The present study adds useful information for both these aspects: Along with dysfunctional and dangerous weight regulation behaviors, weight and especially shape concern, increase the risk of ED symptomatology. Furthermore high emotional dysregulations which describe depressive symptoms and dysfunctional affect regulation, and low self-esteem predict the onset of ED symptomatology. All these factors are applicable for the screening of at-risk individuals and prevention approaches, due to their four months temporal precedence prior to the ED symptomatology.

A meta-analytic review about ED preventive approaches reports that the effects of prevention programs are often more pronounced for at-risk individuals (Stice, Shaw, & Marti, 2007), leading to the development of such tailored programs and the mandatory detection of at-risk individuals. In line with the findings of the present research, a recent prospective study examining risk factors for ED onset and their interactions in a high-risk sample identified weight and shape concern and a history of depression as potent risk factors (Jacobi et al., 2011). The authors conclude that issues such as weight and shape concerns should be expanded to focus on topics of affect and affect regulation.

2 Discussion of methodological issues

Requirements to investigate risk factors (see section I.2.5, p. 27) are explicitly proposed by Kraemer et al. (1997): outcome, risk factors and study sample have to be defined clearly, temporal precedence of the risk factor, and low- vs. high-risk groups have to be established, and potency has to be demonstrated. In the following, these theoretical requirements and their realization in the present study are discussed successively.

Defining the outcome

The outcome criterion in the present study is defined as "*partial* ED" referring to individuals who report only substantial ED symptoms. Certainly, the determination of a full-blown ED diagnosis assessed via structured clinical interviews (SCID; First, Spitzer, Gibbon Miriam, & Williams, 2002) would be the gold standard as an outcome criterion. In the present study this procedure could not be accomplished due to the fact that the data collection was Internet-based where no face-to-face contact occurred. Additionally the small incidence rates of full ED would require an extended large sample which led to the decision to use a less stringent outcome criterion in the present study (for more details see section III.4.3, p. 58). This procedure is justified as there is supporting evidence that subthreshold or partial manifestations of ED are still clinically concerning (The McKnight Investigators, 2003; Wittchen et al., 1998), and associated with substantial impairment (Mond et al., 2005), and that the etiology of full-blown clinical pictures often starts with partial manifestation. Thus, the outcome criterion in the present study was designed to be less stringent.

To estimate how many partial ED cases in the present study approximate the criteria of a full diagnosis of AN or BN, participants were screened for low body weight

(DSM-IV: criterion A for anorexia nervosa, see annex, Table VII.1, p. 126), and both recurrent binge eating and inappropriate compensatory behaviors (DSM-IV: criteria A, B and C for bulimia nervosa, see annex, Table VII.2, p. 127). None of the partial ED cases in the present study reported a body mass index (BMI) less than 17.5, i.e. it can be assumed that non participant met the criteria for AN. Behavioral diagnostic criteria for a full diagnosis of BN requires recurrent episodes of binge eating and recurrent inappropriate compensatory behaviors (vomiting, misuse of laxatives, fasting or excessive exercise) on average, at least twice a week for three consecutive months (see DSM-IV, American Psychiatric Association, 2000). In the present study, the examination of behavioral features for a diagnosis of BN (bingeing, vomiting, use of laxatives), yields nine partial ED cases who reported binge eating and inappropriate compensatory behaviors (vomiting and/or the use of laxatives) twice a week at the same monthly assessment, and only one case who reported both behaviors in three consecutive months. It can be assumed that only few participants approximates the DSM-IV criteria for BN.

Defining risk factors

The clear a priori definition of risk factors presents another requirement by Kraemer et al. (1997). However, the body of empirical evidence about risk factor for the development of ED is spare and inconsistent (Jacobi, Hayward et al., 2004). Thus, based on the available literature, in the present study a wide range of variables were included in the analyses to test for their risk factor status. Both variables measuring eating related behaviors (e.g. controlled, restraint and emotional eating) and shape/weight related factors (e.g. shape and weight concern, body embarrassment), and unspecific factors such as self-esteem, emotional dysregulation, and perfectionism were examined.

Defining the sample

The sampling procedure of the present study targeted a university population both for feasibility reasons and the assumption that the period of transition from high school to university might present an exciting but difficult time of change for young adults (Vohs, Heatherton, & Herrin, 2001), with an associated increased risk for the development of mental illnesses (Dyrbye, Thomas, & Shanafelt, 2006). However, the sampling procedure involved some problematic issues, which are discussed in the following.

From the 470 participants who registered for study participation (see Figure IV.1, p. 65), only 57.79 percent (n = 272) completed two or more full assessments. Due to the nature of the study, longitudinal data (≥ 2 assessments) are required. The percentage of participants who completed less than 2 assessments – approx. 42 percent – appears to be rather high. However, in a meta-analysis Cook et al. (2000) reported low response rates for Internet-based surveys in general. The authors suggest that follow-up contacts with non-responders yield higher response rates. In the present study, non-responders received two messages reminding them of the monthly assessments in addition to the standard email. Nevertheless, it appears that for some participants initial curiosity faded after the first completed questions.

Although male participants were excluded from data analyses (see chapter III.5.3, p. 62), the number of male participants should be reported (n = 56). From the participants who completed two or more assessments, approx. 80 percent were female. It can be assumed that the topic of the study seems to attract predominately female participants, which appears to be consistent with the male to female ratio in ED in general, and the topics eating behavior, weight and shape in particular. From the 56 male participants, 15 met at least one partial ED criterion (26.8%), while only 4 fulfilled the criteria later than at the first assessment (26.7% from the male partial ED cases).

On average included participants were 22 years old (see subsample 1, Table IV.1, and subsample 2, Table IV.2, pp. 66) which reflects the recruitment process in a university student population. However, the age of participants ranged from 18 to 50 years (12.7 percent of subsample 1 and 6 percent of subsample 2 are older then 25 years). This might be due to the open access of this online study, which allows everybody to register and participate.

The reason for excluding participants who reported partial ED criteria at the first assessment are described in chapter III.5.3 (p. 62) in detail. From the 216 female participants who completed 2 or more assessments, 90 met partial ED criteria, while 65 individuals already fulfilled these criteria at the first assessment. The high percentage of partial ED cases (41.7% of the participants who completed 2 or more assessments) could be explained by the attraction of the study for individuals who are already occupied with both food and eating, and the relationship to weight and shape, respectively. Evidence for this assumption is the high percentage of individuals who reported at least one partial ED criterion at their first assessment (72.2% of the 90 partial ED cases). In addition, these participants showed more severe ED psychopathology, i.e. a higher EDE-Q total score (see Table III.2, p. 63).

Most of the 25 cases fulfill the partial ED criterion of binge eating behavior at least once per week (n = 22), whereas around a fourth used laxatives (n = 6), and 12 percent reported vomiting or underwent ED treatment (n = 3). More than one fourth of the binge eaters (n = 6) fulfilled simultaneously at least one other partial ED criterion (see Table IV.3, p. 68).

Summarizing the sampling procedure and the analyses of the included samples, the potential for a self-selecting bias could not be ruled out. Generalization of the results to other populations should be regarded cautiously.

Establishing temporal precedence

A further requirement in the accurate investigation of risk factors, is the establishment of temporal precedence of the risk factor prior to the associated outcome (Kraemer et al., 1997). The present longitudinal observational study design allowed the verification of the temporal order of the association between risk factor and outcome. This stands in line with Kraemer et al. (1997) and Kazdin et al. (1997) who strongly recommend establishing the risk factor status within a prospective design.

Establishing low- and high-risk groups

In order to identify high-risk individuals, a dichotomization of the total sample was conducted using a median split of the continuous variables (total, global and subscale scores of the EDE-Q, CR-EAT, and DASS-21). Generally, dichotomization of a population always bears the expense of loss of information about individual differences. This could lead to a loss of effect size and power in the case of bivariate relationships, and the potential overlooking of nonlinear relationships (Altman & Royston, 2006; MacCallum, Zhang, Preacher, & Rucker, 2002). Moreover, in the context of risk factor research a dichotomization seems appropriate: It facilitates the explanation and prediction of the onset of a psychiatric disorder, and additionally encourages the identification of individuals who are particularly vulnerable because they possess risk factors, and who then may be specially targeted in prevention programs (Kraemer et al., 2001). Nevertheless, the arbitrariness of how to dichotomize a given sample produces some problematic issues. In the absence of a priori cutpoints, the methods for cutpoint determination fall into two broad categories: data-oriented and outcome-oriented methods. A data-oriented approach dichotomizes the continuous variable based on certain quantiles such as the median or upper quartile (Schulgen, Lausen, Olsen, & Schumacher, 1994), whereas the

outcome-oriented methods provide a cutpoint value that corresponds to the most significant relation with outcome (Altman, 1998). To establish this “optimal” outcome-oriented cutpoint, all observed values of the quantitative factor are considered as potential cutpoints (excluding only the extreme values), then the one that corresponds to the most significant relation with outcome (minimizing the *P*-value relating the prognostic factor to outcome) is chosen. Although, the outcome-oriented methods are expected to have better statistical indicators than data-oriented methods (Kuo, 1997), this technique is barely “optimal” because of the well-known problem of multiple testing (Altman, Lausen, Sauerbrei, & Schumacher, 1994). On the other hand, data-oriented cutpoint determination also involves a serious problem: the arbitrary use of a certain cutpoint based on the data (e.g. the median, a quantile or one standard deviation above the mean, etc.). In the end, in the present study, a data-oriented dichotomization approach with the median as a cutpoint was applied. Clearly, an advantage of this technique is that the division of the present sample produces two comparable sized groups (low- vs. high-risk) which in return is an advantage for the survival analytic approach.

Examining potency

As already indicated, it is crucial to investigate a risk factor beyond mere statistical significance between factor and outcome. A wide range of different indicators to demonstrate the potency or strength of a risk factor are available. Kraemer et al. (1999) explicitly describe the trade offs between the different potency measures. The odds ratio which was used in the present study presents the most widely used statistic employed in risk factor research and a predominant effect size to describe increased risk for disorders in epidemiological studies (Bland & Altman, 2000).

Statistical analyses

In order to test risk factors for the onset of ED symptoms, a survival analytic approach was conducted – a standard procedure for such research questions. In the present study, right censoring of the data occurs commonly due to the small incidence rates of ED symptoms. Methods of estimation taking into account whether an individual's observations are censored are an effective approach to deal with censored data. Under the assumption that censoring occurs independently from the survival time, the Kaplan-Meier estimator of the survival function and the log-rank test for testing equality of two survival functions are such methods. Both techniques were used in the present analyses. However, the heavy right censored data, did not allow the calculation of meaningful mean survival times.

In order to explore the temporal course of factors prior to the partial ED onset, hierarchical linear modeling was applied. Hierarchical linear modeling has several advantages over a standard general linear model approach: it can deal with unbalanced data, missing observations and varying measurement occasions across individuals. In the present longitudinal data set, the repeated measurements were considered as data points nested within subjects. Thus, with this approach, the intercepts and slopes were modeled allowing that both vary across individuals.

3 *Limitations and strengths*

The present study represent one of the few longitudinal risk factor studies in the ED field. In particular, several strong points are included: a prospective study design with several measurement points (on average more the seven), the examination of potency of the risk factors, and the detailed exploration of temporal occurrence and course of risk factors prior to ED symptoms.

The Internet-based measurements allowed for completely anonymous data collection. Among other advantages of online assessments (e.g. an extended reach), it can be assumed that individuals tend to disclose more information about themselves in an online setting due to perceived anonymity while completing Internet-based questionnaires (Joinson, 1999).

Beyond the identification of risk factors, the potencies of these factors were measured using the odds ratio. This potency measure provides valuable information about the strength of influence of the risk factors.

To the author's knowledge, the present work includes the first study about "early" vs. "late" risk factors for ED symptomatology. The detailed examination of the temporal course of risk factors prior to the onset of partial ED, offers useful evidence for early detection and prevention programs. In this regard, the concept of "early" vs. "late" risk factors presents an innovative approach to extend the scientific evidence with detailed information about the pathway from an early risk factor to the onset of a psychiatric disorder.

The present study involves some shortcomings as well, such as a limited sample size, and a lax outcome criterion (*partia*l ED), and the therewith associated lacking possibility of generalizing the results to individuals with a full ED.

Out of the 470 registered participants only 151 (subsample 1) and 25 (subsample 2) individuals, were included for data analyses. The reasons that such a high percentage of individuals were not eligible include insufficient data, male gender, and presence of the partial ED criteria at the first measurement point.

Due to the limited sample size, and both the recruitment procedure which was mainly accomplished at university campuses, and the Internet-based data collection, a broad generalization of the results is not possible. University students were chosen as study population due to the fact that college students report notably high rates of body dissatisfaction, dieting, and problematic eating (Heatherton, Mahamedi, Striepe, Field, & Keel, 1997). Primarily, an Internet-based data collection procedure was applied to facilitate the repeated monthly assessments.

Finally, the non stringent outcome criteria “partial ED” limits the scope of the results to partial manifestation of symptoms such as binge eating, vomiting and the inappropriate use of laxatives. However, in a broader sense, these (partial) symptoms could be considered early indicators towards a possible etiology of a full ED.

4 *Conclusion and outlook*

The prevalence rates of ED are relatively low (Hoek & van Hoeken, 2003). However, for those who are affected, ED are serious conditions with substantial impact on both physical and mental health (Treasure et al., 2010). ED are also associated with a profound loss in quality of life in affected individuals (Mond et al., 2005) and their close others (de la Rie et al., 2005), as well as with substantial direct and indirect health care costs.

Even though effective treatment for ED exists, the situation is far from satisfactory (Keel & Brown, 2010): A considerable proportion of patients does not respond to treatment or experiences relapses and multiple illness episodes (Fichter & Quadflieg, 2004; Grilo et al., 2007). A subgroup of patients even shows a chronic course of illness (Fichter, Quadflieg, & Hedlund, 2006). Thus, the need for prevention and early intervention is evident. However, this requires the identification of individuals who are at-risk for the development of an ED. Even though a number of risk factors have been studied over the past decade and our understanding of the development of ED has improved (Jacobi, Hayward et al., 2004; Stice, 2002), there is a clear need for longitudinal risk factor studies that take into account a spectrum of potentially relevant factors in order to identify those that may predict illness onset.

As discussed before, such research needs to meet a number of methodological requirements (Kraemer et al., 1997) which have not been taken into account in many studies so far. The present study followed these recommendations and used a prospective observational design which is one of the requirements in order to establish the temporal precedence of risk factors. Additionally, the potency of each risk factor was scrutinized with an adequate analytic measure which allowed to quantify the impact of each of the identified risk factors.

Clearly, future studies in this field would benefit from a more stringent outcome criterion which would ideally be assessed with structured clinical interviews (First et al., 2002), and over longer observation periods. It would also be desirable to explore different risk factors and their pathways for the onset of AN and BN separately. However, given the low prevalence rates for AN and BN (e.g. Hoek & van Hoeken, 2003) large samples (> 3.000 subjects for BN, > 10.000 for AN) would be needed to detect enough individuals with full-blown ED (according to DSM criteria) within a community-based population (Jacobi, Hayward et al., 2004).

Such future studies might also want to consider the concept of “early” vs. “late” risk factors that was introduced by the present work. This concept provides particularly valuable information for the early detection of ED symptomatology. Taking into account that “early” risk factors such as shape concern, unhealthy weight regulation behaviors, emotional dysregulation, and low self-esteem that were identified by the present study appears to be especially useful for the development of prevention programs that may address individuals at-risk early enough to successfully counteract the development of substantial ED related impairment. Despite the above mentioned limitations and the need for replication of the results of the present study, the findings are promising and contribute to an improved understanding of risk factors for ED.

