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EATING PATHOLOGY
A Challenge in School Health Care

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To my family

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EATING PATHOLOGY – A CHALLENGE IN SCHOOL HEALTH CARE
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ABSTRACT

This is a prospective study of self-reported eating disorder symptoms (EDS) in youth. The aim was to enhance the detection of EDS and the use of health records in identifying individuals with the highest risk of EDS.

In the outset, the 8th (n=1036) and 9th (n=855) graders and their school nurses (n=14) in Finnish-language secondary schools in Turku provided data for testing the feasibility of the SCOFF questionnaire in measuring EDS. The 8th grade data were also used to investigate gender differences in EDS. The course of EDS was investigated with a 1-year follow-up of the 9th graders (n=372), and a 4-year follow-up of the initially EDS-positive participants (n=208) and their non-symptomatic counterparts (n=514).

SCOFF proved to be a feasible screen for all mid-adolescents. With SCOFF, the detection of EDS increased fourfold. In mid-adolescence, girl preponderance in EDS was small and associated with appearance and weight dissatisfaction. At the 1-year follow-up, almost one half of initially EDS-positive adolescents were still symptomatic. Prolonged EDS was associated with anxiety. At the 4-year follow-up, one half of the girls and a significant number of boys with EDS in mid-adolescence were still EDS-positive. The strongest predictor of prolonged EDS was depressiveness in mid-adolescence. On population level, the estimated prevalence of protracted EDS was 6%–11% in females and 0.7%–2% in males in late adolescence.

The detection of EDS may be enhanced by including SCOFF in health examinations throughout adolescence. To identify individuals most in need for clinical attention, health records provide useful information.

Key words: eating disorders, screening, questionnaire, adolescence, school nursing

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HÄIRIINTYNYT SYÖMISKÄYTTÄYTYMINEN – HAASTE KOULUTERVEYDENHUOLLOLLE
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TIIVISTELMÄ

Tämä on prospektiivinen tutkimus nuorten itse ilmoittamista syömishäiriöön viittaavista oireista. Tutkimuksen tarkoituksena oli edistää syömishäiriöön viittaavien oireiden havaitsemista ja terveyskertomustietojen hyödyntämistä suurimman syömishäiriöriskin omaavien yksilöiden tunnistamiseksi.

Tutkimuksen ensivaiheessa testattiin SCOFF-syömiskäyttäytymiskyselyn soveltuvuutta syömishäiriöön viittaavan oireilun mittaamiseen. Tutkimukseen osallistuivat Turun suomenkielisten peruskoulujen 8- (n=1036) ja 9-luokkalaiset (n=855) oppilaat ja kouluterveydenhoitajat (n=14). Kahdeksasluokkalaisilta kerättyä aineistoa käytettiin lisäksi kuvattaessa sukupuolten välisiä eroja syömishäiriöön viittaavassa oireilussa. Yhdeksäsluokkalaiset puolestaan kutsuttiin vuoden kuluttua seurantakyselyyn (n=372) syömishäiriöön viittaavan oireilun kulun kuvaamiseksi. Tutkimuksen lopuksi toteutettiin 4-vuotisseurantakysely nuorille aikuisille, joilla tutkimuksen ensivaiheessa oli syömishäiriöön viittaavia oireita (n=208), ja joukolle oireettomia verrokkeita (n=514) syömishäiriöön viittaavan oireilun pidempiaikaisen kulun kuvaamiseksi.

Tulosten mukaan SCOFF soveltui syömishäiriöön viittaavan oireilun seulontaan keskimurrosikäisillä nuorilla. SCOFF:n käyttö nelinkertaisti havainnot syömishäiriöön viittaavasta oireilusta. Keskimurrosikäisillä tytöillä ilmeni syömishäiriöön viittaavaa oireilua hieman enemmän kuin pojilla. Sukupuolten välinen ero oireilussa oli yhteydessä tytöillä yleisempään ulkonäkö- ja painotyytymättömyyteen. Yhden vuoden seurantajaksolla syömishäiriöön viittaava oireilu jatkui lähes puolella tutkimuksen alussa oireilleista nuorista. Oireilun jatkuminen oli yhteydessä ahdistuneisuuteen. Nelivuotisseurannassa syömishäiriöön viittaava oireilu jatkui puolella tytöistä ja merkittävällä osalla pojista, joilla oli kyseisiä oireita jo tutkimuksen alussa. Syömishäiriöön viittaavan oireilun jatkuminen oli todennäköisintä nuorilla, joilla oli masentuneisuutta keskimurrosiässä. Väestötasolla pitkittynyttä syömishäiriöön viittaavaa oireilua arvioitiin esiintyvän 6–11 %:lla myöhäismurrosikäisistä tytöistä ja 0.7–2 %:lla vastaavan ikäisistä pojista.

Syömishäiriöön viittaavan oireilun havaitsemista voidaan edistää käyttämällä SCOFF-syömiskäyttäytymiskyselyä kaikkien murrosikäisten nuorten terveystarkastusten yhteydessä. Terveyskertomustiedoista on puolestaan hyötyä hoitoon ohjausta koskevassa päätöksen teossa.

Avain sanat: syömishäiriö, seulonta, kysely, murrosikä, kouluterveydenhuolto

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ABBREVIATIONS

Add Health	National Longitudinal Study of Adolescent Health
AIC	Akaike's information criterion
ARFID	Avoidant/restrictive food intake disorder
AN	Anorexia nervosa
AN-R	Restricting type of AN
BED	Binge eating disorder
BMI	Body mass index (kg/m^2)
BN	Bulimia nervosa
CBT-A	Cognitive-behavioral therapy adjusted for adolescent BN
CFA	Confirmatory factor analysis
CI	Confidence interval
df	Degrees of freedom
DSM	Diagnostic and Statistical Manual of Mental Disorders
ED	Eating disorder
EDNOS	Eating disorders not otherwise specified
EDS	Eating disorder symptoms
EDS1	Self-reported eating disorder symptoms only at baseline
EDS12	Self-reported eating disorder symptoms at baseline and follow-up
EDS2	Self-reported eating disorder symptoms only at follow-up
GUTS	Growing Up Today Study
ICC	Intra-class correlation
ICD	International Classification of Diseases
KR-20	Kuder Richardson formula 20
LCI	Lower level of 95% confidence interval
LR–	Negative likelihood ratio
LR+	Positive likelihood ratio
MLCH	Mannerheim League for Child Welfare
noEDS	No self-reported eating disorder symptoms at baseline or follow-up
NPV	Negative predictive value
OR	Odds ratio
p	*p*-value
PBI	Parental Bonding Instrument
PPV	Positive predictive value
OSFED	Other specified feeding or eating disorders
Project EAT	Eating Among Teens project
r	Correlation coefficient
Raine	Western Australian Pregnancy Cohort Study
RBDI	Raitasalo's modification of the Beck Depression Inventory
RMSEA	Root-mean-square error of approximation
SCOFF	Acronym of the words 'sick', 'control', 'one', 'fat', 'food'
SD	Standard deviation
Se	Sensitivity
SES	Socioeconomic status
Sp	Specificity
T1	Baseline assessment in mid-adolescence
T2	Follow-up assessment in late adolescence
UCI	Upper level of 95% confidence interval

UFED	Other unspecified feeding or eating disorders
WHO	World Health Organization
χ^2	Chi-square

LIST OF ORIGINAL PUBLICATIONS

This thesis is based on the following original publications, which are referred to in the text by the corresponding Roman numerals I–IV. In addition, some unpublished findings are presented.

I   Hautala L, Junnila J, Alin J, Grönroos M, Maunula AM, Karukivi M, Liuksila PR, Räihä H, Välimäki M, Saarijärvi S. Uncovering hidden eating disorders using the SCOFF questionnaire: cross sectional survey of adolescents and comparison with nurse assessments. International Journal of Nursing Studies 2009;46:1439–1447.

II   Hautala L, Junnila J, Helenius H, Väänänen AM, Liuksila PR, Räihä H, Välimäki M, Saarijärvi S. Towards understanding gender differences in disordered eating among adolescents. Journal of Clinical Nursing 2008;17:1803–1813.

III Hautala L, Junnila J, Helenius H, Väänänen AM, Liuksila PR, Räihä H, Välimäki M, Saarijärvi S. Adolescents with fluctuating symptoms of eating disorders: a 1-year prospective study. Journal of Advanced Nursing 2008;62:674–680.

     Corrigendum. Journal of Advanced Nursing 2013;69:981.

IV   Hautala L, Helenius H, Karukivi M, Maunula AM, Nieminen J, Aromaa M, Liuksila PR, Räihä H, Välimäki M, Saarijärvi S. The role of gender, affectivity and parenting in the course of disordered eating: a 4-year prospective case-control study among adolescents. International Journal of Nursing Studies 2011;48:959–972.

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1   INTRODUCTION

Eating disorders are among the most challenging mental illnesses in adolescence [1]. In Finland, eating disorders are one of the most common primary diagnoses of 13–17-year-old patients in specialized psychiatric care [2]. In Finnish population, approximately 2% of females [3] and 0.2% of males [4] are diagnosed with anorexia nervosa (AN). The prevalence of bulimia nervosa (BN) is at the same level for females [5] and at 0.1% for males [6]. Due to the strong female preponderance, eating pathology is sometimes considered as a female problem [7]. However, various eating disturbances apart from full-blown eating disorders are rather common among both genders in youth. For example, loss of control over eating is experienced by 15% of girls [8], and unhealthy weight control behaviors such as fasting or self-induced vomiting are reported for 12% and 10% of girls, respectively [9]. Among boys, the occurrence of these problems is somewhat lower (9% [8], 10% and 7% [9], respectively). One possible explanation for the gender difference is suggested to be due to the female orientation in the diagnostic criteria of eating disorders [7] and consequently, in instruments measuring eating disorders. Gender-specific questions may give an impression that eating pathology is specific to girls, thus increasing the risk of denial and non-response among boys. Another explanation might be found in the different ways girls and boys are exposed to the risk factors of eating pathology. Despite the large body of literature, reasons for the gender disparity in the occurrence of eating pathology are not well known.

Eating pathologies typically emerge in adolescence [10] in the form of abnormal behaviors and attitudes related to eating and weight. In many cases, the problems remain temporary and emerge as isolated episodes during adolescence. However, sometimes pathological eating becomes a persistent part of the individual's life, causing significant impairment to one's health and functioning, and even to the well-being of the whole family. Typically, the progression from prodromes into a full-blown eating disorder takes at least several months, which allows for early detection and intervention to steer the adolescent's development from a detrimental path to a healthy one. Unfortunately, eating disturbances are difficult to detect and only a minority of those suffering from these problems receive adequate and timely clinical attention [3; 5]. To improve the situation, school health care professionals are encouraged to pay special attention to the possible signs and symptoms of eating pathologies in youth [11; 12; 13]. To identify those who are at risk or already have the problem screening for eating disorders is recommended [14; 15].

Screening may help to detect eating pathology, but a key question is in which cases the problem is likely to continue if no intervention takes place. The multifactorial nature of eating disturbances is well recognized. However, we still have limited understanding about the role and effects of the factors associated with the course of eating pathology. Today, most of the evidence from prospective studies concerns the onset of eating pathology. Female gender and adolescence are consistently rated as risk factors for eating disturbances. The findings are less consistent in terms of other factors, e.g., body mass index (BMI), weight concerns, negative emotionality, and family relationships. [for a review, see 16.] In addition, there is scarce evidence concerning the factors associated with the maintenance of eating pathology. Community-based studies of the topic are few, and thus, the characteristics of adolescents with prolonged eating problems are not well known.

Taken together, more evidence is needed to shed light on the gender differences and the predictors of the course of eating pathology. In addition, we lack evidence of the utility of

screening for eating pathology within Finnish school health care. The primary aims of extending the research of eating pathology to cover the health examinations of students were to enhance the detection of eating disorder symptoms (EDS) in adolescent population and to explore the possible benefits of the use of health-record information for school health care personnel for the purpose of identifying individuals most in need for intervention. Thus, the overall orientation of this study was to provide evidence-based information for preventive health care. In other words, the nature of this study was preventive with the focus on primary prevention. School health care was seen as an ideal setting for the study because of annual health examinations of all adolescents attending compulsory education and the resulting extensive health care records. To enhance the understanding of the development of eating pathology, the study searched for prospective evidence on the course of EDS from middle to late adolescence and the contributing factors as implicated in the previous research of eating pathology.

2   REVIEW OF THE LITERATURE

2.1   School health care

In Finland, all youth attending basic education are entitled to school health care as part of the free student welfare services. School health care includes: 1) annual health examinations, 2) oral health care, 3) early identification of children and youth with a need for special support or testing, and referral to treatment if necessary, 4) special examinations required to ascertain an individual's health status, 5) support for the parents, and 6) health and safety inspections of the school environment. [17.]

Municipalities are responsible for the provision of school health care as stipulated in the Health Care Act (1326/2010) [17] and the Government Decree on maternity and child health clinic services, school and student health services and preventive oral health services for children and youth (380/2009, updated 338/2011) [18]. Other laws and regulations governing school health care services are numerous [19]. For example, according to the Act on the Status and Rights of Patients (785/1992) [20], depending on the age or level of development, a minor person has the right to forbid the health care personnel from disclosing health-related information to the parents or guardians. The Child Welfare Act (417/2007) [21] states that schools must be provided with psychologist and social worker services to support the school attendance of children and youth and to prevent and eliminate difficulties affecting their development. The Basic Education Act (628/1998) [22] declares that the student welfare services shall be provided in cooperation with students and their parents. The Student Welfare Act (1287/2013) [23], in turn, states that the school nurse's working hours shall be arranged to provide students with access to services during school days without appointment. The number, content and scope of health examinations are regulated by the Government Decree (380/2009, updated 338/2011) [18], whereas more detailed instructions for the school health care are given by the Ministry of Social Affairs and Health [11; 19; 24] and the National Institute for Health and Welfare [25; 26].

The foundation for Finnish school health care was laid in the 1920s when a civic organization, the Mannerheim League for Child Welfare (MLCH), started the systematic education of public health nurses. In the 1930s, the education was passed on to the State. However, the MLCH continued supervising school nurses up to 1944, when the health care of school-age children was assigned to municipalities on the basis of the Act on the Public Health Nurses. In the early decades of school health care, the main interest was on physical health, infections, hygiene, and the nutritional status of pupils. Emphasis was put on the prevention and screening of diseases as well as nursing services as part of health care for the whole family. Cooperation with parents was realized through home visits. In the 1970s, the focus shifted from physical health to psychological well-being of children and youth with a more individual orientation. [27.]

Nowadays, school health care services contribute to both the physical and psychosocial health of children and youth, and special attention is paid to the early detection of mental health problems. Annual health examinations are performed by the school nurses. More extensive health examinations, conducted jointly by the school nurse and the school doctor, take place at the beginning of the school (1st grade), in early adolescence (5th grade), and in mid-adolescence (8th grade) [24]. The compulsory basic education age is from 7 to 16 years, the usual school starting age is 7 years, 5th graders are 11–12 and 8th

graders are 14–15 years old. During the extensive health examinations, the well-being of the whole family is assessed and strengthened in close cooperation with the student and the parents [24; 28]. In addition to providing support for an individual's health, development and well-being, the health examinations aim at primary and secondary prevention by means of screening [29]. Regular health examinations offer school health care personnel opportunities to detect the early signs and symptoms of various health-related problems and identify the needs that children and youth may have [24]. This facilitates early interventions, which sometimes are enough in preventing eating pathologies [15] from taking a protracted course that would have a detrimental influence on an individual [30; 31] and the whole family [32]. However, the benefits of health examinations depend on the accuracy and timing of detection and treatment – the sooner, the better [33].

2.2 Eating pathology

2.2.1 Terminology

The first medical report of a condition now recognized as an eating disorder, was published in the 17th century by English physician Richard Morton [34] who described 'nervous consumption' as a physiological disorder characterized by extreme underweight and fear of weight gain, which now is seen to resemble anorexia nervosa. [35; 36.] The first clinical descriptions of eating disorders are from the 19th century, when French psychiatrists Louis-Victor Marcé [see 37] and Ernest C. Laséque, and British physician William W. Gull independently published reports of anorexia [38]. Marcé [39] defined anorexia as a form of 'hypochondriacal delirium', and Laséque [40] characterized it as a 'nervous disease'. The term 'anorexia nervosa' was coined by Gull [41], while Laséque [40] used the term 'hysterical anorexia'. The word 'anorexia' is of Greek origin and it means lack of appetite [42].

The concept 'eating disorder' was introduced in scientific articles in the 1960s by Hilde Bruch [43] who used it to indicate pathologic weight disturbances, such as AN and obesity. Later, eating disorders have become a diagnostic category that includes several syndromes. The best known are anorexia nervosa and bulimia nervosa, the latter of which is characterized by repeated binge eating and inappropriate behaviors to counteract its weight increasing influence. Binge eating was first characterized by Stunkard [44] as a 'pattern of overeating with orgiastic quality' in which enormous amounts of food are consumed in relatively short periods, followed by severe discomfort and expressions of self-condemnation. The first description of the development of a syndrome where dieting, binge eating, and purging are interrelated was provided in the 1970s by American psychologist Marlene Boskind-Lodahl [45] who named it as 'bulimarexia'. However, the credit of introducing the syndrome as a clinical entity called 'bulimia nervosa', is granted to British psychiatrist Gerald Russell [46], who described BN as 'an omnious variant of AN'. The word 'bulimia' derives from Greek and it means ravenous hunger [42].

Despite the large body of literature generated by researchers and clinical experts over several decades, an unambiguous definition of the concept of 'eating disorder' is still missing [47]. General and medical dictionaries define eating disorder(s) as presented in Table 1.

Table 1. Dictionary definitions of the concept of eating disorder

Dictionary	Definition of eating disorder
General	
The American Heritage® Science Dictionary [48]	Any of several patterns of severely disturbed eating behavior, especially anorexia nervosa and bulimia, seen mainly in female teenagers and young women
Encarta® World English Dictionary [49]	Any emotional disorder, e.g., bulimia, that manifests itself in an irrational craving for or avoidance of food.
Macmillan Dictionary [50]	A medical condition such as anorexia or bulimia in which someone tries to control their weight in a way that makes them ill
Cambridge Advanced Learner's Dictionary [51]	A mental illness in which people eat far too little or far too much food and are unhappy with their bodies
Medical	
The American Heritage® Stedman's Medical Dictionary [52]	A potentially life-threatening neurotic condition, such as anorexia nervosa or bulimia, usually seen in young women
The Merriam-Webster's Medical Dictionary [53]	Any of several psychological disorders (as anorexia nervosa or bulimia) characterized by serious disturbances of eating behavior
National Institute of Mental Health [54]	Serious behavior problems. They include Anorexia nervosa, in which you become too thin, but you don't eat enough because you think you are fat; Bulimia nervosa, involving periods of overeating followed by purging, sometimes through self-indused vomiting or using laxatives; Binge-eating, which is out-of-control eating…
National Library of Medicine [55]	A group of disorders characterized by physiological and psychological disturbances in appetite or food intake

As these definitions show, there are various ways of understanding eating disorders ranging from behavioral problems through mental illnesses to a potentially life-threatening condition. The definitions tend to equate eating disorders with AN and BN, which are categorical diagnoses based on an expert opinion of the presence of the disorder, as described in the International Classification of Diseases (ICD) published by the World Health Organization (WHO), or the Diagnostic and Statistical Manual of Mental Disorders (DSM) published by the American Psychiatric Association (APA). In the ICD, the term 'eating disorder' was used for the first time in ICD-9 [56], but without an explicit definition. The diagnostic criteria of eating disorders were described in the next revision, ICD-10 [57], which includes three broad categories: AN, BN, and an atypical eating disorder, which

comprises atypical forms of AN and BN, overeating and vomiting associated with other psychological disturbances, other eating disorders, and unspecified eating disorders. In accordance with the general consensus on the multidimensional nature of these syndromes [58; 59], the category of eating disorders is included in 'Behavioral syndromes associated with psychological disturbances and physical factors'.

In the DSM, the term 'eating disorder' was first used in DSM-III [60] together with the symptom-based criteria of disorders. Prior to this, the symptoms did not denote disease entities, but were seen as manifestations of broad underlying conditions or reactions to problems in life [61]. DSM-III [60] included the criteria of AN, bulimia, pica, rumination disorder of infancy, and atypical eating disorders; in later revisions the last one was further named as eating disorders not otherwise specified (EDNOS), indicating a residual category for eating disorders of clinical severity that do not meet the criteria for AN or BN [62]. In DSM-IV [63] and its text-revision, DSM-IV-TR [64], the category of 'Eating Disorders' consisted of 'severe disturbances in eating behavior', i.e., AN, BN, and EDNOS, including the preliminary criteria for binge eating disorder (BED), whereas 'persistent feeding and eating disturbances' including pica, rumination disorder, and feeding disorder of infancy or early childhood were separated into the category entitled 'Disorders usually first evident in infancy, childhood, or adolescence'. Today, in DSM-5 [65], the two categories are combined and named as 'Feeding and Eating Disorders', including three primary eating disorders: AN, BN, and BED, and three feeding disorders: pica, rumination disorder, and avoidant/restrictive food intake disorder (ARFID). In addition, it includes the preliminary criteria for other specified as well as unspecified feeding or eating disorders (OSFED and UFED).

In sum, each revision of the ICD and DSM has presented some modifications to the previous one with more or less changing criteria for eating disorders. As an example of this, Loeb and co-writers [66] present that binge eating without inappropriate compensatory behaviors has resulted in the diagnosis of BN in DSM-III [60], no clear diagnosis in DSM-III-R [67], and the diagnosis EDNOS in DSM-IV-TR [64]. Today, in DSM-5 [65], BED is a full-standing diagnosis of its own category. On one hand, this reflects the evolution of knowledge about eating disorders. On the other hand, it indicates the incompleteness of these classifications which are not fully evidence-based, but based on experts' opinions [68; 69]. Thus, one can agree with the notion stated already 30 years ago: "*…we are still fumbling for appropriate definitions of 'caseness' for the eating disorders…*" [70].

In spite of the instability of the categorical classification system, assessing eating disorders on the basis of the diagnostic criteria may be warranted in, for example, clinical trials. However, in population-based epidemiological studies, such classification systems may be less useful [e.g., 71]. In the community, there is a wide range of eating disturbances that fall out of the diagnostic categories of eating disorders [72; 73], particularly in adolescents [74]. This has raised a concern that the formal diagnostic criteria and thresholds may not adequately capture eating pathologies and be applicable for adolescents, in particular [74; 75]; sub-syndromes in adolescence may actually be eating disorders in evolution or even 'age-specific manifestations of full diagnosis' [76]. Therefore, a broader definition of eating disorders and lower thresholds of symptom severity are recommended for application in adolescent populations [77; 78; 79].

In the child and adolescent version of the diagnostic and statistical manual for primary care (DSM-PC) [80] this has been taken into account by adopting the continuum hypothesis [see 81; 82] approach to adolescents' eating-related behaviors and cognitions, such as

dieting, body image problems, purging, and binge eating, in relation to AN, BN and EDNOS. The basic assumption is that the symptoms demonstrated by adolescents vary along a continuum from normal developmental variations through problems to disorders. Developmental variations are within the range of expected behaviors for the age, although they may raise concern in parents. Problems reflect behaviors that are serious enough to disturb the individual's growth, development, and social functioning. They may be resolved with short-term interventions within primary health care or by referral to mental health care. Disorders, in turn, cause clinically significant distress or impairment and, in most cases, warrant referral to mental health care. [80.]

Some definitions reflect the broad spectrum of eating disturbances associated with harmful consequences to an individual. For example, Fairburn and Walsh [83] define an eating disorder as *"a persistent disturbance of eating behavior or behavior intended to control weight, which significantly impairs physical health or psychosocial functioning".* Similarly, the Finnish Current Care Guideline [15] defines eating disorders as mental disorders that are most typical in youth and young adult females showing abnormal eating behavior with impairment in mental, physical or social functioning. As the causal relationship between eating pathology and associated impairment often remains unknown due to the methodological limitations of the research, the term 'eating disorder' is mostly used to refer to abnormal concerns and behaviors related to eating and weight [84]. Researchers use also other terms such as: 'subthreshold disorder', which indicates a situation where all the features of eating disorders are present but not severe enough to reach the defined diagnostic threshold of a full syndrome; 'partial syndrome', where one [83] or several [31] of the diagnostic features of AN or BN are missing; 'subclinical eating disorder', which refers to situations when more than one of the diagnostic criteria of AN or BN are not fulfilled, thus corresponding to 'partial syndrome' [31]; 'prodrome', which is an early symptom or cluster of symptoms that indicate the initiation of onset of an eating disorder; 'eating disorder symptom', which refers to the defining features of eating disorders and, in surveys, is typically used in composites to indicate a broader spectrum of disordered eating than the DSM eating disorders; 'eating pathology', 'disordered eating' [85], and 'eating disturbances' [72], which are used in the broad sense of abnormal eating behavior. In the end, the purpose of an individual study dictates the terms and definitions to be used.

In this study, the term 'eating disorder symptoms' (EDS) was chosen to reflect the core features of AN and BN. The definition of EDS adopted in this study covers eating- and weight-related behavioral and attitudinal problems, such as intentional vomiting, loss of control over eating, weight loss, body dissatisfaction, and food intrusive thoughts. Other terms used here in the same meaning are 'disordered eating', 'eating disturbance', and 'eating problems'. The term 'eating pathology' is used for all abnormal or risky behaviors and attitudes related to eating and weight including full-blown eating disorders.

2.2.2 Theories and models of eating pathology

This section provides an overview into some theories and models of the etiology of eating pathology. The section is divided into individual, environmental, and integrated approaches on the basis of the main factor(s) emphasized in each theory. In the individual approach, the focus is on physiological, psychological and/or behavioral factors. The environmental approach focuses on family and culture as the proximal and distal environment, respectively. The integrated approach includes models with factors from both individual and environmental domains, thus representing a broad perspective to eating pathology. Advances in biological knowledge have contributed to the development of models that

focus on genetic heritability, epigenetic changes to DNA, and alterations in brain function. However, these are beyond the scope of this study and, therefore, biological models are excluded here.

Individual approach

In the 1970s and 1980s, Herman and colleagues [86; 87] described the association between restraint (dieting) and unrestraint (binge) eating, which has thereafter been called the restrained model [88]. The model suggests that restriction of dietary intake decreases sensitivity to internal cues for hunger and satiety and increases reliance on situational cues for eating. Relying on cognitive control instead of physiological cues increases the risk for uncontrolled eating in situations when cognitive control is disrupted by so-called 'dis-inhibitors', for example emotional stress. Thus, dieting may result in an elevated risk for binge eating. Binge eating episodes, in turn, may lead to increased dietary efforts and purgative techniques, such as vomiting and abuse of laxatives, which further increase the risk for subsequent binge eating, hence resulting in the maintaining of binge-purge cycles. [87.] The restraint model has influenced the development of later ideas about the etiology, treatment, and prevention of eating disorders, e.g., the dual pathway model of bulimic pathology [89].

Another model focusing on eating regulation on physiological bases was developed by Bennett [90] with an emphasis on so-called biologically determined 'set point', first presented by Nisbett [91] in his model of obesity. The set-point model suggests that each individual has a biologically dictated set point weight that the body strives to achieve and maintain by regulation of metabolic rate, physical activity and food intake. The set point is not permanent, but is modulated over time by several factors, for example, normal development (e.g., puberty, pregnancy), the intake of chemical regulators (e.g., appetite suppressants), and the physical activity habits. However, attempts to lower the set point by prolonged dieting typically fail, and what is more, dieting tends to lead to the loss of normal physiological signals of hunger and satiety. [90.] Thus, according to the set point model, binge eating may be the body's natural response to the individual's weight-loss attempts [92].

In the 1980s, the over-concern about shape and weight and the related control behaviors were given an important role in the cognitive behavioral theory of AN [93; 94; 95] and BN [94; 96]. In this theory, the onset of an eating disorder is based on the individual's need for self-control, which originates in low self-esteem and perfectionism. These persons evaluate their self-worth mainly in terms of eating, shape and weight. Over-evaluation of these features leads to extreme dieting and strict food restrictions. [96; 97.] In AN, successful weight loss with an enhanced sense of self-control and self-worth links the person's identity with the disorder in a way that resists later attempts to behavioral change [93; 95]. At some point, however, the weight loss declines. This may be perceived as an evidence of failure, which leads to the use of more severe weight control techniques, e.g., excessive exercise, vomiting, and the misuse of laxatives or diuretics. [95.] In many cases, the behavior rules and dietary restrictions become impossible to keep. This typically leads one to abandon all control over eating, with a result of a binge eating episode. The risk for binge eating is increased particularly when in negative mood; binge eating may be used as a means to decrease negative affect. However, the relief gained by binge eating is temporary. After a binge eating episode, concerns about self-control over eating, shape and weight increase and reinforce the harmful weight loss behaviors. [96.] These, in turn, lead to further binge eating episodes, thus creating a self-perpetuating vicious circle

between binge eating and compensatory behaviors which are found both in BN [96] and the purging type of AN [95]. In this theory, overloaded concerns about and control over eating, weight, and shape are the factors that maintain disordered eating [96; 97].

The cognitive behavioral theory was the leading evidence-based theory of the maintenance of eating disorders up to the end of the 20th century [98]. However, the unsatisfactory results of the therapies based on this theory raised concerns that the theory might be too narrow because it focuses mainly on the eating disorder-specific psychopathology and draws only little attention to other circumstances. This encouraged Fairburn and colleagues [98] to develop the trans-diagnostic theory of eating disorders, which extends the previous theory by providing a broader view of the factors that maintain AN, BN and EDNOS. As in cognitive behavioral theory, over-evaluation of and control over eating, weight and shape play the central role in the trans-diagnostic theory. Beside these, the theory includes four additional factors that are suggested to contribute to the maintenance of eating disorders. These are clinical perfectionism, core low self-esteem, mood intolerance, and interpersonal difficulties. The theory suggests, first that individuals with clinical perfectionism judge their self-worth mainly on the basis of high-standard achievements in several domains of life, including control over eating, shape and weight. They tend to evaluate their performance negatively and are afraid of failure. The negative self-evaluation encourages them to strive harder for the self-made goals and self-control, which results in maintaining eating disorder. Secondly, core low self-esteem as a part of the individual's identity increases the sense of hopelessness and the resistance against behavior change attempts. Thirdly, mood intolerance, reflecting poor coping skills with intensive emotional states, e.g., anger, anxiety, and depression, leads to 'dysfunctional mood modulatory behaviors', such as binge eating and compensatory behaviors. These can become habitual means for neutralising mood states, which further maintains the problematic behavior. Fourthly, difficulties in interpersonal life undermine the individual's self-esteem and further increase the need for self-control with a result of maintaining eating disorder. The applicability of the trans-diagnostic theory to the full range of eating disorders is suggested on the basis of the findings that AN, BN and EDNOS share the core psychopathology; individuals typically migrate across these diagnostic categories over time, and the shared clinical features of these categories tend to be maintained by similar processes. [98.]

Environmental approach

For several decades, the etiological explanations of eating pathology focused on family. Eating disorders were believed to originate in the early parent-child relationship and were interpreted in relation to sexuality as suggested by the psychoanalytic approach [see 99]. For example, AN was seen as a rejection of femininity and a fear of 'oral impregnation' with the symbolism of the gastrointestinal tract as a womb, eating as impregnation, getting fat as becoming pregnant, and constipation as a child in the abdomen. Amenorrhea was seen either as a reflection of the pregnancy fantasies or as a proof of an attempt to reject femininity. [100.] In the 1960s and 70s, these symbolic interpretations were increasingly criticized for unproven etiologic concepts, diagnostic unreliability, and the lack of efficacy of treatment [101]. Adopting a critical view to the psychoanalytic approach, Minuchin and colleagues [102] developed a conceptual model of psychosomatic illness in children, called the open system family model or the Minuchin model. This challenged the earlier tendency to focus on changing the individual alone, and separate one from the environmental influence, i.e., from the family. In the Minuchin model, the focus turned to the family processes that were believed to trigger and maintain severe psychosomatic

problems such as eating disorders. In the model, Minuchin describes the conditions that he suggests as being necessary for the development and maintenance of psychosomatic symptoms in children. First, the individual has 'vulnerability' to transform emotional conflicts into somatic symptoms like AN. Second, the family has a so-called 'psychosomatic family' organization, which is characterized by transactional enmeshment, overprotectiveness, rigidity, and lack of conflict resolution. Third, the child's symptoms maintain family homeostasis, increase parental control and protection, and increase the sense of dependency and incompetence in the offspring, thus creating an autonomous process that maintains eating disorder symptoms.

In the 1980s, clinicians at the Maudsley Hospital in London questioned the family process as a cause of eating disorders [103]. The view of a dysfunctional family being responsible for an adolescent's illness was rejected and replaced by the new idea where the illness was externalised outside of the family and personified as an outsider that, by taking control over the adolescent, has a negative influence on the whole family. The Maudsley model adopted an agnostic approach to the etiological factors of eating disorders, released parents from the feelings of guilt and criticism of each other and the adolescent, and encouraged them to conjoin to enhance the recovery process. Thus, the focus shifted to seeing the parents as a resource to resolve the eating problem rather than causing or being the problem itself. [104.]

An increasing interest in the influence of culture led to the development of sociocultural models, which emphasize the importance of environmental pressures to thinness in the development of eating disorders [105; 106; 107]. Body image was given a central role in AN already in the 1970s. Since then, the pursuit of thinness has been seen as the female's response to the conflict that she experiences when the environmental expectations are not in harmony with her identity. [108.] In Western like cultures, females are exposed to environmental pressures, which declare that a thin body image and a slender appearance are central to the female's role and success in society. The likelihood of a female to internalize these ideas increases during identity confusion, especially in individuals with low self-esteem and when the pressures are transmitted by family, peers and the media. Internalizing an extremely thin body ideal results in an experience of a discrepancy between the ideal and self and leads to body dissatisfaction, especially in heavier females. [105; 106; 107.] Body dissatisfaction, in turn, may result in restraint eating and negative affect, which in some individuals lead to overeating, followed by increased body image concerns and further compensatory behaviors [105; 107], and BN [106].

In the 1990s, Fredrickson and Roberts [109] developed the objectification theory to provide a broader understanding of female experiences and mental health risks, including eating disorders. The objectification theory argues that our culture socializes females to internalize an observer's perspective as a primary way to view and judge themselves in terms of appearance. This 'self-objectification', characterized by preoccupation with physical appearance, is viewed as a survival strategy that helps the female to assess how others will view and treat her, thus contradicting the typical interpretation of body checking as narcissism. The habitual checking and comparisons between the individual's own appearance and the cultural beauty ideal create the feelings of shame and anxiety, and decrease the quality of life. In addition, they are suggested to decrease the person's sensitivity to internal bodily cues. These negative experiences and affects, together with the cultural messages telling that body weight and shape are the matter of the individual's own choice, provoke disturbed attitudes toward food, and promote restraint and disordered eating. Thus, the objectification theory sees eating disorders as pathological strategies,

which reflect the female's lack of power to better control the objectification of her own body. [109; 110.]

Integrated approach

The approaches to integrate all sorts of factors that contribute to eating disorders are called biopsychosocial models [111]. The multidimensional approach to eating disorders was adopted in the 1980s [108]. Since then, several versions of biopsychosocial models have been developed, each of which differ from the next to some extent [111].

In the 1980s, Johnson and Maddi [112] presented a model of bulimia as a result of an interrelationship of biological, familial and sociocultural factors. The model suggests that young females who have a biological vulnerability to affective instability, a conflictual family background, and who live in a confusing sociocultural environment that emphasizes achievement and thinness, tend to experience difficulties in self-esteem and self-regulation. A belief that thinness is a solution to these difficulties leads to dieting which, in contrast to expectations, exacerbates affective instability and low self-esteem. In this situation, any life stressor may lead to disinhibited eating in order to regulate tensions, to get a temporary relief from over-controlling or self-sacrificing activities, or to express opposition. To avoid weight gain, binge eating is followed by purging behaviors, which, in turn, reinforce disturbed eating behaviors by allowing both restraint eating and binge eating for an individual striving for thinness. Finally, the binge-purge cycle is suggested to turn from a means used by the individual to control her life to a process that controls over the individual and further worsens her affective instability and self-esteem.

In the 1990s, Heatherton and Polivy [113] offered the spiral model as a pathway from dieting through psychological distress to the development of eating disorders. The model is in line with the restraint model [86; 87] but acknowledges also the importance of affective factors. The spiral model suggests that, in adolescence, the individual starts to compare one's own body to the ideal in the given culture, becomes dissatisfied with one's own body, and undertakes dieting in order to attain the ideal weight and shape. Unsuccessful dieting leads to a negative affect and lowered self-esteem, which further increase the likelihood of a failure in future dieting attempts, thus creating a spiral of the heightened negative affect, more extreme dieting, more severe binge eating and purging, and finally the onset of an eating disorder.

Another model from the 1990s is the dual pathway model of bulimic pathology, [89; 114], which is based on the synthesis of the socio-cultural [see 105], dietary restraint [87], and affect regulation [115] approaches to eating disorders, and has some similarities also with the spiral model [113]. According to the dual pathway model, sociocultural pressures towards a thin body ideal and internalizing this ideal contribute to body dissatisfaction, which may lead to the onset of bulimic pathology via two pathways. First, body dissatisfaction contributes to the negative affect, which may lead to bulimic symptoms due to the common belief that eating alleviates negative emotions. Second, body dissatisfaction contributes to dieting as a means to control weight, which, however, often remains unsuccessful, causes negative affect, and increases the risk for bulimic pathology, because both caloric deprivation and breaking the strict dietary rules contribute to disinhibited eating. Thus, the dual pathway model proposes that dieting and negative affect are the proximal predictors of the onset of bulimic pathology. The effects of other risk factors are suggested to be mediated by these two factors. [89; 114.]