VI. References

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VII. Annex

Table VII.1 Diagnostic criteria: Anorexia nervosa

DSM-IV Criteria for Anorexia Nervosa (307.10)	A	Refusal to maintain body weight at or above a minimally normal weight for age and height (e.g., weight loss leading to maintenance of body weight less than 85% of that expected or failure to make expected weight gain during period of growth, leading to body weight less than 85% of that expected).
	B	Intense fear of gaining weight or becoming fat, even though underweight.
	C	Disturbance in the way in which one's body weight or shape is experienced, undue influence of body weight or shape on self-evaluation, or denial of the seriousness of the current low body weight.
	D	In postmenarchal females, amenorrhea i.e., the absence of at least three consecutive cycles. (A woman is considered to have amenorrhea if her periods occur only following hormone, e.g., estrogen administration.)
		Specify type: *Restricting Type:* During the current episode of anorexia nervosa, the person has not regularly engaged in binge-eating or purging behavior (i.e., self-induced vomiting or the misuse of laxatives, diuretics, or enemas). *Binge-Eating/Purging Type:* During the current episode of anorexia nervosa, the person has regularly engaged in binge-eating or purging behavior (i.e., self-induced vomiting or the misuse of laxatives, diuretics, or enemas).
ICD-10 Criteria for Anorexia Nervosa (F50.0)	A	There is weight loss or, in children, a lack of weight gain, leading to a body weight at least 15% below the normal or expected weight for age and height
	B	The weight loss is self-induced by avoidance of “fattening foods”
	C	There is self-perception of being too fat, with an intrusive dread of fatness, which leads to a self-imposed low weight threshold
	D	A widespread endocrine disorder involving the hypothalamic-pituitary-gonadal axis is manifested in women as amenorrhoea and in men as a loss of sexual interest and potency. (An apparent exception is the persistence of vaginal bleeds in anorexic women who are on replacement hormonal therapy, most commonly taken as a contraceptive pill)
	E	The disorder does not meet criteria A or B for bulimia nervosa
ICD-10 Criteria for Atypical Anorexia Nervosa (F50.1)		Disorder that fulfills some of the features of anorexia nervosa but in which the overall clinical picture does not justify that diagnosis. For instance, one of the key symptoms, such as amenorrhoea or marked dread of being fat, may be absent in the presence of marked weight loss or weight-reducing behavior. This diagnosis should not be made in the presence of known physical disorders associated with weight loss

Note: DSM-IV = Diagnostic and Statistical Manual IV (American Psychiatric Association, 2000), ICD-10 = International Classification of Diseases 10 (WHO, 1992)

Table VII.2 Diagnostic criteria: Bulimia nervosa

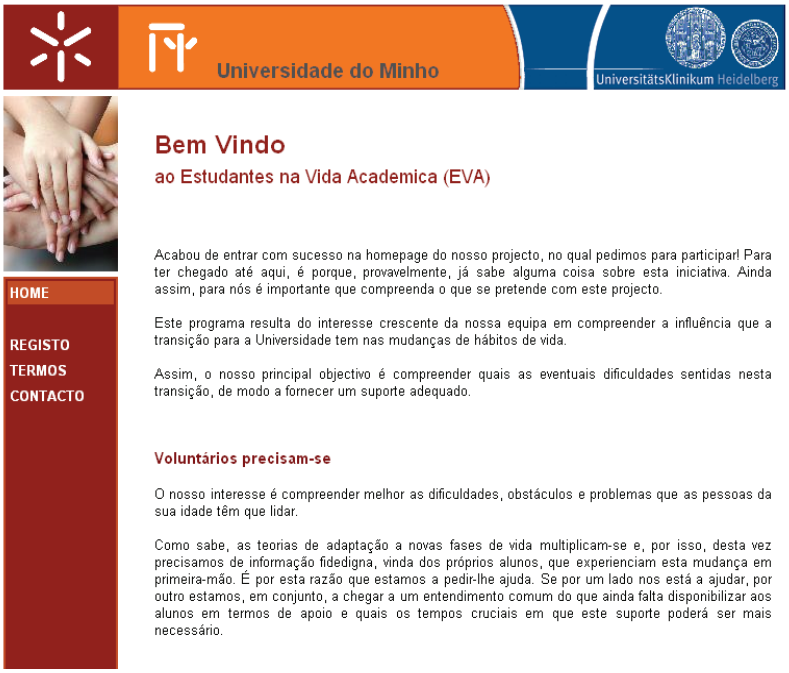
DSM-IV Criteria for Bulimia Nervosa (307.51)	A Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following: B Eating, in a discrete period of time (e.g., within any 2-hour period), an amount of food that is definitely larger than most people would eat during a similar period of time and under similar circumstances (1) A sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating) (2) Recurrent inappropriate compensatory behavior in order to prevent weight gain, such as self-induced vomiting; misuse of laxatives, diuretics, enemas, or other medications; fasting or excessive exercise C The binge eating and inappropriate compensatory behaviors both occur, on average, at least twice a week for 3 months D Self-evaluation is unduly influenced by body shape and weight E The disturbance does not occur exclusively during episodes of anorexia nervosa Specify type: *Purging type:* During the current episode of bulimia nervosa, the person has regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics, or enemas *Nonpurging type:* During the current episode of bulimia nervosa, the person has used inappropriate compensatory behaviors, such as fasting or excessive exercise, but has not regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics, or enemas
ICD-10 Criteria for Bulimia Nervosa (F50.2)	A There are recurrent episodes of overeating (at least twice a week over a period of 3 months) in which large amounts of food are consumed in short periods of time B There is persistent preoccupation with eating, and a strong desire or sense of compulsion to eat (craving) C The patient attempts to counteract the “fattening” effects of food by one or more of the following: (1) self-induced vomiting (2) self-induced purging (3) alternating periods of starvation (4) use of drugs such as appetite suppressants, thyroid preparations, or diuretics; when bulimia occurs in diabetic patients they may choose to neglect their insulin treatment D There is self-perception of being too fat, with an intrusive dread of fatness (usually leading to underweight)
ICD-10 Criteria for Atypical Bulimia Nervosa (F50.3)	Disorder that fulfills some of the features of bulimia nervosa, but in which the overall clinical picture does not justify that diagnosis. For instance, there may be recurrent bouts of overeating or overuse of purgatives without significant weight change, or the typical overconcern about body shape and weight may be absent

Note: DSM-IV = Diagnostic and Statistical Manual IV (American Psychiatric Association, 2000), ICD-10 = International Classification of Diseases 10 (WHO, 1992)

Table VII.3 Diagnostic criteria: Binge eating disorder

DSM-IV Criteria for Binge Eating Disorder (307.50)	A Recurrent episodes of binge eating. An episode of binge eating is characterized by both of the following: A Eating, in a discrete period of time (e.g., within any 2-hour period), an amount of food that is definitely larger than most people would eat in a similar period of time under similar circumstances B The sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating) C Binge-eating episodes are associated with three (or more) of the following: (1) eating much more rapidly than normal (2) eating until feeling uncomfortably full (3) eating large amounts of food when not feeling physically hungry (4) eating along because of being embarrassed by how much one is eating (5) feeling disgusted with oneself, depressed, or very guilty after overeating D Marked distress regarding binge eating is present E The binge eating occurs, on average, at least 2 days a week for 6 months Note: The method of determining frequency differs from that used for bulimia nervosa; future research should address whether the preferred method of setting a frequency threshold is counting the number of days on which binges occur or counting the number of episodes of binge eating F The binge eating is not associated with the regular use of inappropriate compensatory behavior (e.g., purging, fasting, excessive exercise, etc.) and does not occur exclusively during the course of anorexia nervosa or bulimia nervosa

Note: DSM-IV = Diagnostic and Statistical Manual IV (American Psychiatric Association, 2000)

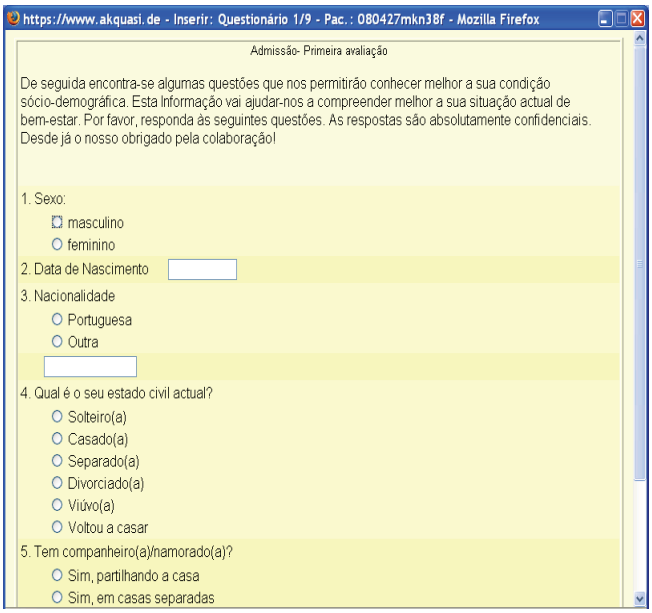


Portuguese study homepage

French study homepage

Figure VII.1 Study homepages

Email with link to the data collection software WEB-Akquasi



Socio-demographic questions assessed with WEB-Akquasi

Figure VII.2 Online assessment procedure

Table VII.4 Comparison: Case at first assessment vs. case at a later assessment II

		Case at first assessment			Case at > first assessment			Statistic	
		n	*M*	*SD*	*n*	*M*	*SD*	*t-value*	*p-value*
EDE-Q	DR	60	2.28	1.74	25	1.61	1.50	1.70	.093
	EC	58	1.91	1.63	25	1.31	1.33	1.73	.089
	WC	58	3.00	1.59	25	2.11	1.37	2.43	.018
	SC	58	3.51	1.51	24	2.61	1.44	2.53	.014
CR-EAT	EBD	62	3.33	1.05	24	2.87	0.98	1.84	.070
	ACI	62	3.52	1.03	24	3.41	1.03	0.43	.666
	PER	62	2.80	1.01	24	2.45	0.80	1.52	.131
DASS	DE	22	21.18	10.58	11	13.09	10.71	2.06	.048
	AN	22	22.09	10.48	11	10.36	7.94	3.26	.003
	ST	22	18.00	10.03	11	19.64	9.79	-0.45	.659

Note: n = number of participants, M = mean, SD = standard deviation, EDE-Q = Eating Disorder Examination Questionnaire, CR-EAT = Clinical and Research Inventory for Eating Disorders, DASS = Depression Anxiety Stress Scale (21 item version), DR = Dietary Restraint, EC = Eating Concern, WC = Weight Concern, SC = Shape Concern, EDB = Eating Behavior Disturbance, ACI = Affective/Cognitive Impairment, DE = Depression, AN = Anxiety, ST = Stress

Table VII.5 Comparison: Goodness of fit indices for models with case at first assessment vs. case at a later assessment

		Case at first assessment			Case at > first assessment		
		AIC	*BIC*	*logLik*	*AIC*	*BIC*	*logLik*
EDE-Q	TO	387.30	404.55	-187.65	174.77	188.52	-81.39
	DR	457.35	474.74	-222.67	216.28	230.11	-102.14
	EC	399.80	417.05	-193.90	174.63	188.38	-81.32
	WC	426.25	443.50	-207.13	202.30	216.04	-95.15
	SC	443.36	460.61	-215.68	224.57	238.31	-106.28
CR-EAT	TO	287.27	304.74	-137.63	120.26	134.09	-54.13
	EBD	311.93	329.41	-149.97	125.96	139.78	-56.98
	ACI	329.32	346.79	-158.66	150.44	164.27	-69.22
	PER	319.12	336.59	-153.56	139.07	152.89	-63.53
	WP	390.40	407.87	-189.20	177.43	191.25	-82.72
	CE	406.67	424.15	-197.34	191.96	205.78	-89.98
	ED	414.05	431.53	-201.03	199.06	212.88	-93.53
	AE	407.64	425.11	-197.82	188.53	202.35	-88.26
	SE	359.24	376.72	-173.62	163.49	177.31	-75.74
	CN	418.53	436.00	-203.26	199.22	213.05	-93.61
	BE	391.88	409.35	-189.94	176.03	189.85	-82.01
	RE	371.65	389.13	-179.83	156.80	170.62	-72.40
	SO	362.79	380.27	-175.40	160.15	173.97	-74.07
	PF	397.74	415.21	-192.87	203.12	216.94	-95.56
	HW	370.84	388.32	-179.42	164.80	178.62	-76.40
	PP	351.37	368.85	-169.69	176.26	190.09	-82.13
DASS-21	TO	470.37	481.96	-229.19	254.23	262.44	-121.12
	DE	375.20	386.79	-181.60	204.93	213.13	-96.46
	AN	377.02	388.61	-182.51	201.84	210.04	-94.92
	ST	374.75	386.34	-181.38	210.59	218.79	-99.29

Note: AIC = Akaike information criterion, BIC = Bayesian Information Criterion, logLik = log Likelihood, EDE-Q = Eating Disorder Examination Questionnaire, CR-EAT = Clinical and Research Inventory for Eating Disorders, DASS-21 = Depression Anxiety Stress Scale (21 item version), TO = Total Score, DR = Dietary Restraint, EC = Eating Concern, WC = Weight Concern, SC = Shape Concern, EBD = Eating Behavior Disturbances, ACI = Affective/Cognitive Impairment, PER = Perfectionism total, WP = Weight Preoccupation, CE = Control over Eating, ED = Emotional Dysregulation, AE = Affect-Regulatory Eating, SE = Self-Esteem, CN = Concern about Negative Evaluation, BE = Body Embarrassment, RE = Restraint Eating Behavior, SO = Societal Expectations of Shape and Weight, PF = Perfectionism: Familial Expectations, HW = Harmful Weight Regulations, PP = Perfectionism: Personal Expectations, DE = Depression, AN = Anxiety, ST = Stress

Table VII.6 Comparison: Intercepts & slopes

for models with case at first assessment vs. case at a later assessment

		Case at first assessment		Case at > first assessment	
		Intercept	*Slope*	*Intercept*	*Slope*
EDE-Q	TO	2.44	0.01312	1.91	0.00854
	DR	2.06	0.00914	1.53	0.00540
	EC	1.71	0.01328	1.27	0.00857
	WC	2.74	0.00843	2.15	0.00571
	SC	3.22	0.01471	2.61	0.00957
CR-EAT	TO	3.38	0.00458	3.11	0.00316
	EBD	3.19	0.00772	2.84	0.00570
	ACI	3.46	0.00300	3.32	0.00276
	PER	2.71	0.00137	2.50	0.00111
	WP	3.61	0.01023	3.27	0.00807
	CE	3.91	0.01006	3.47	0.00724
	ED	3.31	0.00209	3.07	0.00154
	AE	3.30	0.00640	3.25	0.00615
	SE	3.34	0.00214	3.33	0.00247
	CN	3.87	0.00205	3.64	0.00178
	BE	3.40	0.00849	3.08	0.00687
	RE	2.68	0.00461	2.27	0.00200
	SO	2.90	0.00611	2.67	0.00475
	PF	2.83	0.00030	2.76	-0.00003
	HW	2.59	0.00304	2.24	0.00176
	PP	4.11	0.00173	3.81	0.00137
DASS-21	TO	53.00	0.18126	41.81	0.05910
	DE	17.72	0.04688	12.92	-0.00353
	AN	17.29	0.08573	11.60	0.02102
	ST	17.07	-0.00864	15.65	-0.01502

Note: EDE-Q = Eating Disorder Examination Questionnaire, CR-EAT = Clinical and Research Inventory for Eating Disorders, DASS-21 = Depression Anxiety Stress Scale (21 item version), TO = Total Score, DR = Dietary Restraint, EC = Eating Concern, WC = Weight Concern, SC = Shape Concern, EBD = Eating Behavior Disturbances, ACI = Affective/Cognitive Impairment, PER = Perfectionism total, WP = Weight Preoccupation, CE = Control over Eating, ED = Emotional Dysregulation, AE = Affect-Regulatory Eating, SE = Self-Esteem, CN = Concern about Negative Evaluation, BE = Body Embarrassment, RE = Restraint Eating Behavior, SO = Societal Expectations of Shape and Weight, PF = Perfectionism: Familial Expectations, HW = Harmful Weight Regulations, PP = Perfectionism: Personal Expectations, DE = Depression, AN = Anxiety, ST = Stress

Table VII.7 Comparison: ED case vs. non ED case

	ED case			Non ED case			Statistic	
	n			*n*			χ^2	*p-value*
Site	PT: 15		F: 10	PT: 79		F: 47	0.65	.799
	n	*M*	*SD*	*n*	*M*	*SD*	*t-value*	*p-value*
Age	25	22.42	3.0	125	22.27	4.6	0.16	.876
Assessment	25	7.48	6.2	126	7.43	5.8	0.04	.968

Note: n = number of participants, M = mean, SD = standard deviation, χ^2 = χ^2 –test statistic, t-value = t-test statistic, PT = Portugal, F = France

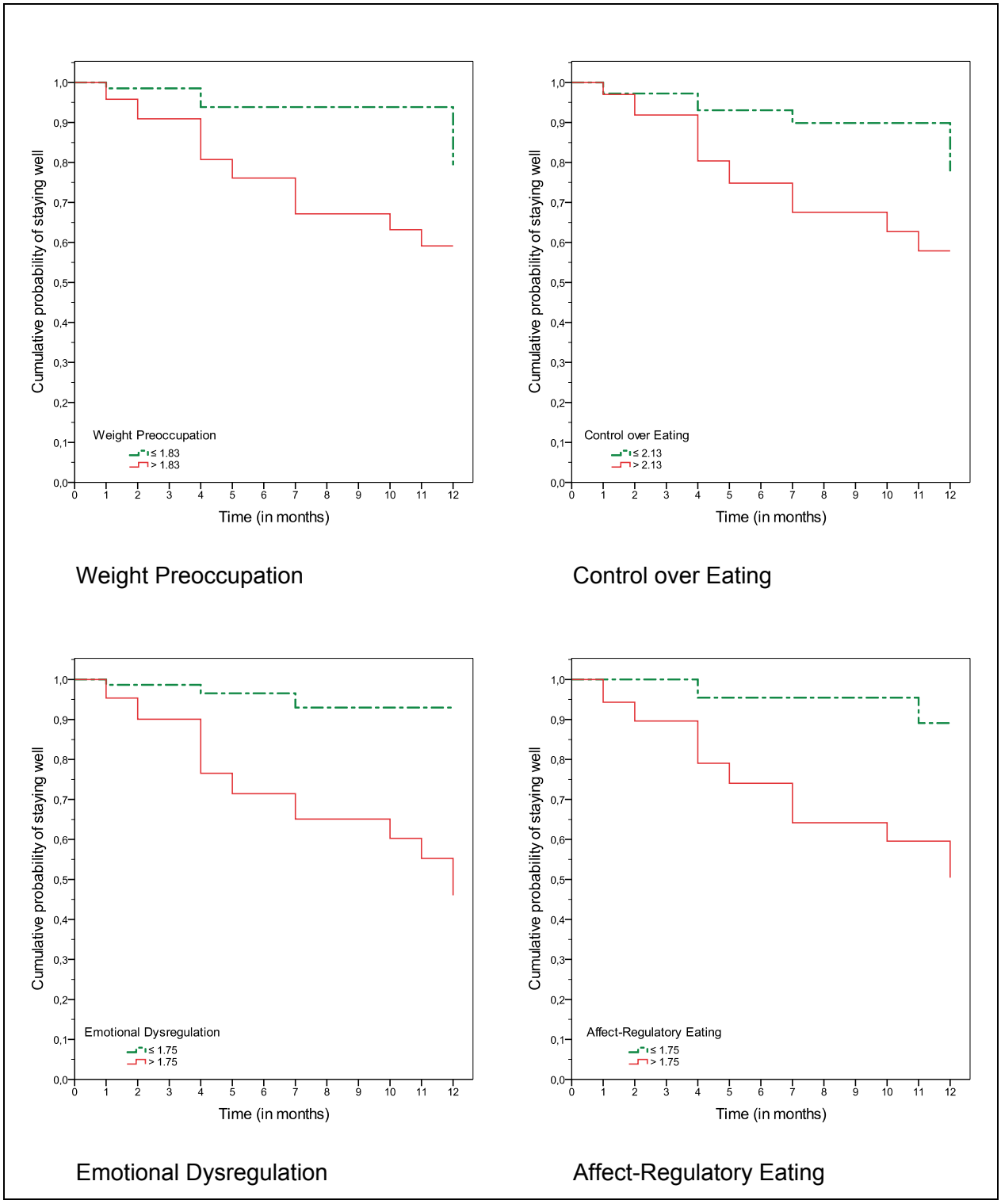


Figure VII.3 Probability of staying well: CR-EAT subscales I

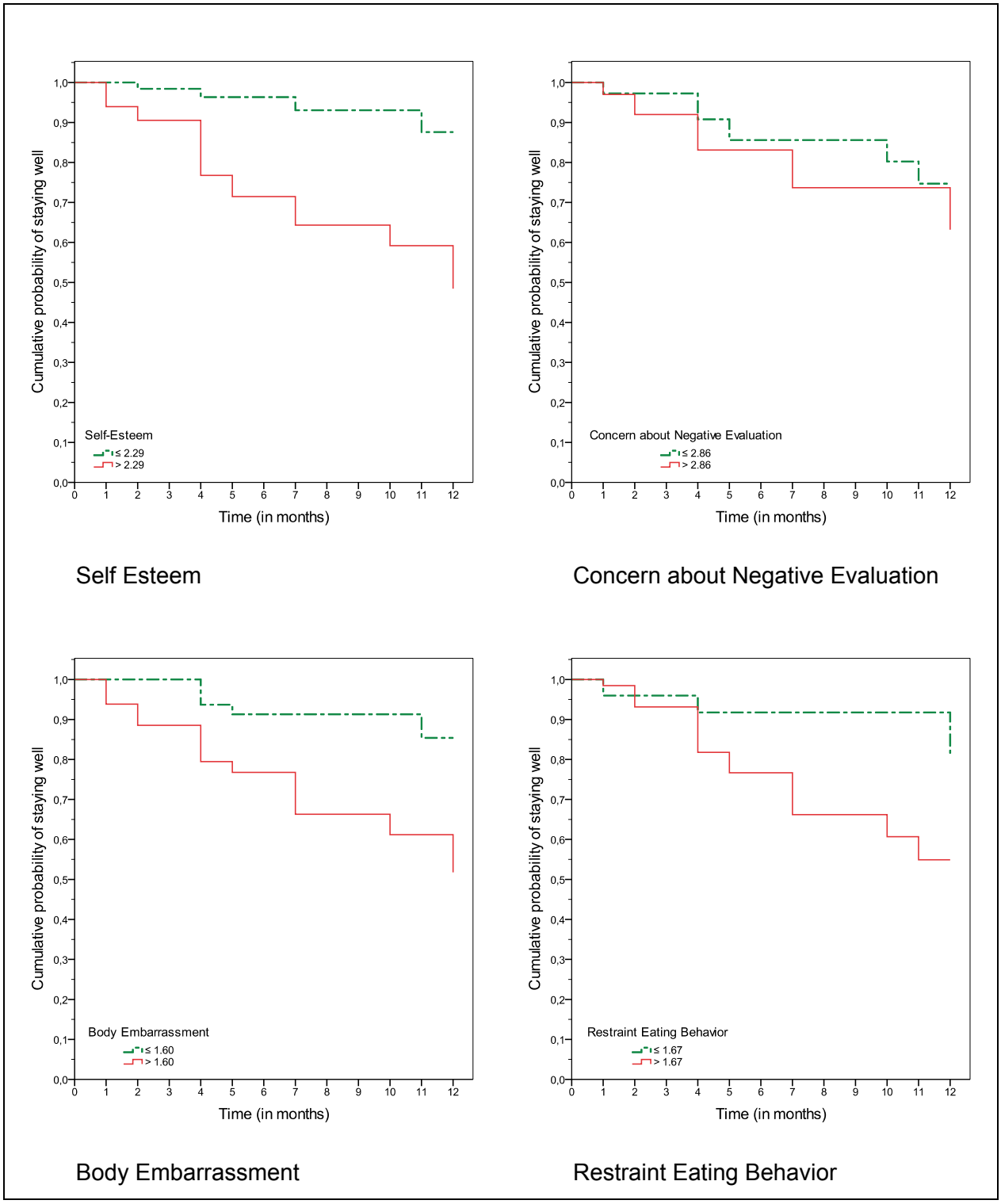


Figure VII.4 Probability of staying well: CR-EAT subscales II

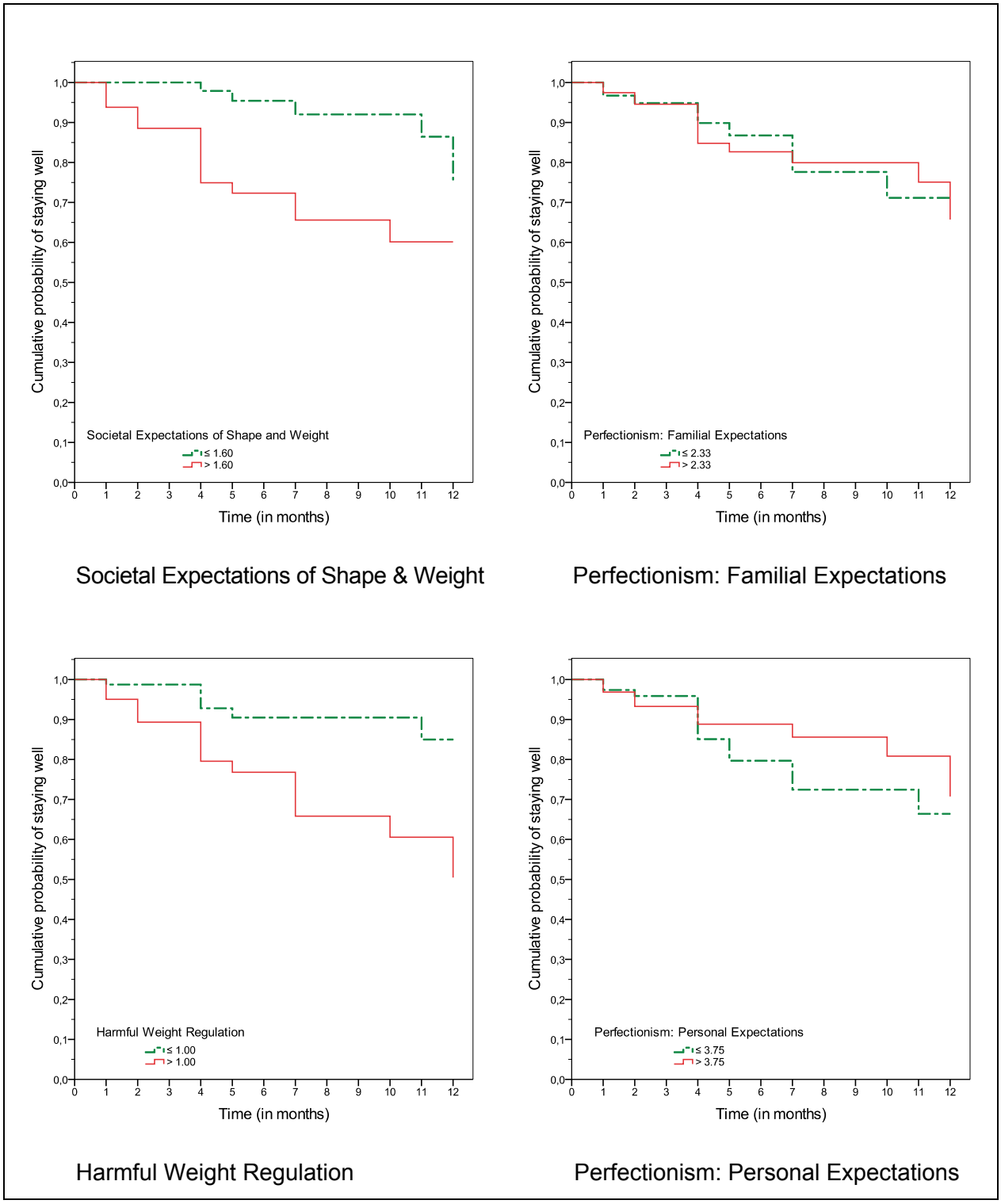


Figure VII.5 Probability of staying well: CR-EAT subscales III

Table VII.8 Difference in the cumulative probability of staying well: CR-EAT subscales

	Cut off	Cumulative probability of staying well		χ^2	p-value
		low risk	*high risk*		
WP	> 1.83	0.79	0.59	8.56	.003
CE	> 2.13	0.78	0.58	6.74	.009
ED	> 1.75	0.93	0.46	15.67	.000
AE	>1.75	0.89	0.50	13.25	.000
SE	>2.29	0.88	0.48	13.11	.000
CN	>2.86	0.75	0.63	1.09	.297
BE	> 1.60	0.85	0.52	9.69	.002
RE	> 1.67	0.82	0.55	7.11	.008
SO	> 1.60	0.76	0.60	10.38	.001
PF	> 2.33	0.71	0.66	0.04	.849
HW	> 1.00	0.85	0.50	8.91	.003
PP	> 3.75	0.66	0.71	0.73	.394

Note: Cut off = cut off scores for high-risk groups, χ^2 = log-rank test statistic, WP = Weight Preoccupation, CE = Control over Eating, ED = Emotional Dysregulation, AE = Affect-Regulatory Eating, SE = Self-Esteem, CN = Concern about Negative Evaluation, BE = Body Embarrassment, RE = Restraint Eating Behavior, SO = Societal Expectations of Shape and Weight, PF = Perfectionism: Familial Expectations, HW = Harmful Weight Regulation, PP = Perfectionism: Personal Expectations

Table.VII.9 Risk potencies: CR-EAT subscales

	n	N of events		OR	95% CI
		low risk	*high risk*		
WP	150	4 / 72	18 / 78	4.15	1.34 – 12.86
CE	150	6 / 77	16 / 73	2.81	1.04 – 7.58
ED	150	3 / 79	19 / 71	7.05	2.00 – 24.82
AE	150	3 / 76	19 / 74	6.50	1.85 – 22.91
SE	150	4 / 78	18 / 72	4.88	1.58 – 15.09
CN	150	9 / 78	13 / 72	1.56	0.63 – 3.88
BE	150	5 / 79	17 / 71	3.78	1.33 – 10.78
RE	150	6 / 79	16 / 71	2.97	1.10 – 8.00
SO	150	5 / 80	17 / 70	3.89	1.36 – 11.08
PF	150	9 / 66	13 / 84	1.13	0.46 – 2.82
HW	150	6 / 84	16 / 66	3.39	1.26 – 9.15
PP	150	13 / 82	9 / 68	0.83	0.34 – 2.07

Note: n = number of participants, N of events = number of participants experiencing partial ED onset, OR = odds ratio, 95% CI = 95% confidence interval of odds ratio, WP = Weight Preoccupation, CE = Control over Eating, ED = Emotional Dysregulation, AE = Affect-Regulatory Eating, SE = Self-Esteem, CN = Concern about Negative Evaluation, BE = Body Embarrassment, RE = Restraint Eating Behavior, SO = Societal Expectations of Shape and Weight, PF = Perfectionism: Familial Expectations, HW = Harmful Weight Regulation, PP = Perfectionism: Personal Expectations

Table VII.10 Life table: EDE-Q Total

	Time in months	N staying well	N terminated	N exposed to risk	N events	% staying well	Cumulative % staying well	Hazard rate
EDE-Q Total ≤ 0.63	0	69	0	69.0	0	1.00	1.00	.00
	1	69	9	64.5	0	1.00	1.00	.00
	2	60	8	56.0	0	1.00	1.00	.00
	3	52	8	48.0	0	1.00	1.00	.00
	4	44	7	40.5	1	.98	.98	.03
	5	36	2	35.0	1	.97	.95	.03
	6	33	8	29.0	0	1.00	.95	.00
	7	25	3	23.5	0	1.00	.95	.00
	8	22	3	20.5	0	1.00	.95	.00
	9	19	3	17.5	0	1.00	.95	.00
	10	16	1	15.5	0	1.00	.95	.00
	11	15	1	14.5	0	1.00	.95	.00
	12	14	13	7.5	1	.87	.82	.00
EDE-Q Total > 0.63	0	78	0	78.0	0	1.00	1.00	.00
	1	78	12	72.0	4	.94	.94	.06
	2	62	5	59.5	2	.97	.91	.03
	3	55	6	52.0	0	1.00	.91	.00
	4	49	10	44.0	6	.86	.79	.15
	5	33	3	31.5	1	.97	.76	.03
	6	29	3	27.5	0	1.00	.76	.00
	7	26	4	24.0	2	.92	.70	.09
	8	20	1	19.5	0	1.00	.70	.00
	9	19	5	16.5	0	1.00	.70	.00
	10	14	1	13.5	1	.93	.65	.08
	11	12	0	12.0	1	.92	.59	.09
	12	11	11	5.5	0	1.00	.59	.00

Note: EDE-Q TO = Eating Disorder Examination Questionnaire Total score, number of participants at time point 0 = 149, Missing n = 2, N staying well = number of participants who stay well, N terminated = number of participants who terminate the study, N exposed to risk = number of participants who are exposed to the risk of experiencing partial ED onset, N events = number of partial ED onsets, % staying well = percentage of participants who stay well, Cumulative % staying well = cumulative percentage of participants who stay well

Table VII.11 Life table: EDE-Q Dietary Restraint

	Time in months	N staying well	N terminated	N exposed to risk	N events	% staying well	Cumulative % staying well	Hazard rate
EDE-Q DR ≤ 0.40	0	82	0	82.0	0	.00	1.00	.00
	1	82	10	77.0	0	.00	1.00	.00
	2	72	8	68.0	0	.00	1.00	.00
	3	64	11	58.5	0	.00	1.00	.00
	4	53	6	50.0	2	.04	.96	.03
	5	45	2	44.0	1	.02	.94	.03
	6	42	7	38.5	0	.00	.94	.03
	7	35	5	32.5	0	.00	.94	.03
	8	30	4	28.0	0	.00	.94	.03
	9	26	5	23.5	0	.00	.94	.03
	10	21	1	20.5	0	.00	.94	.03
	11	20	1	19.5	1	.05	.89	.06
	12	18	17	9.5	1	.11	.80	.10
EDE-Q DR > 0.40	0	65	0	65.0	0	.00	1.00	.00
	1	65	11	59.5	4	.07	.93	.03
	2	50	5	47.5	2	.04	.89	.04
	3	43	3	41.5	0	.00	.89	.04
	4	40	11	34.5	5	.14	.76	.06
	5	24	3	22.5	1	.04	.73	.07
	6	20	4	18.0	0	.00	.73	.07
	7	16	2	15.0	2	.13	.63	.09
	8	12	0	12.0	0	.00	.63	.09
	9	12	3	10.5	0	.00	.63	.09
	10	9	1	8.5	1	.12	.56	.10
	11	7	0	7.0	0	.00	.56	.10
	12	7	7	3.5	0	.00	.56	.10