One of the latest models, the cognitive-interpersonal model of the maintenance of the restricting type of AN (AN-R), developed by Schmidt and Treasure [116], is based on the assumption of a distinct and separate phenotype of AN-R. Thus, it differs from the trans-diagnostic theory of eating disorders [98], which highlights the similarities in AN, BN, and EDNOS. In addition, unlike many other models, the cognitive-interpersonal model does not emphasize the role of weight- and shape-related factors in maintaining AN-R and suggests that AN-R is not a culture-bound syndrome, but has adaptive and defensive functions in reducing social threats. Essentially, AN-R is suggested to include several possible motivations for restraint eating, e.g., inappetence, ascetic and religious ideals, as well as weight and shape concerns. The model presents four domains of maintaining factors: pro-AN beliefs (e.g., AN helps in managing emotions), emotional style (e.g., avoidance), thinking style (e.g., rigidity), and interpersonal factors (e.g., reassurance). It is hypothesized that in the early stages of AN-R, dietary restrained is maintained by the positive consequences of starvation (e.g., temporary improvement in mood and well-being, the feelings of control and mastery), which may be moderated by vulnerability due to unknown biological factors. Later on, as starvation becomes a chronic state, eating leads to unpleasant consequences such as constipation, bloating, and nausea. In addition, eating anything outside the limited range of 'safe foods' causes negative emotions, although at the same time, starvation provokes an intense preoccupation of food and eating. When resisting the drive to eat, the individual focuses exclusively on food and eating and becomes somewhat numb to any emotions. This leads to pro-AN belief that AN-R helps in managing emotions. The interpersonal factors, i.e. the reactions of close others, may reinforce these behaviors and beliefs in several ways. First, compliments about weight loss may encourage the individual to further restrict food intake and lead to pro-AN beliefs about being attractive and special due to AN-R. As individual's emaciation proceeds, it typically provokes negative emotions, criticism, and overprotection in family members, especially in the parents, who may feel helplessness and blame themselves for causing the illness. These highly expressed emotions of family members may be perceived as dangerous and intolerable by the individual with AN-R, thus fostering self-starvation as a defence mechanism to avoid negative emotions. Alternatively, the illness may lead the family to organize its functions around the needs of the individual with AN. This, in turn, may maintain the illness by creating a pro-AN belief, that due to AN-R the person attains more care and attention than she would get without the illness. Thus, the intra-individual and interpersonal factors interact in several ways which tend to maintain AN-R. [116; 117.]

In sum, the etiology of eating pathology has been explained by various theories and models each of which have their pros and cons. The models focusing on physiological and cognitive factors present a commonly accepted notion of eating pathology accompanied by physiological consequences. However, these models do not explain why the cognitive factors begin to dominate some individuals' behavior in a way that disturbs their normal physiology. This question remains unanswered also in the cognitive behavioral theories, which, served as the best evidence-based theories for many years [98]. These theories propose that maladaptive cognitions, thoughts and beliefs contribute to the onset and maintenance of eating pathology, but they do not give a thorough explanation for the occurrence of eating disorders, in other words, why some persons choose disturbed eating rather than some other means to cope with their negative emotions [118]. The models focusing on environmental factors explain the etiology of eating pathology by social influences; eating problems occur as a reaction to experiences perceived in the proximal and/or distal environment. Social events and circumstances are also seen to maintain eating pathology. Although these models broaden the view of the potential risk factors of

eating pathology, there still remains the question why only some individuals of all those exposed to the same environmental forces react through eating disturbance, while others do not. Integrated models have the advantage to approach the origins of eating pathology from the multifactorial point of view, thus providing the most sophisticated descriptions of the onset and maintenance of eating problems. However, the empirical evidence of these models is still limited [e.g., 116; 119] and both longitudinal and intervention studies are required to establish the pathways from normal eating behavior to the onset and maintenance of eating pathology. In addition, there is a need to integrate biological factors into these models in order to gain a comprehensive multifactorial model that represents all risk domains [120]. While the required studies are expensive and time-consuming, the majority of risk-factor studies have focused on testing different variables from different risk domains rather than testing any particular theory or model [107].

In this study, EDS and associated factors were investigated without using a background of any particular theory or model. This was due to the practical approach adopted in order to test whether the health-related factors generally measured among adolescents in school health care could be useful for the purpose of identifying individuals with increased risk for EDS. The lack of a comprehensive universally sound theory of the etiology of eating disorders [111; 121] justified this approach, and the need of prospective studies with a broad range of putative risk factors [121] made this approach even preferable to a theoretically limited one.

2.3 Measurement of eating pathology

2.3.1 Rationale of measurement

Prolonged disturbances in eating behavior result in several signs that can be observed in health examinations, e.g., weight loss or failure of growth, bradycardia, hypotension, lanugo hair, parotitis, the loss of tooth enamel, and calluses on the dorsum of hand. In addition, it is typical that those who experience eating pathology use high levels of health care services to get treatment for problems such as constipation, dizziness, fatigue, headache, or lack of regular periods. [59; 122.] Before the occurrence of these signs, eating disturbances are difficult to detect [123]. Epidemiological studies show that the majority of individuals with disturbed eating behaviors remain undetected in primary health care [3; 5; 124; 125]. Especially individuals with bulimic eating disorders tend not to receive treatment for their eating problems [126; 127; 128; 129]. This may be due to several reasons. First, individuals with disturbed eating behaviors typically feel shame and try to hide their problems while being afraid of judgment and stigmatization [130; 131]. This concerns especially those with bulimic type pathology [132]. Second, persons with eating pathology may lack the feeling of sickness [130; 131], or, although admitting the seriousness of their condition, they may want to maintain the status quo. This is typical of individuals with anorectic behaviors. [132.] Third, some do not understand how serious and permanent consequences their behavior may have [133; 134]. This concerns particularly children and adolescents. [78.] Fourth, many are not aware of available treatments [131], they may be skeptical about the benefits of the treatment [134; 135], or think that no one can help them in eating problems [136]. The vast majority of people with a diagnosable or subthreshold eating disorder do not seek eating disorder specific treatment [137; 138]. Finally, primary health care professionals may have overoptimistic beliefs about their knowledge and capability to recognize eating pathology [139], which results underdiagnosis and treatment delays [134]. In order to improve the situation,

screening for eating pathology is recommended as a routine part of adolescents' health examinations [75; 127; 140; 141] with an emphasis on early detection and intervention [11; 12; 15; 24; 59].

In the following, the justification of screening for eating pathology is assessed on the basis of the WHO principles for screening. First, the condition to be screened should be an important problem with serious consequences to the individual and the family. Second, there should be an acceptable and available treatment, which affects the course and prognosis of the condition better at its early stages than later, and the condition must have early symptoms that can be detected by screening. Third, there must be a suitable screen that is easy and quick to use and acceptable to the population. Fourth, the development of the condition is understood, and the policy on whom to treat is clear. Finally, the costs of screening and treatment are economically balanced in relation to other health care expenditure, and screening should be a regular, routine part of examination of the population at risk. [142.]

First, eating disorders are important health problems irrespective of their relatively low prevalence in population [143; 144]. They cause significant burden on the individual, familial, and societal levels in and of themselves [for a review of cost of disorders, see 145; for a review of quality of life, see 146] and when accompanied by severe complications, [147; 148], co-morbidity [e.g., 30; 124; 138; 140; 149; 150; 151; 152] and premature mortality [153; 154; 155; 156; 157]. In addition, non-threshold eating pathology also jeopardizes an individual's mental [31; 138; 150; 158; 159; 160; 161; 162; 163; 164; 165] and physical health [31; 150; 161; 166] and development [79; 148; 167; 168], social functioning [138; 159; 160; 161; 167], and quality of life [160; 169; 170]. If a family member suffers from an eating disorder, the entire family experiences high levels of distress [32; 171; 172; 173], isolation, lack of social support, lack of information, lack of understanding [174], and impaired quality of life [175; 176]. Society, in turn, carries the financial burden of the provision of treatment, social services, and the loss of productive years [for a review, see 177].

Second, there are several commonly accepted treatment methods for eating disorders in adolescents [for a review, see 178], including, for example, cognitive-behavioral therapy adjusted for adolescent BN (CBT-A) [179], interpersonal psychotherapy [180], and different family therapies for the treatment of both AN and BN [e.g., 66], of which the Maudsley model of family-based treatment for adolescent AN [181] is reported as having the strongest evidence base [178]. Although the treatment outcomes on eating disorders leave room for improvement [for reviews, see 130; 182; 183; 184; 185; 186] there is evidence suggesting a better prognosis, especially for AN, if the illness onset occurs in adolescence (instead of childhood or adulthood) [151; 187], if it is detected before adulthood [188; 189] and if it is treated with an early intervention [186] so that the symptomatic period before the treatment remains short [151; 190; 191]. For BN, the evidence of the prognostic value of these factors is still scarce [192]. However, there are some findings of the effect of early interventions targeting the symptoms associated with BN and BED [191]. In addition, there is evidence of the continuity of eating problems [e.g., 159; 193; 194; 195; 196; 197; 198; 199; 200; 201; 202; 203; 204; 205] and the development from milder to more severe eating pathology [31; 191; 206]. Early features of eating pathology typically occur several months or even years before the onset of a full-blown syndrome [e.g., 207]. In this phase, even relatively short interventions may be effective in reducing the symptoms [for reviews, see 208; 209; 210] and risk factors of eating disorders [208; 211; 212; 213]. Screening programs with an educational impact are

found to be relatively effective in secondary prevention of eating pathology [133; 214] and increasing evidence suggests that early intervention is effective also in full-blown eating disorders [for a review, see 186] especially in adolescents [215].

Third, screening for eating pathologies is possible because there are several self-report questionnaires developed for this purpose (see section 2.3.2.). In a two-phase screening process, individuals who screened positive in the first phase will undergo a more thorough examination in the second phase to determine if an intervention or referral to treatment is needed, or whether some follow-up assessments are enough. As stated by Wilson and Junger [142], the sensitivity of a screen is most important in order to find the symptomatic individuals, while a relatively high proportion of false positives can be accepted.

Fourth, although the natural history of eating disorders is still partly unknown, the serious consequences of eating disorders both to the individual and the family create a need to minimize the delay of the treatment. In practice, clinical guidelines [14; 15; 216; 217; 218] and statements [e.g., 75; 77; 219] direct the policy of early detection, intervention, and treatment of eating disorders, thus supporting health care personnel in their decisions about how to proceed with screen-positive individuals.

Finally, despite the lacking empirical evidence of the economics of screening for eating pathologies in students, the estimates based on a decision-analytic microsimulation model in a hypothetical cohort of adolescents support the cost-effectiveness of school-based annual screening for eating disorders in adolescents up to the age of 18 years [220]. As the majority of the treatment costs are associated with hospitalization [e.g., 221], actions for early detection and intervention before the indicators for hospitalization occur are warranted, although the treatment of AN [e.g., 222; 223] and BN [e.g., 224] is found cost-effective as well. In school health care, screening for eating pathologies can be performed as a part of an annual health examination, thus facilitating a continuing process to reach the vast majority of adolescent population over the years of the highest risk for the occurrence of eating problems. Universal screening also reduces the possible risk of stigmatizing some adolescents as 'ED cases'. In sum, when assessed on the basis of the WHO's classic criteria for screening [142], school-based screening for eating pathology is justified.

2.3.2 Utility of different measures

Numerous self-report questionnaires heve been designed to measure eating pathology. In this section, the utility of some instruments is discussed in the light of the intended purpose of this study, for use as a primary screening tool for detecting eating pathology in adolescents in school health care settings. The utility of an instrument is evaluated in terms of its relevancy and feasibility. The presentation starts with the focus on relevancy and continues with the discussion about feasibility, with reference to different scales that have been used in adolescent populations. The scales were identified from the reviews of Jacobi and co-writers [225], Peterson and Mitchell [226], and Túry and co-writers [227] and are presented in Table 2. A more detailed description of the questionnaire (SCOFF) that was used in this study is provided at the end of the section.

In order to be a relevant tool for the primary screening of eating problems in non-clinical adolescent populations, the instrument should capture a wide variety of eating pathologies. This limits the utility of such questionnaires that yield only a certain type of eating disturbance or focus on the categorical diagnosis of eating disorders. For example, the revised Restrained Scale (RS) [228] measures dietary restraint, but not binge-purge

behavior, the Forbidden Food Survey (FFS) [229], evaluates individuals' emotional reactions to different food types and caloric levels, but not the related behavior, and the Mizes Anorectic Cognitions Questionnaire (MAC) [230; 231] measures the typical cognitions of AN and BN, but not the behavioral components of these disorders. The Eating Attitude Test (EAT) [232], the first questionnaire designed to measure the symptoms of eating disorders [233], and its later version, EAT-26 [234], were developed for evaluating behaviors and attitudes found in AN. Bulimic type eating pathology, in turn, was of interest to the designers of the Binge Scale (BS) [235], the Bulimia Cognitive Distortions Scale (BCDS) [236], the Bulimia Test (BULIT) [237] and its revision (BULIT-R) [238]. The Binge Eating Scale (BES) [239], the Bulimic Investigatory Test, Edinburgh (BITE) [240], and the adolescent version of the Questionnaire on Eating and Weigth Pattern (QEWP-A) [241] were developed with a focus on binge eating. The Survey for Eating Disorders (SEDs) [242] and the Eating Disorder Diagnostic Scale (EDDS) [243] were designed for detecting and diagnosing full-blown eating disorders. (Table 2.) Consequently, the abovementioned instruments are not the most relevant for use as a primary screen in non-clinical populations.

Feasibility of a scale consists of several factors that enable the screening procedure to be carried out rapidly and widely. A feasible questionnaire is simple, brief, acceptable, and inexpensive [see 244]. Its items and response options are unambiguous and explicit, and the scoring procedure is clear and easy. If the questionnaire is not simple enough, it may lead to diminished validity. In multi-choice response formats, respondents are required to choose between several options with fine distinctions [245]. In order to be able to give an elaborated response, the respondent needs enough experience of the topic, which may be limited in youth. In contrast, dichotomous items are usually easy to complete and therefore, people often prefer them as compared to formats that place more burden on the respondent by demanding concentration on fine discriminations [245]. Accordingly, questionnaires with, e.g., Likert scale response options may be not the best choice, although not prohibited [see 246], for use in adolescent populations. The length of the instrument may also decrease its validity; girls tend to be patient in completing questionnaires, but boys are found to become easily frustrated with lengthy instruments [247]. In addition, it is important that the questionnaire is acceptable to both genders. If the respondents find the items irrelevant, they may not answer at all [244]. Thus, gender-specific questions may result a validity risk [248]. The feasibility of the instrument may also be limited due to fees charged for their use by the copyright holder. Public-domain measures, which are available for use at no cost with a permission of the designer, may be preferable tools for use in public health care.

Taking into account these notions, many of the questionnaires that otherwise might be suitable for measuring a wide range of eating pathology fail in terms of feasibility. First, some of them are quite long consisting of tens of items. For example, the Stirling Eating Disorder Scales (SEDS) [249], which measure cognitive/emotional and dietary behavioral aspects of AN and BN, include 80 items. Second, some of the instruments are both lengthy and use multi-choice response formats. For example, the Anorexia Nervosa Inventory for Self Rating (ANIS) [250], a measure of deviant eating behavior, includes 32 statements with a 6-point Likert scale; the Dutch Eating Behavior Questionnaire (DEBQ) [251] concerning restraint, emotional, and external eating includes 33 Likert-formatted questions; and the Eating Disorder Examination-Questionnaire (EDE-Q) [252], a measure of eating disorder symptoms, consists of 38 items with a 7-point forced-choice rating scale. The Setting Conditions for Anorexia Nervosa Scale (SCANS) [253], the Multifactorial Assessment of Eating Disorders Symptoms (MAEDS) [254], the Branched Eating

Disorders Test (BET) [255], the Questionnaire for Eating Disorder Diagnosis (Q-EDD) [256], and the Three-Factor Eating Questionnaire (TFEQ) [257], also known as the Eating Inventory [226], and its shorter version, TFEQ-18 [258], all consist of numerous items with Likert scales.

Third, some of the questionnaires have a cumbersome scoring procedure. For example, the Short Evaluation of Eating Disorders (SEED) [259] provides the scores and total severity indices for the symptoms of AN and BN on the basis of four out of six items, which can be completed quickly, but the scoring and calculating process is complicated [see 259], thus hindering its usefulness in everyday practice.

Fourth, the feasibility of some measures is limited by their scope on females only. For example, the Weight Concerns Scale [261; 262] was designed to assess adolescent girls' preoccupation with thinness and body shape; the Rapid Screen [263] was made for use in primary health care to identify female adolescents at risk for developing eating disorders; and the three-item screen developed by McKnight Investigators [264] to detect partial and full-blown eating disorders, is based on the McKnight Risk Factor Survey [265], which is designed for use among females only.

Finally, some of the questionnaires are both cumbersome, set special professional requirements for their users, and also liable to a charge. For example, one of the most commonly used self-report eating disorder questionnaires, the Eating Disorder Inventory (EDI) [266; 267] and its later versions, EDI-2 [268] and EDI-3 [269], include tens of items rated on a Likert scale. The recommendation is that the results be interpreted only by qualified professionals who have formal assessment training, knowledge of psychometric statistical methods, and experience with eating disorder psychopathology. Lower professional requirements are set for the use of the EDI-3 Referral Form (EDI-3 RF) scale, which was developed to assist with screening in order to ascertain whether an individual should be referred to treatment. Therefore, it could be used, for example, in schools. [269.] However, all versions of EDI are copyrighted and royalties are charged for their use. (Table 2.) In sum, none of the instruments mentioned above can be deemed to the first choice for primary screening in a busy practice of school health care.

Table 2. Measures of behaviors, attitudes, and cognitions related to eating and weight

Measure, Designer(s)	Dimensions measured	Items, scale	Remarks
Anorexia Nervosa Inventory for Self Rating (ANIS), Fichter & Keeser [250]	Deviant eating behavior, especially AN and BN	32 items: 31 for total score, 1 for reliability, 6-point Likert scale	Copyrighted
Binge Eating Scale (BES), Gormally et al. [239]	Behaviors, feelings, and cognitions associated with a binge episode	16 items, multiple-choise scale	Copyrighted, originally not for adolescents
Binge Scale (BS), Hawkins & Clement [235]	Behavioral and attitudinal parameters of bulimia	19 items: 9 for the total score, 10 for the prevalence and characteristics of binge eating, Likert scale and multiple-choice response formats	Available upon request from Raymond C. Hawkins
Branched Eating Disorders Test (BET), Selzer et al. [255]	Symptoms of eating disorders (DSM-III-R)	47 items: a minimum of 20, nine of which branch to further items, forced choice response options	A computerized measure designed and implemented using HyperCard software
Bulimia Cognitive Distortions Scale (BCDS), Schulman et al. [236]	Cognitive distortions and irrational beliefs associated with bulimia	25 items, divided into two factors: 1) automatic behaviors, 2) cognitive distortions, 5-point Likert scale	A treatment outcome measure
Bulimia Test (BULIT), Smith & Thelen [237]	Symptoms of bulimia (DSM-III)	36 items: 32 for the total score, 2 for the symptom frequency, 2 for females only, 5-point Likert and multiple-choice scales	Copyrighted
Revised version of the Bulimia Test (BULIT-R), Thelen et al. [238]	Symptoms of bulimia (DSM-III-R and DSM-IV)	36 items: 28 for the total score, 8 for weight-control behaviors, 5-point Likert scale	Copyrighted
Bulimic Investigatory Test, Edinburgh (BITE), Henderson & Freeman [240]	Symptoms of bulimia and binge eating over the past 3 months	33 items, dichotomous and multiple-choice scales	In the public domain
Dutch Eating Behavior Questionnaire (DEBQ), van Strien et al. [251]	Restrained, emotional, and external eating	33 items, 5-point Likert scale	Copyrighted
Eating Attitude Test (EAT), Garner & Garfinkel [232]	Symptoms of AN	40 items, 6-point Likert scale	Copyrighted
Shorter version of the Eating Attitude Test (EAT-26), Garner et al. [234]	Symptoms of AN	26 items: 13 for dieting, 6 for bulimia and food preoccupation, 7 items for oral control, 6-point Likert scale	Copyrighted

Table 2. (continues)

Measure, Designer(s)	Dimensions measured	Items, scale	Remarks
Eating Disorder Diagnostic Scale (EDDS), Stice et al. [243]	Symptoms for AN, BN, and BED (DSM-IV) over the past 3 months	22 items, 7-point Likert scale, dichotomous, multiple-choice, and write-in response formats	Copyrighted
Eating Disorder Examination Questionnaire (EDE-Q), Fairburn & Beglin [252]	Main behavioral features of eating disorders over the past 4 weeks	38 items divided into three subscales: 1) restraint, 2) shape concern, and 3) weight concern, 7-point Likert scale	Copyrighted, recommended in the Finnish Current Care Guideline as a secondary screen to assess the severity of eating pathology [15]
Eating Disorder Inventory (EDI), Garner et al. [266; 267]	Behavioral and attitudinal characteristics of AN and bulimia	64 items: 7 for drive for thinness, 10 for interoceptive awareness, 7 for bulimia, 9 for body dissatisfaction, 10 for ineffectiveness, 8 for maturity fears, 6 for perfectionism, 7 for interpersonal distrust, 6-point Likert scale	Copyrighted, recommended to be interpreted only by qualified professionals who have formal assessment training, knowledge of psychometric statistical methods, and experience with eating disorder psychopathology
First revision of the Eating Disorder Inventory (EDI-2), Garner [268]	Psychological traits and constructs in individuals with eating disorders	91 items: 6-point Likert scale	Copyrighted, recommended in the Finnish Current Care Guideline as a secondary screen to assess the severity of eating pathology [15]
Second revision of the Eating Disorder Inventory (EDI-3), Garner [269]	Drive for thinness, bulimia, body dissatisfaction, self-esteem, personal alienation, interpersonal insecurity, interpersonal alienation, interoceptive deficits, emotional dysregulation, perfectionism, ascetism, and maturity fears	91 items: 25 specific to eating disorders (= Eating Disorder Risk Scales), 66 for general psychological factors related to eating disorders, 6-point Likert scale	Copyrighted
EDI-3 Referral Form (EDI-3 RF), Garner [269]	Dieting concerns, body weight, height, weight and menstrual history, and behavioral symptoms of eating disorders	39 items: 25 specific to eating disorders, 9 for BMI, 5 for behavioral symptoms	Copyrighted , lower professional requirements, intended to assist in ascertaining whether an individual should be referred to treatment
Forbidden Food Survey (FFS), Ruggiero et al. [229]	Emotional reactions to different food types and caloric levels	45 items divided into 5 food groups and 3 caloric levels, 5-point Likert scale	In the public domain, food items represent the foods consumed in the American diet
Mizes Anorectic Cognitions Questionnaire (MAC), Mizes [230], Mizes & Klesges [231]	Typical cognitions of AN and BN	33 items divided into three factors: 1) rigid weight and eating regulation, 2) weight and eating behavior as the basis of approval from others, 3) self-esteem based on excessive self-control, 5-point Likert scale	Copyrighted

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Table 2. (continues)

Measure, Designer(s)	Dimensions measured	Items, scale	Remarks
Revised version of the Mizes Anorectic Cognitions Questionnaire (MAC-R), Mizes et al. [270]	Typical cognitions of AN and BN	24 items: 8 for self-control and self-esteem, 8 for weight and approval, 8 for rigid weight regulation and fear of weight gain, 5-point Likert scale	Copyrighted
Modified Survey for Eating Disorders (SEDs), Ghaderi & Scott [242]	Eating disorders (DSM-IV)	39 items: 18 for diagnosis, 4 for demographic, 17 for additional information, dichotomous, multiple-choice, and write-in response formats	In the public domain
Multifactorial Assessment of Eating Disorders Symptoms (MAEDS), Anderson et al. [254]	Cognitive and behavioral components relevant to treatment outcome for AN and BN	63 items: 11 for depression, 8 for binge eating, 7 for purgative behavior, 11 for fear of fatness, 9 for restrictive eating, 10 for avoidance of forbidden foods, 7-point rating scale	Available upon request from Donald A. Williamson
Questionnaire for Eating Disorder Diagnosis (Q-EDD), Mintz et al. [256]	Eating disorder criteria (DSM-IV) over the past 3 months	50 questions, dichotomous, Likert-type, multiple-choice, and write-in response formats	Available upon request from Laurie B. Mintz
Questionnaire on Eating and Weight Pattern, adolescent version (QEWP-A), Johnson et al. [241]	Binge eating disorder	12 items for an adolescent and 12 for a parent, dichotomous and multiple-choice scales	In the public domain
Rapid Screen, Anstine & Grinenko [263]	Disordered eating	4 items, 6-point Likert scale	In the public domain, for females only
Revised Restrained Scale (RS), Herman & Polivy [228]	Dietary restraint	10 items, 4–5-point Likert scale and multiple-choice response formats	Designed to identify dieters, not to assess eating pathology
Risk Factor Screen, McKnight Investigators [264]	Thin body preoccupation and social pressure	4 items, 5-point Likert scale	For females only
Setting Conditions for Anorexia Nervosa Scale (SCANS), Slade & Dewey [253]	Risk of developing AN or BN	40 items: 14 for general dissatisfaction, 10 for social and personal anxiety, 8 for perfectionism, 3 for adolescent problems, 2 for weight control, 5-point Likert scale	In the public domain
Short Evaluation of Eating Disorders (SEED), Bauer et al. [259]	Key symptoms of AN and BN	6 items, multi-choice rating scale, and write-in response formats	In the public domain

(continues)

Table 2. (continues)

Measure, Designer(s)	Dimensions measured	Items, scale	Remarks
Stirling Eating Disorder Scales (SEDS), Williams et al. [249]	Dietary behavioral, cognitive and emotional aspects of AN and BN	80 items in 8 scales: anorexic dietary cognitions and behavior, bulimic dietary cognitions and behavior, perceived external control, low assertiveness, low self-esteem, self-directed hostility, dichotomous scale	Copyrighted
Three-Factor Eating Questionnaire (TFEQ), Stunkard & Messick [257]	Cognitive restraint of eating, susceptibility to periodic disinhibition of control over eating, and perceived hunger	51 items divided into three factors: 1) dietary restraint, 2) disinhibition, and 3) perceived hunger, dichotomous and 4-point Likert-type response formats, and a rating scale of 0 to 5	Copyrighted as the Eating Inventory, for professionals with relevant training and experience in assessment
Shorter version of the Three-Factor Eating Questionnaire (TFEQ-R18), Karlsson et al. [258]	Cognitive and behavioral components of eating	18 items: 6 for cognitive restraint, 3 for emotional eating, 9 for uncontrolled eating, 4-point forced-choice Likert-type response formats, and a rating format of 1 to 8	Copyrighted, tested among Finnish adolescent girls [260]
Weight Concerns Scale, Killen et al. [261; 262]	Preoccupation with thinness and body shape	5 items, Likert-type and multi-choice scales	For adolescent girls only

The instrument used in this study, the SCOFF questionnaire [271], captures several symptoms of eating disorders. It is simple and brief, its response options and scoring procedure are clear and easy and its items are suitable for both genders. In addition, as a public domain measure, it is inexpensive.

SCOFF was designed in England in 1999 by Morgan and coworkers to screen for the core features of AN and BN in primary health care. SCOFF (acronym for Sick, Control, One stone, Fat, Food) consists of five questions concerning intentional vomiting, the loss of control over eating, weight loss, body dissatisfaction, and food intrusive thoughts. Focus groups of eating disorder patients and specialists contributed to the refining of the questions into their present form. [272.] The items of SCOFF are responded to on a dichotomous scale (Yes/No). One point is given for every 'Yes' answer, and zero point for every 'No' answer. Total score is obtained by summing up the points (range 0–5). A threshold of two or more points is interpreted as indicating a likely case of an eating disorder. [271.]

Originally, SCOFF was intended to be completed face-to-face. However, due to enhanced disclosure via written format as compared to face-to-face interview, the written version of the SCOFF is recommended for routine use. It takes less than two minutes to administer SCOFF. [272.] The instrument is available in the public domain.

The psychometric properties of SCOFF have been evaluated in several studies [e.g., 273] conducted in community and/or clinical settings. The results obtained in community samples of adolescents and young adults [274; 275; 276; 277; 278; 279; 280; 281; 282;

283; 284; 285; 286] are presented in Table 3. The different methods used in these studies to evaluate the reliability and validity of SCOFF are discussed in the following.

Firstly, the reliability of SCOFF has been assessed in terms of internal consistency by computing coefficient alfa, i.e., Cronbach's alfa or Kuder Richardson formula 20 (KR-20). For the reliability assessment, coefficient alfa is suggested to be suitable in most situations [287] where the scale items measure the same construct, i.e., the latent variable underlying the items. Coefficient alfa values exceeding 0.60 are acceptable, and those between 0.80–0.90 are considered to indicate very good internal consistency. In adolescent samples [275; 276; 277; 278; 279; 280; 281; 282; 283; 285], the internal consistency of SCOFF has been low (< 0.60) with one exception (0.62 in a sample of male students) [283] (Table 3.). As the alfa value depends on the number of items in the scale, the variance within each item, and the variance within the scale [288; 289], the low values in these studies may partly be due to the small number of items and the dichotomous response format in SCOFF. On the other hand, the low alfa values may raise the question whether the items of SCOFF are manifestations of the same unidimensional construct; in questionnaires consisting of items that measure different constructs which, however, may belong to the same category, the items are not expected to covary the same way [see 245; 290].

Secondly, SCOFF has been assessed in terms of the stability of the measure over time, which is expressed as a test-retest coefficient. This method is recommended generally not to be used to estimate reliability [287] as it may have serious defects. For example, the trait being measured may fluctuate even over short periods of time, and changes may occur as a result of the first measurement, independently of the stability of the instrument [287; 291]. For SCOFF, the intra-class correlation (ICC) between the two sets of scores with a 2-week interval has been acceptable (r = 0.66) [280], and the concordance of the scores obtained on two administrations of the test with an interval of 1–2 weeks has been high (91.6%) with Cohen's kappa value of 0.813 [276] indicating very good stability [see 291]. (Table 3.)

Thirdly, the concurrent validity of SCOFF has been evaluated in terms of convergent and divergent validity by analyzing the degree of relationship between the SCOFF score and the scores attained by means of other eating disorder questionnaires (convergency), and CAGE [292], a measure of alcoholism (divergency). The associations are described by correlation coefficients. Correlation is suggested to be moderate with the scores between 0.40–0.60, high when scores are between 0.60–0.80, and very high with scores above 0.80 [288]. All correlations between SCOFF and the other eating disorder measures have been statistically significant varying between low and high [279; 285]. Mostly, correlations have been on a moderate level [279; 280; 282; 284; 285], supporting the convergent validity of SCOFF. Low correlations (r = 0.280 for girls and 0.256 for boys) between the scores of SCOFF and CAGE, in turn, have supported the divergency of these measures [278]. (Table 3.)

Fourthly, the criterion validity of SCOFF has been assessed by comparing the performance of SCOFF against other psychometric tools and diagnostic interviews (see Table 3). The results are reported as Cohen's kappa value, the sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), positive likelihood ratio (LR+), and negative likelihood ratio (LR–) of SCOFF. The strength of agreement between the two measures expressed with the values of kappa statistic is recommended to be interpreted as follows: poor (< 0.00), slight (0.00–0.20), fair (0.21–0.40), moderate (0.41–0.60), substantial (0.61–0.80), and almost perfect (≥ 0.81) [293]. Sensitivity is the ability of

a screen to correctly identify those with the condition, i.e. the outcome of concern. Specificity is the ability of the instrument to correctly exclude those without the condition. PPV is the proportion of those screen-positives who have the condition, whereas NPV is the proportion of those screen-negatives who do not have the condition. Both PPV and NPV depend on the prevalence of the condition; with a falling prevalence also PPV falls whereas NPV rises. [294.] LR+ is the ratio of true-screen-positives to false-screen-positives computed as sensitivity/(1-specificity), thus expressing the probability of a positive screening result for a person with the condition compared to one without the condition. LR–, in turn, is the ratio of false-screen-negatives to true-screen-negatives computed as (1-sensitivity)/specificity, thus reflecting the probability of a negative screening result for a person with the condition compared to an individual without the condition. [291.] For the purpose of screening, the high sensitivity of a test is regarded as ideal; even a high proportion of 'false' positives is suggested as being acceptable in order not to miss affected individuals [142].

The level of agreement between SCOFF and psychiatric interviews has ranged from moderate (Cohen's kappa = 0.433) [276] to substantial (Cohen's kappa = 0.74) [281]. When evaluated against psychiatric interviews, SCOFF has provided somewhat higher values of sensitivity and PPV than when evaluated against other eating disorder questionnaires. This indicates that, with a threshold of two or more points, SCOFF may perform better in detecting the full-blown eating disorders than eating pathology in general. In line with this, computed on the basis of the reported sensitivity and specificity of SCOFF [274; 276; 277; 279; 281; 282; 284], the LR+ scores suggest that it is from 2 to 18 times more likely to find that a person who scores two points in SCOFF already has an eating disorder than to find that she/he has no eating disorder. With the threshold of one or more points, SCOFF has provided high sensitivity (> 90%) in assessments against EDI-2 [279] and EAT-26 [282]. The specificity, in turn, has been as low as 43% [282] and 54% [279], which reflects a high number of individuals that were screen positives in SCOFF but screen negatives in the reference measures. This may partly be due to the different focus of these measures; EDI-2 measures a variety of factors some of which are specific to eating disorders while others are non-specific psychological features found to be related to eating disorders, and EAT-26 measures behaviors and attitudes typical in AN, whereas SCOFF focuses on the key features of both AN and BN. Thus, EDI-2 and EAT-26 may fail to measure some of the features detected by the means of SCOFF and vice versa. Accordingly, the LR+ scores suggest that it is about twice as likely to find that a person who scores one or more points in SCOFF is screen positive also in EDI-2 and EAT-26 as to find that the person is screen negative in these measures. In addition, according to LR– scores, the likelihood of people who self-report no EDS in SCOFF to be screen positive in EDI-2 and EAT-26 is only 1/7 of the likelihood that they are screen negative in these measures as well. (Table 3.)

All the studies discussed above used community-based samples of adolescents. Therefore, the tests' properties were comparable without a risk of a spectrum bias. In studies assessing the sensitivity and specificity of an instrument among clearly diseased and not diseased people, the ability of the instrument to discern these two groups tends to be better than in studies conducted among general population with a wide spectrum of possible symptoms [295].

Table 3. Psychometric properties of SCOFF in community samples of adolescents

Author(s)	Sample (n, females %); age in years; country	Tests administered	Internal consistency	Test-retest stability (interval)	Strength of agreement / correlation	Se %	Sp %	PPV %	NPV %	LR+	LR–
Berger et al.282]	Students (n = 807, 53%); 12 years; Germany	German version of SCOFF ≥1 points and SCOFF ≥2 points vs. EAT-26D	Cronbach's alfa = 0.45	r = 0.73 (7–28 days)	Pearson's r = 0.53	94 / 79	43 / 74	16 / 25	98 / 97	1.65[a] / 3.04[a]	0.14[b] / 0.28[b]
Caamaño [274]	Students (n = 289); 11–13 years; Spain	SCOFF ≥2 points vs. EAT	-	-	-	64.1	87.2	43.9	94.0	5.0[a]	0.41[b]
Campo-Arias et al. [278]	Students (n = 4871, 49%); 10–19 years (mean 14.47 y); Colombia	Spanish version of SCOFF vs. CAGE	KR-20 = 0.584 for girls, 0.521 for boys	-	Pearson's r = 0.280 for girls; 0.256 for boys	-	-	-	-	-	-
Garcia et al. [281]	Students (n = 400, 100%); 18–35 years (mean 20.9 y); France	SCOFF-F ≥1 points and SCOFF-F ≥2 points vs. MINI scales of AN and BN (DSM-IV)	KR-20 = 0.432	-	Cohen' s kappa = 0.74	97.3 / 94.6	66.1 / 94.8	- / 65	- / 99	2.87[a] / 19.0[a]	0.04[b] / 0.06[b]
Hansson et al. [285]	Students (n = 1265, 51.6%); mean 16.19 y; Sweden	Swedish version of SCOFF vs. EDE-Q	KR-20 = 0.48	-	Point biseral r = 0.66 for girls; 0.60 for boys	-	-	-	-	-	-
Jung et al. [275]	Students (n = 417, 100%); mean 20.73 y; Korea	K-SCOFF ≥2 points vs. KEAT-26	Cronbach's alfa = 0.40	Spearman r = 0.58 (14 days)	-	-	-	-	-	-	-
Leung et al. [280]	Students (n = 954, 46.2%); 12–25 years (mean 14.9 y); Hong Kong	Chinese version of SCOFF ≥2 points vs. EDE-Q	Cronbach's alfas = 0.44–0.57	ICC r = 0.66 (2 weeks)	Pearson's r = 0.5	76.1*	97.1*	-	-	-	-
Muro-Sans et al. [279]	Students (n = 954, 49.8%); 10.9–17.3 years (mean 13.5 y); Spain	SCOFF-c ≥1 points and SCOFF-c ≥2 points vs. EDI-2	Cronbach's alfa = 0.54	-	Pearson's r = 0.111–0.633	92.31 / 73.08	54.11 / 77.74	-	-	2.01[a] / 3.28[a]	0.14[b] / 0.35[b]

(continues)

Table 3 (continues)

Author(s)	Sample (n, females %); age in years; country	Tests administered	Internal consistency	Test-retest stability (interval)	Strength of agreement / correlation	Se %	Sp %	PPV %	NPV %	LR+	LR–
Richter et al. [286]	General population (n = 423, 51%); 14–29 years; Germany	German version of SCOFF ≥2 points vs. a subscale of ISR-E	-	-	-	31 F 20 M	97 F 97 M	88 F 68 M	63 F 77 M	10.33 F 6.67 M	0.71 F 0.82 M
Rueda et al. [276]	Students (n = 385, 100%); 17–35 years (mean 21.2 y); Colombia	Spanish version of SCOFF ≥2 points vs. CIDI (DSM-IV)	Cronbach's alfa = 0.480	Cohen's kappa = 0.813 (7–14 days)	Cohen's kappa = 0.433	78.4	75.8	46.5	92.9	3.24[a]	0.28[b]
Rueda Jaimes et al. [277]	Students (n = 241, 100%); 10–19 years (mean 14 y); Colombia	Spanish version of SCOFF ≥2 points vs. CIDI (DSM-IV-TR)	Cronbach's alfa = 0.436	-	Cohen's kappa = 0.554	81.9	78.7	62.1	91.1	3.85	0.23
Sánchez-Armass et al. [283]	University applicants (n = 3594, 55.6%); 16–21 years (mean 18.1 y); Mexico	Spanish version of SCOFF ≥2 points	KR-20 = 0.49 for females; 0.62 for males	-	-	-	-	-	-	-	-
Sánchez-Armass et al. [284]	Students (n = 1057, 67%); 17–56 years (mean 21.0 y); Mexico	SCOFF ≥2 points vs. EDI-2; SCOFF ≥2 points vs. EDE (DSM-IV)	-	-	Bivariate r = 0.32–0.61	78	84	58	93	4.88[a]	0.26[b]

Notes: AN = anorexia nervosa; BN = bulimia nervosa; CAGE = an acronym of the four questions on alcohol screening [292]; CIDI = Composite International Diagnostic Interview [300]; DSM-IV = Diagnostic and Statistical Manual of Mental Disorders 4th revision [63]; DSM-IV-TR = Diagnostic and Statistical Manual of Mental Disorders text revision [64]; EAT = Eating Attitude Test [232]; EAT-26D = German version of the Eating Attitude Test [298]; EDE = Eating Disorder Examination [301]; EDE-Q = Eating Disorder Examination-Questionnaire [252]; EDI-2 = first revision of the Eating Disorder Inventory [268]; F = female; ICC = intraclass correlation coefficient; ISR-E = ICD-10-Symptom-Rating questionnaire [297]; KR-20 = Kuder Richardson's formula 20; KEAT-26 = Korean version of EAT-26 [275]; K-SCOFF = Korean version of SCOFF [275]; LR– = negative likelihood ratio; LR+ = positive likelihood ratio; M = male; MINI = Mini-international neuropsychiatric interview [299]; NPV = negative predictive value; PPV = positive predictive value; r = correlation; SCOFF-c = Catalan version of SCOFF [279]; SCOFF-F = French version of SCOFF [281]; Se = sensitivity; Sp = specificity; * = correct data not available; [a] the value was computed as sensitivity/(1–specificity); [b] the value was computed as (1–sensitivity)/specificity

Fifthly, the content validity of SCOFF has been investigated by using a panel of experts to evaluate the relevancy of items. The experts found the items of SCOFF relevant with an agreement from 86% to 100%. [280.]

Sixthly, the construct validity of SCOFF has been assessed by analyzing the factor structure of SCOFF [279; 283; 285]. In a sample of Spanish adolescents, the exploratory factor analyses revealed a two-factor structure of SCOFF. Factor 1, entitled 'Loss of Control Over Food', consisted of items measuring the loss of control over eating, body dissatisfaction, and food intrusive thoughts. Factor 2, entitled 'Purging Behaviors', consisted of items focusing on intentional vomiting and weight loss. Yet, the factor structure was not invariant; two factors were obtained for girls and only one factor for boys. [279.] In Sweden, both one- and two-factor models showed good fit for girls while only one-factor structure was obtained for boys [285]. In Mexico, exploratory factor analyses resulted in a one-factor model for both genders, but the confirmatory factor analysis indicated a better fit of the two-factor model for males [283].

Finally, in a recent study [296], the screening value of SCOFF was assessed in clinical practice. The study was conducted in France among 15–50-year-old women visiting their general practitioner for reasons unrelated to body image, weight or dietary concerns. First, the participants completed the SCOFF questionnaire before meeting the GP. Blinded to women's responses to SCOFF, the GP considered their clinical notes and impressions to deem if the women had an eating disorder. Then, the investigators interviewed each woman for an eating disorder diagnosis using the DSM-IV criteria of eating disorders. About two thirds of women with an eating disorder were detected by means of SCOFF using a cut-off criterion of two points. This was two times more than the number of cases identified by the general practitioners.

In sum, on the basis of studies conducted among adolescents, SCOFF is a valid instrument that correlates as predicted with other measures of eating pathology. With a threshold of two or more points, SCOFF detects the vast majority of individuals with an eating disorder. With a threshold of one or more points, SCOFF is highly sensitive to identify also those adolescents who report eating pathology not meeting the diagnostic criteria of eating disorders. The low scores of internal consistency of SCOFF support the use of the items of SCOFF as one-item scales which measure different constructs (features of AN and BN) of the same category (EDS). To date, SCOFF has been used as a unidimensional scale where the item points are summed up to obtain a single variable indicating the degree of eating pathology as a number of specific features belonging to EDS. However, the finding of a two-factor model of SCOFF for girls and one-factor model for boys raises a question of the optimal factor solution for use in clinical practice and also warrants further testing of SCOFF in different settings.

In this study, the orientation was preventive, and therefore, priority was placed on the sensitivity of SCOFF to ensure that no adolescents with any EDS would be missed. Accordingly, the cut-off was set at one point with a dichotomy of 'no symptoms' (0 points) and 'one or more symptoms' (1–5 points). With a higher threshold, the sensitivity of SCOFF would have suffered, thus hampering the purpose of the study. Prior to this study, there were no published reports of any eating disorder questionnaire tested among Finnish adolescents.

2.4   Descriptive epidemiology of eating pathology

The description of the epidemiology of eating disorders is challenging as attempts to compare findings across different studies are hindered by several reasons. First, there is great variation in the criteria for case definition ranging from the strict diagnostic criteria of full-blown eating disorders to the broader criteria of subthreshold and partial eating disorders and eating pathology in general. The diagnostic criteria vary depending on the version of the classification system applied in the study.

Second, the measurement of these criteria varies between studies. Some rely on self-report questionnaires, the number and variety of which is extensive (see section 2.3.2.), while others prefer interviewing techniques, which include several options both for professionals and lay interviewers [e.g., 302]. The approach currently recommended for investigating the prevalence of eating disorders is a two-phase procedure that combines these two techniques in order to obtain diagnostic accuracy with economical efforts [72]. Problems in both types of studies include poor response rates, the restricted size of the groups interviewed, and flaws in assessment. [62].

Third, the study populations derive from different settings, e.g., registers, records, communities, and general population, each of which have their inherent problems in terms of validity. [303]. Weaknesses in register studies include possible variations in the registration policy, the faulty inclusion of information, and demographic differences between populations [62]. In record-based studies, findings are influenced by the accessibility of services, professionals' ability to detect eating pathology, help-seeking behavior among those with eating problems, and the quality of records. Community- and population-based studies, in turn, show enormous variation in terms of the sampling methodology, participation rate, measurement technique, and sample diversity. [303; 304.] In addition, they may not have enough differential power for the cases due to the low prevalence of eating disorders in the population and concealment of illness among those who have it [303]. In general, large population-based studies are preferable in order to obtain a representative sample of the whole spectrum of eating pathology in the general public. [305].

Keeping in mind the limited comparability of the findings across studies, the next section provides an overview of the prevalence and course of eating pathology. The overview is based on reviews [62; 144; 303; 305] and original articles of the epidemiology of eating pathologies published in the last decade. As the research of the topic is scarce in Finland, findings for Finnish samples are described starting from the 1990s. Priority is given to larger studies due to the relatively low prevalence of eating disorders in general population. Thus, many methodologically sound small-scale studies are excluded from this overview. The focus is on eating pathology in Western countries, because studies on non-Western populations are still few [305], and the presentations of eating pathology in non-Western countries are slightly different from those in Western countries and may not be captured by the methods and classification criteria used in the West [306]. The results obtained among adolescents are emphasized, although it is well known that eating disorders may have their onset already in childhood [e.g., 307; 308] or later in adulthood [e.g., 309; 310].

2.4.1 Eating disorders

Anorexia nervosa

Using the DSM-IV criteria, the estimates of the lifetime prevalence of AN range from 0.9% to 2.2% among females and from 0.2% to 0.3% in males [305]. Recent findings from Europe support these estimates for females, but fail to provide evidence for AN in males [311]. In adolescent samples, the prevalence is more equal between girls and boys. For example, in the USA, a nationally representative study of 13–18-year-old adolescents revealed the lifetime prevalence of 0.3% for AN in both genders [138]. In the DSM-5, the diagnostic criteria of AN were broadened, resulting in an increase in the prevalence. For example, in a Dutch cohort of 20-year-olds, the lifetime prevalence of 1.2% for DSM-IV AN increased to 1.7% for DSM-5 AN in females and from 0% to 0.1% in males, respectively [312]. In Finland, population-based studies have reported lifetime prevalence of roughly 2% [3; 313; 314; 315; 316] for AN and from 4% [3] to almost 8% [313] for broad AN in young females, and 0.24% for AN in young males [4].

The rate of recovery from AN in general population is relatively high. In Finland, more than two out of three females with broad AN are found to reach clinical recovery within five years after the onset of disorder. On the other way round, almost every third female with adolescent onset AN still has the disorder in young adulthood. In addition, many of the recovered persons have been ill for several years. Furthermore, after clinical recovery, these females still show psychosomatic symptoms and body dissatisfaction. [3.]

Bulimia nervosa

The lifetime prevalence of BN is estimated to range from 1% to 2% in young females [305] and from 0.1% [303] to 0.5% in males [305]. In line with these are the recent findings of population-based studies conducted in the USA [138], Australia [317], New Zealand [318], and Europe [311]. However, in some countries, the findings are more favorable. For example, in the Netherlands, the lifetime prevalence of BN is 0.6% for DSM-IV BN and 0.8% for DSM-5 BN in females, and 0% and 0.1% in males, respectively [312]. In Finland, the lifetime prevalence of BN is around 1% in girls aged 17.5 years [314], and about 2% [5; 315] in young adults. The lifetime prevalence of broad BN is roughly 2% [5]. In adolescents, the prevalence is suggested to be lower [313; 316]. Estimates for males are still missing in the literature.

The majority of the individuals with BN may recover. In Finland, 55% of women with broadly defined BN are found to reach clinical recovery within five years from the onset of the disorder. However, the psychological problems tend to be long-lasting: many women still have psychosomatic symptoms and body dissatisfaction at five years after their clinical recovery. In other words, more than two out of five females with onset of BN in adolescence still have BN in young adulthood. In addition, although more than every other case recover in terms of behavioral symptoms, psychological problems tend to sustain. [305.] Furthermore, while the recovery rate may increase along with a longer follow-up period, a significant number of individuals develop a chronic disorder. For example, in a 20-year follow-up study of students in the USA, 28% of the females with BN in late adolescence failed to recover by mid-life [309].

Binge eating disorder and other eating disorders

The prevalence of BED is suggested to be at least 1% [303]. In line with this, recent findings suggest the lifetime prevalence of 2.3% for BED and for subthreshold BED in 13–18-year-old girls in the USA, and 0.8% and 2.6% in boys, respectively [138]. In Europe, the lifetime prevalence of 1.92% is estimated for BED and of 0.55% for subthreshold BED in females aged 18 years and older, and 0.26% and 0.91% in males, respectively [311]. Using the DSM-5 diagnostic criteria for BED, the prevalence is somewhat higher. For example, the lifetime prevalence of 2.3% for 20-year-old females and 0.7% for males is reported in the Netherlands [312.] In Finland, a population-based study of young adult female twins found the lifetime prevalence of 0.7% for BED (DSM-5) [319].

Avoidant/restrictive food intake disorder

To date, studies of the prevalence of ARFID in general non-clinical population are still few. In Switzerland, a community-based study of children aged 8–13 years found a point prevalence of 3.2% for ARFID with no significant gender or age differences [320]. In another study conducted in Australia, the 3-month prevalence of ARFID was 0.3% in general population aged 15 years and older. As in the Swiss study, the gender distribution of Australians with ARFID was relatively even. [321.]

Other eating disorders

A large number of eating disorder cases in clinical practice do not meet in full the criteria for any specific eating disorder, but are classified as EDNOS (DSM-IV), or as OSFED or UFED (DSM-5). In population, the occurrence of these disorders is somewhat unclear due to the lack of positive diagnostic criteria – the diagnosis is based on the clinical severity of the disorder and the exclusion of the specific eating disorders. [64; 65.] Thus, determining the caseness depends significantly on individual consideration [322]. However, there is some evidence of higher prevalence of EDNOS than AN and BN in community [305; 323]. For example, with the lifetime prevalence of 4.78% EDNOS is the most common eating disorder diagnosis in adolescent population in the USA [323]. In addition, studies using expressions such as subthreshold, partial, subclinical, or atypical AN and BN when referring to EDNOS [see 324] have shown that the forms of AN and BN that do not reach the diagnostic threshold are more common than the full-blown eating disorders [62; 303; 305]. Using the DSM-5 diagnostic criteria, the prevalence of OSFED/UFED is found to be 0.8% for female and 0.3% for male adolescents in the Netherlands [312], 2.0% for students in Portugal [325], and 5% (OSFED) [326] and 4.7% (UFED) [327] for female adolescents in Australia.

In Finland, bulimic type pathology has been measured by means of a questionnaire survey as part of the large-scale School Health Promotion Studies of adolescents aged 14–16 years. In 1995, self-reported bulimia (corresponding to DSM-III-R diagnostic criteria) was detected in almost 2% of girls and 0.3% of boys and bulimic behavior in about 17% of girls and 12% of boys [328]. In 1998, the prevalence of self-reported BN (DSM-IV) as well as bulimic behavior was approximately at the same level as in 1995 [329]. At the beginning of the 21st century, a population-based study of young adults revealed the lifetime prevalence of 2% for EDNOS in females aged 20–35 years [315]. Another population-based study of young adult female twins found the lifetime prevalence of 3.9% for EDNOS and 1.5% for OSFED/UFED [330].

The rates of recovery from EDNOS vary from moderate to high. In the USA, roughly one third of the girls and more than one half of the boys with subthreshold eating disorder symptoms in mid-adolescence are found to become asymptomatic in five years [200], and three out of four females with EDNOS at the age of 20, on average, show recovery within 20 years. [309]. In Finland, 60% of the females with OSFED/UFED are found to recover within five years [330]. In partial eating disorders, the course may be even better. For example, in Australia, 85% of the females with partial syndromal eating disorder at the age of 15–17 years are found to be free from eating disorders at the age of 21–24 years [161]. In other words, in approximately two thirds of females and two out of five males with subthreshold eating disorder symptoms in mid-adolescence, the symptoms still sustain in late adolescence [200], one quarter of women with onset of EDNOS in adolescence still have it in mid-life [309], two out of five females with OSFED/UFED in late adolescence have not recovered by young adulthood, and in around every seventh case of partial eating disorders the problem continues from adolescence to young adulthood [161].

2.4.2 Eating pathology apart from eating disorders

The forms of eating pathology that do not meet the diagnostic criteria of eating disorders are numerous and so common that, to some degree, they may be considered as normative [73]. For example, dissatisfaction with body and weight is typical in adolescence [80]. In a large cross-national study of health behavior in school-aged children (HBCS), conducted in a random sample of youth in 42 countries in Europe and North America, 43% of girls and 22% of boys aged 15 years felt too fat, while 13% of girls and 22% of boys were actually overweight or obese according to their BMI. Weight reduction behavior was reported by 26% and 11%, respectively. [331.] Comparable findings of common weight loss attempts in youth are reported in several studies [e.g., 332; 333; 334; 335; 336; 337; 338].

Unfortunately, the weight control practices used by youth may be somewhat or extremely unhealthy. For example, in a population-based 10-year longitudinal study of eating among teens and young adults (EAT-III), weight control behaviors, such as fasting, eating very little food, using a food substitute, skipping meals, and smoking more cigarettes, were found in 60.7% of girls and 27.9% of boys in mid-adolescence. In young adulthood, these behaviors were still reported by over 50% of females and over 25% of males. Extreme weight control behaviors (vomiting, using diet pills, laxatives, or diuretics) were less common among mid-adolescent girls (12.6%) and boys (2.1%), but, by young adulthood, these behaviors almost doubled in females and tripled in males. [335.] In other large-scale studies, the findings of unhealthy weight control behaviors in youth, for example self-induced vomiting, has ranged from less than 4% [339; 340] to about 10 % [334; 336; 338] in girls and from less than 1% [339; 340] to around 3% in boys [334; 338].

Unhealthy weight control behaviors typically co-occur with binge eating [341]. Thus, while extreme weight control behaviors tend to increase towards adulthood, binge eating may do the same. In mid-adolescence, binge eating occurs from 7% [339] to over 15% [334; 337; 340] of girls and from 2% [339] to almost 14% [340] of boys, and towards adulthood, it is found to become more common in both genders [335]. In addition, there are findings showing a detrimental course from binge eating and compensatory behaviors in adolescence to continued or worsening problem by young adulthood in both females and males [200].

Most studies measuring eating pathology by means of the SCOFF questionnaire have used a cut-off criterion of two or more points. With this threshold, eating pathology has

been found from roughly one quarter [160; 285; 342; 343] to almost one third [342; 344; 345; 346] of girls and from one out of fifteen [345] to around one out of seven [160; 285; 342; 343; 344; 346] boys in mid-adolescence. The findings are comparable to those of early adolescent girls, whereas in boys self-reported eating pathology is found to be more common in early [282; 344] than middle adolescence [160; 285; 342; 343; 344; 345; 346]. In late adolescents and young adults, the estimated prevalence of eating pathology has varied from 1/10 in females and 1/30 in males in the USA [347] to four-fold and ten-fold rates obtained in Greece [348], respectively. (Table 4.)

In Finland, the School Health Promotion Study of 14–16-year-old adolescents conducted in 1995 reported that roughly 16% of the girls were over-concerned about body weight, i.e., they felt fat and were afraid of putting on weight. Altogether 9% of the girls used extreme methods (vomiting, strict dieting, fasting, heavy exercise, abusing laxatives/diuretics) to control weight, 8% felt fear of losing control over eating, and almost 7% reported frequent binges. These behaviors were 2–3 times more common in girls than boys with one exception: frequent binges were more typical of boys than girls. [328.] In 1998, another school-based survey of Finnish adolescents aged 13–17 years revealed that roughly 37% of the girls wanted to be thinner, 27% were dissatisfied with own body shape, 17% were dieting, and 15% often lost control over eating. Altogether 3% of the girls reported using frequent self-induced vomiting after eating. In boys, about 10% of the respondents were often dissatisfied with own body shape, desired to be thinner, and reported loss of control over eating. Compared to girls, dieting and vomiting were less common in boys. [8.]

In the early 2000's, dissatisfaction with weight or shape was reported by almost every other girl and one out of five boys (mean age 17.5 years) [314]. Among younger adolescents aged 13 and 15 years, the HBSC study revealed that, in Finland, body weight dissatisfaction was perceived by roughly 40% of girls and 20% of boys. In girls, dissatisfaction with body weight tended to increase with age, whereas among boys, the opposite occurred. [349.] Altogether 12% of non-overweight girls and 3% of boys reported current attempts to lose weight. Almost one half of the non-overweight girls had used some weight control practises during the past year. Fasting as well as self-induced vomiting was reported by roughly 10% of the girls. In boys, 10% had used some weight control practises during the past year. Fasting and self-induced vomiting were almost as common in boys as in girls. Using diet pills and/or laxatives were uncommon among both genders. [9.] In a nationally representative School Health Promotion Study conducted in 2003, binge eating with the loss of control over eating was found in about 10% of girls and 5% of boys in the 8th and 9th grades. In girls, dieting or fasting was reported by 9% of girls and 3% of boys. Intentional vomiting, and using laxatives or diuretics were uncommon. [350.] The most recent HBSC survey in 2013–2014 found that about 40% of the girls aged 13 or 15 years feel too fat, and from 18% to 24% of the girls use diet or some other methods to lose weight [331]. Corresponding findings are reported in the School Health Promotion Study in 2015. In Finnish secondary and upper secondary schools, about every third girl feels overweight. [351.] In boys, feeling too fat and trying to lose weight are about half as common as in girls [331; 351]. Feeling overweight is still common in later adolescence and young adulthood. About every third female undergraduate university student feels overweight. In males, the feeling is almost as common. However, uncontrolled dieting remains relatively rare in young adults. [352.] In the MOPO study among young men participating in the call-ups for military service in 2010, 2011 and 2013, 6.9% reported symptoms of disordered eating, i.e., drive for thinness or bulimic behavior [353].

Table 4. Prevalence of eating pathology in adolescents and young adults measured with SCOFF

Author(s)	Sample; setting, years	Age (years); gender	Cut-off point; SCOFF version	Eating pathology	Prevalence (%)
Baechle et al. [343]	Participants of the KiGGS study represent noninstitutionalized children aged ≤18y (n = 6813); Germany in 2003–06	11–17 years (mean 15.3 y); 48.7% females	≥2 points; German version of SCOFF	Disordered eating	28.9 for F 15.2 for M
				Intentional vomiting	5.0 for F 4.9 for M
				Losing control over food	29.7 for F 14.6 for M
				Lost >6kg/3 months	5.6 for F 5.6 for M
				Body dissatisfaction	31.9 for F 15.7 for M
				Food intrusive thoughts	25.6 for F 22.1 for M
Berger et al. [282]	School-based sample of early adolescents (n = 807); Germany in 2007	12 years; 53% females	≥2 points; German version of SCOFF	Disordered eating	37.4 for F 25.1 for M
Eisenberg et al. [347]	A random sample of undergraduate university students (n = 1181); USA in 2005	18–31+ years, 94% were 18–22 year-old; 57% females	≥3 points; USA version of SCOFF	EDS	13.5 for F 3.6 for M
				Intentional vomiting	13.7 for F 4.3 for M
				Losing control over eating	26.4 for F 8.4 for M
				Lost >14 pounds/3 months	4.9 for F 5.2 for M
				Body dissatisfaction	33.8 for F 9.2 for M
				Food intrusive thoughts	16.8 for F 7.1 for M
Fragkos & Frangos [348]	A random sample of students (n = 1865); Greece in 2010–11	16–26 years (mean 21.2 y); 54.5% females	≥2 points; version of SCOFF not specified	Eating disorder risk	44.6 for F 33.9 for M
Hansson et al. [285]	Students (n = 1265); Sweden in 2014	Mean 16.19 years; 54.5% females	1 point; Swedish version of SCOFF	Disordered eating	24.4 for F 14.8 for M
				Intentional vomiting	4.9 for F 1.4 for M
				Losing control over eating	18.6 for F 4.5 for M
				Lost >6.8kg/3 months	6.1 for F 6.2 for M
				Body dissatisfaction	18.8 for F 4.9 for M
				Foos intrusive thoughts	18.0 for F 10.5 for M

(continues)

Table 4 (continues)

Author(s), year	Sample; setting, years	Age (years); gender	Cutpoint; SCOFF version	Eating pathology	Prevalence (%)
Herpertz-Dahlmann et al. [160]	Youth in the BELLA study were drawn from the random sample of families participating in the KiGGS study (n = 1843); Germany in 2003–06	11–17 years (mean 14.6 y); 48.7% females	≥2 points; German version of SCOFF	Disordered eating	29.4 for F 14.4 for M
Hölling & Schlack [344]	Participants of the KiGGS study (n = 7498); Germany in 2003–06	11–17 years; gender ratio not specified	≥2 points; German version of SCOFF	EDS	Aged 11–13 y: 23.5 for F 17.8 for M Aged 14–17 y: 32.3 for F 13.5 for M
Jáuregui Lobera et al. [345]	Students (n = 318); Spain in 2007	12–18 years (mean 14.41 y); 53.83 % females	>2 points; Spanish version of SCOFF	Eating disorders	29.66 for F 6.66 for M
Nurkkala et al. [354]	Population-based sample of young men attending call-ups for military service (n = 922); Finland in 2013	17.9 y (mean); 100% males	≥2 points; Finnish version of SCOFF	Body image distortion / Fear of losing control over eating	8.8 / 8.7
Solmi et al. [355]	Population-based sample in the SELCoH study (n = 1645); UK in 2008–10	16+ years of which 21.4% 16–24-year-olds; 56.6% females	≥2 points; original version of SCOFF	Aged 16–24 y: Disordered eating / Deliberate vomiting / Loss of control over eating / Lost >1 stone/3 months / Body image distortion / Preoccupation with food-related thoughts	16.1 / 5.5 / 16.1 / 18.9 / 11.9 / 12.2
Tavolacci et al. [356]	University students (n = 3457); France in 2009–12	18–25 years, (mean 20.5 y); 57% females	≥2 points; French version of SCOFF	ED	26.4 for F 10.3 for M
Veses et al. [342]	Adolescents participating in the AFINOS study (n = 2017); Spain in 2007–08	13–17 years, (mean 14.8 y); 51.4% females	≥2 points; Spanish version of SCOFF	At risk for ED / Deliberate vomiting / Loss of control over eating / Lost >7kg/3 months / Body image distortion / Impact of food on life	32.3 for F 17.6 for M / 43.6 for F 31.9 for M / 18.8 for F 12.6 for M / 8.3 for F 9.9 for M / 28.2 for F 11.7 for M / 13.0 for F 8.9 for M

(continues)

Table 4 (continues)

Author(s), year	Sample; setting, years	Age (years); gender	Cutpoint; SCOFF version	Eating pathology	Prevalence (%)
Veses et al. [342]	Adolescents participating in the AVENA study (n = 1554); Spain in 2000–02	13–18.5 years, (mean 15.4 y); 53.3% females	≥2 points; Spanish version of SCOFF	At risk for ED	22.9 for F 12.5 for M
				Deliberate vomiting	38.6 for F 30.0 for M
				Loss of control over eating	13.2 for F 9.8 for M
				Lost >7kg/3 months	4.4 for F 4.9 for M
				Body image distortion	26.3 for F 9.0 for M
				Impact of food on life	6.1 for F 4.1 for M
Zeiler et al. [346]	School-based sample of students participating in the MHAT study (n = 3610); Austria	10–18 years; 55.3% females	≥2 points; German version of SCOFF	At risk for EDs	30.92 for F 14.63 for M
				Intentional vomiting	6.14 for F 5.12 for M
				Losing control over eating	31.75 for F 14.06 for M
				Lost >6kg/3 months	10.52 for F 12.20 for M
				Body dissatisfaction	28.15 for F 10.98 for M
				Food intrusive thoughts	31.00 for F 23.28 for M

Note: AFINOS = Physical Activity as a Preventive Measure of the Development of Overweight, Obesity, Allergies, Infections, and Cardiovascular Risk Factors in Adolescence; AVENA = Food and Assessment of the Nutritional Status of Adolescents; BELLA = Behavior and wellbeing of children and adolescents in Germany; ED = eating disorder; EDS = eating disorder symptoms ; F = female; KiGGS = Health Interview and Examination Survey for Children and Adolescents; M = male; MHAT = Mental Health in Austrian Teenagers; SELCoH = South East London Community Health

Taken together, in Western countries, a significant number of individuals experience eating pathology in adolescence and young adulthood – females more commonly than males since early adolescence [see 357]. However, the gender discrepancy in eating pathology among youth seems to be smaller than that often quoted for adults (10:1). In the reviewed studies, the female:male ratio showed wide variation ranging from 1:1 [138] to 17:1 [312; 314] for AN, from 2–4:1 [305] to 8:1 [312] for BN, and from 3:1 [138; 311 312] to 7:1 [311] for BED in adolescent and young adult populations. In addition, the ratio between girls and boys varied from 1:1 [340; 350] to 4:1 [339] for binge eating, from 4:1 [328] to 6:1 [335] for using extremely unhealthy weight control behaviors, and from 1–2:1 [8; 9] to 9:1 [335] for self-induced vomiting. Furthermore, using two or more points in SCOFF as a threshold of eating disorder risk, girls outnumbered boys with a gender ratio from 1.3:1 [344; 348] to 4.5:1 [345] for being at risk for an eating disorder. Thus, attention to both genders is warranted in future studies of eating pathology.

In this study, EDS was measured with a self-report questionnaire. This approach was chosen due to economical and practical reasons and because previous studies [e.g., 358; 359] have evidenced a higher disclosure of eating pathology in self-report questionnaires than in face-to-face interviews. The chosen approach was supported by the focus of the study being confined to the detection of eating pathology by means of screening among adolescents.

2.5   Analytical epidemiology of eating pathology

The causal mechanisms leading to eating disorders are still unknown. However, it is obvious that the origin of eating pathology is multifactorial [14; 111]. Tens of factors are found to be associated with eating disorders. Some of them are labeled as risk factors, some as protective factors, and some as correlates of eating pathology. By definition, a risk factor is any measurable characteristic, event, or experience that precedes the onset of the outcome in question and can be used to indentify individuals whose probability of the outcome is higher than its base rate in the general population [360]. Thus, knowing the risk factors may help to identify the high-risk groups for eating pathology [e.g., 361]. A protective factor is any measurerable antecedent condition that decreases the risk of an adverse outcome or increases the likelihood of a desirable outcome among high-risk individuals. Correlate, in turn, means any factor associated with the outcome in question without evidence of the temporal order. [360; 362.] Thus, a correlate may be a risk factor or a protective factor, a sign or a symptom, or a consequence of the outcome. [16].

In the following, the role of different factors in relation to eating pathology are described on the basis of previous research. The first part of this section presents previous reviews of studies investigating the potential predictors of the onset or maintenance of eating pathology. Reviews in which the methods of the study were described are included. The second part of the section consists of a new descriptive review of relevant observational studies investigating factors that may have an effect on the onset or maintenance of eating pathology. In both parts, the included factors were limited to those which reflect adolescents' experiences in order to allow the use of the findings for appropriate interventions against eating pathology within school health care. In line with this, the findings of the factors occurring prior adolescence, e.g., eating problems in early childhood, were excluded. In addition, findings based on other persons' assertions of the characteristics of an individual or the family, were not included because, instead of revealing the predictive power of the studied characteristics, the findings may reflect the predictive ability of the subjective interpretation of the observer [363]. In addition, although it is well acknowledged that vulnerability to the development and maintenance of eating pathology may partly be due to biological factors, such as genetic and neurobiological mechanisms [364] which may be activated by pubertal development [365], such factors were beyond the scope of this study and were excluded from the section.

2.5.1 Review of previous reviews

To date, there are numerous reviews describing a wide range of factors associated with eating pathology. However, only few of them focus on the risk and protective factors for the onset or maintenance of eating pathology. The most comprehensive is the descriptive review by Jacobi and co-writers [16]. It comprises about 320 cross-sectional and longitudinal studies and reviews published prior to April 2002 that investigate psychosocial and biological risk factors for the onset of eating disorders. Of all the studies included in the review, 15 were longitudinal with a follow-up period of at least one year. Ten of these had adolescent samples, while three investigated children and two focused on young adults. Risk factor status was given to factors which had a statistically significant association with the outcome in at least half of the studies. [16.] Another review discussed in this section is the meta-analytic review by Stice [366], which comprises about 70 prospective and experimental studies from 1968 to 2002. It focused on putative risk factors for an increase in or the maintenance of eating pathology. Factors investigated in at least two studies were included as risk factors. Altogether three prospective and eight

experimental studies investigated the maintenance factors of eating pathology. Two of the prospective studies were conducted among adolescents and one in a sample of young adults. Participants in experimental studies were adults. The third review by Stice and Shaw [367] assessed the effect of body dissatisfaction on the onset and maintenance of eating pathology. Prospective and experimental studies that controlled for the initial level of the outcome in question were included. Empirical findings were drawn from 25 articles. Of these, 6 papers were not included in the review of Jacobi and co-writers [16] or in the meta-analysis of Stice [366].

2.5.1.1 Predictors of the onset of eating pathology

Of the biological factors affecting the onset of eating disorders, the most consistent evidence was found for the role of gender and age. According to Jacobi and co-writers [16], findings from clinical samples and non-clinical populations consistently show that, in eating disorders, females outnumber males in the prevalence, and the peak onset of the syndromes occurs in adolescence and young adulthood. Thus, the female gender and age of adolescence were rated as risk factors for eating disorders. Findings of the role of other biological factors were less consistent. For example, in five longitudinal studies pubertal timing was not found to be predictive for later eating disturbances. In contrast, a number of cross-sectional studies using either retrospective or current reporting of pubertal timing, found a significant association between early pubertal timing and eating disorder symptomatology. The reviewers concluded that, on the basis of cross-sectional evidence, early pubertal timing is a risk factor for eating disorders, but, on the basis of longitudinal studies, it cannot be given risk factor status. Conclusions of the role of BMI and other weight related variables were based on five longitudinal studies. Two of the studies reported higher BMI being predictive of partial syndromes, while three studies found no such evidence. Accordingly, the reviewers inferred that higher BMI cannot be classified as a risk factor for eating disorders. The role of ethnicity on the development of eating disorders was assessed on the basis of the rates of eating pathology in people with different ethnic origin. While the findings varied between studies, ethnicity (other than Asian) was classified as a risk factor, the effect of which depends on the cultural context and the type of an eating disorder. Male homosexuality, although reported to be associated with a number of symptoms related to eating disorders, was classified as a noncorrelate on the grounds that full-blown eating disorders have not been found to be higher among homosexual men compared to heterosexual males. [16.]

Among psychological and behavioral factors, the most consistent evidence of the risk effect on the onset of eating disorders was reported for a factor named weight concerns including the fear of weight gain, negative body image, dieting, and specific eating disorder symptoms and attitudes. It was found to predict the development of eating disturbances in nine out of ten longitudinal studies, which controlled for initial eating symptomatology. [16.] Additional three out of four prospective studies and two experimental trials were found to support the effect of body dissatisfaction on bulimic pathology [367]. As a result, body dissatisfaction [367], elevated weight concerns, negative body image, and dieting were given a risk factor status in relation to eating disorders [16] and bulimic pathology [367]. Low self-esteem was found to be predictive for the onset of an eating disorder in two out of four studies. Ineffectiveness and low interoceptive awareness were found to predict eating disorders in one out of four studies, respectively. Taking into account findings from univariate analyses supporting the presence of low self-esteem, higher ineffectiveness, and low interoception prior to the onset of an eating disorder, the reviewers classified these factors as risks for eating disorders. However, they highlighted the need for more

studies to confirm the conclusion. Seven longitudinal studies were used to assess the effect of general psychiatric disturbance and negative emotionality on eating disorders. In three of the studies, a significant predictive effect of these factors was found on later eating disorders, whereas four studies reported nonsignificant effects. Considering also univariate comparisons, the reviewers rated prior psychiatric morbidity and negative emotionality as risk factors for eating disorders. In four longitudinal studies investigating a relationship between perfectionism and eating disturbances, no evidence was found of the predictive role of perfectionism on subsequent eating disturbances. However, a significant association between perfectionism and co-occuring eating disorders was obtained in cross-sectional studies as well as in univariate analyses at baseline assessment in longitudinal studies. Thus, perfectionism was rated as a correlate of eating disorders. [16.]

Assessment of behavioral predictors other than dieting was limited to those concerning physical activity, health risk behavior, and coping strategies. All but one of the studies of athletic competition, participation in the weight-related subculture, and exercise were cross-sectional by nature. The only prospective study did not provide information enough for the assessment of the risk effect of esthetic sports on the onset of eating disorders. Thus, based on cross-sectional studies, the reviewers classified athletic competition or participation in a competitive weight-related subculture as a correlate of an eating disorder. The high level of exercise was rated as a retrospective correlate. Studies with larger sample sizes and prospective designs were called for in order to obtain more precise findings. The higher consumption of alcohol, higher scores indicating aggressive as well as unpopular behavior, and the high use of escape-avoidance coping were rated as risk factors for eating disorders on the basis of one prospective study per each factor. Attention was called to a need of replication studies. [16.]

Evidence of the effect of environmental factors on the onset of eating disorders was drawn mainly from cross-sectional studies. The role of acculturation in the development of an eating disorder was found to depend on the cultural context and the type of eating pathology. Based on cross-sectional studies, it was rated as a retrospective correlate. Only cross-sectional studies were also used in the assessment of the association between adverse life events and a later onset of eating disorders. Although the findings in the seven included studies differed, depending on the contrasted groups (e.g., AN patients vs. psychiatric patients) and severity as well as the temporal distance of an adverse event, the reviewers found evidence enough to rate general adverse life events as retrospective correlates for eating disorders. The assessment of the effect of sexual abuse on later eating disorders was based on one prospective and five cross-sectional studies. The only longitudinal study evaluated childhood adversities as potential predictors for later problems with eating or weight. Of the cross-sectional studies, two focused on sexual abuse in childhood and three were reported without information of the age of participants at the time of the occurrence of sexual trauma. On the basis of the longitudinal study, the reviewers classified sexual abuse as a risk factor for eating disorders, reminding, however, the readers of the need for further prospective studies. The rating was supported by four out of five cross-sectional studies, which found an association between prior sexual abuse and an eating disorder. [16.]

The effect of family structure or functioning on later eating problems was assessed on the basis of four longitudinal studies. Only one of these reported that communication, roles, or general functioning in the family was associated with later eating disturbances. In this study, initial eating disturbances were not taken into account. In the other studies, family relationships did not predict later eating problems. Thus, family relationship factors were

not given a risk factor status but, on the basis of cross-sectional studies, they were rated as correlates of eating disorders. Low perceived social support was rated as a risk factor for subsequent eating disorders based on one prospective study and with a note of the need for replication. Teasing, in turn, was not given any role in relation to eating disorders owing to the lack of appropriate studies. [16.]

2.5.1.2 Predictors of the maintenance of eating pathology

The number of factors assessed for their effect on the maintenance of eating pathology was small and so was the number of studies used in these review assessments. Of the biological factors, the individual's initial body mass was rated not to be a maintenance factor for eating pathology. The conclusion was based on two prospective studies. [366.] Of the psychological and behavioral factors, body dissatisfaction was found to predict the maintenance of bulimic symptoms in one prospective study [366; 367]. Support for this was found in another prospective study where weight and shape concerns were found to predict the persistence of bulimic symptoms [367]. Contradictory findings were not reported. Thus, body dissatisfaction was classified as a maintenance factor for bulimic pathology. Based on findings from one prospective study, perfectionism was also rated as a maintenance factor for eating pathology. In two prospective studies, thin-ideal internalization was found to predict the maintenance of bulimic symptoms. One of these studies provided support for the predictive role of negative affect as well, in contrast to another study in which the association remained nonsignificant. Taking into account two studies where negative affect was experimentally manipulated, the negative affect was rated as a causal maintenance factor for binge eating but not for caloric intake among people with an eating disorder. The predictive role of dieting was found to be supported in one prospective study, but not in another one. Contradictory findings from experimental studies exist as well. In three studies, experimentally manipulated dieting was found to increase caloric intake and binge eating among participants with BN and BED. On the other hand, in four experimental studies conducted in the natural environment, long-term low-calorie diet was found to decrease binge eating among obese participants with BED. As a conclusion, dieting was rated not to be a maintenance factor for eating pathology. The only environmental factor, sociocultural pressure to be thin, was rated as a nonsignificant maintenance factor for bulimic pathology on the basis of one prospective study. [366.]