Note: EDE-Q DR = Eating Disorder Examination Questionnaire Dietary Restraint score, number of participants at time point 0 = 149, Missing n = 2, N staying well = number of participants who stay well, N terminated = number of participants who terminate the study, N exposed to risk = number of participants who are exposed to the risk of experiencing partial ED onset, N events = number of partial ED onsets, % staying well = percentage of participants who stay well, Cumulative % staying well = cumulative percentage of participants who stay well

Table VII.12 Life table: EDE-Q Eating Concern

	Time in months	N staying well	N terminated	N exposed to risk	N events	% staying well	Cumulative % staying well	Hazard rate
EDE-Q EC ≤ 0.20	0	100	0	100.0	0	1.00	1.00	.00
	1	100	12	94.0	1	.99	.99	.01
	2	87	11	81.5	0	1.00	.99	.00
	3	76	9	71.5	0	1.00	.99	.00
	4	67	14	60.0	3	.95	.94	.05
	5	50	4	48.0	1	.98	.92	.02
	6	45	9	40.5	0	1.00	.92	.00
	7	36	6	33.0	0	1.00	.92	.00
	8	30	4	28.0	0	1.00	.92	.00
	9	26	5	23.5	0	1.00	.92	.00
	10	21	2	20.0	0	1.00	.92	.00
	11	19	1	18.5	1	.95	.87	.06
	12	17	16	9.0	1	.89	.77	.12
EDE-Q EC > 0.20	0	47	0	47.0	0	1.00	1.00	.00
	1	47	9	42.5	3	.93	.93	.07
	2	35	2	34.0	2	.94	.87	.06
	3	31	5	28.5	0	1.00	.87	.00
	4	26	3	24.5	4	.84	.73	.18
	5	19	1	18.5	1	.95	.69	.06
	6	17	2	16.0	0	1.00	.69	.00
	7	15	1	14.5	2	.86	.60	.15
	8	12	0	12.0	0	1.00	.60	.00
	9	12	3	10.5	0	1.00	.60	.00
	10	9	0	9.0	1	.89	.53	.12
	11	8	0	8.0	0	1.00	.53	.00
	12	8	8	4.0	0	1.00	.53	.00

Note: EDE-Q EC = Eating Disorder Examination Questionnaire Eating Concern score, number of participants at time point 0 = 147, Missing n = 4, N staying well = number of participants who stay well, N terminated = number of participants who terminate the study, N exposed to risk = number of participants who are exposed to the risk of experiencing partial ED onset, N events = number of partial ED onsets, % staying well = percentage of participants who stay well, Cumulative % staying well = cumulative percentage of participants who stay well

Table VII.13 Life table: EDE-Q Weight Concern

	Time in months	N staying well	N terminated	N exposed to risk	N events	% staying well	Cumulative % staying well	Hazard rate
EDE-Q WC ≤ 0.80	0	82	0	82.0	0	1.00	1.00	.00
	1	82	10	77.0	0	1.00	1.00	.00
	2	72	8	68.0	0	1.00	1.00	.00
	3	64	11	58.5	0	1.00	1.00	.00
	4	53	6	50.0	2	.96	.96	.04
	5	45	2	44.0	1	.98	.94	.02
	6	42	7	38.5	0	1.00	.94	.00
	7	35	5	32.5	0	1.00	.94	.00
	8	30	4	28.0	0	1.00	.94	.00
	9	26	5	23.5	0	1.00	.94	.00
	10	21	1	20.5	0	1.00	.94	.00
	11	20	1	19.5	1	.95	.89	.05
	12	18	17	9.5	1	.89	.80	.00
EDE-Q WC > 0.80	0	65	0	65.0	0	1.00	1.00	.00
	1	65	11	59.5	4	.93	.93	.07
	2	50	5	47.5	2	.96	.89	.04
	3	43	3	41.5	0	1.00	.89	.00
	4	40	11	34.5	5	.86	.76	.16
	5	24	3	22.5	1	.96	.73	.05
	6	20	4	18.0	0	1.00	.73	.00
	7	16	2	15.0	2	.87	.63	.14
	8	12	0	12.0	0	1.00	.63	.00
	9	12	3	10.5	0	1.00	.63	.00
	10	9	1	8.5	1	.88	.56	.13
	11	7	0	7.0	0	1.00	.56	.00
	12	7	7	3.5	0	1.00	.56	.00

Note: EDE-Q WC = Eating Disorder Examination Questionnaire Weight Concern score, number of participants at time point 0 = 147, Missing n = 4, N staying well = number of participants who stay well, N terminated = number of participants who terminate the study, N exposed to risk = number of participants who are exposed to the risk of experiencing partial ED onset, N events = number of partial ED onsets, % staying well = percentage of participants who stay well, Cumulative % staying well = cumulative percentage of participants who stay well

Table VII.14 Life table: EDE-Q Shape Concern

	Time in months	N staying well	N terminated	N exposed to risk	N events	% staying well	Cumulative % staying well	Hazard rate
EDE-Q SC ≤ 1.13	0	76	0	76.0	0	1.00	1.00	.00
	1	76	10	71.0	0	1.00	1.00	.00
	2	66	8	62.0	0	1.00	1.00	.00
	3	58	10	53.0	0	1.00	1.00	.00
	4	48	8	44.0	1	.98	.98	.02
	5	39	1	38.5	1	.97	.95	.03
	6	37	8	33.0	0	1.00	.95	.00
	7	29	4	27.0	0	1.00	.95	.00
	8	25	2	24.0	0	1.00	.95	.00
	9	23	3	21.5	0	1.00	.95	.00
	10	20	2	19.0	0	1.00	.95	.00
	11	18	1	17.5	1	.94	.90	.06
	12	16	15	8.5	1	.88	.79	.13
EDE-Q SC > 1.13	0	71	0	71.0	0	1.00	1.00	.00
	1	71	11	65.5	4	.94	.94	.06
	2	56	5	53.5	2	.96	.90	.04
	3	49	4	47.0	0	1.00	.90	.00
	4	45	9	40.5	6	.85	.77	.16
	5	30	4	28.0	1	.96	.74	.04
	6	25	3	23.5	0	1.00	.74	.00
	7	22	3	20.5	2	.90	.67	.10
	8	17	2	16.0	0	1.00	.67	.00
	9	15	5	12.5	0	1.00	.67	.00
	10	10	0	10.0	1	.90	.60	.11
	11	9	0	9.0	0	1.00	.60	.00
	12	9	9	4.5	0	1.00	.60	.00

Note: EDE-Q SC = Eating Disorder Examination Questionnaire Shape Concern score, number of participants at time point 0 = 147, Missing n = 4, N staying well = number of participants who stay well, N terminated = number of participants who terminate the study, N exposed to risk = number of participants who are exposed to the risk of experiencing partial ED onset, N events = number of partial ED onsets, % staying well = percentage of participants who stay well, Cumulative % staying well = cumulative percentage of participants who stay well

Table VII.15 Life table: CR-EAT Total

	Time in months	N staying well	N terminated	N exposed to risk	N events	% staying well	Cumulative % staying well	Hazard rate
CR-EAT TO ≤ 2.14	0	75	0	75.0	0	1.00	1.00	.00
	1	75	8	71.0	1	.99	.99	.01
	2	66	9	61.5	0	1.00	.99	.00
	3	57	8	53.0	0	1.00	.99	.00
	4	49	11	43.5	1	.98	.96	.02
	5	37	2	36.0	0	1.00	.96	.00
	6	35	7	31.5	0	1.00	.96	.00
	7	28	4	26.0	0	1.00	.96	.00
	8	24	3	22.5	0	1.00	.96	.00
	9	21	4	19.0	0	1.00	.96	.00
	10	17	1	16.5	0	1.00	.96	.00
	11	16	1	15.5	1	.94	.90	.07
	12	14	14	7.0	0	1.00	.90	.00
CR-EAT TO > 2.14	0	75	0	75.0	0	1.00	1.00	.00
	1	75	13	68.5	3	.96	.96	.04
	2	59	4	57.0	3	.95	.91	.05
	3	52	6	49.0	0	1.00	.91	.00
	4	46	6	43.0	6	.86	.78	.15
	5	34	3	32.5	2	.94	.73	.06
	6	29	4	27.0	0	1.00	.73	.00
	7	25	3	23.5	3	.87	.64	.14
	8	19	1	18.5	0	1.00	.64	.00
	9	18	4	16.0	0	1.00	.64	.00
	10	14	1	13.5	1	.93	.59	.08
	11	12	0	12.0	0	1.00	.59	.00
	12	12	11	6.5	1	.85	.50	.00

Note: CR-EAT TO = Clinical and Research Inventory for Eating Disorders total score, number of participants at time point 0 = 150, Missing n = 1, N staying well = number of participants who stay well, N terminated = number of participants who terminate the study, N exposed to risk = number of participants who are exposed to the risk of experiencing partial ED onset, N events = number of partial ED onsets, % staying well = percentage of participants who stay well, Cumulative % staying well = cumulative percentage of participants who stay well

Table VII.16 Life table: CR-EAT Eating Behavior Disturbance

	Time in months	N staying well	N terminated	N exposed to risk	N events	% staying well	Cumulative % staying well	Hazard rate
CR-EAT EBD ≤ 1.76	0	75	0	75.0	0	1.00	1.00	.00
	1	75	9	70.5	1	.99	.99	.01
	2	65	7	61.5	0	1.00	.99	.00
	3	58	8	54.0	0	1.00	.99	.00
	4	50	10	45.0	2	.96	.94	.05
	5	38	1	37.5	0	1.00	.94	.00
	6	37	7	33.5	0	1.00	.94	.00
	7	30	5	27.5	0	1.00	.94	.00
	8	25	4	23.0	0	1.00	.94	.00
	9	21	3	19.5	0	1.00	.94	.00
	10	18	2	17.0	0	1.00	.94	.00
	11	16	1	15.5	1	.94	.88	.07
	12	14	13	7.5	1	.87	.76	.00
CR-EAT EBD > 1.76	0	75	0	75.0	0	1.00	1.00	.00
	1	75	12	69.0	3	.96	.96	.04
	2	60	6	57.0	3	.95	.91	.05
	3	51	6	48.0	0	1.00	.91	.00
	4	45	7	41.5	5	.88	.80	.13
	5	33	4	31.0	2	.94	.75	.07
	6	27	4	25.0	0	1.00	.75	.00
	7	23	2	22.0	3	.86	.64	.15
	8	18	0	18.0	0	1.00	.64	.00
	9	18	5	15.5	0	1.00	.64	.00
	10	13	0	13.0	1	.92	.59	.08
	11	12	0	12.0	0	1.00	.59	.00
	12	12	12	6.0	0	1.00	.59	.00

Note: CR-EAT EBD = Clinical and Research Inventory for Eating Disorders Eating Behavior Disturbance global score, number of participants at time point 0 = 150, Missing n = 1, N staying well = number of participants who stay well, N terminated = number of participants who terminate the study, N exposed to risk = number of participants who are exposed to the risk of experiencing partial ED onset, N events = number of partial ED onsets, % staying well = percentage of participants who stay well, Cumulative % staying well = cumulative percentage of participants who stay well

Table VII.17 Life table: CR-EAT Affective/Cognitive Impairment

	Time in months	N staying well	N terminated	N exposed to risk	N events	% staying well	Cumulative % staying well	Hazard rate
CR-EAT ACI ≤ 2.23	0	76	0	76.0	0	1.00	1.00	.00
	1	76	10	71.0	0	1.00	1.00	.00
	2	66	11	60.5	0	1.00	1.00	.00
	3	55	7	51.5	0	1.00	1.00	.00
	4	48	9	43.5	1	.98	.98	.02
	5	38	3	36.5	0	1.00	.98	.00
	6	35	6	32.0	0	1.00	.98	.00
	7	29	4	27.0	0	1.00	.98	.00
	8	25	2	24.0	0	1.00	.98	.00
	9	23	4	21.0	0	1.00	.98	.00
	10	19	1	18.5	0	1.00	.98	.00
	11	18	0	18.0	1	.94	.92	.06
	12	17	17	8.5	0	1.00	.92	.00
CR-EAT ACI > 2.23	0	74	0	74.0	0	1.00	1.00	.00
	1	74	11	68.5	4	.94	.94	.06
	2	59	2	58.0	3	.95	.89	.05
	3	54	7	50.5	0	1.00	.89	.00
	4	47	8	43.0	6	.86	.77	.15
	5	33	2	32.0	2	.94	.72	.06
	6	29	5	26.5	0	1.00	.72	.00
	7	24	3	22.5	3	.87	.62	.14
	8	18	2	17.0	0	1.00	.62	.00
	9	16	4	14.0	0	1.00	.62	.00
	10	12	1	11.5	1	.91	.57	.09
	11	10	1	9.5	0	1.00	.57	.00
	12	9	8	5.0	1	.80	.46	.00

Note: CR-EAT ACI = Clinical and Research Inventory for Eating Disorders Affective/Cognitive Impairment global score, number of participants at time point 0 = 150, Missing n = 1, N staying well = number of participants who stay well, N terminated = number of participants who terminate the study, N exposed to risk = number of participants who are exposed to the risk of experiencing partial ED onset, N events = number of partial ED onsets, % staying well = percentage of participants who stay well, Cumulative % staying well = cumulative percentage of participants who stay well

Table VII.18 Life table: CR-EAT Perfectionism Total

	Time in months	N staying well	N terminated	N exposed to risk	N events	% staying well	Cumulative % staying well	Hazard rate
CR-EAT PER ≤ 1.83	0	66	0	66.0	0	1.00	1.00	.00
	1	66	9	61.5	2	.97	.97	.03
	2	55	5	52.5	0	1.00	.97	.00
	3	50	5	47.5	0	1.00	.97	.00
	4	45	9	40.5	2	.95	.92	.05
	5	34	3	32.5	1	.97	.89	.03
	6	30	7	26.5	0	1.00	.89	.00
	7	23	3	21.5	1	.95	.85	.05
	8	19	4	17.0	0	1.00	.85	.00
	9	15	2	14.0	0	1.00	.85	.00
	10	13	0	13.0	0	1.00	.85	.00
	11	13	0	13.0	1	.92	.78	.08
	12	12	12	6.0	0	1.00	.78	.00
CR-EAT PER > 1.83	0	84	0	84.0	0	1.00	1.00	.00
	1	84	12	78.0	2	.97	.97	.03
	2	70	8	66.0	3	.95	.93	.05
	3	59	9	54.5	0	1.00	.93	.00
	4	50	8	46.0	5	.89	.83	.11
	5	37	2	36.0	1	.97	.81	.03
	6	34	4	32.0	0	1.00	.81	.00
	7	30	4	28.0	2	.93	.75	.07
	8	24	0	24.0	0	1.00	.75	.00
	9	24	6	21.0	0	1.00	.75	.00
	10	18	2	17.0	1	.94	.70	.06
	11	15	1	14.5	0	1.00	.70	.00
	12	14	13	7.5	1	.87	.61	.00

Note: CR-EAT PER = Clinical and Research Inventory for Eating Disorders Perfectionism Total global score, number of participants at time point 0 = 150, Missing n = 1, N staying well = number of participants who stay well, N terminated = number of participants who terminate the study, N exposed to risk = number of participants who are exposed to the risk of experiencing partial ED onset, N events = number of partial ED onsets, % staying well = percentage of participants who stay well, Cumulative % staying well = cumulative percentage of participants who stay well