To conclude, 17 factors were given a risk factor status in relation to the onset of eating disorders, whereas four potential predictors were found to be non-risk factors. None of the factors were classified as a causal risk factor. Altogether three predictors were rated as maintenance factors, one was classified as a causal maintenance factor, and two remained nonsignificant in terms of the maintenance of eating pathology. Some of the factors were rated both as risk and maintenance factors for eating pathology, whereas some were given a risk factor but not a maintenance factor status, or vice versa. On the basis of cross-sectional studies, five potential risk factors were classified as correlates or retrospective correlates. Due to the lack of appropriate studies, the predictive role of several putative exposures was not assessed.

In general, the number of studies reviewed to assess the putative predictors for the onset of eating disorders exceeded the number of those used to estimate the maintenance factors of eating pathology. For some factors rated as a risk for eating disorder onset, the rating was based on consistent findings from several prospective studies, but more often, the number of longitudinal studies included in the review was small and the findings were inconsistent. In several cases, the risk factor status was based on a single longitudinal

study besides cross-sectional findings. In addition, for some factors, all prospective evidence of the risk effect was based on findings among children. In such cases, caution is warranted for the generalization of the results to adolescents. Similarly, ratings which based on retrospective findings among eating disorder patients may not be generalizable to people not seeking treatment. Furthermore, while the majority of the prospective studies included girls only or the assessment was made without gender stratification, caution is warranted in generalizing the results to boys. Studies that were used to review the maintenance factors of eating pathology were even more limited. Most of the factors were classified on the basis of only one or two prospective studies, in which the reviewer himself was the first or the second author. In addition, all studies were conducted among female samples mostly comprised of adults. Taking into account that the effect of different factors on eating pathology may differ between genders and change during the course of development [16], caution is warranted in generalizing the results to males and adolescents. As stated by the reviewers, more studies are needed to confirm the findings. Based on the extensive work of the previous reviewers, a new review was performed to find further evidence of the etiology of eating pathology and to override some of the limitations discussed above.

2.5.2 New descriptive review

The aim was to review the research of individual and environmental factors that have an effect on the onset and/or maintenance of eating pathology among adolescents in Western countries. The search for the review focused on definitive risk studies with an interest in the development and natural course of eating pathology. Therefore, cross-sectional and retrospective studies, clinical trials, and studies among patients were excluded. All relevant population and community based studies with a prospective design were considered with no publication date restrictions. However, studies conducted in specific groups (e.g., ballet dancers) were excluded to uphold comparability between studies. In addition, because eating pathology is a culture-bound problem, that is, its presentations and associated factors vary between cultures [306], only studies undertaken in Western countries were considered in order to enhance comparability between studies and to avoid construct bias, which would be present if eating pathology was not an equally valid phenomenon across countries [368]. The length of the follow-up period was set the minimum of one year for two reasons; because the probability of the recurrence of eating pathology is found to reach a steady rate after a year [369], and because one year was the follow-up period used in the second phase of the present study. Unpublished material was not considered.

Studies conducted among adolescents were emphasized, although the search was extended into young adulthood, as recommended by previous reviewers [120]. Studies with heterogenous samples representing a wide range of age were included if the analyses were conducted separately for different age groups (children, adolescents, adults). This was warranted because the effect of different factors on eating pathology may change during the course of development [370; 371; 372]. Although the factors affecting the course of eating pathology may differ between genders [373], gender stratification was not used as an inclusion criterion in order to enable the comparison of the findings with those obtained in the second phase of the present study.

The review was limited to studies investigating risk and protective factors occurring in adolescence. This restriction was based on the view of adolescence as a critical period during which the aggregation and interaction of several risk and protective factors have strong influence on whether a person develops or avoids eating pathology [121].

The outcome used in the review was self-reported eating pathology. Those studies were included in which similar methods and reference standards to ascertain the outcome were used at baseline and follow-up assessments. No other restrictions on the measurement of eating pathology were imposed. Studies which reported the size of the effect of exposures, with 95% confidence intervals as recommended in the PRISMA statement [374], were considered. To describe the predictors of the onset of eating pathology, those studies were included in the review in which participants who reported eating pathology at baseline were excluded from the analyses. Thus, the findings of these studies result from differences between participants who become cases and those who remain non-symptomatic. In line with this, the description of the predictors of the maintenance of eating pathology included studies of individuals who reported eating disorder symptoms already at baseline assessment. Accordingly, these results are based on differences between individuals with prolonged or recurring eating pathology versus the recovered ones. Studies where all participants were analyzed as one group, despite their level of eating pathology at baseline, were excluded because they reflect the possible change in eating pathology instead of the onset or maintenance of the problem.

There are no universal rules for the appraisal of studies. Instead, it is generally accepted to develop assessment criteria for each review [375] giving priority to the assessment of key sources of bias [376]. In this review, the methodological quality of studies was assessed on the basis of actions taken during sampling, data collection, and analyses to control for confounding and to limit bias.

Studies were identified by searching six electronic databases and screening the reference lists of all included papers. Search was applied to EMBASE (from <1966), PsychINFO (from 1806), SCOPUS (from 1960), and Web of Science (from 1900) on 15 August 2016, and to CINAHL (from 1981) and Medline (via PubMed, from 1946) on 17 August 2016. The search was limited to articles of original studies. The search strategy was based on the outcome variable, explanatory variables, and the study design. For example, in Medline, titles and abstracts were searched using the following terms and phrases (in quotes) of the outcome variable: eating disorder (MeSH Terms), "eating disorder", "eating pathology", "abnormal eating", "atypical eating", "disordered eating", "disturbed eating", "eating disturbance", "eating problems", "eating disorder not otherwise specified", EDNOS, "binge eating", anore*, and bulimi* (Text Word). The asterisk (*) is used to search for all terms that begin with the root term but continue with multiple endings (i.e., noun, adjective, or plural forms). Terms and phrases selected for explanatory variables were as follows: causality and etiology (MeSH Terms), causal*, "risk factor*", predict*, predispos*, protect*, prevent*, precipita*, and etiology (Text Word). The appropriate study design was searched with the following terms: longitudinal studies, prospective studies, follow-up studies (MeSH Terms), follow-up, longitudinal, and prospective (Text Word). Besides the search field (title/abstract), the following filters were used in the search: journal article, humans, English, adolescent, and young adult. The Boolean operators ('AND", "OR") were used to combine the search terms as described in Table 5. The same terms and phrases were searched in CINAHL, EMBASE, PsychINFO, SCOPUS, and Web of Science.

Table 5. Search strategies

Database	Search terms and filters/limitations	Results
Medline	Search field: title/abstract 1. eating disorder (MeSH Terms) 2. "eating disorder" OR "eating pathology" OR "abnormal eating" OR "atypical eating" OR "disordered eating" OR "disturbed eating" OR "eating disturbance" OR "eating problems" OR "eating disorder not otherwise specified" OR EDNOS OR "binge eating" OR anore* OR bulimi* (Text Word) 3. causality OR etiology (MeSH Terms) 4. causal* OR "risk factor*" OR predict* OR predispos* OR protect* OR prevent* OR precipita* OR etiology (Text Word) 5. follow-up studies OR longitudinal studies OR prospective studies (MeSH Terms) 6. follow-up OR longitudinal OR prospective (Text Word) 7. (#1 OR #2) AND (#3 OR #4) AND (#5 OR #6) 8. #7 filters: Article type: journal article; Species: humans; Languages: English; Ages: adolescent (13–18 years), young adult (19–24 years)	1 487
Web of Science	Searched in topic: "eating disorder" OR "eating pathology" OR "abnormal eating" OR "atypical eating" OR "disordered eating" OR "disturbed eating" OR "eating disturbance" OR "eating problems" OR "eating disorder not otherwise specified" OR EDNOS OR "binge eating" OR anore* OR bulimi* AND causal* OR "risk factor*" OR predict* OR predispos* OR protect* OR prevent* OR precipita* OR etiology AND follow-up OR longitudinal OR prospective AND adolescen* OR "young adult*" Limitations: Research domains: social sciences, science technology; Document type: article; Selected language: English; Research areas: behavioral sciences, family studies, health care sciences services, nursing, nutrition dietetics, pediatrics, psychology, women's studies	787
SCOPUS	"eating disorder" OR "eating pathology" OR "abnormal eating" OR "atypical eating" OR "disordered eating" OR "disturbed eating" OR "eating disturbance" OR "eating problems" OR "eating disorder not otherwise specified" OR EDNOS OR "binge eating" OR anore* OR bulimi* AND causal* OR "risk factor*" OR predict* OR predispos* OR protect* OR prevent* OR precipita* OR etiology AND follow-up OR longitudinal OR prospective OR cohort Limitations: Subject area: medicine, psychology, neuroscience, nursing, social sciences, health professions; Document type: article, article in press; Language: English; Source type: journal; Keyword: human, humans, adolescent, adolescents, young adult, follow up, follow-up studies, prospective studies, prospective study, longitudinal studies, longitudinal study, cohort studies; Exclude keyword: aged, aged 80 and over, middle aged, preschool child, child preschool, infant, major clinical study, clinical article, clinical trial, randomized controlled trial, controlled clinical trial, cross-sectional studies, cross-sectional study, retrospective studies, retrospective study, treatment outcome, feces incontinence, fecal incontinence	601

Table 5 (continues)

Database	Search terms and filters/limitations	Results
PsychINFO	Searched in abstract: eating disorder" OR "eating pathology" OR "abnormal eating" OR "atypical eating" OR "disordered eating" OR "disturbed eating" OR "eating disturbance" OR "eating problems" OR "eating disorder not otherwise specified" OR EDNOS OR "binge eating" OR anorexia OR anorectic OR anorectics OR bulimi* AND causal* OR "risk factor*" OR predict* OR predispos* OR protect* OR prevent* OR precipita* OR etiology Limitations: Peer reviewed; Record type: journal article; Population: human; Methodology: follow-up study, longitudinal study, prospective study; Language: English; Age group: adolescence (13–17 years), young adulthood (18–29 years);	536
EMBASE	Searched in abstract/title: ("eating disorder" OR "eating pathology" OR "abnormal eating" OR "atypical eating" OR "disordered eating" OR "disturbed eating" OR "eating disturbance" OR "eating problems" OR "eating disorder not otherwise specified" OR EDNOS OR "binge eating" OR anore* OR bulimi*) AND (causal* OR "risk factor*" OR predict* OR predispos* OR protect* OR prevent* OR precipita* OR etiolog*) AND (follow-up OR longitudinal study OR prospective) Study types: longitudinal OR prospective Limits: Publication types: article, article in press; Humans; Only in English; Age: adolescent (13–17 years), young adult (18–24 years)	233
CINAHL	Searched in abstract/title: 1. eating disorder" OR "eating pathology" OR "abnormal eating" OR "atypical eating" OR "disordered eating" OR "disturbed eating" OR "eating disturbance" OR "eating problems" OR "eating disorder not otherwise specified" OR EDNOS OR "binge eating" OR anore* OR bulimi* 2. causal* OR "risk factor*" OR predict* OR predispos* OR protect* OR prevent* OR precipita* OR etiology 3. follow-up OR longitudinal study OR prospective #1 AND #2 AND #3 Limitations: peer reviewed; research article; journal article; academic journals; human, English language; adolescent (13–18 years)	186

Data of all relevant articles were extracted and tabulated on a pilot-tested sheet on the details of the putative predictors (i.e., factors and measures), publication (authors and year of publication), the sample of the study (sample size, gender division, and mean or range of age of the participants), the duration of the follow-up, the outcome (types of eating pathology and measures), and findings (odds ratios with 95% confidence intervals).

The search provided altogether 3830 records: 1413 in Medline, 787 in Web of Science, 601 in SCOPUS, 536 in PsycINFO, 233 in EMBASE, and 186 in CINAHL. Of these, 1077 records were found in more than one databases, which reduced the number of potentially relevant articles to 2753. The selection of studies for review, as described in Figure 1, was conducted in four phases. In the first phase, the titles and abstracts of the retrieved articles were screened against the inclusion criteria to exclude irrelevant papers (n = 2592). In the second phase, the full papers of all potentially relevant studies (n = 161) were obtained and examined in more detail for inclusion. Of these, 13 articles met the inclusion criteria. In the third phase, a manual search was performed of the bibliographies of the eligible articles, which revealed one more study that met the inclusion criteria. In the fourth phase, the reports that were eligible for inclusion were further assessed in terms of duplicate data. A study was considered a duplicate if the same data had been used in another study that also fulfilled the inclusion criteria for the review. Consideration was also focused on studies where the data was drawn from the same cohort. Multiple reports of the same cohort were not considered as duplicates if they provided findings with the follow-up periods of different

length or with different explanatory or outcome variables. In the case of a duplicate study, the most recent report was selected. Finally, altogether 14 reports concerning 6 study projects were identified for inclusion in the review.

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┌─────────────────────────────────┐
│ Altogether 3830 records identified │
│ through CINAHL, Embase, PsycINFO, │
│ PubMed, SCOPUS, and Web of       │
│ Science searching                │
└─────────────────────────────────┘
                │          ┌──────────────────────────────────┐
                ├─────────▶│ Duplicate records (n = 1077) removed │
                │          └──────────────────────────────────┘
┌─────────────────────────────────┐
│ Potentially relevant articles (n = 2753) │
│ screened on basis of title/abstract │
└─────────────────────────────────┘
                │          ┌──────────────────────────────────┐
                ├─────────▶│ Papers excluded after evaluation of │
                │          │ title/abstract (n = 2592)        │
                │          └──────────────────────────────────┘
┌─────────────────────────────────┐
│ Full-text articles (n = 161) assessed │
│ for eligibility                  │
└─────────────────────────────────┘
                │          ┌──────────────────────────────────┐
                │          │ Articles excluded (n = 148):     │
                │          │ - no relevant outcome (n = 104)  │
                │          │ - no relevant measurement or report of │
                │          │   the results (n = 16)           │
                ├─────────▶│ - no relevant sample (n = 15)    │
                │          │ - no relevant exposure (n = 5)   │
                │          │ - follow-up < 1 year (n = 5)     │
                │          │ - not prospective study (n = 2)  │
                │          │ - duplicate data (n = 1)         │
                │          └──────────────────────────────────┘
┌─────────────────────────────────┐
│ Articles included in the review (n = 13 │
│ + 1 additional paper identified by │
│ screening the bibliographies of the │
│ identified articles)             │
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Figure 1. Selection of studies for review

2.5.2.1 Description of the reviewed studies

The papers selected for the review were published in 1999–2015. Most of them were based on prospective studies conducted in the USA; only one took place in Australia [372]. Five articles [339; 377; 378; 379; 380] reported the findings of the Growing Up Today Study (GUTS), a large prospective cohort study among the children of female nurses participating in the Nurses' Health Study II. GUTS was established in 1996 in USA to investigate the association of diet and activity to excessive weight gain. Four articles [204; 333; 381; 382] reported the results of Project EAT (Eating Among Teens), a large longitudinal study of eating behaviors, weight outcomes, and related psychosocial factors among youth in the USA. EAT was established in the academic year of 1998–1999 at 31

schools in Minnesota, USA. Two articles [383; 384] reported the findings of a longitudinal community-based study established at eight schools in a metropolitan area in the Southwestern United States. The time of the onset of the study was not reported. One paper [385] presented the findings of the National Longitudinal Study of Adolescent Health (Add Health), the largest national study of adolescents in the USA. The Add Health was founded in 1994–1995 with a representative sample of 132 schools. One article [386] reported the results of a prospective study of health and eating patterns in young adults conducted in two non-clinical samples of late adolescents recruited in 1982 and 1992 in the USA. Findings from Australia were reported in a paper [372] presenting the results of the Western Australian Pregnancy Cohort (Raine) Study, a population-based cohort study of the offspring of women recruited through antenatal booking clinics in 1989–1991. The participants were followed up from prebirth to young adulthood.

In two studies, the sampling procedure included randomization. In the Add Health study [385], participants were recruited from a random sample of 80 high schools and 52 middle schools, and in the study by Keel and Heatherton [386], the participants were randomly selected from one college [387]. All other studies used non-random sampling. In GUTS, sampling was restricted to children aged 9–14 years at the time the study was initiated [377]. In one project [383; 384], sampling was restricted to girls, and in two papers [377; 380], the analyzed sample included girls only.

In the reviewed studies, data were collected by means of self-report questionnaires and/or diagnostic interviews. In GUTS, questionnaires were posted to participants every 12–24 months [380]. In Project EAT, an in-class survey was conducted at baseline and postal surveys were used at the follow-ups with 5-year intervals [335]. Two papers [383; 384] used data that were collected annually with a self-report questionnaire and a structured interview on the school campus or at participants' home. In the Add Health study, in-home surveys were conducted with an interval of seven years [385]. Ten-year follow-up period was used in the study by Keel and Heatherton [386] where, after the baseline survey, a two-stage case-control design was applied at follow-up. In the Raine Study, adolescents responded to self-report questionnaires three times with three-year intervals. In the articles included in the review, the follow-up periods ranged from one to ten years.

Predictors

The factors investigated in association with eating pathology were divided into individual and/or environmental factors. The individual category comprises biological, psychological, and behavioral factors. The environmental category centers on interpersonal elements by focusing on an individual's relationship with the family and friends. Two projects (GUTS and EAT), used several individual and environmental factors as putative predictors of eating pathology, while in other projects, the number of predictors was smaller [372; 384] or focused on individual characteristics [383; 385; 386]. Of the individual factors, biological factors, were often used as covariates in the analyses. Of the psychological factors, the most commonly reported were the effects of depressiveness and weight concerns. The effects of adolescent's feelings, beliefs, and thoughts about oneself, own body, and weight on eating pathology were also discussed in several papers, whereas participants' thoughts about health were used as a predictor in one paper only. Behavioral factors reported in the studies included those concerning eating, substance use, physical exercise, and sedentary behavior. Of these, the effect of dieting was the most commonly reported one.

The majority of the environmental factors concerned others' behaviors and attitudes, such as teasing and negative comments about weight. Effects of potential protective factors, such as the frequency of family meals and family connectedness, were less reported.

Outcomes

The types of eating pathology investigated as the outcomes were binge eating, purging, bulimic pathology, full-blown eating disorders, unhealthy weight control behaviors, dieting, extreme weight loss behaviors, and overeating. Only three papers reported on the effect of factors on the maintenance of eating pathology, i.e., binge eating, bulimic syndrome, and unhealthy weight control behaviors.

In GUTS, the questions measuring dieting, binge eating, and purging in the past 12 months were adapted from the Youth Risk Behavior Surveillance System questionnaire [388] developed by the Centers for Disease Control and Prevention (CDC) to monitor the most important health risk behaviors among youth, icnluding unhealthy dietary behaviors [389]. Dieting was assessed with one question about the frequency of dieting to lose weight or to avoid weight gain. The response options ranged from less than once a month to every day. Those who reported having dieted at least once a week were classified as frequent dieters. Binge eating was ascertained with a two-part question; first, asking about the frequency of the behavior in question with response options ranging from "never" to "more than once a week", and secondly, asking about the feeling of out of control during the eating episode [339]. Binge eating was defined as eating a very large amount of food in a short period of time and having the feeling of out of control during the eating episode at least weekly [379] or monthly [378; 380] or at least once in the past year [339]. Overeating was defined as eating a large amount of food in a short period of time at least once a month but without losing control over eating [380]. Purging was assessed with two questions concerning the frequency of the use of laxatives and vomiting, with response options ranging from "never" to "daily" [339]. Purging was defined as using either laxatives or self-induced vomiting to control weight at least weekly [379] or monthly [377; 378] or at least once in the past year [339]. The questions were validated in the GUTS cohort [379].

In Project EAT, researchers developed a survey to assess multiple factors of potential relevance to nutritional health and obesity among adolescents [390]. In the Project EAT survey, binge eating with the loss of control was assessed with two dichotomous (yes/no) questions. Binge eating with the loss of control was defined as ever in the past year eating so much food in a short period of time that would be embarrassed if were seen by others, and feeling unable to stop eating or control the amount of or type of food during eating this way. [204; 333; 381.] Unhealthy weight control behaviors were measured with one question about the use of nine different weight-loss methods (fasting, eating very little, using a food substitute, using laxatives, skipping meals, smoking more cigarettes, taking diet pills, making oneself vomit, using diuretics) with dichotomous (yes/no) response options for each method. Unhealthy weight control behaviors were defined as having engaged in at least one of the given weight-loss methods to lose weight or avoid weight gain in the past year. [333; 382.] The frequency of dieting was ascertained with a single question, with response options ranging from "never" to "I am always dieting". Participants were classified as dieters unless they chose the response option "never" [381.] Frequent dieting was defined as going on a diet at least five times a year. [333]. The reliability of the survey was tested at the baseline of the study [381].

In the article by Stice and co-workers [383], bulimic pathology was assessed with the Eating Disorder Examination (EDE), a semi-structured psychiatric interview designed by

Cooper and Fairburn [391] to assess the specific psychopathology of eating disorders. Bulimic pathology was assessed on the basis of the diagnostic criteria for BN according to DSM-IV [63]. It was defined as the presence of all the symptoms of BN in the past year at threshold or subthreshold level. In the paper by Rhode and co-workers [384], eating disorders (DSM-5) were measured by means of the Eating Disorders Diagnostic Interview (EDDI) adapted from EDE [391]. The reliability of the measures was tested in a subsample of participants.

In the Add Health study, dieting was measured with one question (yes/no) concerning dieting in the past seven days to lose weight. Extreme weight control behaviors were assessed with three items assessing the use of diet pills, laxatives, or vomiting in the past seven days to lose or control weight. Affirmative response to at least one of the items indicated the use of extreme weight control behaviors. The reliability and validity of the measures were not reported. [385.]

In the study of Keel and Heatherton [386], bulimic symptoms were measured with the Eating Disorder Inventory (EDI) designed by Garner and co-workers [267] and with a set of questions assessing BN symptoms (DSM-III-R) for establishing the diagnosis of the bulimic syndrome. The bulimic syndrome consisted of BN, BN-type EDNOS, BED-type EDNOS, and/or purging disorder. BN diagnosis was set in the presence of current binge eating with lack of control over eating and purging behaviors at least once a week to control weight, and overconcern with weight or shape. The criteria of subthreshold BN-type EDNOS consisted of overconcern with weight or shape, recurrent binge-eating, and compensatory behaviors not fulfilling the criteria of a full-blown BN. The criteria of BED-type EDNOS included binge eating at least once a week with worry and lack of control over eating. Purging disorder, in turn, consisted of self-induced vomiting, using laxatives, and/or diuretics at least once a week to control weight, and overconcern with weight or shape. Survey-based bulimic syndrome diagnoses were confirmed at the follow-up using the Structured Clinical Interview for DSM-IV Axis I Disorders [392].

In the Raine Study [372], eating disorder symptoms were measured by means of 24 self-report items adapted from the Eating Disorder Examination-Questionnaire (EDE-Q) designed by Fairburn and Beglin [252]. The response options ranged from "not at all" to "most of the time". Diagnoses of BN and purging disorder (DSM-5) based on the participants' responses to the items and information of height and weight. For the psychometric properties of the survey, the researchers referred to the findings of other studies.

Confounding and bias

In all studies, the risk of bias was limited by using the original methods and reference standards to ascertain the outcome throughout the study. Other actions to control confounding and limit bias were taken during sampling and data analyses and were slightly different between the studies.

In GUTS, the confounding effect of age was limited by age-restricted sampling and by controlling the age effect in multivariable analyses [339; 377; 378; 380]. In addition, in one article [379] the researchers tested interactions between the age-group variable and other predictors with respect to outcomes and reported age-stratified results when appropriate. The confounding effect of gender was controlled either by including girls only in the study [377; 380], or by analysing girls and boys, separately [339; 378; 379]. Other potential confounders considered in GUTS were weight status [378; 380], Tanner stage of pubic

hair development [377; 378], age at menarche, follow-up time [380], race/ethnicity [339], dieting [378; 379], and maternal history of an eating disorder [377; 379]. In this review, the findings of Field and co-workers [377] are presented for models showing the effect of each predictor adjusted separately for age and the Tanner stage of pubic hair development. Attrition, a potential source of selection bias, ranged from 9% [378] to 19% [377] among girls and from 12% [378] to 28% [377] among boys at 1-year follow-up. In the 3-year follow-up of females [380], attrition ranged from 8% to 12%. At 7-year follow-up, drop-out rates were 23% in females and 28% in males [379]. In one article [339], drop-out rates were not specified. None of the papers reported attrition analyses. Missing data, another potential risk of selection bias, was handled with the deletion approach, i.e., participants with missing values were excluded from the analyses.

In Project EAT, the sample included two age cohorts of adolescents with a difference of approximately three years between the younger and the older. Three papers [204; 381; 382] took into account the confounding effect of age by controlling the cohort in the analyses. In one paper [333], the researchers investigated interactions between the age cohort and predictors. As the results indicated no need for cohort-stratified analyses, the final models were estimated as age-adjusted. Other anthropometric and demographic factors controlled in EAT were sex [204], weight status [382], overweight status [381], BMI [333], ethnicity/race [204; 333; 381; 382], and socioeconomic status (SES) [204; 333; 381]. Three papers [333; 381; 382] reported the results of gender-stratified multivariable analyses. Two papers [333; 381] fitted multivariable models with all predictors adjusted for the confouders presented above. In two other papers [204; 382], the models estimated each predictor, separately, including the variable of change in the predictor in question. These models were adjusted for potential counfounders as well. Drop-out rates ranged from 47% at 5-year follow-up [333; 382] to 60% at 10-year follow-up [204; 381]. All papers reported non-random attrition from the baseline sample to follow-up. To limit selection bias, data were weighted using the propensity method. Missing data for predictor variables were handled by multiple imputation in one article [381]. In other papers [204; 333; 382], missing data were not discussed.

In the article by Stice and co-workers [383], it was verified that age, ethnicity and parental education had no significant effect on the outcome. Thus, they were not controlled in the analyses. While the sampling was restricted to girls only, gender was not a confounding issue. As a result, the multivariable model consisted of two predictors and the outcome variable. Attrition was 1% at 2-year follow-up and occurred randomly across the background, predictive and outcome variables. Missing data were not discussed. Rhode and co-workers [384], in turn, presented bivariable associations between each predictor and the outcome variable. During the 7-year follow-up period, the attrition rates ranged from 1% to 6% in annual assessments. Attrition was not associated with the predictors and outcome variable. Missing data were managed with multiple imputation. [384.]

In the Add Health study [385], the analyses were controlled for the participants' age, race/ethnicity, parents' education, family structure, and BMI. Prospective multivariable models, which included all predictors as presented in Tables 6 and 7, were fitted as gender stratified due to findings in previous studies and interaction between the gender variable and one predictor with respect to outcome variables. Attrition was not discussed. Missing data were handled with the deletion approach.

In the study of Keel and Heatherton [386], the sample consisted of two cohorts recruited with a 10-year interval. This was taken into account in the multivariable analyses by controlling for the cohort. Sex was used as one of the predictors in the analyses, thus

providing sex-adjusted results for all other predictors in the multivariable analyses. In the 10-year follow-up assessment, the attrition rate was 18% among females and 27% in males contacted for follow-up survey, and 31% among those called for clinical interviews. Missing data were not discussed.

In the Raine Study [372], the number of early adolescent predictors of a later onset eating pathology was large. Therefore, the researchers conducted the analyses in three steps: first, they examined bivariate associations between each predictor and the outcome, then, they fitted domain-specific models including variables that were significant ($p < 0.10$) in the first step, and finally, they performed multivariable analysis using predictors that remained significant ($p < 0.05$) in step two. The final analysis was controlled for sex. Since parental reports of the child's externalizing problems and maternal mental health problems were included in the multivariable analysis, the findings concerning predictors based on the adolescents' responses are adjusted for these two variables. The missing data of the outcome variables were handled by means of EM imputation.

2.5.2.2 Predictors of the onset of eating pathology

The results of the studies on factors associated with the onset of eating pathology are displayed in Tables 6–9. In general, estimates for boys were less reported and based on a smaller number of cases than those for girls due to the lower incidence of eating pathology among boys than girls. The role of individual factors was reported in 13 of the 14 articles. Biological factors were used as predictors in five articles. In line with the previous review [16], female gender was found to be associated with the onset of binge eating and purging disorders in the Raine Study, which followed participants from middle to late adolescence [372]. In contrast, a study [386] investigating late adolescents till young adulthood failed to find any gender effect on the onset of bulimic syndrome. BMI was shown to be associated with the onset of binge eating and purging disorders [372], dieting, and extreme weight loss behaviors [385], but not with the bulimic syndrome [386] or DSM-5 eating disorders [384]. However, in individuals reporting weight suppression in young adulthood, the probability of the onset of bulimic syndrome was higher than in others [386]. The effect of the menarcheal status of girls and the height of boys on the onset of weekly binge eating and/or purging was assessed in one paper [379]. Age-stratified analyses showed that the odds of the onset of purging were higher among postmenarcheal than premenarcheal girls younger than 14 years. In older girls, associations remained nonsignificant. In boys, the association between height and the onset of binge eating was nonsignificant. (Table 6.)

Table 6. Associations of biological factors with the onset of eating pathology in the prospective studies of non-clinical populations

Factor, measure	Study	Sample size (% of females)	Age in years, mean	Follow-up years	Eating pathology outcome (measure)	OR (95% CI)
BMI / change in BMI						
Measured height and weight	Allen et al. [372]	1 383 (51%)	14.01	3–6	DSM-5 BE and purging eating disorders (EDE-Q)	1.08 (1.02–1.12)
Self-reported height and weight	Keel & Heatherton [386]	1 337 (72%)	20	10	Bulimic syndrome (EDI and a separate set of questions for DSM-III-R BN symptoms)	1.07 (0.93–1.23) Weight supression: 1.05 (1.01–1.08)
Self-reported height and weight	Liechty & Lee [385]	14 322 (50.9%)	15.9	7	Dieting (1 item, yes/no) Extreme weight loss behaviors (3 items, yes/no)	1.74 (1.58–1.92) for F 2.52 (2.20–2.89) for M 1.48 (1.28–1.73) for F 2.52 (1.92–3.31) for M
Measured height and weight	Rhode et al. [384]	496 (100%)	13.5	7	DSM-5 eating disorders (EDDI)	1 SD change: 1.16 (0.63–2.12) for 13y 1.08 (0.73–1.60) for 14y 1.20 (0.84–1.70) for 15y 1.14 (0.84–1.52) for 16y
Female gender						
	Allen et al. [372]	1 383 (51%)	14.01	3–6	DSM-5 BE and purging eating disorders (EDE-Q)	5.06 (2.88–8.89)
	Keel & Heatherton [386]	1 337 (72%)	20	10	Bulimic syndrome (EDI and a separate set of questions for DSM-III-R BN symptoms)	1.28 (0.43–3.79)
Postmenarcheal status						
Self-reported	Field et al. [379]	12 534 (55%)	12	7	Binge eating (YRBSS) Purging (YRBSS) Both binge eating and purging (YRBSS)	Post- vs. premenarche: 1.6 (1.0–2.6) for <14y 1.6 (0.4–6.1) for ≥14y 2.3 (1.4–4.1) for <14y 1.5 (0.5–4.5) for ≥14y 3.0 (0.8–11.1)
Height (males only)						
Self-reported	Field et al. [379]	12 534 (55%)	12	7	Binge eating (YRBSS)	1.1 (1.0–1.2)

Note: BE = binge eating; BMI = body mass index; BN = bulimia nervosa; CI = confidence interval; EDDI = Eating Disorders Diagnostic Interview [212]; EDE-Q = Eating Disorder Examination-Questionnaire [252]; EDI = Eating Disorder Inventory [267]; OR = odds ratio; YRBSS = Youth Risk Behavior Surveillance System questionnaire [388; 389]

The effect of psychological factors on the onset of eating pathology was described in 13 papers. Seven of them described the predictive power of depressiveness. In five articles, the findings supported the harmful effect of depressiveness on eating behavior, and in two papers the association of depressiveness with binge eating and purging [372] and with dieting [385] was positive but nonsignificant. In girls, depressive symptoms predicted the onset of bulimic pathology [383]. Girls with high level of depressive symptoms were more likely to start binge eating as well as overeating within the next two years than girls with no or low level of depressive symptoms. [380.] In both genders, depressed mood increased the odds of developing unhealthy weight control behaviors within five years [382]. What is more, depression symptoms still predicted the onset of extreme weight loss behaviors after seven years [385]. In line with this, the higher the level of depression symptoms was [381] or the higher it increased from middle to late adolescence [204], the more likely participants started binge eating within the next five years. The effect of depression symptoms perceived in adolescence sustained in girls till young adulthood and predicted the onset of binge eating, whereas in boys, the association attenuated when dieting was taken into account. However, boys who reported both dieting and high depression symptoms in adolescence were more likely to start binge eating in young adulthood than dieters with lower level of depression symptoms. In nondieting boys, the level of depression symptoms had no effect on the onset of binge eating in young adulthood. [381.] (Table 7.)

In six articles, the predictor variables included weight concerns which, in this review, showed the strongest effect on the onset of eating pathology. An elevated level of weight concerns was rated as a risk factor for eating disorders in the previous review [16] as well. Adolescents who reported a higher level of concern with weight were more likely to start binge eating [378] as well as purging within a year [377; 378] or 1.5 years [379] than those with a lower level of such concerns. After a 3-year follow-up, the onset of binge eating, purging, obesity, and overweight in girls and binge eating in boys were still more likely in those who reported weight concerns at baseline than in others [339]. In addition, adolescent girls and boys who reported thinking about being thinner and worrying about gaining weight or perceived increase in such concerns had higher odds than others for the onset of unhealthy weight control behaviors within the next five years [382]. With an even longer follow-up period, weight concerns had a significant effect on the onset of binge eating and purging [372]. (Table 7.)

The effect of body dissatisfaction, body image distortion, or weight perception was investigated in six papers. In four of them, the findings supported an adverse effect of body dissatisfaction/distortion or a protective effect of body satisfaction on eating behavior, which is in keeping with the previous reviews [16; 367]. According to the findings, body dissatisfaction predicted the onset of unhealthy weight control behaviors within five years [382], and the onset of DSM-5 eating disorders within four years [384]. Furthermore, nononverweight adolescents who overestimated their own weight had increased odds for the onset of dieting, but not for the onset of extreme weight loss behaviors, after seven years [385]. In line with these, increase in body satisfaction from middle to late adolescence increased the odds of youth for avoiding binge eating in young adulthood [204]. Among young adults [386], the association between perception of greater weight and bulimic syndrome was positive, but nonsignificant. In one paper [379], body dissatisfaction was excluded from the final model because it showed no association with the onset of binge eating or purging when potential confounders were taken into account. (Table 7.)