Table VII.19 Life table: CR-EAT Weight Preoccupation

	Time in months	N staying well	N terminated	N exposed to risk	N events	% staying well	Cumulative % staying well	Hazard rate
CR-EAT WP ≤ 1.83	0	72	0	72.0	0	1.00	1.00	.00
	1	72	8	68.0	1	.99	.99	.01
	2	63	7	59.5	0	1.00	.99	.00
	3	56	8	52.0	0	1.00	.99	.00
	4	48	12	42.0	2	.95	.94	.05
	5	34	1	33.5	0	1.00	.94	.00
	6	33	7	29.5	0	1.00	.94	.00
	7	26	4	24.0	0	1.00	.94	.00
	8	22	4	20.0	0	1.00	.94	.00
	9	18	4	16.0	0	1.00	.94	.00
	10	14	2	13.0	0	1.00	.94	.00
	11	12	0	12.0	0	1.00	.94	.00
	12	12	11	6.5	1	.85	.79	.17
CR-EAT WP > 1.83	0	78	0	78.0	0	1.00	1.00	.00
	1	78	13	71.5	3	.96	.96	.04
	2	62	6	59.0	3	.95	.91	.05
	3	53	6	50.0	0	1.00	.91	.00
	4	47	5	44.5	5	.89	.81	.12
	5	37	4	35.0	2	.94	.76	.06
	6	31	4	29.0	0	1.00	.76	.00
	7	27	3	25.5	3	.88	.67	.13
	8	21	0	21.0	0	1.00	.67	.00
	9	21	4	19.0	0	1.00	.67	.00
	10	17	0	17.0	1	.94	.63	.06
	11	16	1	15.5	1	.94	.59	.07
	12	14	14	7.0	0	1.00	.59	.00

Note: CR-EAT WP = Clinical and Research Inventory for Eating Disorders Weight Preoccupation score, number of participants at time point 0 = 150, Missing n = 1, N staying well = number of participants who stay well, N terminated = number of participants who terminate the study, N exposed to risk = number of participants who are exposed to the risk of experiencing partial ED onset, N events = number of partial ED onsets, % staying well = percentage of participants who stay well, Cumulative % staying well = cumulative percentage of participants who stay well

Table VII.20 Life table: CR-EAT Control over Eating

	Time in months	N staying well	N terminated	N exposed to risk	N events	% staying well	Cumulative % staying well	Hazard rate
CR-EAT CE ≤ 2.13	0	77	0	77.0	0	1.00	1.00	.00
	1	77	9	72.5	2	.97	.97	.03
	2	66	8	62.0	0	1.00	.97	.00
	3	58	7	54.5	0	1.00	.97	.00
	4	51	9	46.5	2	.96	.93	.04
	5	40	1	39.5	0	1.00	.93	.00
	6	39	7	35.5	0	1.00	.93	.00
	7	32	6	29.0	1	.97	.90	.04
	8	25	4	23.0	0	1.00	.90	.00
	9	21	4	19.0	0	1.00	.90	.00
	10	17	2	16.0	0	1.00	.90	.00
	11	15	1	14.5	0	1.00	.90	.00
	12	14	13	7.5	1	.87	.78	.14
CR-EAT CE > 2.13	0	73	0	73.0	0	1.00	1.00	.00
	1	73	12	67.0	2	.97	.97	.03
	2	59	5	56.5	3	.95	.92	.05
	3	51	7	47.5	0	1.00	.92	.00
	4	44	8	40.0	5	.88	.80	.13
	5	31	4	29.0	2	.93	.75	.07
	6	25	4	23.0	0	1.00	.75	.00
	7	21	1	20.5	2	.90	.68	.10
	8	18	0	18.0	0	1.00	.68	.00
	9	18	4	16.0	0	1.00	.68	.00
	10	14	0	14.0	1	.93	.63	.07
	11	13	0	13.0	1	.92	.58	.08
	12	12	12	6.0	0	1.00	.58	.00

Note: CR-EAT CE = Clinical and Research Inventory for Eating Disorders Control over Eating score, number of participants at time point 0 = 150, Missing n = 1, N staying well = number of participants who stay well, N terminated = number of participants who terminate the study, N exposed to risk = number of participants who are exposed to the risk of experiencing partial ED onset, N events = number of partial ED onsets, % staying well = percentage of participants who stay well, Cumulative % staying well = cumulative percentage of participants who stay well

Table VII.21 Life table: CR-EAT Emotional Dysregulation

	Time in months	N staying well	N terminated	N exposed to risk	N events	% staying well	Cumulative % staying well	Hazard rate
CR-EAT ED ≤ 1.75	0	79	0	79.0	0	1.00	1.00	.00
	1	79	8	75.0	1	.99	.99	.01
	2	70	11	64.5	0	1.00	.99	.00
	3	59	7	55.5	0	1.00	.99	.00
	4	52	11	46.5	1	.98	.97	.02
	5	40	3	38.5	0	1.00	.97	.00
	6	37	8	33.0	0	1.00	.97	.00
	7	29	4	27.0	1	.96	.93	.04
	8	24	3	22.5	0	1.00	.93	.00
	9	21	4	19.0	0	1.00	.93	.00
	10	17	1	16.5	0	1.00	.93	.00
	11	16	1	15.5	0	1.00	.93	.00
	12	15	15	7.5	0	1.00	.93	.00
CR-EAT ED > 1.75	0	71	0	71.0	0	1.00	1.00	.00
	1	71	13	64.5	3	.95	.95	.05
	2	55	2	54.0	3	.94	.90	.06
	3	50	7	46.5	0	1.00	.90	.00
	4	43	6	40.0	6	.85	.77	.16
	5	31	2	30.0	2	.93	.71	.07
	6	27	3	25.5	0	1.00	.71	.00
	7	24	3	22.5	2	.91	.65	.09
	8	19	1	18.5	0	1.00	.65	.00
	9	18	4	16.0	0	1.00	.65	.00
	10	14	1	13.5	1	.93	.60	.08
	11	12	0	12.0	1	.92	.55	.09
	12	11	10	6.0	1	.83	.46	.00

Note: CR-EAT ED = Clinical and Research Inventory for Eating Disorders Emotional Dysregulation score, number of participants at time point 0 = 150, Missing n = 1, N staying well = number of participants who stay well, N terminated = number of participants who terminate the study, N exposed to risk = number of participants who are exposed to the risk of experiencing partial ED onset, N events = number of partial ED onsets, % staying well = percentage of participants who stay well, Cumulative % staying well = cumulative percentage of participants who stay well

Table VII.22 Life table: CR-EAT Affect-Regulatory Eating

	Time in months	N staying well	N terminated	N exposed to risk	N events	% staying well	Cumulative % staying well	Hazard rate
CR-EAT AE ≤ 1.75	0	76	0	76.0	0	1.00	1.00	.00
	1	76	14	69.0	0	1.00	1.00	.00
	2	62	7	58.5	0	1.00	1.00	.00
	3	55	7	51.5	0	1.00	1.00	.00
	4	48	8	44.0	2	.95	.95	.05
	5	38	2	37.0	0	1.00	.95	.00
	6	36	7	32.5	0	1.00	.95	.00
	7	29	4	27.0	0	1.00	.95	.00
	8	25	4	23.0	0	1.00	.95	.00
	9	21	4	19.0	0	1.00	.95	.00
	10	17	2	16.0	0	1.00	.95	.00
	11	15	0	15.0	1	.93	.89	.07
	12	14	14	7.0	0	1.00	.89	.00
CR-EAT AE > 1.75	0	74	0	74.0	0	1.00	1.00	.00
	1	74	7	70.5	4	.94	.94	.06
	2	63	6	60.0	3	.95	.90	.05
	3	54	7	50.5	0	1.00	.90	.00
	4	47	9	42.5	5	.88	.79	.13
	5	33	3	31.5	2	.94	.74	.07
	6	28	4	26.0	0	1.00	.74	.00
	7	24	3	22.5	3	.87	.64	.14
	8	18	0	18.0	0	1.00	.64	.00
	9	18	4	16.0	0	1.00	.64	.00
	10	14	0	14.0	1	.93	.60	.07
	11	13	1	12.5	0	1.00	.60	.00
	12	12	11	6.5	1	.85	.50	.17

Note: CR-EAT AE = Clinical and Research Inventory for Eating Disorders Affect-Regulatory Eating score, number of participants at time point 0 = 150, Missing n = 1, N staying well = number of participants who stay well, N terminated = number of participants who terminate the study, N exposed to risk = number of participants who are exposed to the risk of experiencing partial ED onset, N events = number of partial ED onsets, % staying well = percentage of participants who stay well, Cumulative % staying well = cumulative percentage of participants who stay well

Table VII.23 Life table: CR-EAT Self-Esteem

	Time in months	N staying well	N terminated	N exposed to risk	N events	% staying well	Cumulative % staying well	Hazard rate
CR-EAT SE ≤ 2.29	0	78	0	78.0	0	1.00	1.00	.00
	1	78	9	73.5	0	1.00	1.00	.00
	2	69	11	63.5	1	.98	.98	.02
	3	57	5	54.5	0	1.00	.98	.00
	4	52	10	47.0	1	.98	.96	.02
	5	41	3	39.5	0	1.00	.96	.00
	6	38	7	34.5	0	1.00	.96	.00
	7	31	3	29.5	1	.97	.93	.03
	8	27	3	25.5	0	1.00	.93	.00
	9	24	6	21.0	0	1.00	.93	.00
	10	18	1	17.5	0	1.00	.93	.00
	11	17	0	17.0	1	.94	.88	.06
	12	16	16	8.0	0	1.00	.88	.00
CR-EAT SE > 2.29	0	72	0	72.0	0	1.00	1.00	.00
	1	72	12	66.0	4	.94	.94	.06
	2	56	2	55.0	2	.96	.91	.04
	3	52	9	47.5	0	1.00	.91	.00
	4	43	7	39.5	6	.85	.77	.16
	5	30	2	29.0	2	.93	.71	.07
	6	26	4	24.0	0	1.00	.71	.00
	7	22	4	20.0	2	.90	.64	.11
	8	16	1	15.5	0	1.00	.64	.00
	9	15	2	14.0	0	1.00	.64	.00
	10	13	1	12.5	1	.92	.59	.08
	11	11	1	10.5	0	1.00	.59	.00
	12	10	9	5.5	1	.82	.48	.20

Note: CR-EAT SE = Clinical and Research Inventory for Eating Disorders Self-Esteem score, number of participants at time point 0 = 150, Missing n = 1, N staying well = number of participants who stay well, N terminated = number of participants who terminate the study, N exposed to risk = number of participants who are exposed to the risk of experiencing partial ED onset, N events = number of partial ED onsets, % staying well = percentage of participants who stay well, Cumulative % staying well = cumulative percentage of participants who stay well

Table VII.24 Life table: CR-EAT Concern about Negative Evaluation

	Time in months	N staying well	N terminated	N exposed to risk	N events	% staying well	Cumulative % staying well	Hazard rate
CR-EAT CN ≤ 2.86	0	78	0	78.0	0	1.00	1.00	.00
	1	78	10	73.0	2	.97	.97	.03
	2	66	11	60.5	0	1.00	.97	.00
	3	55	4	53.0	0	1.00	.97	.00
	4	51	12	45.0	3	.93	.91	.07
	5	36	2	35.0	2	.94	.86	.06
	6	32	7	28.5	0	1.00	.86	.00
	7	25	4	23.0	0	1.00	.86	.00
	8	21	2	20.0	0	1.00	.86	.00
	9	19	3	17.5	0	1.00	.86	.00
	10	16	0	16.0	1	.94	.80	.06
	11	15	1	14.5	1	.93	.75	.07
	12	13	13	6.5	0	1.00	.75	.00
CR-EAT CN > 2.86	0	72	0	72.0	0	1.00	1.00	.00
	1	72	11	66.5	2	.97	.97	.03
	2	59	2	58.0	3	.95	.92	.05
	3	54	10	49.0	0	1.00	.92	.00
	4	44	5	41.5	4	.90	.83	.10
	5	35	3	33.5	0	1.00	.83	.00
	6	32	4	30.0	0	1.00	.83	.00
	7	28	3	26.5	3	.89	.74	.12
	8	22	2	21.0	0	1.00	.74	.00
	9	20	5	17.5	0	1.00	.74	.00
	10	15	2	14.0	0	1.00	.74	.00
	11	13	0	13.0	0	1.00	.74	.00
	12	13	12	7.0	1	.86	.63	.15

Note: CR-EAT CN = Clinical and Research Inventory for Eating Disorders Concern about Negative Evaluation score, number of participants at time point 0 = 150, Missing n = 1, N staying well = number of participants who stay well, N terminated = number of participants who terminate the study, N exposed to risk = number of participants who are exposed to the risk of experiencing partial ED onset, N events = number of partial ED onsets, % staying well = percentage of participants who stay well, Cumulative % staying well = cumulative percentage of participants who stay well

Table VII.25 Life table: CR-EAT Body Embarrassment

	Time in months	N staying well	N terminated	N exposed to risk	N events	% staying well	Cumulative % staying well	Hazard rate
CR-EAT BE ≤ 1.60	0	79	0	79.0	0	1.00	1.00	.00
	1	79	9	74.5	0	1.00	1.00	.00
	2	70	9	65.5	0	1.00	1.00	.00
	3	61	9	56.5	0	1.00	1.00	.00
	4	52	9	47.5	3	.94	.94	.07
	5	40	2	39.0	1	.97	.91	.03
	6	37	7	33.5	0	1.00	.91	.00
	7	30	5	27.5	0	1.00	.91	.00
	8	25	3	23.5	0	1.00	.91	.00
	9	22	4	20.0	0	1.00	.91	.00
	10	18	2	17.0	0	1.00	.91	.00
	11	16	1	15.5	1	.94	.85	.07
	12	14	14	7.0	0	1.00	.85	.00
CR-EAT BE > 1.60	0	71	0	71.0	0	1.00	1.00	.00
	1	71	12	65.0	4	.94	.94	.06
	2	55	4	53.0	3	.94	.89	.06
	3	48	5	45.5	0	1.00	.89	.00
	4	43	8	39.0	4	.90	.79	.11
	5	31	3	29.5	1	.97	.77	.03
	6	27	4	25.0	0	1.00	.77	.00
	7	23	2	22.0	3	.86	.66	.15
	8	18	1	17.5	0	1.00	.66	.00
	9	17	4	15.0	0	1.00	.66	.00
	10	13	0	13.0	1	.92	.61	.08
	11	12	0	12.0	0	1.00	.61	.00
	12	12	11	6.5	1	.85	.52	.17

Note: CR-EAT BE = Clinical and Research Inventory for Eating Disorders Body Embarrassment score, number of participants at time point 0 = 150, Missing n = 1, N staying well = number of participants who stay well, N terminated = number of participants who terminate the study, N exposed to risk = number of participants who are exposed to the risk of experiencing partial ED onset, N events = number of partial ED onsets, % staying well = percentage of participants who stay well, Cumulative % staying well = cumulative percentage of participants who stay well

Table VII.26 Life table: CR-EAT Restraint Eating Behavior

	Time in months	N staying well	N terminated	N exposed to risk	N events	% staying well	Cumulative % staying well	Hazard rate
CR-EAT RE ≤ 1.67	0	79	0	79.0	0	1.00	1.00	.00
	1	79	9	74.5	3	.96	.96	.04
	2	67	8	63.0	0	1.00	.96	.00
	3	59	7	55.5	0	1.00	.96	.00
	4	52	13	45.5	2	.96	.92	.04
	5	37	1	36.5	0	1.00	.92	.00
	6	36	7	32.5	0	1.00	.92	.00
	7	29	3	27.5	0	1.00	.92	.00
	8	26	4	24.0	0	1.00	.92	.00
	9	22	3	20.5	0	1.00	.92	.00
	10	19	2	18.0	0	1.00	.92	.00
	11	17	0	17.0	0	1.00	.92	.00
	12	17	16	9.0	1	.89	.82	.12
CR-EAT RE > 1.67	0	71	0	71.0	0	1.00	1.00	.00
	1	71	12	65.0	1	.98	.98	.02
	2	58	5	55.5	3	.95	.93	.06
	3	50	7	46.5	0	1.00	.93	.00
	4	43	4	41.0	5	.88	.82	.13
	5	34	4	32.0	2	.94	.77	.06
	6	28	4	26.0	0	1.00	.77	.00
	7	24	4	22.0	3	.86	.66	.15
	8	17	0	17.0	0	1.00	.66	.00
	9	17	5	14.5	0	1.00	.66	.00
	10	12	0	12.0	1	.92	.61	.09
	11	11	1	10.5	1	.90	.55	.10
	12	9	9	4.5	0	1.00	.55	.00

Note: CR-EAT RE = Clinical and Research Inventory for Eating Disorders Restraint Eating Behavior score, number of participants at time point 0 = 150, Missing n = 1, N staying well = number of participants who stay well, N terminated = number of participants who terminate the study, N exposed to risk = number of participants who are exposed to the risk of experiencing partial ED onset, N events = number of partial ED onsets, % staying well = percentage of participants who stay well, Cumulative % staying well = cumulative percentage of participants who stay well

Table VII.27 Life table: CR-EAT Societal Expectations of Shape and Weight

	Time in months	N staying well	N terminated	N exposed to risk	N events	% staying well	Cumulative % staying well	Hazard rate
CR-EAT SO ≤ 1.60	0	80	0	80.0	0	1.00	1.00	.00
	1	80	10	75.0	0	1.00	1.00	.00
	2	70	10	65.0	0	1.00	1.00	.00
	3	60	7	56.5	0	1.00	1.00	.00
	4	53	11	47.5	1	.98	.98	.02
	5	41	3	39.5	1	.97	.95	.03
	6	37	7	33.5	0	1.00	.95	.00
	7	30	4	28.0	1	.96	.92	.04
	8	25	4	23.0	0	1.00	.92	.00
	9	21	2	20.0	0	1.00	.92	.00
	10	19	2	18.0	0	1.00	.92	.00
	11	17	1	16.5	1	.94	.86	.06
	12	15	14	8.0	1	.88	.76	.13
CR-EAT SO > 1.60	0	70	0	70.0	0	1.00	1.00	.00
	1	70	11	64.5	4	.94	.94	.06
	2	55	3	53.5	3	.94	.89	.06
	3	49	7	45.5	0	1.00	.89	.00
	4	42	6	39.0	6	.85	.75	.17
	5	30	2	29.0	1	.97	.72	.04
	6	27	4	25.0	0	1.00	.72	.00
	7	23	3	21.5	2	.91	.66	.10
	8	18	0	18.0	0	1.00	.66	.00
	9	18	6	15.0	0	1.00	.66	.00
	10	12	0	12.0	1	.92	.60	.09
	11	11	0	11.0	0	1.00	.60	.00
	12	11	11	5.5	0	1.00	.60	.00

Note: CR-EAT SO = Clinical and Research Inventory for Eating Disorders Societal Expectations of Shape and Weight score, number of participants at time point 0 = 150, Missing n = 1, N staying well = number of participants who stay well, N terminated = number of participants who terminate the study, N exposed to risk = number of participants who are exposed to the risk of experiencing partial ED onset, N events = number of partial ED onsets, % staying well = percentage of participants who stay well, Cumulative % staying well = cumulative percentage of participants who stay well

Table VII.28 Life table: CR-EAT Perfectionism: Familial Expectations

	Time in months	N staying well	N terminated	N exposed to risk	N events	% staying well	Cumulative % staying well	Hazard rate
CR-EAT PF ≤ 2.33	0	66	0	66.0	0	1.00	1.00	.00
	1	66	10	61.0	2	.97	.97	.03
	2	54	5	51.5	1	.98	.95	.02
	3	48	5	45.5	0	1.00	.95	.00
	4	43	10	38.0	2	.95	.90	.05
	5	31	4	29.0	1	.97	.87	.04
	6	26	6	23.0	0	1.00	.87	.00
	7	20	2	19.0	2	.89	.78	.11
	8	16	2	15.0	0	1.00	.78	.00
	9	14	2	13.0	0	1.00	.78	.00
	10	12	0	12.0	1	.92	.71	.09
	11	11	0	11.0	0	1.00	.71	.00
	12	11	11	5.5	0	1.00	.71	.00
CR-EAT PF > 2.33	0	84	0	84.0	0	1.00	1.00	.00
	1	84	11	78.5	2	.97	.97	.03
	2	71	8	67.0	2	.97	.95	.03
	3	61	9	56.5	0	1.00	.95	.00
	4	52	7	48.5	5	.90	.85	.11
	5	40	1	39.5	1	.97	.83	.03
	6	38	5	35.5	0	1.00	.83	.00
	7	33	5	30.5	1	.97	.80	.03
	8	27	2	26.0	0	1.00	.80	.00
	9	25	6	22.0	0	1.00	.80	.00
	10	19	2	18.0	0	1.00	.80	.00
	11	17	1	16.5	1	.94	.75	.06
	12	15	14	8.0	1	.88	.66	.13

Note: CR-EAT PF = Clinical and Research Inventory for Eating Disorders Perfectionism: Familial Expectations score, number of participants at time point 0 = 150, Missing n = 1, N staying well = number of participants who stay well, N terminated = number of participants who terminate the study, N exposed to risk = number of participants who are exposed to the risk of experiencing partial ED onset, N events = number of partial ED onsets, % staying well = percentage of participants who stay well, Cumulative % staying well = cumulative percentage of participants who stay well

Table VII.29 Life table: CR-EAT Harmful Weight Regulation

	Time in months	N staying well	N terminated	N exposed to risk	N events	% staying well	Cumulative % staying well	Hazard rate
CR-EAT HW ≤ 1.00	0	84	0	84.0	0	1.00	1.00	.00
	1	84	10	79.0	1	.99	.99	.01
	2	73	9	68.5	0	1.00	.99	.00
	3	64	8	60.0	0	1.00	.99	.00
	4	56	12	50.0	3	.94	.93	.06
	5	41	3	39.5	1	.97	.90	.03
	6	37	6	34.0	0	1.00	.90	.00
	7	31	5	28.5	0	1.00	.90	.00
	8	26	4	24.0	0	1.00	.90	.00
	9	22	4	20.0	0	1.00	.90	.00
	10	18	1	17.5	0	1.00	.90	.00
	11	17	1	16.5	1	.94	.85	.06
	12	15	15	7.5	0	1.00	.85	.00
CR-EAT HW > 1.00	0	66	0	66.0	0	1.00	1.00	.00
	1	66	11	60.5	3	.95	.95	.05
	2	52	4	50.0	3	.94	.89	.06
	3	45	6	42.0	0	1.00	.89	.00
	4	39	5	36.5	4	.89	.80	.12
	5	30	2	29.0	1	.97	.77	.04
	6	27	5	24.5	0	1.00	.77	.00
	7	22	2	21.0	3	.86	.66	.15
	8	17	0	17.0	0	1.00	.66	.00
	9	17	4	15.0	0	1.00	.66	.00
	10	13	1	12.5	1	.92	.61	.08
	11	11	0	11.0	0	1.00	.61	.00
	12	11	10	6.0	1	.83	.50	.18

Note: CR-EAT HW = Clinical and Research Inventory for Eating Disorders Harmful Weight Regulation score, number of participants at time point 0 = 150, Missing n = 1, N staying well = number of participants who stay well, N terminated = number of participants who terminate the study, N exposed to risk = number of participants who are exposed to the risk of experiencing partial ED onset, N events = number of partial ED onsets, % staying well = percentage of participants who stay well, Cumulative % staying well = cumulative percentage of participants who stay well

Table VII.30 Life table: CR-EAT Perfectionism: Personal Expectations

	Time in months	N staying well	N terminated	N exposed to risk	N events	% staying well	Cumulative % staying well	Hazard rate
CR-EAT PP ≤ 3.75	0	82	0	82.0	0	1.00	1.00	.00
	1	82	12	76.0	2	.97	.97	.03
	2	68	7	64.5	1	.98	.96	.02
	3	60	9	55.5	0	1.00	.96	.00
	4	51	13	44.5	5	.89	.85	.12
	5	33	3	31.5	2	.94	.80	.07
	6	28	5	25.5	0	1.00	.80	.00
	7	23	2	22.0	2	.91	.72	.10
	8	19	3	17.5	0	1.00	.72	.00
	9	16	4	14.0	0	1.00	.72	.00
	10	12	0	12.0	0	1.00	.72	.00
	11	12	0	12.0	1	.92	.66	.09
	12	11	11	5.5	0	1.00	.66	.00
CR-EAT PP > 3.75	0	68	0	68.0	0	1.00	1.00	.00
	1	68	9	63.5	2	.97	.97	.03
	2	57	6	54.0	2	.96	.93	.04
	3	49	5	46.5	0	1.00	.93	.00
	4	44	4	42.0	2	.95	.89	.05
	5	38	2	37.0	0	1.00	.89	.00
	6	36	6	33.0	0	1.00	.89	.00
	7	30	5	27.5	1	.96	.86	.04
	8	24	1	23.5	0	1.00	.86	.00
	9	23	4	21.0	0	1.00	.86	.00
	10	19	2	18.0	1	.94	.81	.06
	11	16	1	15.5	0	1.00	.81	.00
	12	15	14	8.0	1	.88	.71	.13

Note: CR-EAT PP = Clinical and Research Inventory for Eating Disorders Perfectionism: Personal Expectations score, number of participants at time point 0 = 150, Missing n = 1, N staying well = number of participants who stay well, N terminated = number of participants who terminate the study, N exposed to risk = number of participants who are exposed to the risk of experiencing partial ED onset, N events = number of partial ED onsets, % staying well = percentage of participants who stay well, Cumulative % staying well = cumulative percentage of participants who stay well

Table VII.31 Life table: DASS-21 Total

	Time in months	N staying well	N terminated	N exposed to risk	N events	% staying well	Cumulative % staying well	Hazard rate
DASS TO ≤ 26.00	0	33	0	33.0	0	1.00	1.00	.00
	1	33	3	31.5	0	1.00	1.00	.00
	2	30	2	29.0	0	1.00	1.00	.00
	3	28	6	25.0	0	1.00	1.00	.00
	4	22	3	20.5	2	.90	.90	.10
	5	17	2	16.0	0	1.00	.90	.00
	6	15	4	13.0	0	1.00	.90	.00
	7	11	1	10.5	0	1.00	.90	.00
	8	10	0	10.0	0	1.00	.90	.00
	9	10	2	9.0	0	1.00	.90	.00
	10	8	2	7.0	0	1.00	.90	.00
	11	6	0	6.0	0	1.00	.90	.00
	12	6	6	3.0	0	1.00	.90	.00
DASS TO > 26.00	0	26	0	26.0	0	1.00	1.00	.00
	1	26	5	23.5	2	.91	.91	.09
	2	19	1	18.5	2	.89	.82	.11
	3	16	1	15.5	0	1.00	.82	.00
	4	15	3	13.5	1	.93	.76	.08
	5	11	0	11.0	0	1.00	.76	.00
	6	11	1	10.5	0	1.00	.76	.00
	7	10	1	9.5	1	.89	.68	.11
	8	8	0	8.0	0	1.00	.68	.00
	9	8	2	7.0	0	1.00	.68	.00
	10	6	0	6.0	0	1.00	.68	.00
	11	6	0	6.0	0	1.00	.68	.00
	12	6	5	3.5	1	.71	.48	.33

Note: DASS TO = Depression Anxiety Stress Scale (21 item version) Total score, number of participants at time point 0 = 59, Missing n = 92, N staying well = number of participants who stay well, N terminated = number of participants who terminate the study, N exposed to risk = number of participants who are exposed to the risk of experiencing partial ED onset, N events = number of partial ED onsets, % staying well = percentage of participants who stay well, Cumulative % staying well = cumulative percentage of participants who stay well

Table VII.32 Life table: DASS-21 Depression

	Time in months	N staying well	N terminated	N exposed to risk	N events	% staying well	Cumulative % staying well	Hazard rate
DASS DE ≤ 8.00	0	34	0	34.0	0	1.00	1.00	.00
	1	34	4	32.0	1	.97	.97	.03
	2	29	1	28.5	0	1.00	.97	.00
	3	28	5	25.5	0	1.00	.97	.00
	4	23	3	21.5	2	.91	.88	.10
	5	18	2	17.0	0	1.00	.88	.00
	6	16	4	14.0	0	1.00	.88	.00
	7	12	1	11.5	0	1.00	.88	.00
	8	11	0	11.0	0	1.00	.88	.00
	9	11	2	10.0	0	1.00	.88	.00
	10	9	2	8.0	0	1.00	.88	.00
	11	7	0	7.0	0	1.00	.88	.00
	12	7	7	3.5	0	1.00	.88	.00
DASS DE > 8.00	0	25	0	25.0	0	1.00	1.00	.00
	1	25	4	23.0	1	.96	.96	.04
	2	20	2	19.0	2	.89	.86	.11
	3	16	2	15.0	0	1.00	.86	.00
	4	14	3	12.5	1	.92	.79	.08
	5	10	0	10.0	0	1.00	.79	.00
	6	10	1	9.5	0	1.00	.79	.00
	7	9	1	8.5	1	.88	.69	.13
	8	7	0	7.0	0	1.00	.69	.00
	9	7	2	6.0	0	1.00	.69	.00
	10	5	0	5.0	0	1.00	.69	.00
	11	5	0	5.0	0	1.00	.69	.00
	12	5	4	3.0	1	.67	.46	.40

Note: DASS DE = Depression Anxiety Stress Scale (21 item version) Depression score, number of participants at time point 0 = 59, Missing n = 92, N staying well = number of participants who stay well, N terminated = number of participants who terminate the study, N exposed to risk = number of participants who are exposed to the risk of experiencing partial ED onset, N events = number of partial ED onsets, % staying well = percentage of participants who stay well, Cumulative % staying well = cumulative percentage of participants who stay well

Table VII.33 Life table: DASS-21 Anxiety

	Time in months	N staying well	N terminated	N exposed to risk	N events	% staying well	Cumulative % staying well	Hazard rate
DASS AN ≤ 8.00	0	33	0	33.0	0	1.00	1.00	.00
	1	33	3	31.5	2	.94	.94	.07
	2	28	1	27.5	1	.96	.90	.04
	3	26	5	23.5	0	1.00	.90	.00
	4	21	3	19.5	1	.95	.86	.05
	5	17	2	16.0	0	1.00	.86	.00
	6	15	5	12.5	0	1.00	.86	.00
	7	10	1	9.5	0	1.00	.86	.00
	8	9	0	9.0	0	1.00	.86	.00
	9	9	2	8.0	0	1.00	.86	.00
	10	7	1	6.5	0	1.00	.86	.00
	11	6	0	6.0	0	1.00	.86	.00
	12	6	6	3.0	0	1.00	.86	.00
DASS AN > 8.00	0	26	0	26.0	0	1.00	1.00	.00
	1	26	5	23.5	0	1.00	1.00	.00
	2	21	2	20.0	1	.95	.95	.05
	3	18	2	17.0	0	1.00	.95	.00
	4	16	3	14.5	2	.86	.82	.15
	5	11	0	11.0	0	1.00	.82	.00
	6	11	0	11.0	0	1.00	.82	.00
	7	11	1	10.5	1	.90	.74	.10
	8	9	0	9.0	0	1.00	.74	.00
	9	9	2	8.0	0	1.00	.74	.00
	10	7	1	6.5	0	1.00	.74	.00
	11	6	0	6.0	0	1.00	.74	.00
	12	6	5	3.5	1	.71	.53	.33

Note: DASS AN = Depression Anxiety Stress Scale (21 item version) Anxiety score, number of participants at time point 0 = 59, Missing n = 92, N staying well = number of participants who stay well, N terminated = number of participants who terminate the study, N exposed to risk = number of participants who are exposed to the risk of experiencing partial ED onset, N events = number of partial ED onsets, % staying well = percentage of participants who stay well, Cumulative % staying well = cumulative percentage of participants who stay well

Table VII.34 Life table: DASS-21 Stress

	Time in months	N staying well	N terminated	N exposed to risk	N events	% staying well	Cumulative % staying well	Hazard rate
DASS ST ≤ 6.00	0	31	0	31.0	0	1.00	1.00	.00
	1	31	2	30.0	1	.97	.97	.03
	2	28	3	26.5	0	1.00	.97	.00
	3	25	5	22.5	0	1.00	.97	.00
	4	20	3	18.5	1	.95	.91	.06
	5	16	2	15.0	0	1.00	.91	.00
	6	14	4	12.0	0	1.00	.91	.00
	7	10	1	9.5	0	1.00	.91	.00
	8	9	0	9.0	0	1.00	.91	.00
	9	9	2	8.0	0	1.00	.91	.00
	10	7	2	6.0	0	1.00	.91	.00
	11	5	0	5.0	0	1.00	.91	.00
	12	5	5	2.5	0	1.00	.91	.00
DASS ST > 6.00	0	28	0	28.0	0	1.00	1.00	.00
	1	28	6	25.0	1	.96	.96	.04
	2	21	0	21.0	2	.90	.87	.10
	3	19	2	18.0	0	1.00	.87	.00
	4	17	3	15.5	2	.87	.76	.14
	5	12	0	12.0	0	1.00	.76	.00
	6	12	1	11.5	0	1.00	.76	.00
	7	11	1	10.5	1	.90	.68	.10
	8	9	0	9.0	0	1.00	.68	.00
	9	9	2	8.0	0	1.00	.68	.00
	10	7	0	7.0	0	1.00	.68	.00
	11	7	0	7.0	0	1.00	.68	.00
	12	7	6	4.0	1	.75	.51	.29

Note: DASS ST = Depression Anxiety Stress Scale (21 item version) Stress score, number of participants at time point 0 = 59, Missing n = 92, N staying well = number of participants who stay well, N terminated = number of participants who terminate the study, N exposed to risk = number of participants who are exposed to the risk of experiencing partial ED onset, N events = number of partial ED onsets, % staying well = percentage of participants who stay well, Cumulative % staying well = cumulative percentage of participants who stay well