Table 7. Associations of psychological factors with the onset of eating pathology in the prospective studies of non-clinical populations

Factor, measure	Study	Sample size (% of females)	Age in years, mean / range	Follow-up years	Eating pathology outcome (measure)	OR (95% CI)
Depressiveness						
BDI-Y	Allen et al. [372]	1 383 (51%)	14.01	3–6	DSM-5 BE and purging eating disorders (EDE-Q)	1.02 (0.98–1.06)
Kandel and Davies' scale[1]	Goldschmidt et al. [381]	1 827 (56.9%)	12.8 and 15.9	10	Binge eating (Project EAT survey[2])	First 5 years: 1.63 (1.27–2.09) for F 2.20 (1.36–3.54) for M Last 5 years: 1.55 (1.23–1.96) for F 1.35 (1.00–1.84) for M
Kandel and Davies' scale[1]	Goldschmidt et al. [204]	1 902 (not specified)	14.8	10	Binge eating (Project EAT survey[2])	Increase in the first 5 years: 1.15 (1.06–1.24)
19 items from CES-D	Liechty & Lee [385]	14 322 (50.9%)	15.9	7	Dieting (1 item, yes/no) Extreme weight loss behaviors (3 items, yes/no)	1.11 (0.87–1.42) for F 1.01 (0.69–1.48) for M 1.48 (1.28–1.73) for F 2.52 (1.92–3.31) for M
Kandel and Davies' scale[1]	Linde et al. [382]	2 516 (55%)	12.8 and 15.8	5	Unhealthy weight control behaviors (Project EAT survey[2])	1.52 (1.19–1.94) for F 1.57 (1.22–2.03) for M Change in depressed mood: 1.18 (1.10–1.26) for F 1.14 (1.06–1.23) for M
1 item from MRFS	Skinner et al. [380]	4 798 (100%)	14.9	2	Binge eating monthly (YRBSS) Binge eating weekly (YRBSS) Overeating monthly (YRBSS) Overeating weekly (YRBSS)	High vs. no or low 2.3 (1.7–3.0) 2.4 (1.9–3.2) 1.9 (1.4–2.5) 1.8 (1.4–2.5)
K-SADS	Stice et al. [383]	496 (100%)	13	2	Bulimic pathology (EDE)	1.74 (1.18–2.55)
Weight concerns						
14 items from EDE-Q	Allen et al. [372]	1 383 (51%)	14.01	3–6	DSM-5 BE and purging eating disorders (EDE-Q)	2.74 (1.50–5.02)
MRFS	Field et al. [377]	6 982 (100%)	9–14	1	Purging behaviors (YRBSS)	2.3 (1.9–2.9)
MRFS	Field et al. [378]	11 358 (57%)	10–15	1	Binge eating (YRBSS) Purging (YRBSS)	High vs. low: 2.6 (1.4–5.1) for F 3.8 (1.8–8.0) for F

Table 7 (continues)

Factor, measure	Study	Sample size (% of females)	Age in years, mean / range	Follow-up years	Eating pathology outcome (measure)	OR (95% CI)
MRFS	Field et al. [379]	12 534 (55%)	12.0	7	Binge eating (YRBSS) Purging (YRBSS) Both binge eating and purging (YRBSS)	High vs. low 2.7 (1.7–4.4) for F 3.0 (1.4–6.5) for M 2.3 (1.6–3.2) for F 2.5 (0.7–8.5) for M 5.3 (2.1–13.4) for F
MRFS	Haines et al. [339]	7 172 (59%)	11–17	3	Binge eating / purging / obesity / overweight (YRBSS, self-reported height and weight) Binge eating (YRBSS)	1.56 (1.42–1.71) for F 1.65 (1.27–2.13) for M
Project EAT survey[2]	Linde et al. [382]	2 516 (55%)	12.8	5	Unhealthy weight control behaviors (Project EAT survey[2])	5.09 (3.55–7.30) for F 4.84 (3.32–7.04) for M Increase in weight concerns: 5.87 (4.21–8.17) for F 3.64 (2.70–4.91) for M
Body dissatisfaction						
Body Shape Satisfaction Scale[3]	Goldschmidt et al. [204]	1 902 (not specified)	14.8	10	Binge eating (Project EAT survey[2])	Decrease in the first 5 years: 0.97 (0.94–0.99)
Items from Body Shape Satisfaction scale[3] and Body Cathexis Scale[4]	Linde et al. [382]	2 516 (55%)	12.8	5	Unhealthy weight control behaviors (Project EAT survey[2])	1.07 (1.04–1.11) for F 1.05 (1.01–1.08) for M Change in dissatisfaction: 1.08 (1.06–1.11) for F 1.06 (1.03–1.09) for M
9 items from Satisfaction and Dissatisfaction with Body Parts Scale[5]	Rhode et al. [384]	496 (100%)	13.5	7	DSM-5 eating disorders (EDDI)	1 SD change: 1.68 (1.01–2.77) for 13y 1.89 (1.21–2.97) for 14y 1.51 (1.00–2.25) for 15y 1.67 (1.19–2.34) for 16y
Body image distortion						
1 item (range: 1 very underweight – 5 very overweight)	Liechty & Lee [385]	14 322 (50.9%)	15.9	7	Dieting (1 item, yes/no) Extreme weight loss behaviors (3 items, yes/no)	Over- vs. no overestimation: 1.25 (1.02–1.53) for F 1.72 (1.13–2.61) for M 1.04 (0.75–1.45) for F 1.50 (0.67–3.37) for M
Weight perception						
1 item (range: 1 very underweight – 5 very overweight	Keel & Heatherton [386]	1 337 (72%)	20	10	Bulimic syndrome (EDI and a separate set of questions for DSM-III-R BN symptoms)	1.81 (0.82–4.02)

Table 7 (continues)

Factor, measure	Study	Sample size (% of females)	Age in years, mean / range	Follow-up years	Eating pathology outcome (measure)	OR (95% CI)
Self-esteem						
SPPA	Allen et al. [372]	1 383 (51%)	14.01	3–6	DSM-5 BE and purging eating disorders (EDE-Q)	0.61 (0.36–1.03)
Self-Perception Profile for Children[6]	Field et al. [377]	6 982 (100%)	9–14	1	Purging behaviors (YRBSS)	0.8 (0.7–0.8)
6 items from RSE	Goldschmidt et al. [381]	1 827 (56.9%)	12.8 and 15.9	10	Binge eating (Project EAT survey[2])	First 5 years: 0.79 (0.61–1.02) for F 0.86 (0.50–1.48) for M Last 5 years: 0.55 (0.43–0.71) for F 0.68 (0.50–0.93) for M
6 items from RSE	Goldschmidt et al. [204]	1 902 (not specified)	14.8	10	Binge eating (Project EAT survey[2])	Increase in self-esteem in the first 5years: 0.85 (0.79–0.92)
6 items from RSE	Linde et al. [382]	2 516 (55%)	12.8	5	Unhealthy weight control behaviors (Project EAT survey[2])	0.71 (0.55–0.91 for F 0.58 (0.44–0.75) for M Change in self-esteem: 0.86 (0.80–0.92) for F 0.89 (0.83–0.95) for M
Weight and shape importance						
1 item (range: 1 not very important – 4 most important things)	Linde et al. [382]	2 516 (55%)	12.8	5	Unhealthy weight control behaviors (Project EAT survey[2])	2.23 (1.59–2.84) for F 1.71 (1.29–2.25) for M Change in weight and shape importance: 2.30 (1.78–2.97) for F 1.85 (1.45–2.37) for M
Thin ideal internalization						
Ideal Body Stereotype Scale-Revised[7]	Rhode et al. [384]	496 (100%)	13.5	7	DSM-5 eating disorders (EDDI)	1 SD change: 1.43 (0.76–2.66) for 13y 2.04 (1.25–3.32) for 14y 1.01 (0.69–1.48) for 15y 1.03 (0.73–1.43) for 16y

(continues)

Table 7 (continues)

Factor, measure	Study	Sample size (% of females)	Age in years, mean / range	Follow-up years	Eating pathology outcome (measure)	OR (95% CI)
Negative affectivity						
Buss and Plomin's Emotionality Scale[8]	Rhode et al. [384]	496 (100%)	13.5	7	DSM-5 eating disorders (EDDI)	1 SD change: 1.46 (0.84–2.53) for 13y 1.91 (1.25–2.92) for 14y 1.96 (1.35–2.83) for 15y 1.32 (0.96–1.82) for 16y
Health concerns						
Project EAT survey[2]	Linde et al. [382]	2 516 (55%)	12.8	5	Unhealthy weight control behaviors (Project EAT survey[2])	0.99 (0.79–1.25) for F 1.30 (1.03–1.65) for M Increase in health concerns: 1.03 (0.94–1.12) for F 1.10 (1.01–1.19) for M
Perception of healthy eating benefits						
5 items (range: 1 strongly disagree – 5 strongly agree)	Linde et al. [382]	2 516 (55%)	12.8	5	Unhealthy weight control behaviors (Project EAT survey[2])	1.51 (1.18–1.93) for F 1.16 (0.91–1.49) for M Increase in perception: 1.81 (1.33–2.46) for F 1.50 (1.12–2.01) for M

Note: [1] [393]; [2] [390]; [3] [394]; [4] [395]; [5] [396]; [6] [397]; [7] [212]; [8] [398]; BDI-Y = Beck Depression Inventory-Youth [399]; BE = binge eating; BMI = body mass index; BN = bulimia nervosa; CES-D = Center for Epidemiologic Studies Depression Scale [400]; CI = confidence interval; EDDI = Eating Disorders Diagnostic Interview [212]; EDE-Q = Eating Disorder Examination-Questionnaire [252]; EDI = Eating Disorder Inventory [267]; EDI-3 = Eating Disorder Inventory -3 [269]; F = females; K-SADS = Kiddie Schedule for Affective Disorders and Schizophrenia [401]; M = males; OR = odds ratio; RSE = Rosenberg Self-Esteem Scale [402]; SD = standard deviation; SPPA = Self-Perception Profile for Adolescents [403]; y = years-old; YRBSS = Youth Risk Behavior Surveillance System questionnaire [388; 389]

The relationship between self-esteem and the onset of eating pathology was assessed in five papers. In line with the previous review [16], four of them found an association between self-esteem and the onset of eating pathology. Higher initial self-esteem protected girls against purging behaviors one year later [377]. In addition, as the level of their self-esteem increased, the odds of adolescent girls and boys for the onset of unhealthy weight control behaviors decreased. In line with this, the more important weight or shape was for adolescents' feeling about themselves, the more likely they started unhealthy weight control behaviors within five years. [382.] Furthermore, those with a higher self-esteem [381] or greater increase in self-esteem [204] from adolescence to young adulthood were better protected against the onset of binge eating. The inverse association between self-esteem and the onset of binge eating was found already in late adolescence, but it was not significant until in young adulthood [381]. The association of self-esteem in mid-adolescence with binge eating and purging in late adolescence remained nonsignificant also in another study [372]. (Table 7.)

The effects of thin-ideal internalization, negative affectivity, health concerns, and perception of the benefits of healthy eating on the onset of eating pathology were reported in one article, respectively. Girls with internalized thin-ideal at the age of 14 years had increased odds for the onset of DSM-5 eating disorders in late adolescence. However, the association was found in this age-group only; in younger and older girls the association was positive, but nonsignificant. Negative affectivity was associated with DSM-5 eating disorders in girls who were 14 to 15 years of age at the initiation of the study, while in younger and older girls the association remained nonsignificant. [384.] Increase in the level of health concerns was associated with an increased probability to develop unhealthy weight control behaviors among boys but not in girls. On the other hand, unlike boys, the stronger perception of the benefits of healthy eating girls had, the more likely they were to start unhealthy weight control behaviors within five years. Both genders, in turn, had increased odds for the onset of unhealthy weight control behaviors after experiencing an increase in the perception of healthy eating benefits. [382.] (Table 7.)

The effect of behavioral factors on the onset of eating pathology was assessed in ten papers. Eight of these reported findings concerning eating behaviors, such as dieting, objective binge eating, having breakfast, eating fast food, or changing eating habits around peers, in relation to eating pathology. For girls, the results concerning dieting were consistent: dieting had a direct association with the onset of binge eating [378; 379; 381], purging [378; 379], binge eating/purging/obesity/overweight [339], and extreme weight loss behaviors, whereas extreme weight loss behaviors in mid-adolescence had no effect on the onset of dieting in young adulthood [385]. When the analyses were stratified on the basis of the dieting frequency, only those girls who reported frequent dieting were more likely than nondieters to start binge eating or both binge eating and purging within 1–1.5 years; infrequent dieters had no such risk. On the other hand, all dieting regardless of its frequency predicted purging onset. The risk was highest for girls younger than 14 years. [379.] Even so, the predictive effect of dieting on the onset of DSM-5 eating disorders was found only in girls aged 14 at the initiation of the study. In younger and older girls, the findings remained nonsignificant. [384.] (Table 8.)

Table 8. Associations of behavioral factors with the onset of eating pathology in the prospective studies of non-clinical populations

Factor, measure	Study	Sample size (% of females)	Age in years, mean/range	Follow-up years	Eating pathology outcome (measure)	OR (95% CI)
Dieting						
YRBSS	Field et al. [378]	11 358 (57%)	10–15	1	Binge eating (YRBSS) Purging (YRBSS)	1.4 (1.2–1.6) 1.4 (1.1–1.6)
1 item from YRBSS (range: 1 never – 6 every day)	Field et al. [379]	12 534 (55%)	12	7	Binge eating (YRBSS)	Infrequent- vs. nondieter: 1.1 (0.7–1.7) for F Frequent- vs. nondieter: 2.2 (1.4–3.7) for F Dieting vs. nondieting: 1.7 (0.9–3.2) for M
					Purging (YRBSS)	Infrequent- vs. nondieter: 4.1 (2.2–7.8) for F <14y 2.8 (1.9–4.3) for F ≥14y Frequent- vs. nondieter: 7.0 (3.5–14.0) for F <14y 3.1 (1.9–5.2) for F ≥14y Dieting vs. nondieting: 2.4 (0.9–6.1) for M
					Both binge eating and purging (YRBSS)	Infrequent- vs. nondieter: 2.9 (1.0–8.8) for F Frequent- vs. nondieter: 7.4 (2.2–25.0) for F
1 item (range: 1 never – 5 always)	Goldschmidt et al. [381]	1 827 (56.9%)	12.8 and 15.9	10	Binge eating (Project EAT survey[1])	Dieter vs. nondieter: First 5 years: 2.06 (1.24–3.43) for F 1.43 (0.44–4.67) for M Last 5 years: 2.96 (1.78–4.92) for F 2.50 (1.26–4.93) for M
1 item (range: never – always)	Haines et al. [339]	7 172 (59%)	11–17	3	Binge eating / purging / obesity / overweight (YRBSS, self-reported height and weight)	Any vs. none: 1.48 (1.25–1.74) for F 0.94 (0.65–1.34) for M
1 item (yes/no)	Liechty & Lee [385]	14 322 (50.9%)	15.9	7	Extreme weight loss behaviors (3 items, yes/no)	1.63 (1.16–2.29) for F 0.90 (0.29–2.67) for M
Dutch Restrained Eating Scale[2]	Rhode et al. [384]	496 (100%)	13.5	7	DSM-5 eating disorders (EDDI)	1.59 (0.96–2.61) for 13y 1.92 (1.30–2.83) for 14y 1.11 (0.76–1.62) for 15y 1.23 (0.90–1.70) for 16y

Table 8 (continues)

Factor, measure	Study	Sample size (% of females)	Age in years, mean/range	Follow-up years	Eating pathology outcome (measure)	OR (95% CI)
Extreme weight loss behaviors						
3 items (yes/no)	Liechty & Lee [385]	14 322 (50.9%)	15.9	7	Dieting (1 item, yes/no)	0.72 (0.36–1.44) for F 2.01 (0.48–8.38) for M
Objective binge eating						
EDE-Q	Allen et al. [372]	1 383 (51%)	14.01	3–6	DSM-5 BE and purging eating disorders (EDE-Q)	1.70 (1.06–2.75)
Changing eating habits around peers						
MRFS	Field et al. [377]	6 982 (100%)	9–14	1	Purging behaviors (YRBSS)	2.1 (1.7–2.6)
Breakfast frequency						
1 item (range: never / almost never – ≥5 times/week)	Haines et al. [339]	7 172 (59%)	11–17	3	Purging (YRBSS) Binge eating (YRBSS)	0.97 (0.92–1.03) for F 1.03 (0.98–1.09) for F 1.06 (0.94–1.19) for M
Fast-food intake						
1 item (range: never / less than 1/week – daily)	Haines et al. [339]	7 172 (59%)	11–17	3	Binge eating / purging / obesity / overweight (YRBSS, self-reported height and weight) Binge eating (YRBSS)	1.02 (0.97–1.07) for F 1.12 (1.00–1.24) for M
Physical activity						
Youth/Adolescent Activity Questionnaire[3]	Haines et al. [339]	7 172 (59%)	11–17	3	Binge eating / purging / obesity / overweight (YRBSS, self-reported height and weight)	1.05 (1.00–1.11) for F 1.06 (0.99–1.15) for M
TV viewing						
1 item (range: never – ≥31 hours /week)	Haines et al. [339]	7 172 (59%)	11–17	3	Binge eating / purging / obesity / overweight (YRBSS, self-reported height and weight)	1.01 (0.95–1.08) for F 1.01 (0.94–1.09) for M
Reading weight loss articles						
1 item (range: 1 never – 4 often)	Linde et al. [382]	2 516 (55%)	12.8	5	Unhealthy weight control behaviors (Project EAT survey[1])	1.92 (1.49–2.48) for F 1.68 (1.23–2.27) for M Increase in reading articles: 2.07 (1.67–2.58) for F 1.71 (1.36–2.16) for M

Table 8 (continues)

Factor, measure	Study	Sample size (% of females)	Age in years, mean/range	Follow-up years	Eating pathology outcome (measure)	OR (95% CI)
Trying to look like females on TV, movies, and in magazines						
MRFS	Field et al. [377]	6 982 (100%)	9–14	1	Purging behaviors (YRBSS)	1.9 (1.6–2.6)
1 item from MRFS	Field et al. [379]	12 534 (55%)	12	7	Binge eating (YRBSS) Purging (YRBSS) Both binge eating and purging (YRBSS)	Totally / a lot vs. other: 2.2 (1.4–3.4) for F 1.5 (1.1–2.2) for F 1.1 (0.5–2.5) for F
Desire to look like same-sex media figure						
1 item (range: not at all – totally)	Haines et al. [339]	7 172 (59%)	11–17	3	Purging (YRBSS) Binge eating (YRBSS) Binge eating / purging / obesity / overweight (YRBSS, self-reported height and weight)	1.14 (1.01–1.29) for F 1.04 (0.93–1.16) for F 1.04 (0.93–1.16) for M 0.95 (0.82–1.11) for M
Smoking						
Items from YRBSS and the California Tobacco Survey[4]	Field et al. [378]	11 358 (57%)	10–15	1	Binge eating (YRBSS) Purging (YRBSS)	1.4 (0.6–3.0) 0.9 (0.3–2.5)
Getting drunk						
Questions modeled on the items about smoking in YRBSS	Field et al. [378]	11 358 (57%)	10–15	1	Binge eating (YRBSS) Purging (YRBSS)	1.1 (0.5–2.6) 2.7 (1.1–6.9)
Substance abuse symptoms						
Items adapted from Stice et al. [404]	Stice et al. [383]	496 (100%)	13	2	Bulimic pathology (EDE)	1.29 (0.85–1.96)

Note: [1] [390]; [2] [405]; [3] [406]; [4] [407]; BE = binge eating; BET, Branched Eating Disorders Test [255]; CI = confidence interval; EDDI = Eating Disorders Diagnostic Interview [212]; EDE = Eating Disorder Examination [301]; EDE-Q = Eating Disorder Examination-Questionnaire [252]; F = females; M = males; MRFS = McKnight Risk Factor Survey [265]; OR = odds ratio; y = years-old; SD = standard deviation; YRBSS = Youth Risk Behavior Surveillance System questionnaire [388; 389]

For boys, the papers reported nonsignificant associations of dieting with the onset of binge eating, purging [379], binge eating/purging/obesity/overweight [339], as well as extreme weight loss behaviors [385]. However, one article [381] showed that, in boys, the effect of dieting became significant after adolescence; those who were dieting in adolescence were more likely to start binge eating in young adulthood than boys who reported that they never used dieting to lose or control weight. Objective binge eating was found to be associated with the onset of eating disorder (DSM-5) in later adolescence [372]. In addition, girls who changed their eating habits around peers had increased odds for the onset of purging behaviors [377]. No evidence was found of the association of the breakfast frequency or eating fast food with the onset of binge eating or purging [339]. (Table 8.)

The effects of physical activity and sedentary behavior on the onset of eating pathology were reported in four articles. The results showed that neither the weekly hours of physical activity nor the daily hours spent on watching television had any effect on the onset of binge eating/purging/obesity/overweight [339]. On the other hand, an increase in the frequency of reading articles on dieting or weight loss was associated with increased odds of both genders for the onset of unhealthy weight control behaviors [382]. In addition, girls' attempts to look like the females presented in the media increased their odds for the onset of purging [377; 379] as well as binge eating within the next 1–1.5 years [379]. The predictive power of the desire to look like the same-sex media figures on the onset of purging was still significantl after 3 years, whereas the effect on the onset of binge eating attenuated. In boys, such desire was not predictive. [339.]

The role of smoking or substance use on the onset of eating pathology in girls was assessed in two papers. According to the results, neither smoking nor getting drunk was associated with the onset of binge eating. In addition, smoking did not significantly affect the onset of purging. However, girls who had been drunk had a higher probability to start purging to control weight than girls without a history of being drunk. [378.] The association between substance abuse symptoms (obligation impairment, health problems, physically hazardous behavior, legal problems, social difficulties) and the onset of bulimic pathology was positive, but nonsignificant [383]. (Table 8.)

The role of environmental factors on the onset of eating pathology was assessed in eight papers. Six of them reported the estimates for teasing experiences and negative comments about weight. In the previous review [16], the role of teasing in relation to the onset of eating pathology remained unknown due to the lack of prospective studies of the topic. In this review, the findings showed a direct association between teasing and the onset of eating pathology in girls. Only the magnitude and the level of certainty of the effect varied across the articles and outcomes. Girls who had been exposed to weight-related teasing at least a few times a year in adolescence were more likely than others to start frequent dieting [333]. In addition, the more often girls had experienced teasing in adolescence, the more likely they were to start binge eating in young adulthood [381]. On the other hand, teasing frequency had no effect on the onset of unhealthy weight control behaviors in girls [333; 382]. Both their peers and parents had a significant effect on adolescent girls' eating behavior. Teasing and weight related comments by peers increased the odds of the girls for the onset of purging behaviors [377], whereas parental weight-related teasing predicted the onset of binge eating [339]. However, the harmful effect of teasing on eating behavior did not always occur. In one article, the predictive power of teasing on the onset of binge eating increased along with teasing experiences

only in girls who dieted in adolescence. In nondieters, teasing experiences did not effect on the onset of binge eating. [381.] (Table 9.)

For boys, the findings were inconsistent. Teasing experiences [333] and negative comments about weight by father [379] were found to predict the onset of binge eating. However, also nonsignificant associations - both direct and inverse - between teasing and the onset of binge eating were reported [381]. In addition, the higher the frequency of weight-related teasing in mid-adolescence, the higher the odds of the boys for the onset of unhealthy weight control behaviors in late adolescence. Even so, change in the teasing frequency within five follow-up years had no effect on the development of unhealthy weight control behaviors. [382.] (Table 9.)

Altogether six papers reported how attitudes about thinness, weight, and dieting among peers and parents, as well as social pressure to be thin affected eating behavior in adolescents. In one paper [377], girls who reported that thinness was quite important to their peers were more likely than others to start purging behaviors within a year, and in another study [379], girls who perceived that weight was very important to their peers had higher odds than others for the onset of both binge eating and purging within 1–1.5 years. In addition, having friends who were dieting or parents who were concerned about their own or the adolescent's weight predicted the onset of unhealthy weight control behaviors [382]. However, some of the studies failed to find association of the importance of thinness [339; 378] and weight to peers [379] with the onset of binge eating and purging in girls. In addition, neither importance of weight to father [379] nor maternal dieting [339] increased the odds of the girls for the onset of binge eating and/or purging [379] or binge eating/purging/obesity/overweight [339]. The association between social pressure toward thinness and eating disorders was found in girls aged 14 years, but not in younger and older girls [384]. In boys, having dieting friends was associated with an increased probability to start unhealthy weight control behaviors within five years. In addition, boys who reported higher levels of parental concerns about weight were more likely to develop unhealthy weight control behaviors than boys whose parents expressed less concerns about weight. [382.] Furthermore, boys who perceived that weight was very important to their peers tended to be more likely than others to start purging within the next 1–1.5 years [379]. On the other hand, the importance of thinness to peers as well as maternal dieting did not predict the onset of binge eating/purging/obesity/ overweight in boys [339]. (Table 9.)

The favourable effect of environmental factors was reported in two papers. Both of these shed light on the role of the family in protecting adolescents against eating pathology. First, the high frequency of family meals was found to predict decreased odds of the onset of binge eating/purging/obesity/overweight in girls [339]. Second, a girl's perception of the family connectedness, i.e., feeling able to talk about problems to parents and feeling that parents care about her, provided protection against the onset of unhealthy weight control behaviors [382]. In boys, the association of family meal frequency [339] and family connectedness [382] with the onset of eating pathology was inverse, but nonsignificant. (Table 9.)

Table 9. Associations of environmental factors with the onset of eating pathology in the prospective studies of non-clinical populations

Factor, measure	Study	Sample size (% of females)	Age in years, mean/range	Follow-up years	Eating pathology outcome (measure)	OR (95% CI)
Teasing experiences						
2 items from the Perception of Teasing Scale[1] (range: 1 never – 5 at least once a week)	Goldschmidt et al. [381]	1 827 (56.9%)	12.8 and 15.9	10	Binge eating (Project EAT survey[2])	First 5 years: 1.12 (0.90–1.39) for F 1.34 (0.86–2.11) for M Last 5 years: 1.40 (1.13–1.73) for F 0.97 (0.72–1.32) for M
1 item (range: 1 never – 5 at least once a week)	Haines et al. [333]	2 516 (55.1%)	12.8 and 15.8	5	Binge eating (Project EAT survey[2]) Unhealthy weight control behaviors (Project EAT survey[2]) Frequent dieting (Project EAT survey[2])	1.3 (0.9–2.1) for F 3.0 (1.1–8.1) for M 1.2 (0.7–1.8) for F 1.6 (1.0–2.6) for M 1.8 (1.2–2.7) for F 1.1 (0.5–2.3) for M
Teasing and comments about weight by peers						
MRFS	Field et al. [377]	6 982 (100%)	9–14	1	Purging behaviors (YRBSS)	2.0 (1.6–2.6)
Parental weight-related teasing						
2 items (range: never – always)	Haines et al. [339]	7 172 (59%)	11–17	3	Purging (YRBSS) Binge eating (YRBSS) Binge eating / purging / obesity / overweight (YRBSS, self-reported height and weight)	1.10 (0.89–1.37) for F 1.29 (1.08–1.55) for F 1.07 (0.88–1.29) for M
Teasing frequency						
1 item (range: 1 never – 5 at least once a week)	Linde et al. [382]	2 516 (55%)	12.8	5	Unhealthy weight control behaviors (Project EAT survey[2])	1.22 (0.92–1.61) for F 1.40 (1.07–1.85) for M Change in teasing frequency: 1.21 (0.92–1.61) for F 1.14 (0.93–1.39) for M

(continues)

Table 9 (continues)

Factor, measure	Study	Sample size (% of females)	Age in years, mean/range	Follow-up years	Eating pathology outcome (measure)	OR (95% CI)
Negative weight comments by males						
MRFS	Field et al. [379]	12 534 (55%)	12	7	Binge eating (YRBSS) Purging behaviors (YRBSS) Both binge eating and purging YRBSS)	1.3 (0.8–2.1) for F 1.5 (1.0–2.2) for F 1.2 (0.5–3.1) for F
Negative weight comments by father						
MRFS	Field et al. [379]	12 534 (55%)	12	7	Binge eating (YRBSS) Purging behaviors (YRBSS)	2.3 (1.1–4.9) for M 2.1 (0.6–7.2) for M
Pressure to be thin						
Perceived Sociocultural Pressure Scale[3]	Rhode et al. [384]	496 (100%)	13.5	7	DSM-5 eating disorders (EDDI)	1.60 (0.99–2.56) for 13y 2.14 (1.40–3.22) for 14y 1.33 (0.85–2.08) for 15y 1.18 (0.86–1.63) for 16y
Importance of thinness to peers						
MRFS	Field et al. [377]	6 982 (100%)	9–14	1	Purging behaviors (YRBSS)	2.3 (1.8–3.0)
MRFS	Field et al. [378]	11 358 (57%)	10–15	1	Binge eating (YRBSS) Purging (YRBSS)	1.8 (0.8–4.1) 2.1 (0.9–4.9)
3 items (mean score)	Haines et al. [339]	7 172 (59%)	11–17	3	Purging (YRBSS) Binge eating (YRBSS) Binge eating / purging / obesity / overweight (YRBSS, self-reported height and weight)	1.17 (0.96–1.44) for F 1.08 (0.92–1.27) for F 0.92 (0.72–1.17) for M
Importance of weight to peers						
MRFS	Field et al. [379]	12 534 (55%)	12	7	Binge eating (YRBSS) Purging behaviors (YRBSS) Both binge eating and purging (YRBSS)	Very vs. not very important: 1.9 (0.9–4.1) for F 1.6 (0.8–3.1) for F 3.4 (1.0–11.4) for M 3.7 (1.1–12.4) for F

(continues)

Table 9 (continues)

Factor, measure	Study	Sample size (% of females)	Age in years, mean/range	Follow-up years	Eating pathology outcome (measure)	OR (95% CI)
Peer dieting						
1 item (range: 1 don't know – 5 very much)	Linde et al. [382]	2 516 (55%)	12.8	5	Unhealthy weight control behaviors (Project EAT survey[2])	1.57 (1.20–2.04) for F 1.98 (1.45–2.70) for M Increase in peer dieting: 1.38 (1.14–1.68) for F 1.37 (1.10–1.70) for M
Parental concern about weight						
2 items (range: 1 not at all – 4 very much)	Linde et al. [382]	2 516 (55%)	12.8	5	Unhealthy weight control behaviors (Project EAT survey[2])	1.42 (1.07–1.89) for F 1.64 (1.24–2.16) for M Increase in parental concern about weight: 1.03 (0.95–1.12) for F 1.09 (1.01–1.17) for M
Weight importance to father						
MRFS	Field et al. [379]	12 534 (55%)	12	7	Binge eating (YRBSS) Purging behaviors (YRBSS) Both binge eating and purging (YRBSS)	Very vs. not very important: 1.5 (0.8–3.0) for F 1.7 (0.9–3.0) for F 1.0 (0.2–4.6) for F
Maternal dieting						
1 item (range: never – always)	Haines et al. [339]	7 172 (59%)	11–17	3	Binge eating / purging / obesity / overweight (YRBSS, self-reported height and weight)	Any vs. none: 0.89 (0.77–1.02) for F 1.14 (0.94–1.40) for M
Family meal frequency						
1 item (range: never – every day)	Haines et al. [339]	7 172 (59%)	11–17	3	Binge eating / purging / obesity / overweight (YRBSS, self-reported height and weight)	0.90 (0.83–0.98) for F 0.89 (0.78–1.01) for M
Family connectedness						
Voice of Connecticut Youth Survey[4]	Linde et al. [382]	2 516 (55%)	12.8	5	Unhealthy weight control behaviors (Project EAT survey[2])	0.70 (0.56–0.88) for F 0.90 (0.72–1.13) for M Decrease in connetedness: 0.93 (0.87–0.98) for F 0.97 (0.91–1.03) for M

Note: [1] [408]; [2] [390]; [3] [409]; [4] [410]; BET = Branched Eating Disorders Test [255]; CI = confidence interval; EDDI = Eating Disorders Diagnostic Interview [212]; EDE-Q = Eating Disorder Examination-Questionnaire [252]; F = females; M = males; MRFS = the McKnight Risk Factor Survey [265]; OR = odds ratio; y = years-old; YRBSS = Youth Risk Behavior Surveillance System questionnaire [388; 389]

2.5.2.3 Predictors of the maintenance of eating pathology

The findings of the studies on factors associated with the maintenance of eating pathology are presented in Tables 10 and 11. The effect of different predictors was assessed in three articles. In two papers [204; 386], the findings were reported genders combined, while in one article [382] estimates were given for girls and boys separately.

Of the individual factors, the investigation of the biological characteristics was limited to BMI. The findings showed that the association of the respondent's BMI with the maintenance of binge eating [204] and bulimic syndrome [386] was nonsignificant. However, weight suppression from late adolescence to young adulthood predicted the maintenance of bulimic syndrome [386]. (Table 10.)

Of the psychological factors, depressiveness showed a significant effect on the maintenance of eating pathology. The greater the degree of depressed mood reported by girls and boys in early or middle adolescence, the higher the probability that they used unhealthy weight control behaviors five years later [382]. In line with this, those who reported a decrease in depression symptoms from late adolescence to young adulthood were more likely than others to show remission of binge eating. However, baseline depressiveness in middle or late adolescence showed no predictive power on the maintenance of binge eating five years later. [204.] (Table 10.)

The strongest effect on the maintenance of eating pathology was shown by the attitudes and feelings about weight. Weight concerns and importance given on weight and shape predicted the persistence of unhealthy weight control behaviors in both genders. The higher the degree of weight concerns or preoccupation with weight and shape in early or middle adolescence, the higher the probability of still using unhealthy weight control behaviors after five years. [382.] Perception of greater weight in late adolescence, in turn, was associated with increased odds for the maintenance of bulimic syndrome at 10-year follow-up [386]. (Table 10.)

In both genders, body dissatisfaction in adolescence predicted the persistence of unhealthy weight control behaviors [382], but not binge eating [204], at the 5-year follow-up. Self-esteem also had predictive power on the maintenance of unhealthy weight control behaviors among females; those girls who had a higher self-esteem in early or middle adolescence were more likely to stop using unhealthy weight control behaviors within five years. A similar tendency, although not statistically significant, was found in boys. [382.] In line with this, increased self-esteem predicted binge eating cessation in youth from middle to late adolescence and to young adulthood [204]. (Table 10.)

Adolescents' perceptions of the benefits of healthy eating were associated with the persistence of unhealthy weight control behaviors. The stronger perceptions they reported, more likely they were to maintain unhealthy weight control behaviors. Health concerns, in turn, did not predict the persistence of unhealthy weight control behaviors. [382.] (Table 10.)

Table 10. Associations of biological and psychological factors with the maintenance or cessation of eating pathology in the prospective studies of non-clinical populations

Factor, measure	Study	Sample size (% of females)	Age in years, mean	Follow-up years	Eating pathology outcome (measure)	OR (95% CI)
BMI						
Self-reported height and weight	Goldschmidt et al. [204]	1 902 (not specified)	14.8	10	Cessation of binge eating (Project EAT survey[1])	First 5 years: 1.10 (1.00–1.21) Increase in BMI: 0.93 (0.81–1.07) Last 5 years: 0.95 (0.88–1.04) Increase in BMI: 0.98 (0.88–1.09)
Self-reported height and weight	Keel & Heatherton [386]	1 337 (72%)	20	10	Maintenance of bulimic syndrome (EDI and a separate set of questions for DSM-III-R BN symptoms)	0.97 (0.78–1.21) Weight supression: 1.08 (1.02–1.14)
Weight concerns						
Project EAT survey[1]	Linde et al. [382]	2 516 (55%)	12.8	5	Maintenance of unhealthy weight control behaviors (Project EAT survey[1])	3.45 (2.50–4.76) for F 4.55 (2.86–7.14) for M Increase in weight concerns: 3.03 (2.38–4.00) for F 4.55 (2.76–7.14) for M
Weight and shape importance						
1 item (range: 1 not very important – 4 most important things)	Linde et al. [382]	2 516 (55%)	12.8	5	Maintenance of unhealthy weight control behaviors (Project EAT survey[1])	2.44 (1.89–3.23) for F 2.50 (1.79–3.57) for M Change in weight and shape importance: 2.38 (1.89–3.03) for F 1.67 (1.27–2.22) for M
Weight perception						
1 item (range: very underweight – very overweight)	Keel & Heatherton [386]	1 337 (72%)	20	10	Maintenance of bulimic syndrome (EDI and a separate set of questions for DSM-III-R BN symptoms)	Survey data: 11.96 (3.40–42.12) Interview data: 2.98 (1.28–6.94)

(continues)

Table 10 (continues)

Factor, measure	Study	Sample size (% of females)	Age in years, mean	Follow-up years	Eating pathology outcome (measure)	OR (95% CI)
Depressiveness / depression (symptoms)						
Kandel and Davies' scale[2]	Goldschmidt et al. [204]	1 902 (not specified)	14.8	10	Cessation of binge eating (Project EAT survey[1])	First 5 years: 0.96 (0.81–1.13) Increase in symptoms: 0.89 (0.73–1.09) Last 5 years: 0.92 (0.81–1.05) Decrease in symptoms: 0.81 (0.68–0.95)
Kandel and Davies' scale[2]	Linde et al. [382]	2 516 (55%)	12.8	5	Maintenance of unhealthy weight control behaviors (Project EAT survey[1])	1.72 (1.37–2.22) for F 1.85 (1.33–2.50) for M Change in depressed mood: 1.25 (1.16–1.33) for F 1.18 (1.08–1.28) for M
Body dissatisfaction						
Body Shape Satisfaction Scale[3]	Goldschmidt et al. [204]	1 902 (not specified)	14.8	10	Cessation of binge eating (Project EAT survey[1])	First 5 years: 1.00 (0.94–1.06) Decrease in body satisfaction: 1.01 (0.96–1.07) Last 5years: 1.01 (0.95–1.06) Decrease in body satisfaction: 1.06 (1.00–1.13)
Items from Body Shape Satisfaction scale[3] and Body Cathexis Scale[4]	Linde et al. [382]	2 516 (55%)	12.8	5	Maintenance of unhealthy weight control behaviors (Project EAT survey[1])	1.08 (1.05–1.11) for F 1.10 (1.05–1.15) for M Change in dissatisfaction: 1.09 (1.06–1.12) for F 1.06 (1.03–1.10) for M
Self-esteem						
RSE	Goldschmidt et al. [204]	1 902 (not specified)	14.8	10	Cessation of binge eating (Project EAT survey[1])	First 5 years: 1.04 (0.92–1.18) Increase in self-esteem: 1.21 (1.02–1.44) Last 5 years: 1.03 (0.91–1.15) Increase in self-esteem: 1.23 (1.07–1.41)

(continues)

Table 10 (continues)

Factor, measure	Study	Sample size (% of females)	Age in years, mean	Follow-up years	Eating pathology outcome (measure)	OR (95% CI)
RSE	Linde et al. [382]	2 516 (55%)	12.8	5	Maintenance of unhealthy weight control behaviors (Project EAT survey[1])	0.54 (0.42–0.69) for F 0.73 (0.53–1.00) for M Change in self-esteem: 0.84 (0.79–0.89) for F 0.94 (0.87–1.01) for M
Perception of healthy eating benefits						
5 items (range: 1 strongly disagree – 5 strongly agree)	Linde et al. [382]	2 516 (55%)	12.8	5	Maintenance of unhealthy weight control behaviors (Project EAT survey[1])	1.39 (1.10–1.79) for F 1.64 (1.19–2.27) for M Change in perception of healthy eating benefits: 2.27 (1.70–3.03) for F 1.92 (1.32–2.86) for M
Health concerns						
Project EAT survey[1]	Linde et al. [382]	2 516 (55%)	12.8	5	Maintenance of unhealthy weight control behaviors (Project EAT survey[1])	1.01 (0.82–1.25) for F 1.14 (0.86–1.49) for M Change in health concerns: 1.04 (0.96–1.12) for F 1.06 (0.95–1.18) for M

Note: [1] [390]; [2] [393]; [3] [394]; [4] [395]; BN = bulimia nervosa; CI = confidence interval; EDI = Eating Disorder Inventory [267]; F = females; M = males; OR = odds ratio; RSE = Rosenberg Self-Esteem Scale [402]

The behavioral factors investigated in the reviewed studies were dieting and the frequency of reading articles on dieting or weight loss. The findings showed that dieting per se was not associated with the maintenance of bulimic syndrome [386], but those adolescents who frequently read weight loss articles were more likely to use unhealthy weight control behaviors five years later. This concerned both girls and boys. [382.] (Table 11.)

Of the environmental factors, the frequency of perceived teasing predicted the maintenance of unhealthy weight control behaviors for both genders; the more often adolescents were teased about their weight, the higher the probability of persistent unhealthy weight control behaviors. In both girls and boys, having dieting friends also increased the probability of still using unhealthy weight control behaviors after five years. Parental concern about weight, which comprises parental dieting and parental encouragement for an adolescent to diet, affected girls' weight control behaviors. The more girls perceived parental concern about weight, the higher the probability that they used unhealthy weight control behaviors five years later. In boys, the association remained nonsignificant. Adolescents' perceptions about family connectedness did not predict the persistence of unhealthy weight control behaviors, except for girls who perceived a decrease in the family connectedness from early to late adolescence: they were more likely than others to show the cessation of unhealthy weight control behaviors. [382.] (Table 11.)

In sum, of the total of 43 factors investigated for their potential effect on eating pathology altogether 15 showed consistently an association with a later onset of eating pathology; 11 factors for girls, six factors for boys, and five factors for both genders combined. For the remaining 18 factors, the findings were inconsistent or lacked the evidence of their role in the onset of eating pathology.

Moreover, of the 15 factors assessed for the effect on the maintenance of eating pathology, altogether 12 were found to maintain eating pathology; 10 factors for girls, seven for boys, and two for both genders combined. Seven factors were found to have a role both in the onset and maintenance of eating pathology in girls, and correspondingly, three factors in boys. Of all those factors that were found to affect the onset and/or maintenance of eating pathology in boys, only one showed no predictive power in girls, whereas five of the factors that were found predictive in girls, remained nonsignificant in boys. Thus, girls were prone to the detrimental effect of a larger number of exposures than boys.

In general, some of the findings supported the conclusions drawn in the previous reviews, few were contradictory to previous findings, and the majority shed light on factors not assessed in the previous reviews. In line with the previous review [16], two studies found no evidence of the association between BMI in adolescence and a later onset of eating pathology, whereas one study reported on the predictive power of BMI. As a new finding, weight suppression was shown to increase the odds for the onset of bulimic syndrome up until young adulthood, thus supporting the previous conclusion of a detrimental effect of dieting on eating behavior. In this review, female gender increased the odds of adolescents, but not young adults, for the onset of eating pathology, which is in accordance with the finding that eating pathology typically occurs in adolescent girls. In adulthood, the role of gender in the onset of eating pathology is less evident.

Table 11. Associations of behavioral and environmental factors with the maintenance of eating pathology in the prospective studies of non-clinical populations

Factor, measure	Study	Sample size (% of females)	Age in years, mean	Follow-up years	Eating pathology outcome (measure)	OR (95% CI)
Diet frequency						
1 item (range: never – often)	Keel & Heatherton [386]	1 337 (72%)	20	10	Bulimic syndrome (EDI and a separate set of questions for DSM-III-R BN symptoms)	1.06 (0.56–1.99)
Weight loss article reading						
1 item (range: 1 never – 4 often)	Linde et al. [382]	2 516 (55%)	12.8	5	Unhealthy weight control behaviors (Project EAT survey[1])	1.79 (1.41–2.27) for F 1.96 (1.33–2.94) for M Increase in reading: 1.33 (1.10–1.61) for F 1.30 (0.96–1.79) for M
Teasing frequency						
1 item (range: 1 never – 5 at least once a week)	Linde et al. [382]	2 516 (55%)	12.8	5	Unhealthy weight control behaviors (Project EAT survey[1])	1.35 (1.05–1.64) for F 1.61 (1.18–2.22) for M Change in teasing frequency 1.37 (1.14–1.64) for F 1.35 (1.10–1.67) for M
Peer dieting						
1 item (range: 1 don't know – 5 very much)	Linde et al. [382]	2 516 (55%)	12.8	5	Unhealthy weight control behaviors (Project EAT survey[1])	1.56 (1.22–2.00) for F 1.47 (1.04–2.08) for M Increase in peer dieting: 1.47 (1.22–1.79) for F 1.32 (0.99–1.75) for M
Parental concern about weight						
2 items (range: 1 not at all – 4 very much)	Linde et al. [382]	2 516 (55%)	12.8	5	Unhealthy weight control behaviors (Project EAT survey[1])	1.43 (1.12–1.82) for F 1.14 (0.86–1.49) for M Increase in parental concern: 1.11 (1.04–1.21) for F 1.04 (0.96–1.12) for M
Family connectedness						
Voice of Conneticut Youth Survey[2]	Linde et al. [382]	2 516 (55%)	12.8	5	Unhealthy weight control behaviors (Project EAT survey[1])	0.83 (0.66–1.03) for F 0.81 (0.61–1.06) for M Decrease in connectedness: 0.94 (0.89–0.99) for F 0.94 (0.89–1.01) for M

Note: [1] [390]; [2] [410]; BN = bulimia nervosa; CI = confidence interval; EDI = Eating Disorder Inventory [267]; F = females; M = males; OR = odds ratio

In line with the previous findings, the strongest evidence of the effect of all investigated factors on a later onset of eating pathology was found for weight concerns and dieting. However, the evidence concerning dieting was strong only for girls, while the findings were less consistent among boys. Depressiveness and body dissatisfaction or -distortion were found to predict the onset of eating pathology among both genders. The effect of self-esteem in adolescence on a later onset of eating pathology gained support for both genders, although not consistently. In the previous review [16], attention was called to a need for more evidence of the effect of alcohol consumption on the onset of eating pathology. In this review, support was gained for the role of getting drunk in the onset of purging, but not binge eating. However, the finding was based on a single study, so a need for replication remains. In contrast to the previous review [16] showing that family relationships have no effect on the later development of eating problems, a significant inverse association was reported between family connectedness and the onset of unhealthy weight control behaviors among girls, but not in boys. However, also this finding was based on a single paper, raising a need for further research. Factors that were not assessed by the previous reviewers, due to lack of prospective studies, included physical activity and teasing experiences. In this review, physical activity factors showed no significant effect on a later onset of eating pathology, whereas teasing experiences gained some support for the role as a risk factor, but not consistently. Findings concerning several other factors, e.g., reading magazine articles on weight loss, trying to look like same sex figures in the media, and health concerns, each of which were reported in a single article, can be considered as preliminary results that call for more research to be confirmed. The number of studies investigating factors which probably maintain eating pathology was small, as it was already in the previous reviews. In fact, only three of the eligible studies reported on factors that maintain eating pathology and provide support for the previously reported findings of the influence of negative affect and body dissatisfaction on maintaining eating pathology, and the lack of such influence by BMI. The findings of other factors remained tentative and warrant further research.

To conclude, despite the wide range of factors investigated in association with eating pathology, the evidence of the risk effect is strong for very few factors. For most of the putative predictors, the findings are still inconclusive because of the lack of consistency or lack of replication. Inconsistent findings may partly be due to methodological variation between studies, e.g., differences between designs, settings, target populations, and follow-up periods, and variability in selecting, defining, and measuring the factors of interest. Discrepant findings may also be due to differences in modelling the outcome, e.g., the exclusion of important predictors or confounders from the model or overfitting the model. Thus, the conclusions of the role of many factors remain preliminary until confirmed in future studies.

In this study, an atheoretical approach facilitated the investigation of the effect of several factors on the occurrence of EDS in a school-based sample of adolescent girls and boys. The factors were derived from school health care records. Therefore, the number of factors used as putative predictors of eating pathology was smaller in this study than in the review presented above. Factors with a strong empirical evidence of their effect on eating pathology were included, but also factors the role of which was somewhat inconclusive in light of previous studies. Besides these, adolescents' experiences of parenting as perceived during the first 16 years of life were investigated as possible predictors, while the current evidence of the association between parental bonding and eating pathology is limited and inconsistent.

2.6   Conclusions of the reviewed literature

Recommendation to screen for eating disorders in adolescents as a part of school health care is warranted in light of the prevalence and adverse effects of EDS, the high rate of undetected cases, the availability of screening tools and treatment, and better prognosis with early intervention. Taking into account the preventive nature of screening, it is important to use a screen that not only is feasible and acceptable for both genders, but also enhances the detection of eating pathology in its early phases. The fact that the existing instruments originate from other countries and cultures necessitates the assessment of their applicability prior to implementation in the Finnish school health care.

The often quoted gender ratio of eating disorders among adults suggests that the illness occurs mainly in women, which favors females as a target group for screening intervention. However, in adolescent populations, the findings are less consistent; discrepancy in eating pathology between girls and boys varies from nonexistent to even higher than in adults. Consequently, possible gender-based screening limitations should only be considered on the basis of evidence of the gender-ratio of eating pathology in the population for which the screening is intended.

For the purpose of primary prevention, the sensitivity of the screen is a top priority when making a choice between instruments. However, while the sensitivity of an instrument increases, its specificity tends to decrease with an increased number of false positives. After the screening, the question remains which of the screen positives are in actual need for an intervention. To answer this question, a comprehensive picture of the development and course of eating pathology with regard to age and gender would be valuable. Unfortunately, despite the numerous theories and models of eating disorders designed so far, a universally sound theory is still missing. The majority of the etiological research to date is cross-sectional or retrospective in design, thus giving important information of the correlates of eating pathology but no evidence of their possible prognostic effect. Many prospective studies, in turn, use data from clinical samples and provide valuable findings concerning the factors associated with the treatment outcomes, but no information of the natural course of eating pathology. Community studies have their shortcomings as well. Some of them use data from heterogenous samples of females and males with a wide range of age without taking into account possible age and gender differences in the development and course of eating pathology, which complicates the generalization of the results in practice. In some of the studies, the methods or criteria to ascertain the outcome are changed between assessments hindering the interpretation of the results. Some, in turn, analyze both symptomatic and non-symptomatic participants as a single group and control for the level of eating pathology at the baseline. As a result, they shed light on factors associated with the change in eating pathology, but not on factors predicting the onset or maintenance of eating pathology. In fact, prospective community studies providing the evidence of the factors that maintain eating pathology are scarce. There is also a lack of evidence on how the course of these factors affects the course of eating pathology. Hence, several researchers have called for more prospective studies to investigate how the putative prognostic factors contribute to the onset of eating pathology, tend to maintain it, or serve to recover from it [e.g., 107; 411].

3 AIMS OF THE STUDY

The overall aim of the study was to enhance the detection of eating disorder symptoms (EDS) in adolescents and the use of health record information in identifying those with the highest risk for prolonged EDS or a later onset of EDS. The aim was approached by four steps with the following objectives and research questions:

1. **To test the feasibility of the SCOFF questionnaire in screening for EDS in adolescents in the setting of school health care (Paper I).**
   - How do the two alternative factor models of SCOFF fit in the populations of adolescent girls and boys?
   - How efficient is SCOFF, as compared with the established health examination practices in detecting EDS among adolescents?

2. **To describe gender differences in self-reported EDS and associated factors among adolescents (Paper II).**
   - How do adolescent girls differ from boys in terms of engaging in self-reported EDS?
   - How do adolescent girls differ from boys for the occurrence of several health risk factors measured within school health care?
   - How do adolescent girls differ from boys for susceptibility to these health risk factors in respect to self-reported EDS?
   - How are these health risk factors associated with self-reported EDS in adolescent girls and boys?

3. **To describe the course of self-reported EDS and associated factors with a follow-up period of 1 year (Paper III).**
   - How common are the recurrence of EDS and the onset of new presentations of self-reported EDS among adolescents by the 1-year follow-up?
   - How do adolescents with varying courses of EDS differ from each other over a 1-year follow-up period in respect to gender and several health risk factors measured within school health care?
   - How are these factors associated with the recurrence of self-reported EDS by the 1-year follow-up?
   - How are these factors associated with the onset of new presentations of self-reported EDS by the 1-year follow-up? (additional analyses)

4. **To describe the course of self-reported EDS and associated factors with a follow-up period of 4 years (Paper IV).**
   - How common are the recurrence of EDS and the onset of new presentations of self-reported EDS among adolescents by the 4-year follow-up?
   - How common is self-reported EDS in late adolescence at the population level? (additional analyses)
   - How do adolescents with varying courses of EDS differ from each other over a 4-year follow-up period in respect to gender and several health risk factors and experiences of perceived parenting?
   - How are these factors associated with the recurrence of self-reported EDS by the 4-year follow-up?
   - How are these factors associated with the onset of new presentations of self-reported EDS by the 4-year follow-up?

4   METHODOLOGY

4.1   Study design, setting, sampling, and data collection

The study included four stages conducted as observational surveys. The study was approved by the Ethics Committee of the Hospital District of Southwest Finland and the local school health care authority. Summary information of study stages (Papers I–IV) is given in Table 12. A flowchart of the different stages of the study is provided in Figure 2.

At the first stage, during the school year 2003–2004, a cross-sectional questionnaire survey was carried out in the setting of school health care using non-random sampling. Adolescents attending the 9th grade of Finnish-language secondary schools in the City of Turku were invited to participate in the study. Schools or classes for students with a handicap or a severe sensory deficit requiring assistance in responding were excluded from the study. Students received the study questionnaire from the school nurse in connection with their annual health examination. After excluding incompletely responded questionnaires, the adjusted response rate was 62%. (Figure 2.) The response rate was higher among girls than boys (69% vs. 55%, Pearson's chi-square $p < 0.001$). Of the 855 participants in the final sample, 55% (n = 474) were girls. The mean age in the sample was 15.5 years (SD 0.4, range 14.0–17.2). The data were used as a part of a larger dataset to investigate the feasibility and fit of the two alternative factor models of the SCOFF questionnaire in mid-adolescent girls and boys (Paper I). In addition, these data were used in the 1-year and 4-year follow-up assessments of the course of self-reported eating disorder symptoms (EDS) and associated factors (Papers III and IV). (Table 12.)

The second stage was implemented during the school year 2004–2005 with a similar procedure as in the first stage except that, first, the data were collected from adolescents attending the 8th grade of Finnish-language secondary schools and their school nurses, and second, each student returned the self-administered study questionnaire to the school nurse in a sealed envelope for the purpose of obtaining blinded assessments from the nurses regarding the adolescents' risk for an eating disorder (ED). The school nurses (n = 14) from the enrolled schools participated in the study. They were female public health nurses, aged 35 to 57 years (mean 49), and with an average experience of 22 years (range 8–32) in health care and 15 years (range 4–27) within school health care. After excluding incompletely responded questionnaires, the adjusted response rate among the students was 71%. (Figure 2.) The response rate was higher among girls than boys (76% vs. 66%, Pearson's chi-square $p < 0.001$). Of the total of 1036 participants in the final sample, 54% (n = 561) were girls. The mean age was 14.9 years (SD 0.5, range 13.0–18.2). These data were combined with the data collected at the first stage in order to investigate the fit of the two alternative factor models of SCOFF in mid-adolescent populations (Paper I). In addition, the data concerning the 8th graders were used to investigate gender differences in self-reported EDS and associated factors (Paper II). Furthermore, these data were used in the 4-year follow-up assessment of the course of self-reported EDS and its predictors (Paper IV). The school nurses gave an ED risk assessment for a total of 95% of the participating 8th grade students which equals 68% of the source population. (Figure 2.) These data were combined with the data of the 8th graders and the dataset was used to assess the efficiency of the SCOFF questionnaire in comparison to established health examination practice in detecting EDS in adolescents (Paper 1). (Table 12.)

The third stage of the study took place during the school year 2004–2005. A 1-year follow-up survey was carried out in the setting of student health care. Those students who had participated in the first stage and were now receiving upper secondary education either at a general upper secondary school or a vocational institute in Turku were enrolled to the follow-up survey. The school nurses from the total of 14 Finnish-language secondary schools and institutes were recruited to deliver the study questionnaires to students as a part of a health examination. It is not known how many students were actually offered an opportunity to participate in the survey, so it was not possible to calculate the response rate. A total of 372 students completed the questionnaire at follow-up, which equals 44% of the sample of 855 students at the first stage. (Figure 2). Of the respondents, 57% (n = 212) were girls. The mean age was 16.7 years (SD 0.4, range 15.7–18.1). An attrition analysis was conducted, which showed that the attrition rate was higher among individuals without anxiety than those with anxiety (57% vs. 44%, Pearson's chi-square p = 0.026). The data were used to describe the course of self-reported EDS over the follow-up period of one year and factors associated with this course (Paper III). (Table 12.)

Table 12. Description of the study stages (Papers I–IV)

	Paper I	Paper II	Paper III	Paper IV
Design	Cross-sectional survey	Cross-sectional survey	Longitudinal survey including two assessments with a 1-year interval	Longitudinal nested case-control survey including two assessments with a 4-year interval
Setting	School health care	School health care	Student health care	Community
Sampling	Non-random	Non-random	Consecutive	Disproportionate
Sample (number of participants, % of females)	Students (n = 1891, 55%), school nurses (n = 14, 100%)	Students (n = 1036, 54%)	Students (n = 372, 57%)	Late adolescents (n = 722, 74%)
Data collection	Self-administered structured questionnaire and health care records (students), self-administered semi-structured questionnaire (nurses)	Self-administered structured questionnaire and health care records	Self-administered structured questionnaire and health care records	Postal questionnaire survey and health care records
Outcome of interest	Feasibility of the SCOFF questionnaire in school health care	Gender differences in self-reported EDS and associated factors	Course of self-reported EDS within a year and associated factors	Course of self-reported EDS within four years and associated factors

At the fourth stage, a multi-instrument questionnaire was first tested in a pilot study in a sample of 19 late-adolescent girls (n = 12) and boys (n = 7) of approximately the same age (mean 20.7 years, SD 1.3, range 18–22 years) as the population in the final study. The results revealed no need for modifying the items of the questionnaire; all items were rated as clear or rather clear and easy or rather easy to respond. However, some of the response instructions were rephrased to guide participants to choose only one of the possible response options. The average responding time was 25 minutes.
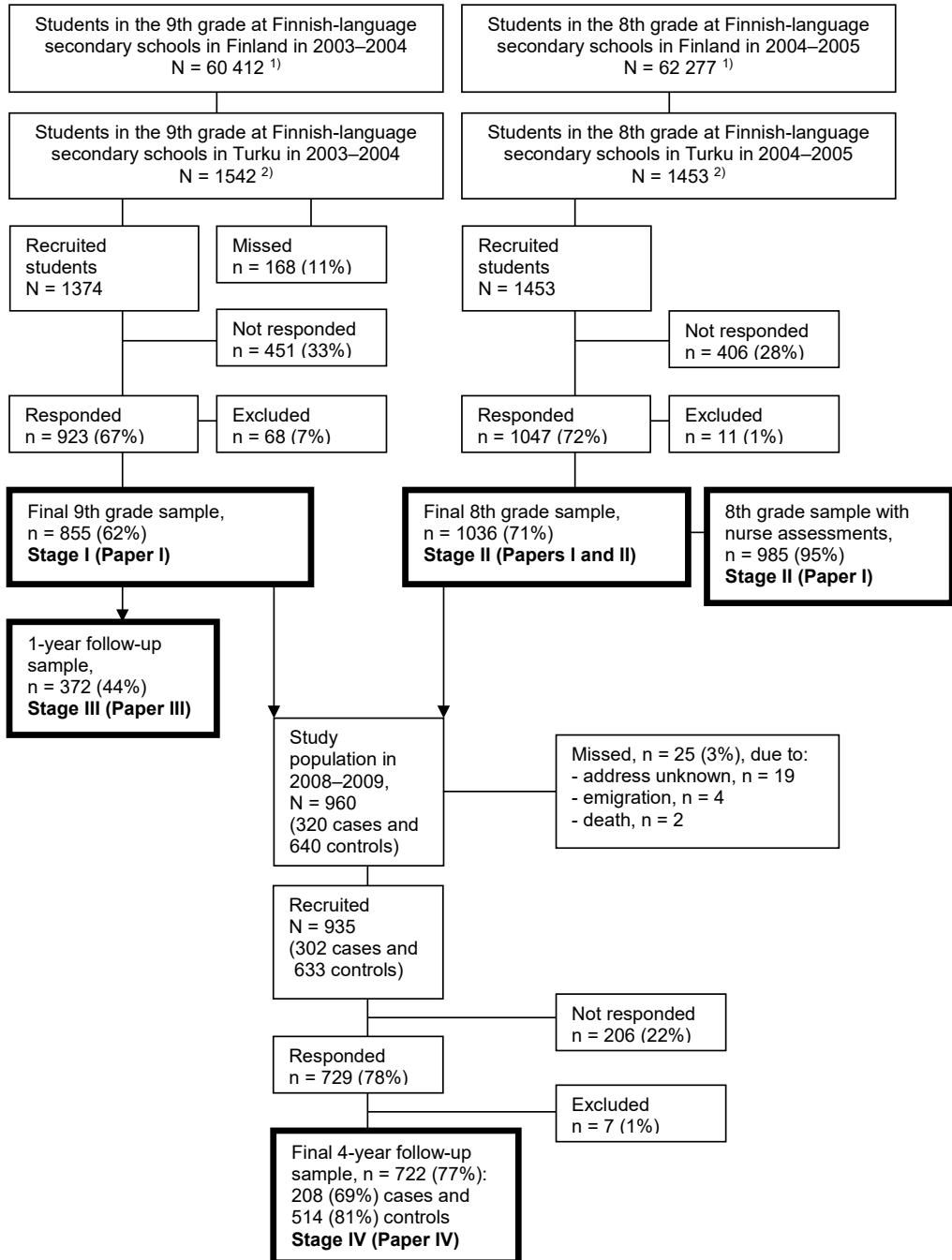
Note: [1] Statistics Finland; [2] School Centre of the City of Turku

Figure 2. Participant flow in the different stages of the study

After the pilot study, a 4-year follow-up postal questionnaire survey was carried out between October 2008 and March 2009. The study population included all those adolescents who had reported at least one EDS in the first or second stage of the study. For each of them, two random sampled non-symptomatic controls were recruited and matched, as a group, to the symptomatic participants by school, grade, and gender. In some schools, the number of available controls was too small, and therefore, 5% of the controls were recruited from randomly selected schools. Participants were asked to return the questionnaire in a separate postage-paid envelope. Those who did not respond to the first mailed survey were sent two reminders with an interval of one month to maximize participation. Of the total of 935 adolescents invited to participate, altogether 729 (78%) responded. After excluding incomplete responses, the adjusted response rate was 77% (Figure 2). According to the attrition analysis, attrition was more typical among boys than girls (37% vs. 17%, Pearson's chi-square $p < 0.001$) and among those with EDS than the non-symptomatic respondents (31% vs. 19%, Pearson's chi-square $p < 0.001$). In addition, attrition tended to be higher among older (Mann-Whitney U test $p = 0.021$) and overweight individuals (29% vs. 21%, Pearson's chi-square $p = 0.026$). Of the 722 participants, 74% (n = 535) were girls. The mean age was 14.9 (SD 0.6, range 13.0–16.9) years at the baseline assessment and 19.0 (SD 1.0, range 17.3–21.6) years at the 4-year follow-up. Two thirds of these late adolescents were living with their parent(s) (66%), while the rest of the respondents were either co-habiting (18%) or living alone (16%). The majority of the participants were students (82%), while the others were either at work (13%), in military or civil service (2%), unemployed (2%), or on a maternity leave (1%). As a token for compensation, the participants received two movie tickets each and took part in a lottery with a 200€ travel voucher as the prize. The data were used to investigate the course of self-reported EDS and associated factors over a period of approximately four years (Paper IV).

4.2   Measures

The outcome variables used in this study measured self-reported EDS and risk for an ED in adolescence. The explanatory variables were selected from those reported as putative predictors of eating pathology in previous studies.

4.2.1 Outcome variables

Finnish version of the SCOFF questionnaire

In Papers I–IV, adolescents' self-reported EDS was measured by means of the Finnish version of the SCOFF questionnaire [412]. The original English version of SCOFF [271] was translated into Finnish with a back-translation procedure [412]. The two versions of SCOFF are presented in Table 13. The clarity of the items of SCOFF, easiness to respond, and response time were assessed in a pilot study [412] conducted among 9th grade students (n = 29) and school nurses (n = 11). The majority of students found items 1–4 very or rather clear (96–100%) and very or rather easy to respond (95–96%). Item 5 was rated as being, at least, rather clear and easy to respond (85% and 79%, respectively). The average response time was 5 minutes. Of the school nurses, 82–100% assessed items 1, 3 and 4 very or rather clear, whereas items 2 and 5 were rated as, at least, rather clear by 64% of school nurses. The stability of the separate items of SCOFF, estimated as the proportion of agreement, was assessed among 8th grade students (n =

81). The proportion of agreement varied between 0.00–0.38 in symptomatic students and between 0.82–0.95 in asymptomatic students with an interval of approximately two weeks. The internal consistency of SCOFF, estimated with Cronbach's alfa, was 0.67. [412.]

As in the original SCOFF, the response options in the Finnish version of SCOFF are dichotomous giving 1 point for every 'Yes' answer and 0 points for every 'No' answer. Due to the preventive nature of this study, any self-reported EDS was considered a threshold for symptom severity. Accordingly, the cut-off was set at one point, with a dichotomy of 'no self-reported EDS' (0 points) and 'one or more self-reported EDS' (1–5 points). (Table 13.)

Table 13. The English and Finnish versions of the SCOFF questionnaire

The original English version of SCOFF [271]

1. Do you make yourself sick because you feel uncomfortably full?
2. Do you worry you have lost control over how much you eat?
3. Have you recently lost more than one stone in a 3-month period?
4. Do you believe yourself to be fat when others say you are too thin?
5. Would you say that food dominates your life?

The Finnish version of SCOFF [412]

1. Yritätkö oksentaa, koska tunnet olevasi epämiellyttävän kylläinen?
2. Huolestuttaako sinua ajatus, ettet enää pysty hallitsemaan syömisesi määrää?
3. Oletko laihtunut lähiaikoina yli kuusi kiloa kolmen kuukauden aikana?
4. Uskotko olevasi lihava, vaikka muut väittävät, että olet liian laiha?
5. Hallitseeko ruoka mielestäsi elämääsi?

Nowadays, there are two other versions of SCOFF in Finnish as well, with slightly different wording. One of them has been tested in a large sample (n = 1316) of general young adult population [273] and has provided the sensitivity of 77.8%, the specificity of 87.6%, positive predictive value of 9.7%, and negative predictive value of 99.6% with the threshold of two or more points in SCOFF. With the cut-off of one or more points, the results were 100%, 70.9%, 5.5%, and 100%, respectively, indicating a good ability of SCOFF to rule out an eating disorder and, on the other hand, a need for further assessment of those who disclose any EDS. [273.] The other Finnish version of SCOFF has been published in the Current Care Guideline for Eating Disorders [15]. No information of its psychometric properties has been reported.

Students' risk for an ED

In Paper I, school nurses gave their assessment of the students' risk for an ED on a semi-structured questionnaire designed for this study. The school nurses were asked to respond ('Yes'/'No') to the question: 'According to your assessment, is the student at risk for an eating disorder?' (Table 14). For every 'Yes' answer, the nurse was further requested to explain her assessment in her own words.

4.2.2 Explanatory variables

Explanatory variables used in this study included health risk factors and parental factors commonly implicated in the previous studies as predictors of eating pathology. At the first, second and third stages of this study, information of the health risk factors was obtained from the adolescents' health care records. At the fourth stage, the relevant information as well as the late adolescents' experiences of perceived parenting were collected with a self-report questionnaire. The health risk factors were classified into four groups: 1) biological factors and health complaints, 2) psychological factors, 3) behavioral factors, and 4) social factors.

Biological health-risk factors and health complaints

Biological factors included adolescents' relative weight and its change (Papers II, III), body mass index (BMI) (Paper IV), and health complaints (Paper III). Relative weight and its change were computed on the basis of the adolescent's height and weight measured by the school nurse. Change in relative weight was based on the comparison of the relative weight measured at the time of the data collection and one year earlier, and was classified as suggested by Näntö-Salonen and Toppari [413]. BMI was calculated of the self-reported height and weight in late adolescence and of the height and weight measured by the school nurse in mid-adolescence. The participants were classified into the 'No overweight' and 'Overweight' groups by using the age- and gender-specific BMI cut-points for children and adolescents, as presented by Cole and colleagues [414]. (Table 14.)

Health complaints comprised of abdominal pain, dizziness, fatigue, headache, and insomnia. These were measured as described in Table 14. An overall measure of health complaints was obtained by summing up the number of positive responses for each participant. The participants were classified into three groups representing their degree of suffering from health complaints: 'No complaints', 'One or two complaints', and 'Three or more complaints'.

Psychological health-risk factors

Psychological factors used in this study were depressiveness, anxiety, dissatisfaction with appearance (Papers II–IV), dissatisfaction with weight, and stress (Papers II, III). Depressiveness, anxiety, and dissatisfaction with appearance were measured with Raitasalo's modification of the 13-item Beck Depression Inventory [415], entitled RBDI [416]. In the questionnaire used in school health care, some response options have been slightly modified from the original RBDI for the purpose of screening mid-adolescents. A list of the modified items and the original ones is presented in Appendix 1. The first 13 items of RBDI assess the respondent's mood on a 5-category scale, showing the intensity of depressive emotions and cognitions on that day. For those who had one to four of these items unanswered, adjusted scores were calculated following instructions presented by Raitasalo [416]. When five or more of the items were unanswered, the respondent's data were excluded from the analysis. The severity of depression is recommended to be classified as presented in Table 14. In clinical practice, the cut-off for further assessment of the symptoms and severity of depression is suggested to be set at five or more points. [416.] Previous studies among adolescents [328] have shown good internal consistency for RBDI (0.84–0.87). In this study, Cronbach's alfa was 0.81 in the 8th grade sample, 0.76 in the 9th grade sample, 0.85 in the sample of students in upper secondary education, and 0.88 in the sample of late adolescents.

Anxiety was measured with the fourteenth item of RBDI. In clinical practice, it is recommended that attention be given to any existing anxiety [416]. Accordingly, responses expressing mild to severe anxiety were labelled as 'Anxiety' and an anxiety-free state as 'No anxiety'. (Table 14.)

The question of self-image also derives from RBDI [416]. The five response options express the intensity of dissatisfaction with appearance, increasing from satisfaction ('I am quite happy with my appearance') to dissatisfaction ('I feel that I am ugly or repulsive-looking'). The ratings of satisfied and neutral feelings about personal appearance were combined as 'Satisfied/Neutral', and the ratings expressing dissatisfaction with appearance were combined as 'Dissatisfied'. Dissatisfaction with one's weight and perceived stress were assessed with dichotomous ('Yes'/'No') questions. (Table 14.)

Behavioral health-risk factors

Behavioral factors focused on substance use and regularity of meals (Paper II). The use of tobacco was measured with two questions, 'Do you smoke?' and 'Do you use chewing tobacco?'. In order to identify every user of tobacco, the responses were combined and all responses indicating the use of tobacco, at least sometimes, were labelled as 'Use of tobacco'. Negative responses to both questions were labelled as 'No use of tobacco'. In line with this, responses to the question concerning alcohol consumption were dichotomized as 'Use of alcohol' and 'No use of alcohol'. Regularity of meals was assessed with a dichotomous ('Yes'/'No') question. (Table 14.)

Social health-risk factors

Social factors included teasing (Paper II), loneliness (Papers II, III), and family communication (Paper II). Teasing was measured with two statements about physical and mental teasing at school. The responses were combined and scored so that at least one positive answer was labelled as 'Teased'. Negative responses to both statements were combined as 'Not teased'. Loneliness and family communication were measured with dichotomous ('Yes'/'No') questions (Table 14).

Table 14. Variables used in the study

Variable (Paper)	Measure/Item(s)	Scale	Response options / Rating	Classification
Outcome variables				
Self-reported eating disorder symptoms (EDS) (Papers I–IV)	SCOFF	5 dichotomous items	'No' = 0 points 'Yes' = 1 point Sum of points range: 0–5	0 points = 'No self-reported EDS' 1–5 points = 'Self-reported EDS'
Eating disorder (ED) risk assessed by the school nurse (Paper I)	'According to your assessment, is the student at risk for an ED?'	3 category Likert scale	'No' 'Yes' 'Do not know'	No = 'No risk for an ED' Yes = 'ED risk' Do not know = 'Unsure risk for an ED'
Explanatory variables				
Relative weight (RW) (Papers II, III)	Height and weight measured by the school nurse	Height (cm), weight (kg)	Individual's weight divided by the mean weight of the standard population in age- and gender-matched height groups	RW < −15% = 'Underweight' −15% ≤ RW ≤ +20% = 'Normal weight' RW > +20% = 'Overweight'
Change in RW (Papers II, III)	Height and weight measured by the school nurse	Height (cm), weight (kg)	Difference between RW at the moment and RW a year ago	RW decreased > 10% = 'Abnormal weight loss' RW ± 10% = 'Normal change in RW' RW increased > 10% = 'Abnormal weight gain'
Body Mass Index (BMI) (Paper IV)	Height and weight measured by the school nurse (Stage I) and self-reported by the participants (Stage IV)	Height (cm), weight (kg)	Individual's weight in kilograms divided by the height in square meters (kg/m^2)	'No overweight' 'Overweight' based on age- and gender-specific cutoffs
Health complaints (Paper III)	'During the last half of the year, have you had some of the following symptoms and how often?' 'Abdominal pain', 'Dizziness', 'Fatigue', 'Headache', 'Insomnia'	4-category Likert scale	'Not at all / Seldom' = 0 points 'About once in a month' = 0 points 'About once in a week' = 1 point 'Almost every day' = 1 point Sum of points range: 0–5	0 points = 'No complaints' 1–2 points = 'One or two complaints' 3–5 points = 'Three or more complaints'
Depressiveness (Papers II–IV)	RBDI	13 items with a 5-category Likert scale scoring 0–3 points in each category	'None or very mild' = 0–4 points 'Mild' = 5–7 points 'Moderate' = 8–15 points 'Severe' = 16–39 points	0–4 points = 'No depressiveness' 5–39 points = 'Depressiveness'
Anxiety (Papers II–IV)	Item 14th of RBDI: 'Are you tense or distress?'	5-category Likert scale	'No anxiety' = 1–2 'Mild anxiety' = 3 'Moderate tension or anxiety' = 4 'Severe tension or anxiety' = 5	1–2 = 'No anxiety' 3–5 = 'Anxiety'
Dissatisfaction with appearance (Papers II–IV)	Item 10th of RBDI: 'How do you feel about your appearance?'	5-category Likert scale	Increasing intensity of dissatisfaction with appearance, range: 1–5	1–2 = 'Satisfied/Neutral' 3–5 = 'Dissatisfied with appearance'
Dissatisfaction with weight (Papers II, III)	'Are you satisfied with your weight?'	Dichotomous	'No' = 0 'Yes' = 1	0 = 'Satisfied' 1 = 'Dissatisfied with weight'

(continues)

Table 14 (continues)

Variable (Paper)	Measure	Scale	Response options/Rating	Classification
Stress (Papers II, III)	'During the last six months, have you been recurrently stressed?'	Dichotomous	'No' = 0 'Yes' = 1	0 = 'No stressed' 1 = 'Stressed'
Use of tobacco (Paper II)	'Do you smoke?' and 'Do you use chewing tobacco?'	4-category Likert scale in each question	'No' = 0 points 'Sometimes' = 1 point 'Once a week' = 1 point 'Daily' = 1 point Sum of points range: 0–2	0 points = 'Not using tobacco' 1–2 points = 'Using tobacco'
Use of alcohol (Paper II)	'Have you used alcohol?'	3-category Likert scale	'No' = 0 points 'Yes, less than 3 times' = 1 point 'Yes, more than 3 times' = 1 point	0 points = 'Not using alcohol' 1 point = 'Using alcohol'
Regularity of meals (Paper II)	'Do you have regular meals?'	Dichotomous	'No' = 0 'Yes' = 1	0 = 'No regular meals' 1 = 'Regular meals'
Teasing (Paper II)	'I have been teased physically at school' and 'I have been teased mentally at school'	3-category Likert scale	'Disagree' = 0 points 'Somewhat agree' = 1 point 'Agree' = 1 point Sum of points range: 0–2	0 points = 'Not teased' 1–2 points = 'Teased'
Loneliness (Papers II, III)	'During the last six months, have you been recurrently lonely?'	Dichotomous	'No' = 0 'Yes' = 1	0 = 'Not lonely' 1 = 'Lonely'
Family communication (Paper II)	'Can you talk to your parents about your problems?'	Dichotomous	'No' = 0 'Yes' = 1	0 = 'Poor communication in the family' 1 = 'Good communication in the family'
Experience of perceived parenting (Paper IV)	PBI: Care subscale and Overprotection subscale	25 statements to be assessed separately for each parent: 12 statements in the Care subscale and 13 statements in the Overprotection subscale, 4-category Likert scale	'Very like' 'Moderately like' 'Moderately unlike' 'Very unlike' scoring 0–3 points in each category Care subscale: sum score range 0–36 Overprotection subscale: sum score range 0–39	Maternal Care: 0–27 points = 'Low care' 28–34 points = 'Average care' 35–36 points = 'High care' Paternal Care: 0–21 points = 'Low care' 22–32 points = 'Average care' 33–36 points = 'High care' Maternal Overprotection: 0–5 points = 'Low overprotection' 6–13 points = 'Average overprotection' 14–39 points = 'High overprotection' Paternal Overprotection: 0–3 points = 'Low overprotection' 4–10 points = 'Average overprotection' 11–39 points = 'High overprotection'

Note: RBDI = Raitasalo's modification of the short form of the Beck Depression Inventory [416]; PBI = Parental Bonding Instrument [417].

Parental factors

Late adolescents' experiences of perceived parenting (Paper IV) were measured with the Parental Bonding Instrument (PBI) [417; 418]. PBI approaches maternal and paternal contribution to parent-child bonding in terms of care (12 items) and overprotection (13 items) as recalled by individuals over the first 16 years of life. The summed scores for the Care subscale indicate the level of affection in the parent-child relationship with higher scores referring to empathy, closeness, and emotional warmth, while lower scores indicate rejection and emotional coldness. For the Overprotection subscale, the summed scores indicate the level of psychological autonomy in parents' involvement with their children. Higher scores indicate control and overprotection and lower scores refer to fostering of autonomy and independence. (Table 14.) When 20% or less of the items were unanswered, adjusted scores were calculated using the average score of the answered items. The data were rejected where more than 20% of the items were unanswered.