Table VII.35 Reference scores: EDE-Q & CR-EAT scales

		Healthy class	Binge Eating class	Classic BN class	Healthy sample	Clinical sample
EDE-Q	TO	1.03	2.28	4.22		
	DR	0.73	1.38	3.58		
	EC	1.48	3.09	5.08		
	WC	1.53	3.13	5.05		
	SC	0.42	1.45	3.17		
CR-EAT	TO				2.55	4.24
	EBD				2.41	4.26
	ACI				2.51	4.24
	PER				3.35	4.10
	WP				2.46	4.24
	CE				2.88	4.83
	ED				2.16	4.39
	AE				2.56	3.99
	SE				2.17	3.88
	CN				3.20	4.68
	BE				2.30	4.34
	RE				2.12	3.66
	SO				2.63	4.36
	PF				2.43	3.46
	HW				1.47	3.66
	PP				4.02	4.67

Note: Healthy, Binge Eating, and Classic BN class = reference scores (Pinheiro et al., 2008), Healthy and Clinical sample = reference scores (Moessner et al., under review), EDE-Q = Eating Disorder Examination Questionnaire, CR-EAT = Clinical and Research Inventory for Eating Disorders, TO = Total Score, RE = Restraint Eating, EC = Eating Concern, WC = Weight Concern, SC = Shape Concern, EBD = Eating Behavior Disturbances, ACI = Affective/Cognitive Impairment, PER = Perfectionism total, WP = Weight Preoccupation, CE = Control over Eating, ED = Emotional Dysregulation, AE = Affect Regulatory Eating, SE = Self Esteem, CN = Concern About Negative Evaluation, BE = Body Embarrassment, RE = Restraint Eating Behavior, SO = Societal Expectations of Shape and Weight, PF = Perfectionism: Familial Expectations, HW = Harmful Weight Regulations, PP = Perfectionism: Personal Expectations

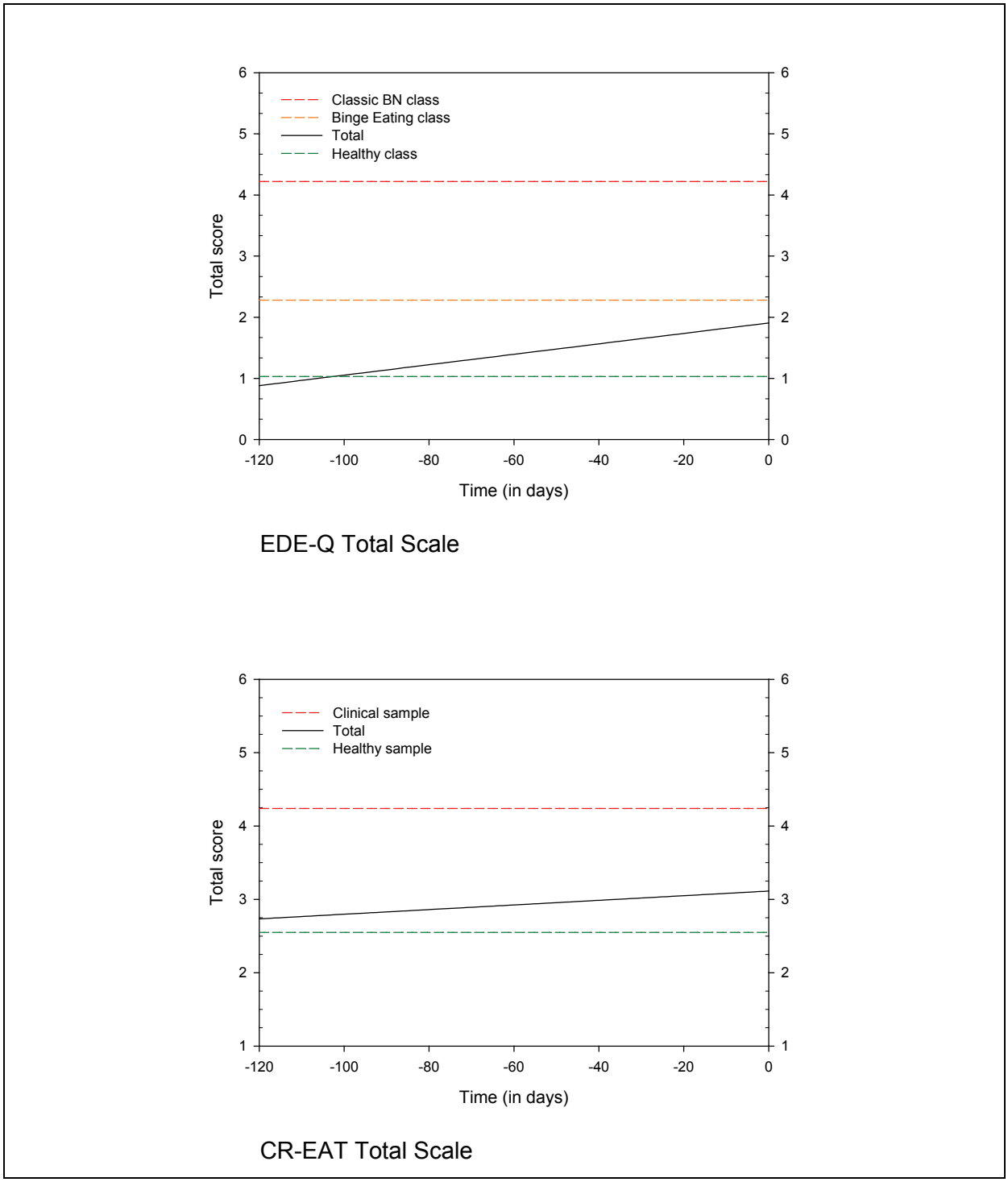


Figure VII.6 Temporal course of the EDE-Q & CR-EAT total scales: Reference scores

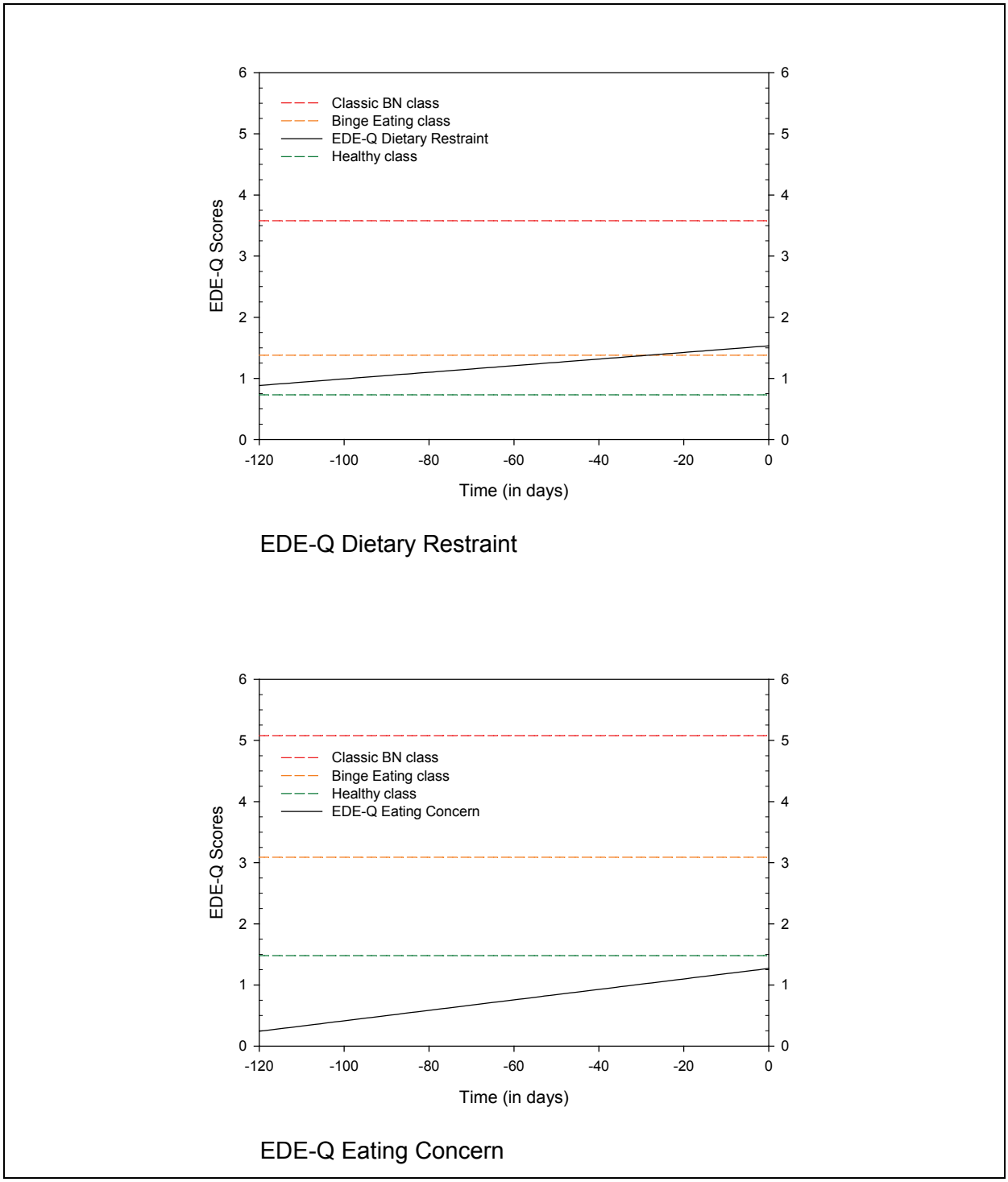


Figure VII.7 Temporal course of the EDE-Q subscales I: References scores

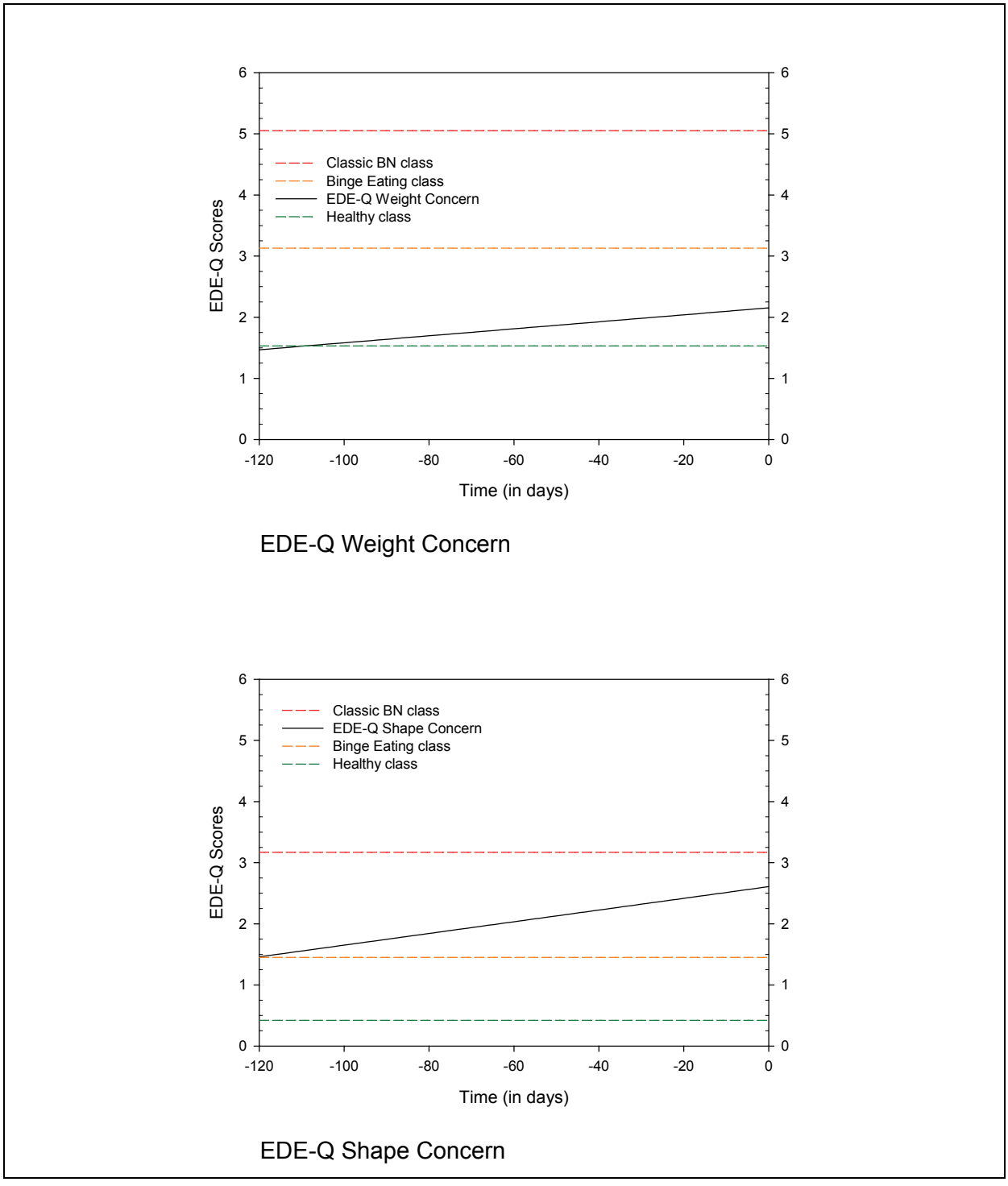


Figure VII.8 Temporal course of the EDE-Q subscales II: References scores

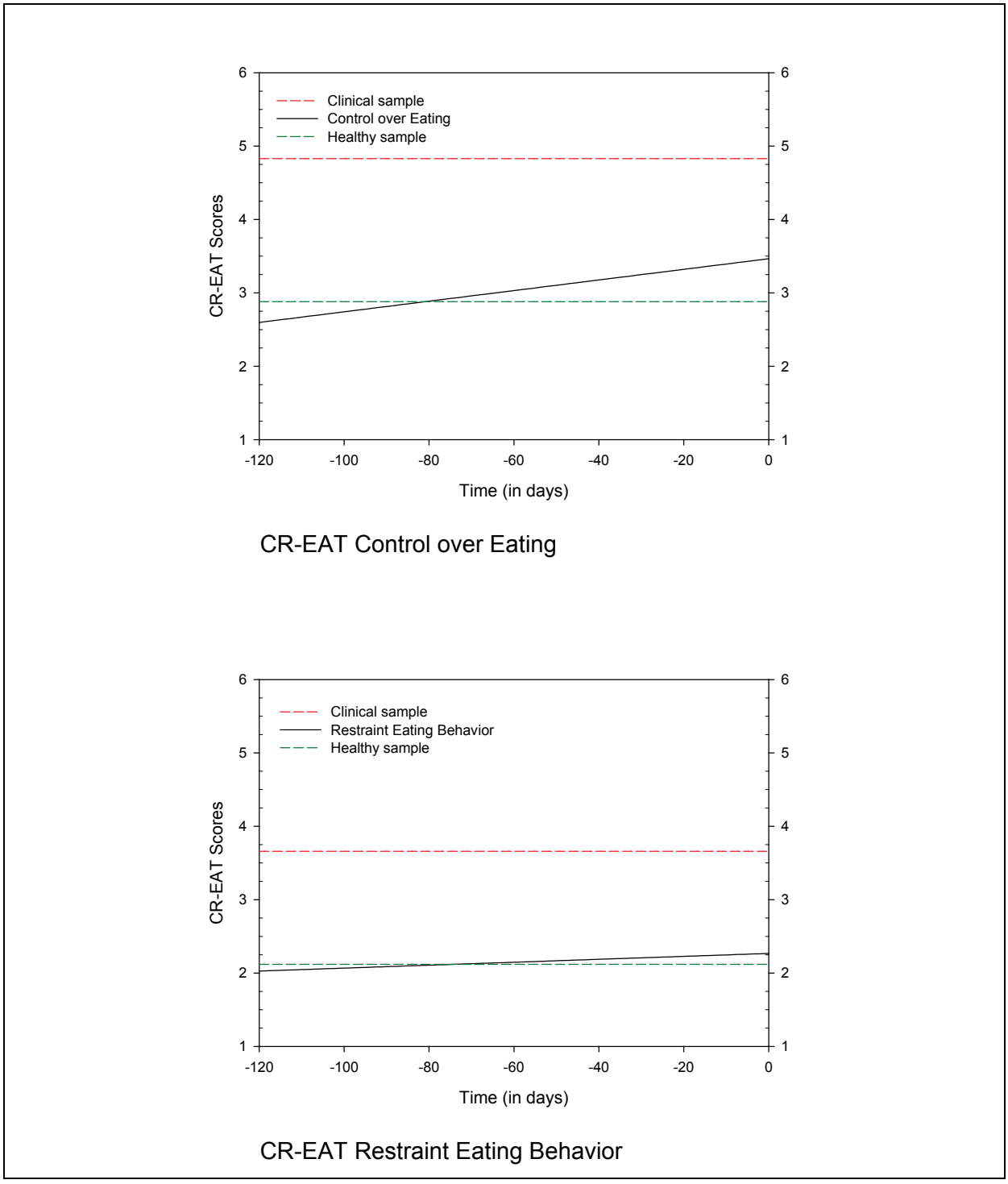
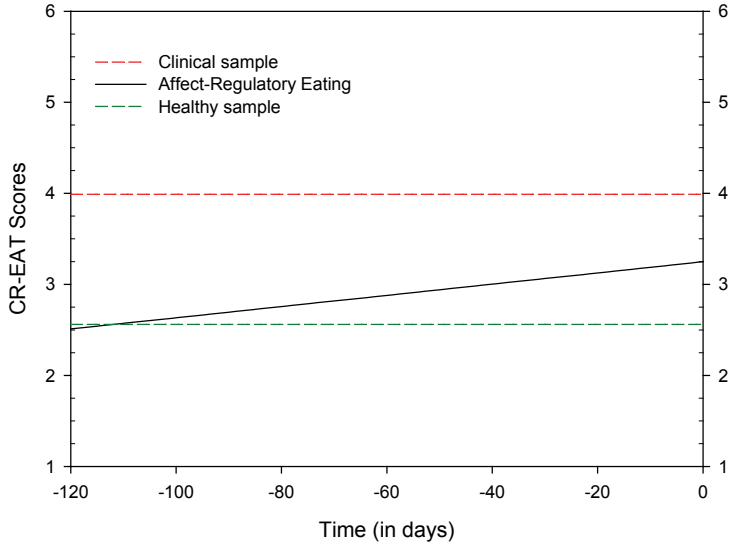
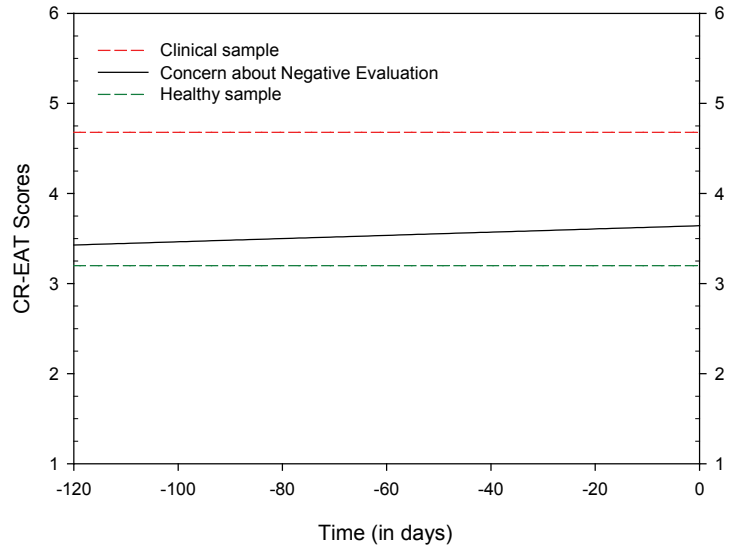