To date, PBI has been used in several ways. Some researchers [e.g., 97; 207; 419; 420] have used the original two-factor-model of PBI, whereas others [e.g., 421; 422] have preferred the three-factor-version of PBI with the subscales of 'care', 'overprotection', and 'authoritarianism'. Furthermore, in some studies, PBI has been used as a continuous variable [e.g., 419; 420; 423; 424], whereas some have assigned responses to four broad parental styles [e.g., 425; 426; 427; 428], or focused on one of these styles [e.g. 97; 207], or used extreme scores [425]. As the designer of PBI does not favor any specific way of using PBI [418], this study approached parental bonding in terms of 'average parenting' with the original two-factor-model of PBI, due to the evidence of its satisfactory reliability and validity [418]. Each subscale was categorized into three intervals on the basis of the quartiles of the summed scores: the upper quartile (the highest 25%), the intermediate quartile (the average-level 50%), and the lower quartile (the lowest 25% scores). Accordingly, the subscales were categorized as presented in Table 14. The internal consistency of PBI subscales has been good (0.87–0.93) in previous studies among youth [429], and the stability of PBI has been acceptable and independent of the respondent's gender [430]. In this study, Cronbach's alphas for the subscales of Maternal Care, Maternal Overprotection, Paternal Care, and Paternal Overprotection were 0.92, 0.85, 0.93, and 0.85, respectively.

4.2.3 Demographic variables

In this study, the demographic data for the adolescents' included age (Paper IV), gender (Papers I–IV), grade (Papers I, IV), school (Papers II–IV), status as a student vs. non-student (Paper IV), and living with parents vs. other (Paper IV). Age and gender were obtained from school health care records, and the school and grade were reported by the school nurses. Participants themselves reported their status as a student vs. non-student and gave information about their living with parents vs. other living arrangements.

Demographic data for the school nurses (Paper I) consisted of age (years), professional education (public health nurse, midwife, nurse, nurse specialist, or other), and the duration of work experience (years) in health care, and particularly in school health care. This information was collected with structured questions designed for this study.

4.3 Data analyses

The data analyses used in Papers I–IV and in the secondary analyses are summarized in Table 15. Non-response analyses were conducted at the first and second stages with the

Pearson's chi-square test of the independence by comparing the gender rate of participants to gender distribution of those who declined to participate in the study. At the third stage, the attrition analysis was performed by comparing those who withdrew from the follow-up assessment with those who participated in both assessments. Comparisons were made with the Pearson's chi-square test regarding gender, self-reported EDS, and the explanatory factors measured in the baseline assessment. In the fourth stage, the Pearson's chi-square test and Mann-Whitney U test, when appropriate, were used for the attrition analyses. Participants who provided data at both assessments were compared with those who did not participate in the follow-up assessment. Comparisons were made for gender, self-reported EDS, and the explanatory factors measured at baseline, and the individuals' age at the beginning of the follow-up assessment. Possible multicollinearity between the explanatory factors was assessed with the variance inflation factor (VIF). The results supported the assumption of no multicollinearity. [431.]

In Paper I, confirmatory factor analysis (CFA) was used to test the two alternative factor models of SCOFF. In prior studies, SCOFF has been used as a unidimensional scale and therefore the one-factor solution was used as the hypothesized model (M1). The two-factor model (M2) consisted of factor one (F1), entitled 'Loss of Control Over Food' (items 2, 4, and 5), and factor two (F2), entitled 'Purging Behaviors' (items 1 and 3) [279]. First, in order to reveal the possible age effect on the screening result, both factor models were tested with CFA for the samples of 8th and 9th graders, separately. Then, the samples were combined and, in order to detect the possible gender dependence in the interpretation of the screening result, CFA was performed separately for girls and boys. Tetrachoric correlations and asymptotic covariance matrices between the items of SCOFF were calculated. Accordingly, the correlation matrices were used to estimate a confirmatory factor model, applying the weighted least squares method [432]. The goodness of fit for the models was assessed with the Satorra-Bentler's scaled chi-square test [433], root-mean-square error of approximation (RMSEA) [434], and Akaike's information criterion (AIC) [435]. A nonsignificant ($p > 0.05$) probability value of chi-square indicates a good fit model [436]. RMSEA estimates the lack of fit in a model compared to a perfect model; the lower the RMSEA, the better the fit model. The value of the RMSEA at 0.05 or less indicates a good-fitting model. [434.] AIC measures the information lost when a given model is used to describe reality. Hence, the lower values of the AIC index indicate the preferred model [435]. CFA were performed with LISREL software, version 8.71. (Table 15.)

Finally, the efficiency of SCOFF in detecting EDS in adolescents was examined against an ED risk assessed by the school nurses on the basis of a health examination and students' health care records. Percentages and frequencies were used for the estimates of the prevalence scores. (Table 15.) These analyses were performed with SPSS 14.0 for Windows. The explanations school nurses gave for their positive ED risk assessments were described as frequencies and categorized into issues involving weight, health care record information, eating behaviors, and health complaints.

In Paper II, cross-tabulations and Pearson's chi-square test of independence were used to investigate the difference between adolescent girls and boys in the prevalence of self-reported EDS and in the occurrence of several health risk factors. Logistic regression analyses were used to assess the possible differences between genders in susceptibility to these health risk factors in respect to self-reported EDS and to investigate the associations between the health risk factors and self-reported EDS. In all models, school was included as a random factor because of possible intra-class correlation within schools, as reported in previous studies [e.g., 437]. Three steps were used in fitting the models.

First, the health risk factors were modelled separately. The explanatory variables consisted of the health risk factor at issue, gender and an interaction term between gender and the health risk factor. Second, the health risk factors were classified into four subgroups: biological, psychological, behavioral and social factors. Variables for these models were selected on the basis of the results obtained in the first step. The models also included gender, but the interaction terms only if they were statistically significant in the first step. Third, the subgroups were combined to form a final model in which the explanatory variables consisted of gender and health risk factors that were significant in the subgroups. As none of the interaction terms between gender and a health risk factor was significant in the subgroups, the final model was based on the combined data of girls and boys. Associations were quantified using odds ratios (OR) with 95% confidence intervals (95% CI) and *p*-values < 0.05 were interpreted as statistically significant. (Table 15.) The statistical analyses were performed with SPSS 12.0.2 for Windows, and with SAS system for Windows, release 9.1, applying the GLIMMIX procedure.

In Paper III, the recurrence of self-reported EDS was assessed by means of descriptive statistics, McNemar's test using the dichotomised score of SCOFF, and the proportions of agreement [438] separately for each item of SCOFF. Change in the sum score of SCOFF from the baseline assessment to the 1-year follow-up was assessed with Wilcoxon's sign rank test. Descriptive statistics were also applied to assess the onset of the new presentations of self-reported EDS. Cross-tabulations and Fisher's exact tests were used to investigate the differences between the groups of adolescents with differing courses of self-reported EDS in respect to gender and health risk factors. Finally, multivariable logistic regression analyses were performed to investigate the factors associated with the course of self-reported EDS. These factors were selected on the basis of Fisher's exact tests at baseline assessment. In the models, associations were quantified using OR with 95 % CI. *P*-values < 0.05 were interpreted as statistically significant. (Table 15.) The analyses were performed with SPSS 12.0.2 for Windows, and with SAS system for Windows, release 9.1.

In Paper IV, the analyses started with preliminary tests to assess whether the course of self-reported EDS differed between girls and boys. A logistic regression analysis was performed with the explanatory variables of time, gender, and an interaction term between time and gender. The result indicated that the time x gender interaction was significant, suggesting a gender difference in the course of self-reported EDS. Therefore, further analyses were performed separately for girls and boys.

Next, descriptive statistics and logistic regression analyses were used to assess the recurrence and the onset of new presentations of self-reported EDS from the baseline to the 4-year follow-up assessments at individual level. Based on the course of self-reported EDS, each participant was placed in one of the following categories: 1) symptom-free at both assessments (noEDS), 2) self-reported EDS at baseline assessment but asymptomatic at follow-up (EDS1), 3) symptom-free at baseline but self-reported EDS at follow-up (EDS2), and 4) self-reported EDS at both assessments (EDS12). Then, logistic regression analyses were performed to investigate differences between the EDS12 versus EDS1 groups, and between the EDS2 versus noEDS groups in relation to health risk and parental factors. Due to the small number of boys showing the recurrence and onset of EDS, these analyses were performed for girls only. In the first step, simple logistic regression analyses were performed using health risk and parental factors one by one as an explanatory variable. Second, multivariable analyses were applied separately for the two subgroups: health risk factors and parental factors. Finally, the effect of factors that remained significant in subgroup analyses was estimated in association with the

recurrence as well as the onset of EDS. School was included as a random factor in all models. *P*-values < 0.05 were interpreted as statistically significant. (Table 15.) The analyses were performed with SAS system for Windows, release 9.1, applying the GLIMMIX procedure, and with SPSS 16.0 for Windows.

Table 15. Statistical analyses used in the study

Paper	Outcomes of interest	Analyses
I	1. Difference between the participants and non-participants 2. Fit of the two alternative factor models of SCOFF 3. Added value of the screening procedure with SCOFF for the detection of ED risk group in comparison to health examination practice without a screening procedure	1. Pearson's chi-square test of the independence 2. Confirmatory factor analyses 3. Descriptive statistics
II	1. Difference between the participants and non-participants 2. Gender differences in the prevalence of EDS 3. Gender differences in the occurrence of the health risk factors 4. Gender differences in susceptibility to the health risk factors with respect to EDS 5. Associations between health risk factors and EDS	1–3. Pearson's chi-square test of the independence 4–5. Logistic regression analyses
III	1. Difference between the participants and non-participants 2. The recurrence of EDS 3. Changes in the total SCOFF score 4. The onset of the new presentation of EDS 5. Associations between the explanatory variables and the course of EDS 6. Predictors of the recurrence of EDS	1. Pearson's chi-square test of the independence 2. Descriptive statistics, Mc Nemar's test, and the proportion of agreement 3. Wilcoxon's signed rank test 4. Descriptive statistics 5. Fisher's exact test 6. Logistic regression analyses
IV	1. Difference between the participants and non-participants 2. Moderating effect of gender in respect to the course of EDS 3. Recurrence of EDS and the onset of new presentations of EDS 4. Associations between the explanatory variables and the course of EDS 5. Predictors of the course of EDS 6. Multicollinearity between the explanatory variables	1. Pearson's chi-square test of the independence and Mann-Whitney-U test 2. Logistic regression analysis 3. Logistic regression analyses and descriptive statistics 4–5. Logistic regression analyses 6. Variance inflation factor (VIF)
Secondary analyses	1. Predictors of the onset of new presentations of EDS 2. Population at risk for EDS in late adolescence	1. Logistic regression analyses 2. Descriptive statistics

The secondary analyses concerned, first, the predictors of the onset of new presentations of self-reported EDS during a 1-year follow-up period and, second, the population at risk for EDS in late adolescence. The putative predictors for the onset of new presentations of self-reported EDS were assessed with logistic regression analyses. While the number of factors that showed significant differences between the group of adolescents who developed EDS during the 1-year follow-up period and those who remained non-symptomatic was relatively high, the analyses were performed in two steps. In the first step, multivariable logistic regression analyses were conducted using backward and forward methods to find the best predictors for the onset of self-reported EDS. Next, the factors that were found statistically significant in the first step were entered into a gender-controlled logistic regression analysis. School was included as a random factor in all

models. The analyses were performed with SAS system for Windows, release 9.3, applying the GLIMMIX procedure, and with SPSS 21.0 for Windows.

The population at risk in late adolescence was computed as follows: 1) the relative number of mid-adolescents with EDS was multiplied with the relative number of late adolescents with recurring EDS, 2) the relative number of mid-adolescents without EDS was multiplied with the relative number of late adolescents with new onset of EDS, and 3) the results of these multiplications were summed up. The population at risk for prolonged EDS was computed by multiplying the relative number of mid-adolescents with EDS with the relative number of late adolescents with recurring EDS. These computations were performed for the total population, and separately for girls and boys. All computations are presented in Appendices 2, 3, and 4. For the computed estimates, 95% confidence intervals were calculated [see 439].

4.4 Ethical issues

The general principles of research ethics [440; 441; 442; 443; 444], and those concerning especially adolescents [445], were taken into account. The study was approved by the Ethics Committee of the Hospital District of Southwest Finland and the local school health care authority. Permission for the use of the SCOFF questionnaire was granted by Doctor J.F. Morgan. Potential participants of the study were given written information about the research, together with the contact information of the researcher for requesting additional information. The information letters were delivered by the school nurses at the same time they informed the students of the forthcoming health examinations. The intention was to avoid interrupting the usual work at school and interfering with the adolescents' regular health care. The researcher informed the school nurses about the research at its various stages in the nurses' usual meetings.

Participation in the study was voluntary. A written informed consent was obtained from individuals aged 15 years and older and, in a case of a younger participant, also from the parents/guardians. The only exception was the third stage of the study where participants were considered to renew their earlier consent by responding to the survey. This was justified by the assumption that the research caused no harm to participants' health. In general, adolescents are able to self-report their behaviors and attitudes [246] and they are found to be unharmed when asked questions concerning risky weight control behaviors and attitudes [446]. A concern has been raised that some individuals might find the questions of SCOFF as intrusive because of the feelings of shame and secrecy inherent to eating disturbances [447]. The fear of judgment and embarrassment may affect adolescents especially in face-to-face interactions [247]. Therefore, when investigating sensitive issues, such as self-induced vomiting, the use of self-report measures is recommended [31; 358; 448]. Instead of causing harm, the research was foreseen to benefit participants and other adolescents by helping clinicians to identify youth who are in need for special attention due to eating pathology.

At stages one to three, all eligible adolescents were recruited in order to avoid giving an impression that participants are someway different from their peers. At the fourth stage, there was no risk of such impression as the data were collected by a postal survey. To protect the privacy of participants and the confidentiality of their personal information, the identification data were encrypted in the analyses.

5 RESULTS

5.1 Description of the participants

The baseline student cohort comprised a total of 1891 mid-adolescents in the 8th (n = 1036, mean age 14.9 years) and 9th (n = 855, mean age 15.5 years) grade of Finnish-language secondary schools in the City of Turku. A total of 55% of the students were females. The sample of school nurses included 14 individuals (mean age 49 years). All of them were females. Each school nurse had the education of a public health nurse. Eight of them also had the qualification of a nurse or a nurse specialist. They had work experience of 4–27 years (mean 15 years) in school health care and 8–32 years (mean 22 years) in health care in general. In the 1-year follow-up survey, 57% of the 372 adolescents (mean age 16.7 years) were females, and in the final 4-year follow-up assessment, 74% of the 722 respondents (mean age 19.0 years) were females. At the time of the final assessment, 66% of the respondents were living with their parents, while 18% were co-habiting and 16% reported living alone. Altogether 82% of the respondents were students, 13% were at work, 2% in military or civil service, 2% reported being unemployed, and 1% were on a maternity leave.

5.2 Utility of the SCOFF questionnaire

Responses to all items of SCOFF were given by 99% of the respondents in the 8th grade and 93% of those in the 9th grade. The rate of missing data across the items was 0.7–0.8% and 1.2–1.5% for 8th and 9th graders, respectively, which supports the assumption that the items of SCOFF are clear and easy to respond. Tetrachoric correlations between the items were higher in older adolescents and girls than in younger participants and boys indicating more diffuse EDS in the latter groups (Tables 16a, 16b, 16c).

Table 16a. Tetrachoric correlations between the items of SCOFF for adolescents in the 8th grade (n = 1036) (above the diagonal line) and the 9th grade (n = 855) (below the diagonal line)

Item	1.	2.	3.	4.	5.
1. Intentional vomiting	1.00	0.40	0.32	0.03	0.25
2. Loss of control over eating	0.55	1.00	0.30	0.38	0.45
3. Weight loss	0.79	0.26	1.00	0.23	0.46
4. Body dissatisfaction	0.71	0.51	0.60	1.00	0.37
5. Food intrusive thoughts	0.71	0.67	0.52	0.55	1.00

Table 16b. Tetrachoric correlations between the items of SCOFF for girls in the 8th grade (n = 561) (above the diagonal line) and the 9th grade (n = 474) (below the diagonal line)

Item	1.	2.	3.	4.	5.
1. Intentional vomiting	1.00	0.42	0.41	0.03	0.41
2. Loss of control over eating	0.56	1.00	0.34	0.38	0.57
3. Weight loss	0.78	0.35	1.00	0.30	0.49
4. Body dissatisfaction	0.64	0.49	0.55	1.00	0.54
5. Food intrusive thoughts	0.65	0.69	0.57	0.54	1.00

Table 16c. Tetrachoric correlations between the items of SCOFF for girls (n = 1035) (above the diagonal line) and boys (n = 856) (below the diagonal line) in the 8th and the 9th grade

Item	1.	2.	3.	4.	5.
1. Intentional vomiting	1.00	0.48	0.63	0.37	0.54
2. Loss of control over eating	0.29	1.00	0.34	0.43	0.62
3. Weight loss	0.52	0.10	1.00	0.42	0.53
4. Body dissatisfaction	0.43	0.26	0.43	1.00	0.52
5. Food intrusive thoughts	0.31	0.40	0.42	0.17	1.00

The factor loadings for the 1-factor and 2-factor models of SCOFF ranged from moderate to high in each sample, expressing acceptable relationships between the items and both constructs of SCOFF (Table 17).

Table 17. Factor loadings for two alternative CFA models (M1, M2) of SCOFF

Sample	Model	Factor	Item 1	Item 2	Item 3	Item 4	Item 5
S1	M1	F1	0.43	0.66	0.57	0.47	0.72
(n = 1036)	M2	F1	–	0.66	–	0.48	0.73
		F2	0.48	–	0.67	–	–
S2	M1	F1	0.98	0.57	0.79	0.74	0.73
(n = 855)	M2	F1	–	0.69	–	0.72	0.79
		F2	0.99	–	0.73	–	–
S1 girls	M1	F1	0.47	0.66	0.56	0.56	0.89
(n = 561)	M2	F1	–	0.64	–	0.57	0.92
		F2	0.58	–	0.70	–	–
S2 girls	M1	F1	0.92	0.62	0.80	0.70	0.74
(n = 474)	M2	F1	–	0.76	–	0.69	0.84
		F2	0.98	–	0.79	–	–
S12 girls	M1	F1	0.71	0.69	0.67	0.60	0.82
(n = 1035)	M2	F1	–	0.71	–	0.61	0.87
		F2	0.82	–	0.77	–	–
S12 boys	M1	F1	0.71	0.36	0.71	0.58	0.50
(n = 856)	M2	F1	–	0.39	–	0.59	0.51
		F2	0.72	–	0.71	–	–

Note: CFA = confirmatory factor analysis; M1 = 1-factor model; M2 = 2-factor model; S1 = 8th graders; S2 = 9th graders; S1 girls = girls in the 8th grade; S2 girls = girls in the 9th grade; S12 girls = girls in the 8th and 9th grades; S12 boys = boys in the 8th and 9th grades

The between-factor correlations were all strong, ranging from 0.75 to 0.95, thus indicating co-occurrence of weight loss behaviors and disordered attitudes to body, eating and food. The goodness-of-fit statistics showed a good fit of both models in all samples (Table 18).

Table 18. Between-factor correlations and the goodness-of-fit indicators for two alternative factor models (M1, M2) of SCOFF

Sample	Model	r	χ^2	df	*p*-value	RMSEA	AIC
S1	M1	–	3.12	5	0.682	<0.001	23.12
(n = 1036)	M2	0.82	2.92	4	0.571	<0.001	24.92
S2	M1	–	8.20	5	0.146	0.027	28.20
(n = 855)	M2	0.83	3.82	4	0.431	<0.001	25.82
S1 girls	M1	–	2.75	5	0.738	<0.001	22.75
(n = 561)	M2	0.75	2.00	4	0.736	<0.001	24.00
S2 girls	M1	–	5.31	5	0.379	0.011	25.31
(n = 474)	M2	0.81	2.01	4	0.735	<0.001	24.01
S12 girls	M1	–	6.79	5	0.237	0.019	26.79
(n = 1035)	M2	0.77	1.56	4	0.817	<0.001	23.56
S12 boys	M1	–	3.43	5	0.634	<0.001	23.43
(n = 856)	M2	0.95	3.02	4	0.554	<0.001	25.02

Note: M1 = 1-factor model; M2 = 2-factor model; S1 = 8th graders; S2 = 9th graders; S1 girls = girls in the 8th grade; S2 girls = girls in the 9th grade; S12 girls = girls in the 8th and 9th grades; S12 boys = boys in the 8th and 9th grades; χ^2 = Satorra-Bentler's chi-square; df = degrees of freedom; *p*-value = Satorra-Bentler's chi-square test; RMSEA = Root-mean-square error of approximation; AIC = Akaike's information criterion

The results of the school nurses' ED risk assessments against adolescents' responses to SCOFF are illustrated in Figure 3. Altogether 21% (204/985) of adolescents self-reported one or more EDS in SCOFF. According to the school nurse assessments, ED risk was present in 5% (47/985) of adolescents; in 11% (22/204) of those who self-reported EDS in SCOFF and in 3% (25/781) of the others. A total of 81% (165/204) of adolescents with self-reported EDS in SCOFF remained undetected in the health examination; 66% (33/50) of those with several self-reported EDS and 86% (132/154) of those with one self-reported EDS. For 5% (46/985) of adolescents, school nurses gave uncertain risk assessment. (Figure 3.)

The rationales school nurses gave to explain their risk ratings involved adolescent's weight (26 statements), prior treatment of disordered eating (9 statements), eating behavior (7 statements), and health complaints (3 statements). Altogether 32% (15/47) of the positive ED risk ratings lacked further explanation.
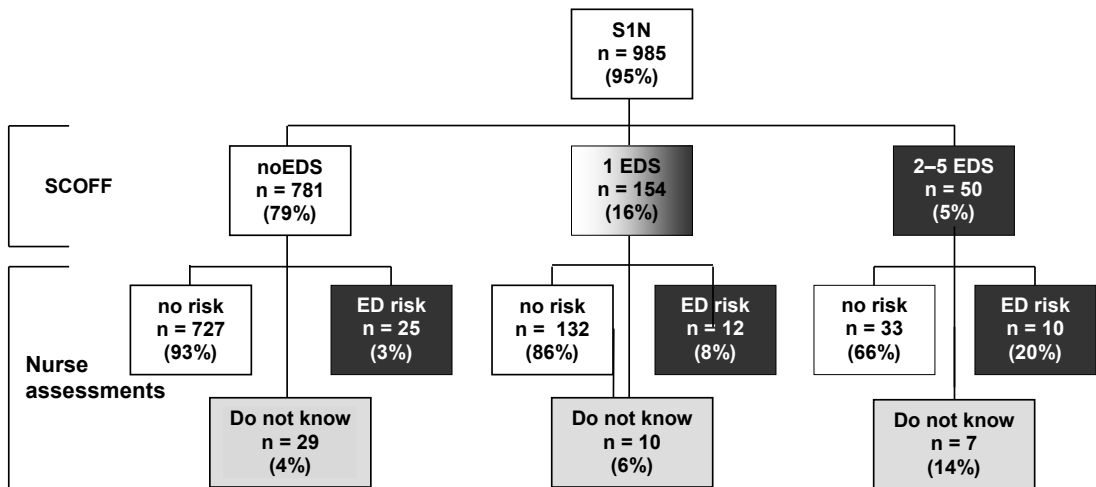
Figure 3. Cross-sectional comparison of self-reported eating disorder symptoms (EDS) in SCOFF and blinded nurse assessments of adolescents' risk for an eating disorder (ED), separately for adolescents without self-reported EDS (noEDS), with one self-reported EDS (1 EDS), and with two or more self-reported EDS (2–5 EDS), in a sample of 8th grade students (S1N)

5.3   Self-reported EDS and associated factors

The prevalence of self-reported EDS was higher in girls than boys (p = 0.001). At baseline, at least one EDS was reported by 20% (209/1036) of adolescents in the 8th grade: 24% (134/561) of girls and 16% (75/475) of boys. Several EDS were reported by 5% (53/1036) of participants: 7% (40/561) of girls and 3% (13/475) of boys. Examination of the separate symptoms showed that fear of losing control over eating was more common among girls than boys (14.3% vs. 6.9%, p < 0.001). Female preponderance was also found in body dissatisfaction (10.9% vs. 2.9%, p < 0.001). Differences between girls and boys in the rates of intentional vomiting (1.4% vs 0.8%, p = 0.405), weight loss (1.8% vs. 1.3%, p = 0.616), and food intrusive thoughts (4.8% vs. 6.5%, p = 0.278) remained statistically nonsignificant. (Figure 4.)

Note: ***, *p* < 0.001; *p*-value for Pearson's chi-square test
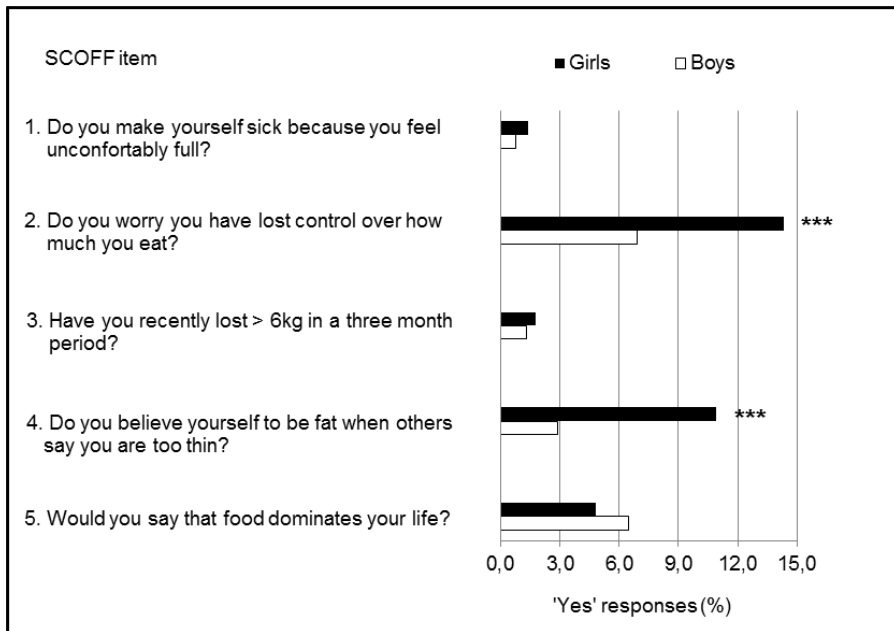
Figure 4. Prevalence (%) of self-reported EDS in girls (n = 561) and boys (n = 475) in the 8th grade. Items with a significant gender difference in the prevalence are marked with asterisks (*)

Gender differences in the health-related risk factors of mid-adolescent girls and boys in the 8th grade are presented in Figure 5. The results of the chi-square test showed that girls differed from boys in terms of change in relative weight (*p* = 0.005). Girls showed less frequently weight decrease (5%) and more commonly weight increase (14%) than boys (7% and 8%, respectively). In addition, girls outnumbered boys in the occurrence of all psychological problems measured in this study, i.e., mild (6% vs. 2%) and moderate (5% vs. 3%) depressiveness (*p* = 0.002), anxiety (15% vs. 9%, *p* = 0.006), and dissatisfaction with appearance (18% vs. 8%, *p* < 0.001). Furthermore, measured in the 7th grade, dissatisfaction with weight (28% vs. 14%, *p* < 0.001), stress (15% vs. 8%, *p* = 0.003), and loneliness (6% vs. 3%, *p* = 0.020) were more common among girls than boys. In contrast, experiences of being teased were less common among girls than boys (3% vs. 6%, *p* = 0.037). Gender differences in the reported substance use, regularity of meals, and perceptions of the quality of communication in the family were statistically nonsignificant. (Figure 5.)
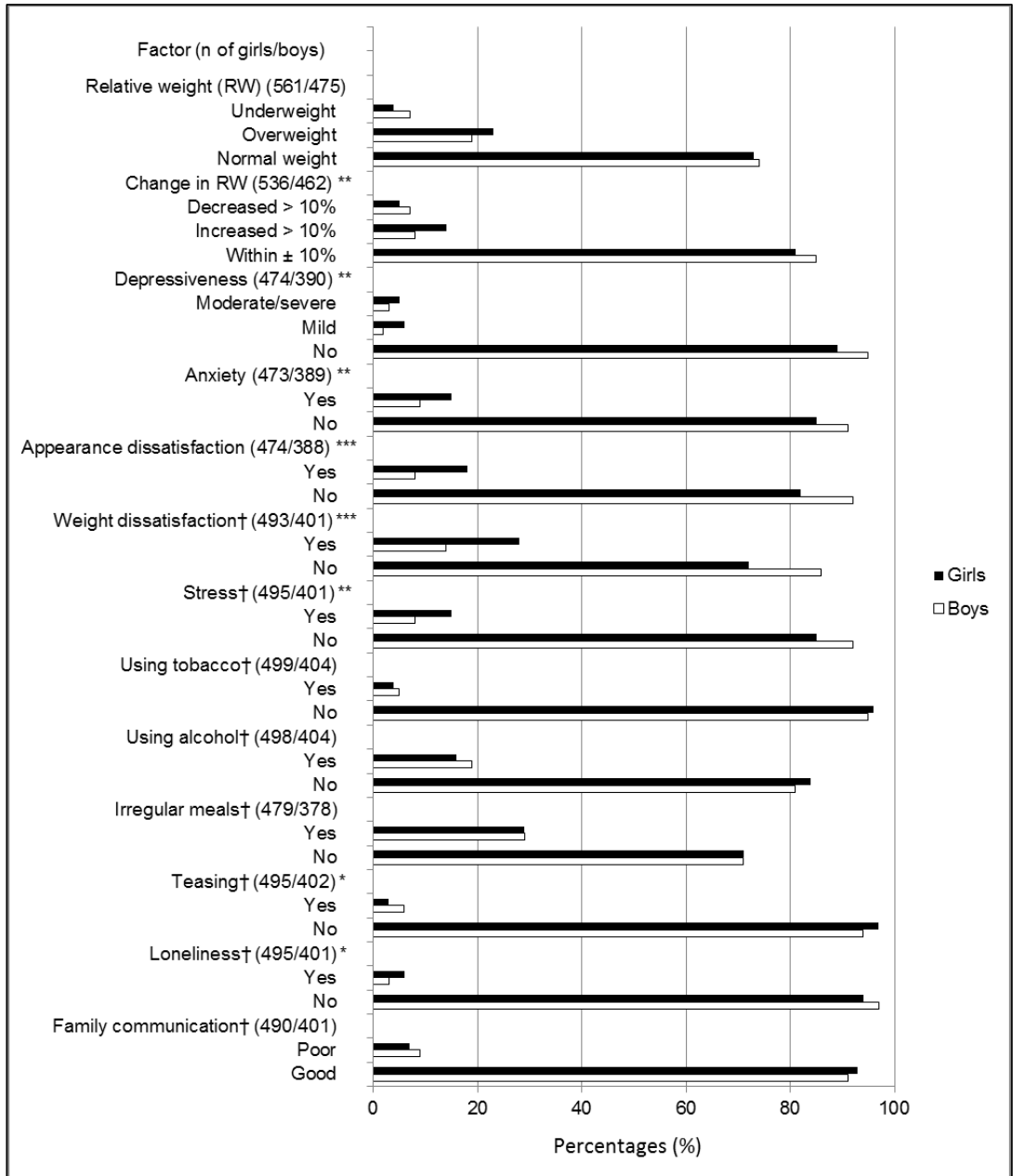
Note: *, *p* < 0.05; **, *p* < 0.01; ***, *p* < 0.001; *p*-value for Pearson's chi-square test; † = measured in the 7th grade

Figure 5. Characteristics of girls (n = 561) and boys (n = 475) in the 8th grade. Each factor is followed, in parentheses, by the number of respondents by gender. Factors with a significant gender difference are marked with asterisks (*)

No evidence was found for gender difference in susceptibility to the health risk factors in relation to self-reported EDS (all *p*-values for interaction terms > 0.05). Therefore, further analyses were performed using the combined data for girls and boys to investigate the associations between the health risk factors and self-repoted EDS. However, due to the gender difference in the occurrence of self-reported EDS and several health risk factors, the analyses were adjusted for gender.

	Adjusted odds ratio with lower and upper level of 95% confidence interval			
		AOR	LCI	UCI
Factor, p-value (n of EDS/noEDS)				
Relative weight (RW), p < 0.001				
Underweight (9/50) vs. normal (131/629)		0.9	0.4	1.8
Overweight (69/148) vs. normal (131/629)		2.2	1.5	3.1
Change in RW, p = 0.008				
Decreased >10% (20/39) vs. no (153/677)		2.4	1.4	4.4
Increased >10% (26/83) vs. no (153/677)		1.3	0.8	2.1
Depressiveness, p < 0.001				
Mild (17/18) vs. (145/650)		3.8	1.9	7.7
Moderate/severe (17/17) vs. (145/650)		4.5	2.2	9.2
Anxiety, p < 0.001				
Yes (45/60) vs. no (134/623)		3.3	2.2	5.2
Dissatisfaction with appearance, p < 0.001				
Yes (53/62) vs. no (126/621)		4.1	2.7	6.3
Dissatisfaction with weight†, p < 0.001				
Yes (73/119) vs. no (108/594)		3.2	2.2	4.6
Stress†, p = 0.014				
Yes (33/73) vs. no (150/640)		1.8	1.1	2.8
Using tobacco†, p < 0.001				
Yes (17/21) vs. no (166/699)		3.7	1.9	7.4
Using alcohol†, p < 0.001				
Yes (47/109) vs. no (136/610)		2.0	1.4	3.0
Irregular meals†, p < 0.001				
Yes (72/176) vs. no (104/505)		2.0	1.4	2.8
Teasing†, p = 0.047				
Yes (13/28) vs. no (171/685)		2.0	1.0	4.1
Loneliness†, p = 0.180				
Yes (12/27) vs. no (171/686)		1.6	0.8	3.3
Family communication†, p < 0.001				
Poor (29/42) vs. good (152/668)		3.1	1.9	5.2

Note: EDS = 1–5 self-reported eating disorder symptoms; noEDS = no self-reported eating disorder symptoms; CI = confidence interval; AOR = adjusted odds ratio (adjusted for gender); LCI = lower level of 95% confidence interval; UCI = upper level of 95% confidence interval; † = measured in the 7th grade

Figure 6. Cross-sectional gender-controlled logistic regression analyses of associations between health risk factors and self-reported EDS among adolescents in the 8th grade (n = 1036). The response options of each factor are followed, in parentheses, by the number of respondents with EDS/noEDS

In the first step of the gender-controlled logistic regression analyses, most of the health risk factors were associated with self-reported EDS (Figure 6).

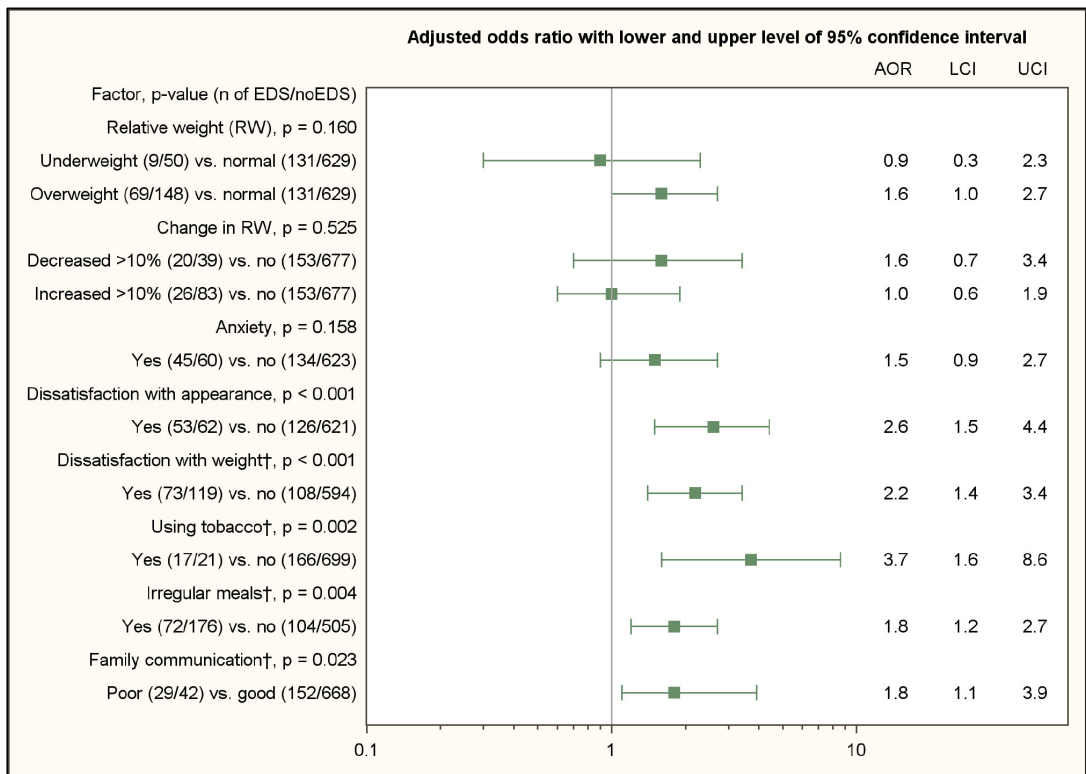| Adjusted odds ratio with lower and upper level of 95% confidence interval			
	AOR	LCI	UCI
Factor, p-value (n of EDS/noEDS)			
SUBGROUP 1: BIOLOGICAL FACTORS			
Relative weight (RW), p < 0.001			
Underweight (9/50) vs. normal (131/629)	0.9	0.4	2.1
Overweight (69/148) vs. normal (131/629)	2.2	1.5	3.2
Change in RW, p = 0.009			
Decreased >10% (20/39) vs. no (153/677)	2.5	1.4	4.4
Increased >10% (26/83) vs. no (153/677)	1.0	0.6	1.6
SUBGROUP 2: PSYCHOLOGICAL FACTORS			
Depressiveness, p = 0.342			
Mild (17/18) vs. no (145/650)	1.8	0.8	4.1
Moderate/severe (17/17) vs. no (145/650)	1.6	0.6	4.1
Anxiety, p = 0.046			
Yes (45/60) vs. no (134/623)	1.6	1.0	3.0
Dissatisfaction with appearance, p = 0.005			
Yes (53/62) vs. no (126/621)	2.1	1.3	3.7
Dissatisfaction with weight†, p < 0.001			
Yes (73/119) vs. no (108/594)	2.7	1.8	4.0
Stress†, p = 0.121			
Yes (33/73) vs. no (150/640)	1.5	0.9	2.6
SUBGROUP 3: BEHAVIORAL FACTORS			
Using tobacco†, p = 0.011			
Yes (17/21) vs. no (166/699)	2.8	1.3	6.0
Using alcohol†, p = 0.056			
Yes (47/109) vs. no (136/610)	1.6	1.0	2.5
Irregular meals†, p < 0.001			
Yes (72/176) vs. no (104/505)	1.9	1.3	2.7
SUBGROUP 4: SOCIAL FACTORS			
Teasing†, p = 0.051			
Yes (13/28) vs. no (171/685)	2.1	1.0	4.3
Family communication†, p < 0.001			
Poor (29/42) vs. good (152/668)	3.0	1.8	5.1

Note: EDS = 1–5 self-reported eating disorder symptoms; noEDS = no self-reported eating disorder symptoms; CI = confidence interval; AOR = adjusted odds ratio (adjusted for gender and factors in the same subgroup); LCI = lower level of 95% confidence interval; UCI = upper level of 95% confidence interval; † = measured in the 7th grade

Figure 7. Subgroup models of the cross-sectional gender-controlled multivariable logistic regression analyses of associations between health risk factors and self-reported EDS among adolescents (n = 1036) in the 8th grade. The response options of each factor are followed, in parentheses, by the number of respondents with EDS/noEDS

The majority of these associations remained significant in the subgroup analyses (Figure 7). The subgroups were as follows: biological factors (relative weight, change in relative weight), psychological factors (depressiveness, anxiety, appearance dissatisfaction, weight dissatisfaction, stress), behavioral factors (using tobacco, using alcohol, having meals irregularly), and social factors (teasing, family communication).