Figure VII.9 Temporal course of the CR-EAT subscales I: References scores



CR-EAT Affect-Regulatory Eating



CR-EAT Concern about Negative Evaluation

Figure VII.10 Temporal course of the CR-EAT subscales II: References scores

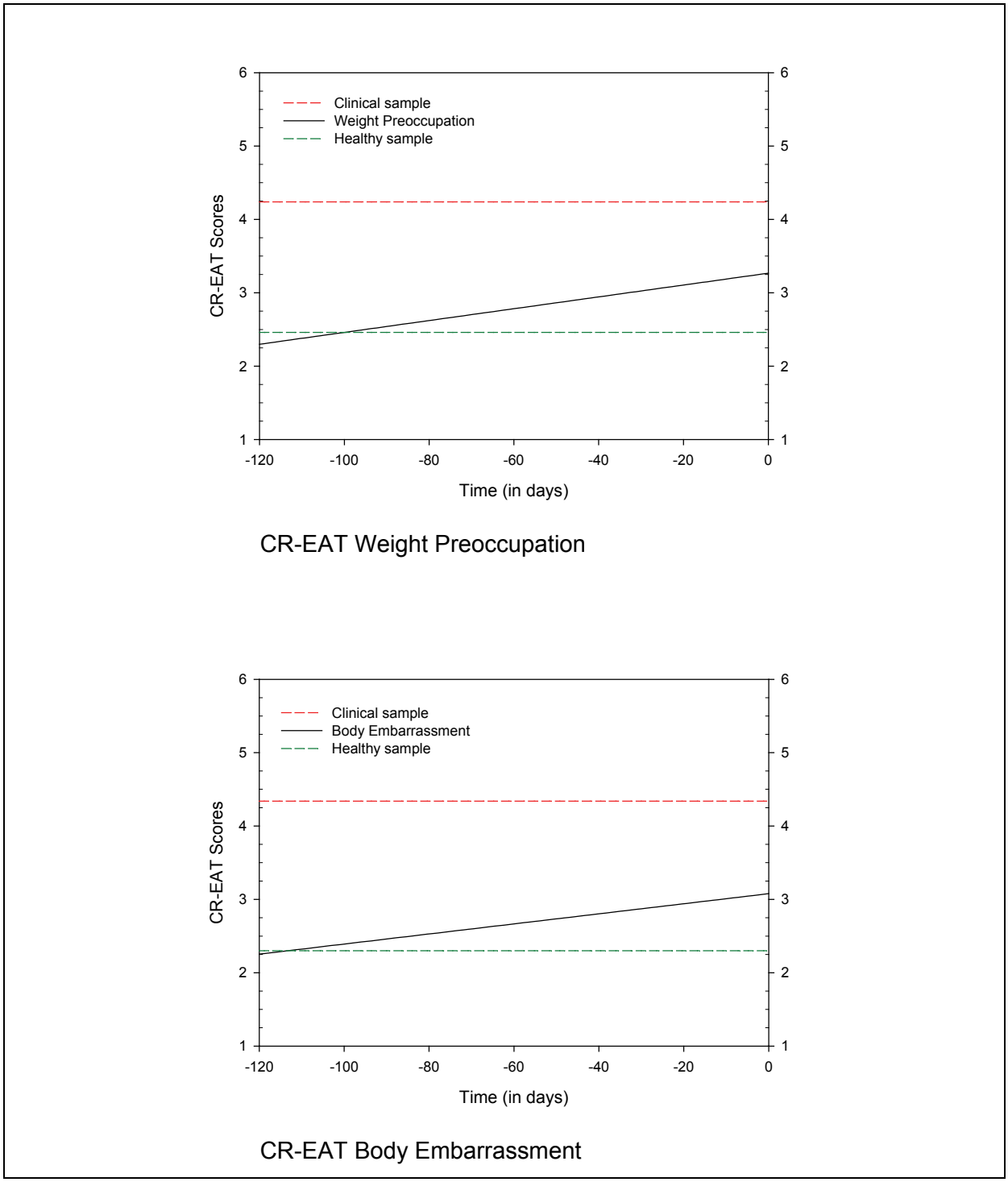
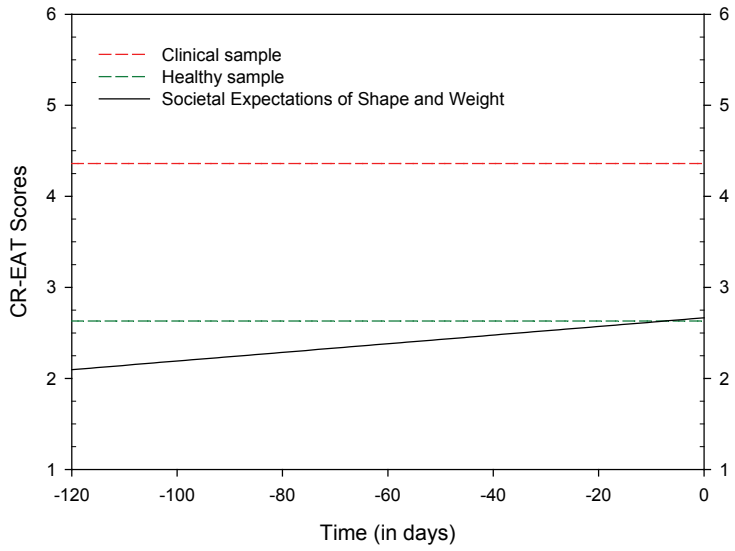
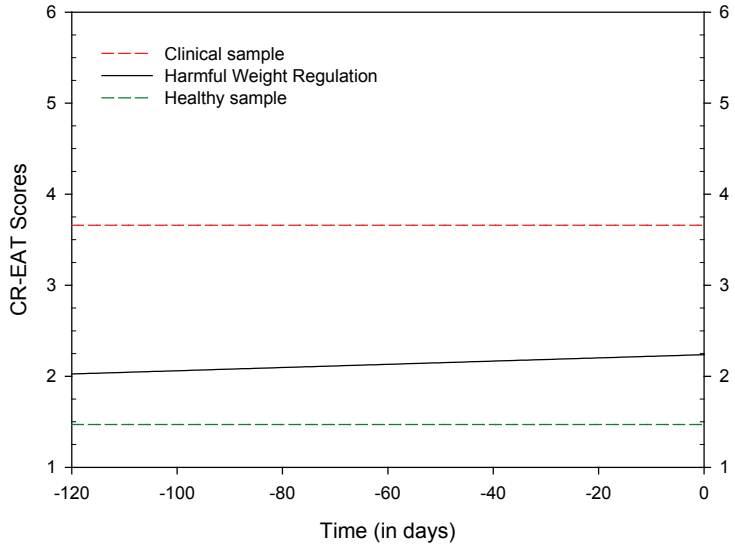


Figure VII.11 Temporal course of the CR-EAT subscales III: References scores



CR-EAT Societal Expectations of Shape and Weight



CR-EAT Harmful Weight Regulation

Figure VII.12 Temporal course of the CR-EAT subscales IV: References scores

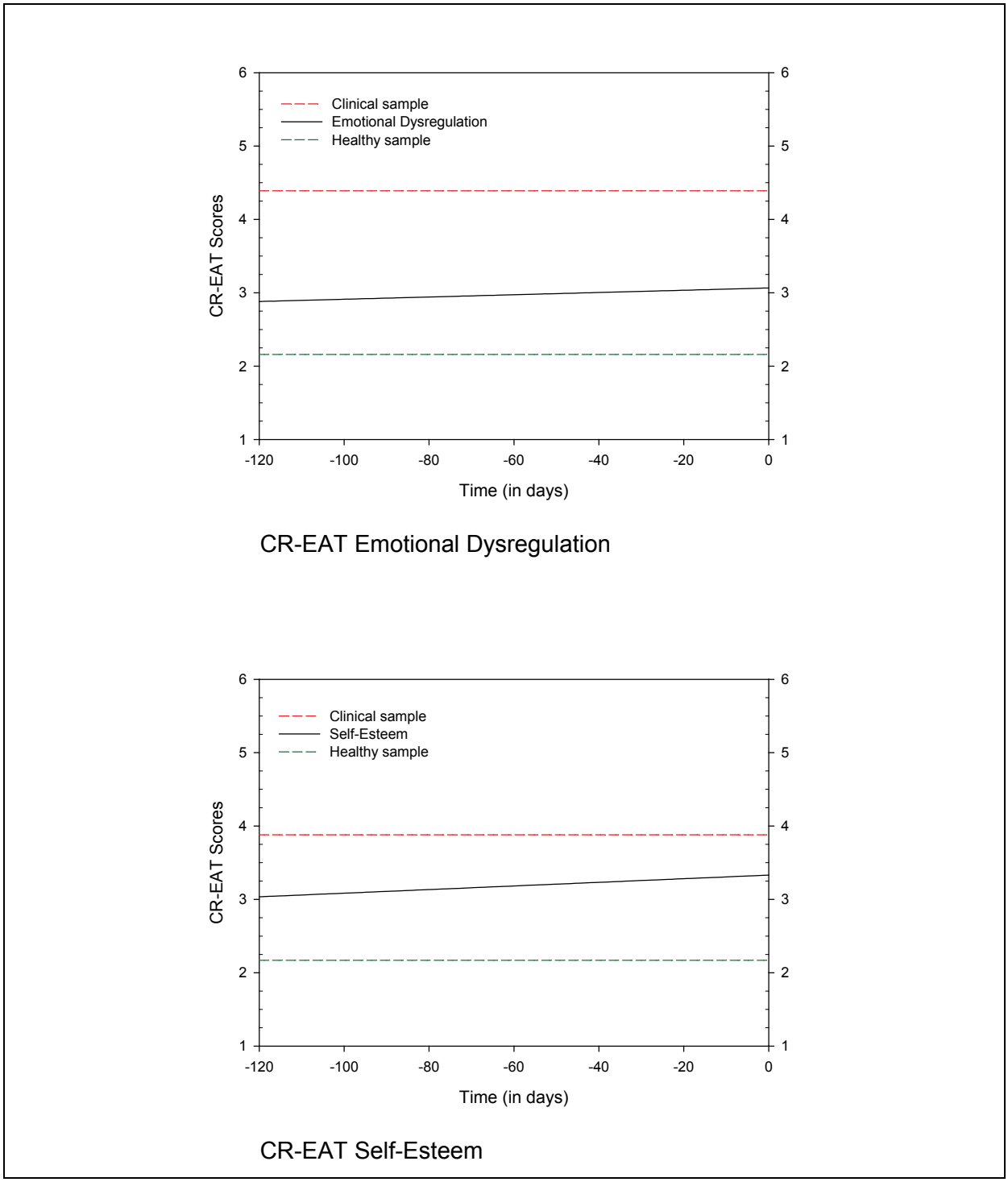
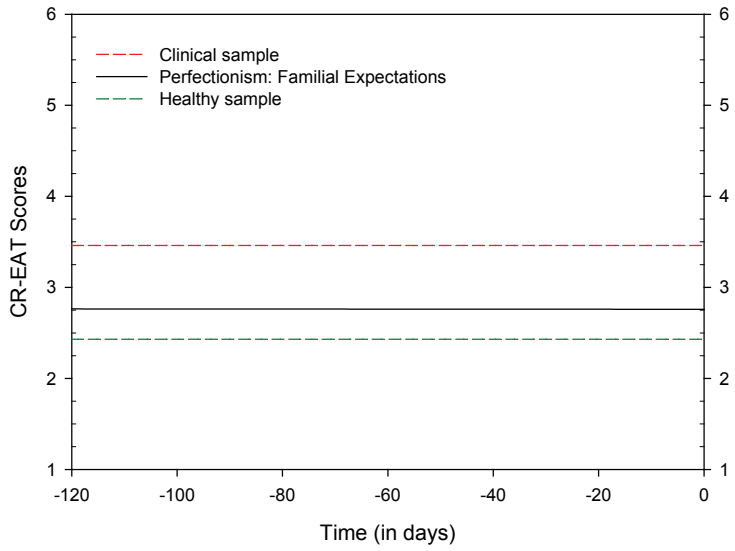
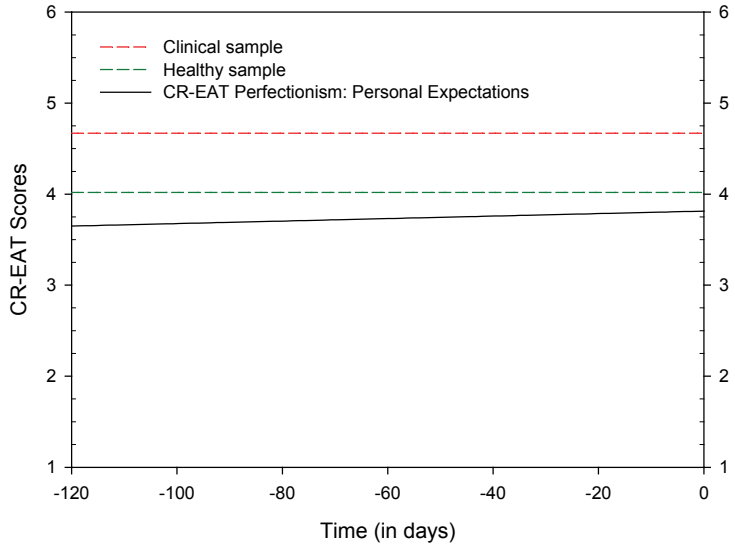


Figure VII.13 Temporal course of the CR-EAT subscales V: References scores



CR-EAT Perfectionism: Familial Expectations



CR-EAT Perfectionism: Personal Expectations

Figure VII.14 Temporal course of the CR-EAT subscales VI: References scores