In the final multivariable analysis adjusted for gender and with the factors listed in Figure 8, dissatisfaction with appearance (OR 2.6, 95% CI 1.5–4.4, *p* < 0.001), dissatisfaction with weight (OR 2.2, 95% CI 1.4–3.4, *p* < 0.001), using tobacco (OR 3.7, 95% CI 1.6–8.6, *p* = 0.002), having meals irregularly (OR 1.8, 95% CI 1.2–2.7, *p* = 0.004), and not being able to talk about problems to parents (OR 1.8, 95% CI 1.1–3.9, *p* = 0.023) showed independent associations with self-reported EDS. However, of these factors only dissatisfaction with appearance and dissatisfaction with weight were more common among girls than boys.


Note: EDS = 1–5 self-reported eating disorder symptoms; noEDS = no self-reported eating disorder symptoms; CI = confidence interval; AOR = adjusted odds ratio (adjusted for gender and other factors in the model); LCI = lower level of 95% confidence interval; UCI = upper level of 95% confidence interval; † = measured in the 7th grade

Figure 8. Final model of the cross-sectional gender-controlled multivariable logistic regression analysis of associations between health risk factors and self-reported EDS among adolescents (n = 1036) in the 8th grade. The response options of each factor are followed, in parentheses, by the number of respondents with EDS/noEDS

5.4 Self-reported EDS over a 1-year follow-up period

During the 1-year follow-up period, the prevalence of self-reported EDS, i.e., 1–5 'Yes' answers in SCOFF, remained unchanged (McNemar *p* > 0.05). The point prevalence of self-reported EDS was 11% (42/372) at baseline and 12% (43/372) at follow-up. The proportions of agreement, reflecting the temporal stability of adolescents' responses to the items of SCOFF, ranged from 0% to 28% in 'Yes' answers for the individual items of SCOFF, and from 93% to 99% in 'No' answers, respectively. The recurrence of the separate self-reported EDS ranged from 0% to 50%: none of those who reported intentional vomiting at baseline, re-reported it at follow-up, whereas one half of those who worried about losing control over eating at baseline reported the same at follow-up. (Figure 9.)
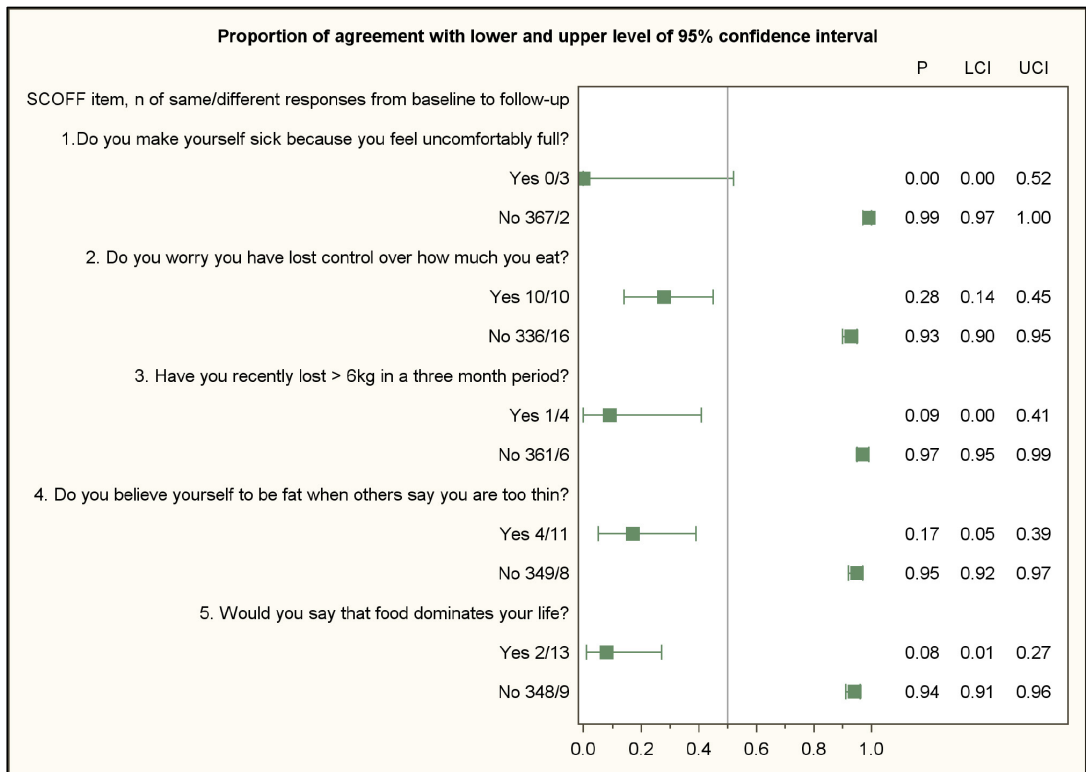
Note: CI = confidence interval; P = proportion of agreement; LCI = lower level of 95% confidence interval; UCI = upper level of 95% confidence interval

Figure 9. Temporal stability of adolescents' (n = 372) responses to the items of SCOFF in a 1-year follow-up period with the 9th grade as the baseline. The response options of each factor are followed by the number of respondents who gave the same/different response at baseline and follow-up

Table 19 shows a transition matrix for self-reported EDS in adolescents during the 1-year follow-up period from the 9th grade of the secondary school to the 1st year in upper secondary education. A total of 45% (19/42) of those respondents who were symptomatic at baseline also reported EDS at follow-up: 36% (10/28) of those with one self-reported EDS, and 64% (9/14) of those with several self-reported EDS. In this sample, the total

SCOFF score remained unchanged (Wilcoxon *p* > 0.05). Thus, altogether 5% (19/372) of students had prolonged EDS and 7% (24/330) of the initially symptom-free participants developed EDS by the 1-year follow-up. (Table 19.)

Table 19. Self-reported eating disorder symptoms (EDS) in adolescents (n = 372) at 1-year follow-up

		Follow-up		
		EDS	noEDS	Total
Baseline	EDS	19 (45%)	23 (55%)	42 (100%)
	noEDS	24 (7%)	306 (93%)	330 (100%)

5.4.1 Recurring versus remitting EDS

Table 20 presents a comparison of adolescents with recurring EDS and those showing remission at the 1-year follow-up in relation to gender and several health risk factors. According to the results, girls outnumbered boys with a gender ratio of approximately 5:1 in recurring EDS. A larger proportion of adolescents with recurring EDS had reported anxiety and dissatisfaction with appearance at the baseline, as contrasted to the respondents with remitting EDS. At follow-up, dissatisfaction with appearance and dissatisfaction with weight were more common in adolescents with recurring EDS than in those with remitting EDS, although the relative weight had increased in the latter group. (Table 20.)

To investigate the possible predictors of the recurrence of self-reported EDS, the explanatory variables were selected on the basis of the comparisons of baseline characteristics between adolescents with recurring EDS and those with remitting EDS. Accordingly, anxiety and dissatisfaction with appearance, which provided statistically significant differences between the groups at baseline, were used as explanatory variables in the multivariable analysis. As a result, anxiety was associated with the recurrence of self-reported EDS (OR 20.3, 95% CI 2.2–187.9, *p* < 0.001), while dissatisfaction with appearance was rendered nonsignificant (*p* = 0.257). Adolescents who reported anxiety in mid-adolescence were about 19 times more likely to have recurring EDS one year later, compared to showing remission, than those without anxiety.

Table 20. Characteristics of the EDS12 (n = 19) and EDS1 (n = 23) groups at baseline and at the 1-year follow-up, with contrasts (*p*-value) based on Fisher's exact test

Factor	Baseline				Follow-up		
	EDS12 n (%)	EDS1 n (%)	EDS12 vs. EDS1 *p*-value		EDS12 n (%)	EDS1 n (%)	EDS12 vs. EDS1 *p*-value
Gender			0.305				
Girl	16 (84)	16 (70)					
Boy	3 (16)	7 (30)					
Relative weight (RW)			1.000				1.000
Underweight	0 (0)	0 (0)			0 (0)	0 (0)	
Overweight	7 (37)	9 (41)			7 (37)	9 (39)	
Normal weight	12 (63)	13 (59)			12 (63)	14 (61)	
Change in RW							**0.026**
Decrease > 10 %	-	-			2 (11)	1 (5)	
Increase > 10 %	-	-			0 (0)	6 (27)	
Within ± 10 %	-	-			17 (89)	15 (68)	
Depressiveness‡			0.139				0.150
Yes	7 (41)	3 (16)			4 (22)	1 (4)	
No	10 (59)	16 (84)			14 (78)	22 (96)	
Anxiety‡			**0.002**				0.098
Yes	9 (53)	1 (5)			8 (42)	4 (17)	
No	8 (47)	18 (95)			11 (58)	19 (83)	
Appearance dissatisfaction ‡			**0.033**				**0.013**
Yes	9 (53)	3 (16)			7 (39)	1 (4)	
No	8 (47)	16 (84)			11 (61)	22 (96)	
Weight dissatisfaction			0.411				**0.025**
Yes	10 (77)	7 (58)			10 (77)	5 (29)	
No	3 (23)	5 (42)			3 (23)	12 (71)	
Loneliness			0.081				0.433
Yes	6 (43)	1 (8)			1 (8)	0 (0)	
No	8 (57)	11 (92)			12 (92)	17(100)	
Stress			0.683				0.676
Yes	10 (71)	7 (58)			3 (23)	3 (17)	
No	4 (29)	5 (42)			10 (77)	15 (83)	
Health complaints			1.000				0.494
Three or more	5 (36)	4 (33)			2 (15)	1 (5)	
One or two	5 (36)	4 (33)			7 (54)	7 (39)	
None	4 (28)	4 (33)			4 (31)	10 (56)	

Note: EDS12 = self-reported eating disorder symptoms both at the baseline assessment and at the follow-up assessment; EDS1 = self-reported eating disorder symptoms only at baseline; ‡ = data obtained from the 8th grade health care records at baseline; statistically significant *p*-values are presented in bold

5.4.2 New onset of EDS versus no EDS

In the group of non-symptomatic adolescents, the gender ratio was about equal, whereas among those who developed EDS after the baseline, girls outnumbered boys with a gender ratio of approximately 4:1 (*p* = 0.018). Already at the baseline, adolescents who later developed EDS differed from their non-symptomatic peers in relation to the majority of the health risk factors. Overweight, psychological problems and health complaints were more common among those who developed EDS than among those who remained non-symptomatic. These differences tended to remain at the follow-up with two additions: weight increase and loneliness became more common among participants who reported the onset of EDS than those who remained symptom-free. (Table 21.)

Table 21. Characteristics of the EDS2 (n = 24) and noEDS (n = 306) groups at baseline and at the 1-year follow-up, with contrasts (*p*-value) based on Fisher's exact test

Factor	Baseline EDS2 n (%)	noEDS n (%)	EDS2 vs. noEDS *p*-value	Follow-up EDS2 n (%)	noEDS n (%)	EDS2 vs. noEDS *p*-value
Gender			**0.018**			
Girl	19 (79)	161 (53)				
Boy	5 (21)	145 (47)				
Relative weight (RW)			**0.028**			**0.014**
Underweight	0 (0)	3 (1)		0 (0)	0 (0)	
Overweight	11 (46)	61 (20)		12 (57)	82 (27)	
Normal weight	13 (54)	240 (79)		9 (43)	218 (72)	
Change in RW						**0.038**
Decrease > 10 %	-	-		2 (10)	5 (2)	
Increase > 10 %	-	-		4 (19)	35 (12)	
Within ± 10 %	-	-		15 (71)	259 (86)	
Depressiveness‡			**0.011**			0.055
Yes	5 (23)	14 (6)		4 (18)	13 (4)	
No	17 (77)	240 (94)		18 (82)	291 (96)	
Anxiety‡			**0.020**			**0.017**
Yes	7 (32)	31 (12)		7 (30)	35 (12)	
No	15 (68)	220 (88)		16 (70)	270 (88)	
Appearance dissatisfaction ‡			**0.001**			**<0.001**
Yes	9 (41)	28 (11)		9 (41)	19 (6)	
No	13 (59)	226 (89)		13 (59)	287 (94)	
Weight dissatisfaction			**0.010**			**<0.001**
Yes	7 (50)	30 (18)		9 (75)	35 (18)	
No	7 (50)	137 (82)		3 (25)	163 (82)	
Loneliness			0.256			**0.009**
Yes	2 (13)	10 (6)		3 (25)	6 (3)	
No	13 (87)	158 (94)		9 (75)	200 (97)	
Stress			**0.011**			**0.014**
Yes	7 (47)	28 (17)		5 (42)	25 (12)	
No	8 (53)	139 (83)		7 (58)	180 (88)	
Health complaints			**0.023**			**0.004**
Three or more	3 (20)	11 (7)		4 (33)	10 (5)	
One or two	8 (53)	59 (35)		4 (33)	67 (33)	
None	4 (27)	98 (58)		4 (33)	128 (62)	

Note: EDS2 = self-reported eating disorder symptoms only at the 1-year follow-up assessment; noEDS = no self-reported eating disorder symptoms in the baseline assessment or in the 1-year follow-up assessment; ‡ = data obtained from the 8th grade health care records at baseline; statistically significant *p*-values are presented in bold

The possible predictors for the onset of new presentations of self-reported EDS were obtained from the comparisons between adolescents who developed EDS during the follow-up period and those who remained non-symptomatic. Multivariable logistic regression analyses using backward and forward methods resulted in models where dissatisfaction with appearance and stress remained associated with the onset of self-reported EDS. When these two factors were entered into a gender-controlled logistic regression analysis the result showed that youth who were dissatisfied with own appearance in mid-adolescence were four times more likely to develop EDS within one year, in comparison to remaining non-symptomatic, than those without appearance dissatisfaction (OR 5.1, 95% CI 1.6–16.5, *p* = 0.006). In addition, students who had stress in mid-adolescence were about twice more likely to develop EDS in a year, compared to remaining symptom-free, than those who reported no stress in mid-adolescence (OR 3.3, 95% CI 1.01–10.8, *p* = 0.048).

5.5   Self-reported EDS over a 4-year follow-up period

The development of self-reported EDS differed between females and males ($p < 0.001$) over the 4-year follow-up period. In late-adolescent respondents, the prevalence of self-reported EDS remained almost unchanged in females (30% vs. 33%, $p = 0.213$), but decreased in males (26% vs. 13%, $p = 0.002$). Altogether 52% (83/160) of the females who reported EDS in mid-adolescence were still symptomatic in late adolescence: 45% (52/115) of those with one self-reported EDS, and 69% (31/45) of those with several self-reported EDS. In males, 17% (8/48) of those with self-reported EDS in mid-adolescence remained symptomatic at the follow-up: 13% (5/39) of those with one self-reported EDS, and 33% (3/9) of those with several self-reported EDS in mid-adolescence. In addition, 26% (96/375) of previously symptom-free females, but only 12% (16/139) of symptom-free males showed the onset of self-reported EDS in late adolescence. (Table 22.) Accordingly, females had higher odds ($p < 0.001$) than males for recurring EDS, compared to showing remission (OR 5.4, 95% CI 2.4–12.3), and for developing EDS, compared to remaining symptom-free (OR 2.6, 95% CI 1.5–4.7).

Table 22. Self-reported eating disorder symptoms (EDS) in females (n = 535) and males (n = 187) in the 4-year follow-up from middle to late adolescence

| | | Late adolescence | | | | | |
| | | Females | | | Males | | |
		EDS	noEDS	Total	EDS	noEDS	Total
Mid-adolescence	EDS	83 (52%)	77 (48%)	160 (100%)	8 (17%)	40 (83%)	48 (100%)
	noEDS	96 (26%)	279 (74%)	375 (100%)	16 (12%)	123 (88%)	139 (100%)

Due to the nested case-control design, it was possible to compute the population at risk for EDS in late adolescence. According to the results presented in Table 23, a quarter of the late-adolescent population (32% of females and 12% of males) were estimated to have at least one EDS. Roughly a third of all reported EDS cases were estimated to be prolonged in nature. Multiform EDS were estimated to occur in about one tenth of late adolescents (16% of females and 1% of males). Of these, about every third individual was estimated to have prolonged EDS.

Table 23. Late-adolescent population at risk for eating disorder symptoms (EDS)

Type of EDS	All % (95% CI)	Females % (95% CI)	Males % (95% CI)
At least one EDS	26 (23.9–27.8)	32 (28.8–34.5)	12 (10.1–14.5)
At least one prolonged EDS	8 (6.4–8.8)	11 (9.2–13.1)	2 (1.0–2.9)
Multiform EDS	12 (10.1–13.0)	16 (13.4–17.9)	1 (0.6–2.1)
Multiform prolonged EDS	4 (3.2–5.0)	6 (4.7–7.6)	0.7 (0.2–1.3)

Table 24. Distribution of the health risk factors and parental factors in all (n = 535) and across the noEDS (n = 279), EDS1 (n = 77), EDS2 (n = 96), and EDS12 (n = 83) groups of females

Factor	All n (%)		NoEDS n (%)		EDS1 n (%)		EDS2 n (%)		EDS12 n (%)	
Health risk factor at T1–T2										
Depressiveness										
No–no	332	(72)	206	(85)	45	(74)	51	(62)	30	(41)
Yes–no	28	(6)	12	(5)	3	(5)	2	(2)	11	(15)
No–yes	76	(17)	21	(9)	10	(16)	24	(29)	21	(28)
Yes–yes	22	(5)	2	(1)	3	(5)	5	(6)	12	(16)
Anxiety										
No–no	250	(55)	158	(66)	30	(49)	34	(42)	28	(38)
Yes–no	23	(5)	10	(4)	4	(7)	3	(4)	6	(8)
No–yes	130	(29)	57	(24)	20	(33)	32	(39)	21	(28)
Yes–yes	51	(11)	13	(6)	7	(11)	12	(15)	19	(26)
Dissatisfaction with appearance										
No–no	311	(68)	199	(83)	40	(66)	44	(54)	28	(38)
Yes–no	62	(14)	30	(13)	11	(18)	4	(5)	17	(23)
No–yes	50	(11)	8	(3)	5	(8)	25	(30)	12	(16)
Yes–yes	34	(7)	3	(1)	5	(8)	9	(11)	17	(23)
Overweight										
No–no	419	(80)	236	(86)	54	(74)	73	(78)	56	(69)
Yes–no	24	(5)	14	(5)	4	(6)	1	(1)	5	(6)
No–yes	22	(4)	5	(2)	1	(1)	8	(8)	8	(10)
Yes–yes	58	(11)	20	(7)	14	(19)	12	(13)	12	(15)
Parental factor at T2										
Maternal Care										
High %	145	(27)	89	(32)	27	(36)	19	(20)	10	(12)
Average %	250	(47)	130	(47)	29	(38)	49	(51)	42	(51)
Low %	136	(26)	58	(21)	20	(26)	28	(29)	30	(37)
Paternal Care										
High %	127	(25)	84	(32)	15	(21)	16	(18)	12	(15)
Average %	243	(49)	123	(47)	39	(55)	48	(54)	33	(42)
Low %	130	(26)	55	(21)	17	(24)	25	(28)	33	(42)
Maternal Over Protection										
Low %	123	(25)	76	(27)	19	(25)	21	(22)	7	(8)
Average %	278	(52)	144	(52)	42	(55)	52	(54)	40	(49)
High %	130	(23)	57	(21)	15	(20)	23	(24)	35	(43)
Paternal Over Protection										
Low %	111	(32)	66	(25)	17	(24)	17	(19)	11	(14)
Average %	228	(46)	132	(50)	34	(48)	34	(38)	28	(36)
High %	161	(22)	64	(24)	20	(28)	38	(43)	39	(50)

Note: T1 = baseline assessment in mid-adolescence; T2 = follow-up assessment in late adolescence; noEDS = no self-reported eating disorder symptoms in mid-adolescence or late adolescence; EDS1 = self-reported eating disorder symptoms only in mid- adolescence; EDS2 = self-reported eating disorder symptoms only in late adolescence; EDS12 = self-reported eating disorder symptoms both in middle and late adolescence

The number of late-adolescent males with self-reported EDS was small. Therefore, further analyses were conducted among females only. Table 24 presents the distribution of the characteristics of all female respondents and across the four groups with or without self-reported EDS. Altogether 45% (204/454) of females reported anxiety, 32% (146/457) felt dissatisfaction with appearance, 28% (126/458) suffered from depressiveness, and 20% (104/523) were overweight in, at least, one assessment during the 4-year period. However, in many cases, these problems were reported only once. In proportion,
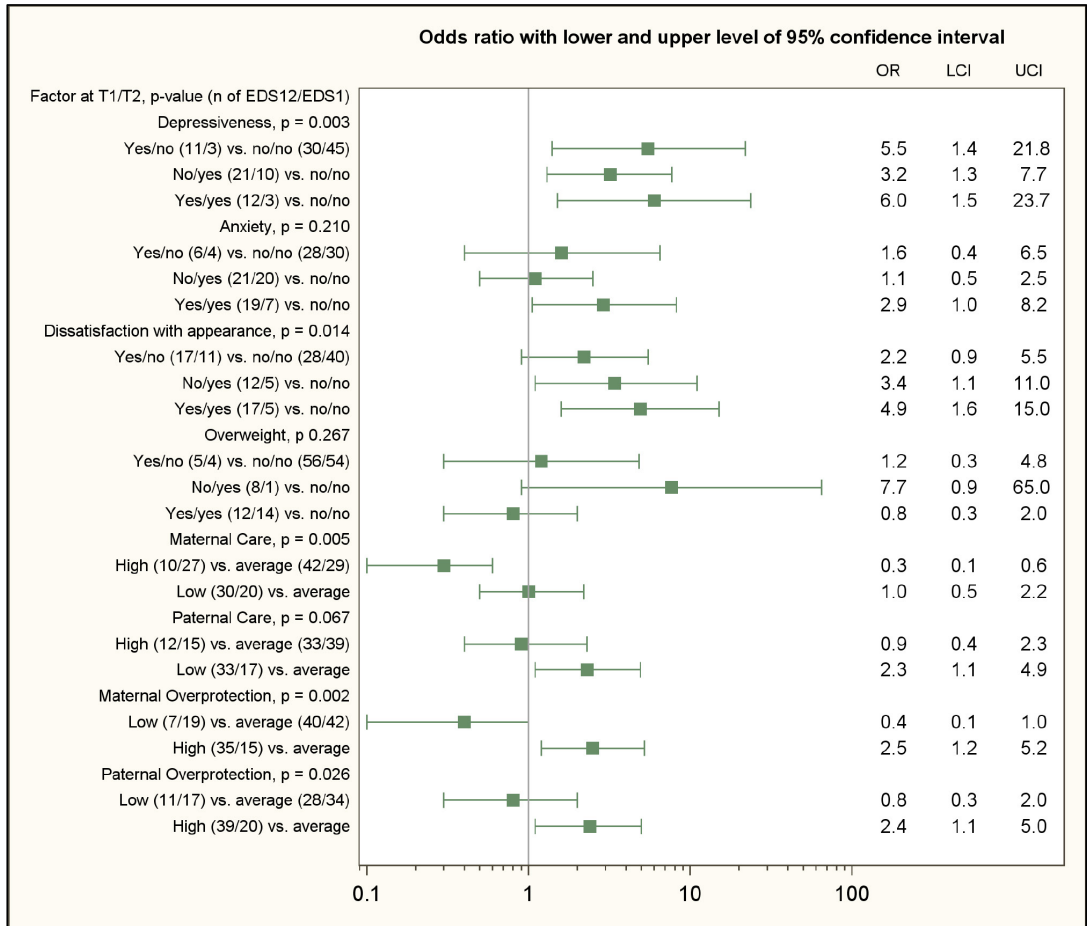
psychological problems and overweight were more common in females with self-reported EDS than in their non-symptomatic peers. In line with this, memories of less caring and more overprotecting parents were more typical of females with self-reported EDS than non-symptomatic females.

5.5.1 Recurring versus remitting EDS

To investigate differences between adolescents with recurring EDS and those showing remission logistic regression analyses were performed. In the simple logistic regression analysis, the odds of a female for recurring EDS in late adolescence, compared to showing remission, were higher among those who reported depressiveness in mid-adolescence, but not thereafter, and among those who reported the recurrence or onset of depressiveness in late adolescence, than in females who never reported depressiveness. In addition, the recurrence and onset of dissatisfaction with appearance and the recurrence of anxiety were associated with the recurrence of self-reported EDS. Furthermore, the odds of a female for recurring EDS in late adolescence, compared to showing remission, were higher among those who reported lower paternal care and higher parental overprotection than among females who reported experiences of average parenting. On the other hand, the odds of a female for recurring EDS in late adolescence were lower among those who reported higher than average maternal care. (Figure 10.)

In the multivariable analysis with health risk factors as explanatory variables, depressiveness perceived in mid-adolescence, but not thereafter, was associated with the recurrence of self-reported EDS (OR 9.7, 95% CI 1.6–58.4, *p* = 0.035) when overweight, anxiety, and dissatisfaction with appearance were taken into account. In the multivariable analysis using parental factors as explanatory variables, maternal care was found to provide protection against the recurrence of self-reported EDS (OR 0.3, 95% CI 0.1–0.7, *p* = 0.028) while other parental factors lost their predictive power.

In the final multivariable analysis with depressiveness and maternal care as explanatory variables, depressiveness remained associated with the recurrence of EDS (*p* = 0.016) while the protective effect of maternal care diminished (*p* = 0.10). Females who reported depressiveness in mid-adolescence, but not thereafter (OR 7.6, 95% CI 1.5–39.0), and those who suffered from depressiveness both in middle and late adolescence (OR 4.5, 95% CI 1.1–19.0), were more likely to have recurring EDS, compared to showing remission, than females who were free from depressiveness at both assessments.

Factor at T1/T2, p-value (n of EDS12/EDS1)	OR	LCI	UCI
Depressiveness, p = 0.003			
Yes/no (11/3) vs. no/no (30/45)	5.5	1.4	21.8
No/yes (21/10) vs. no/no	3.2	1.3	7.7
Yes/yes (12/3) vs. no/no	6.0	1.5	23.7
Anxiety, p = 0.210			
Yes/no (6/4) vs. no/no (28/30)	1.6	0.4	6.5
No/yes (21/20) vs. no/no	1.1	0.5	2.5
Yes/yes (19/7) vs. no/no	2.9	1.0	8.2
Dissatisfaction with appearance, p = 0.014			
Yes/no (17/11) vs. no/no (28/40)	2.2	0.9	5.5
No/yes (12/5) vs. no/no	3.4	1.1	11.0
Yes/yes (17/5) vs. no/no	4.9	1.6	15.0
Overweight, p 0.267			
Yes/no (5/4) vs. no/no (56/54)	1.2	0.3	4.8
No/yes (8/1) vs. no/no	7.7	0.9	65.0
Yes/yes (12/14) vs. no/no	0.8	0.3	2.0
Maternal Care, p = 0.005			
High (10/27) vs. average (42/29)	0.3	0.1	0.6
Low (30/20) vs. average	1.0	0.5	2.2
Paternal Care, p = 0.067			
High (12/15) vs. average (33/39)	0.9	0.4	2.3
Low (33/17) vs. average	2.3	1.1	4.9
Maternal Overprotection, p = 0.002			
Low (7/19) vs. average (40/42)	0.4	0.1	1.0
High (35/15) vs. average	2.5	1.2	5.2
Paternal Overprotection, p = 0.026			
Low (11/17) vs. average (28/34)	0.8	0.3	2.0
High (39/20) vs. average	2.4	1.1	5.0

Note: EDS = 1–5 self-reported eating disorder symptoms; noEDS = no self-reported eating disorder symptoms; CI = confidence interval; LCI = lower level of 95% confidence interval; UCI = upper level of 95% confidence interval; T1 = baseline assessment in mid-adolescence; T2 = follow-up assessment after 4 years in late adolescence; EDS12 = eating disorder symptoms at T1 and T2; EDS1 = eating disorder symptoms only at T1

Figure 10. Longitudinal logistic regression analyses of the associations of health risk and parental factors with the recurrence of self-reported EDS in late adolescent females (n = 160). The response options of each factor are followed, in parentheses, by the number of respondents with EDS at T1 and T2/EDS only at T1

5.5.2 New onset of EDS versus no EDS

To investigate differences between adolescents with new onset of EDS and those who remained asymptomatic logistic regression analyses were performed. The results are presented in Figure 11.
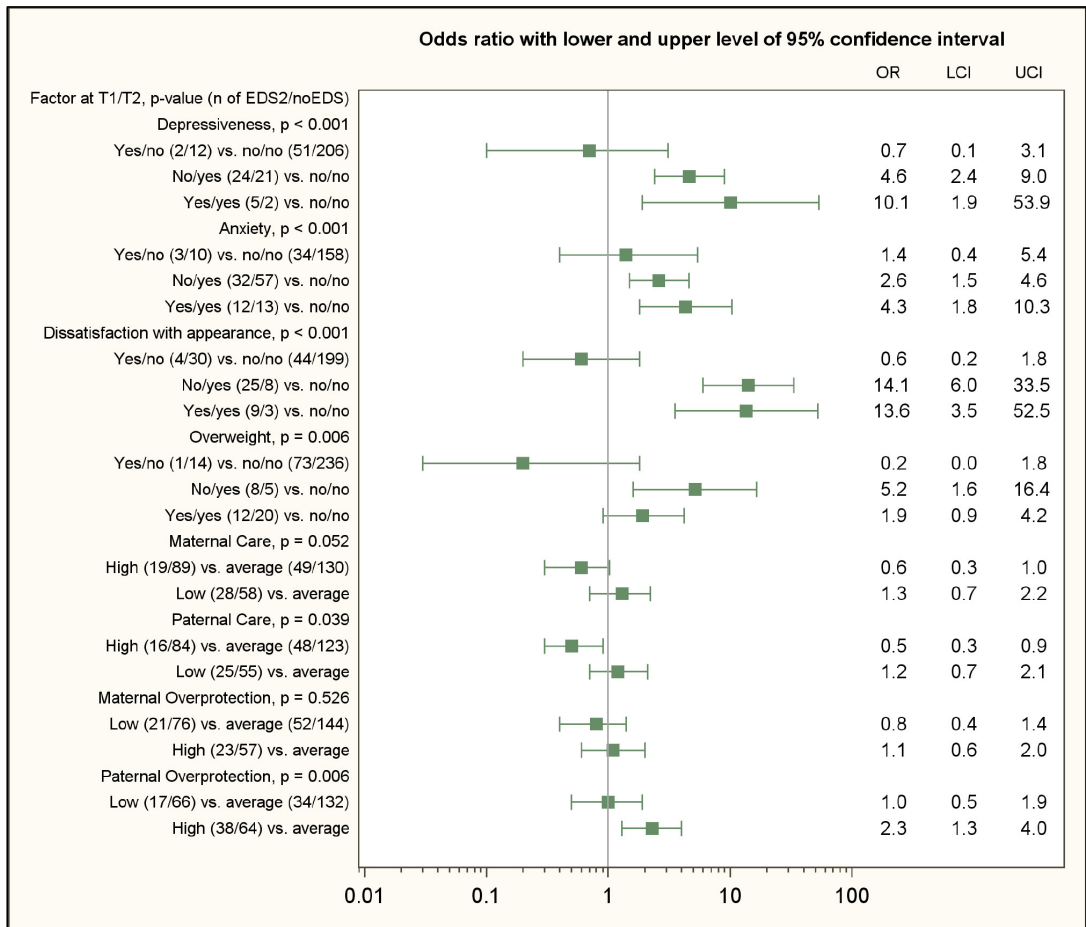
Note: EDS = 1–5 self-reported eating disorder symptoms; noEDS = no self-reported eating disorder symptoms; CI = confidence interval; LCI = lower level of 95% confidence interval; UCI = upper level of 95% confidence interval; T1 = baseline assessment in mid-adolescence; T2 = follow-up assessment after 4 years in late adolescence; EDS2 = eating disorder symptoms only at T2; noEDS,= no eating disorder symptoms

Figure 11. Longitudinal logistic regression analyses of the associations of health risk and parental factors with the onset of self-reported EDS in late adolescent females (n = 375). The response options of each factor are followed, in parentheses, by the number of respondents with EDS only at T2 / noEDS

The analyses showed that the odds of a female for the onset of self-reported EDS in late adolescence, compared to remaining non-symptomatic, were higher among those who reported the recurrence or onset of depressiveness, anxiety, or dissatisfaction with appearance, or the onset of overweight than in females who never reported these problems. In addition, the odds of a female for the onset of self-reported EDS after mid-adolescence increased when paternal overprotection was reported higher than average, but decreased when paternal care exceeded the average level. (Figure 11.).

In the multivariable analysis with health risk factors as explanatory variables, the odds of a female for developing EDS, compared to remaining non-symptomatic, were higher (*p* < 0.001) among those who were dissatisfied with own appearance in both middle and late

adolescence (OR 7.1, 95% CI 1.5–33.5), and among those who reported appearance dissatisfaction after mid-adolescence (OR 12.8, 95% CI 4.6–35.4) than in females who were satisfied with own appearance in middle and late adolescence. The multivariable analysis with parental factors as explanatory variables showed that higher than average paternal overprotection predicted the onset of self-reported EDS (OR 2.3, 95% CI 1.2–4.1, *p* = 0.023) while experiences of paternal care lost predictive power when maternal parenting was taken into account.

In the final multivariable analysis, where dissatisfaction with appearance and paternal overprotection were used as explanatory variables, dissatisfaction with appearance predicted the onset of self-reported EDS (*p* < 0.001), while paternal overprotection was rendered nonsignificant (p = 0.29). Females who remained dissatisfied with own appearance over the follow-up period (OR 18.7, 95% CI 3.7–93.3) and those who reported appearance dissatisfaction after mid-adolescence (OR 12.3, 95% CI 5.1–29.8), were more likely to develop EDS by late adolescence, compared to remaining non-symptomatic, than females who were satisfied with their appearance at both assessments.

6   DISCUSSION

This study aimed to enhance the detection of eating pathology in adolescents and the use of health record information in identifying those with the highest risk of prolonged or later onset of eating disorder symptoms (EDS). The study is grounded on earlier research that has shown that eating disturbances during adolescence may develop on a detrimental trajectory into continued eating pathology and full-blown eating disorders [76]. The aim was approached by the following steps. First, the feasibility of the SCOFF questionnaire was tested among adolescent girls and boys in school health care setting. The purpose was to obtain evidence for or against using SCOFF as a primary screen for eating pathology in health examinations. Second, differences between mid-adolescent girls and boys were examined in relation to EDS and associated factors. This was warranted in order to establish the justification of gender-based vs. universal screening for eating pathology in students at the age when gender differences in eating pathology emerge [see 357]. Finally, the course of EDS and associated factors was investigated with follow-up periods of one year and four years. Previous research indicates that it may be possible to identify youth at risk for later eating pathology on the basis of vulnerabilities they have earlier in adolescence [e.g., 361]. Thus, the intention was to characterize adolescents who might benefit from early intervention.

In the following sections, the main findings of each step of the study are discussed in light of the previous knowledge. This is followed by methodological considerations, including the strengths and weaknesses of the study. Finally, the practical implications of the study and suggestions for future research are discussed.

6.1   Utility of the SCOFF questionnaire

This is the first study to report on the feasibility of an eating disorder questionnaire in screening for EDS among adolescents within the setting of school health care in Finland. The findings support the assumption of the Finnish version of SCOFF as a feasible tool for screening for EDS in mid-adolescent populations without gender limitation. Factor loadings ranging from moderate to high and the goodness-of-fit statistics showed that both the uni- and bi-dimensional factor models of SCOFF fit well in the studied data, with the genders combined and separately. However, higher tetrachoric correlations between the individual items of SCOFF in girls vs. boys and in older vs. younger adolescents raised the questions whether EDS is more diffuse in boys than girls and whether this gender difference increases during the course of adolescence. If so, it is possible that EDS remains somewhat diffuse in boys irrespective of age, whereas in girls, along with age, the forms of EDS may develop into two distinct groups of symptoms: one linked with adverse attitudes to body, eating and weight, and the other related to weight loss behaviors. The bi-dimensional model of SCOFF may thus be a better option for girls after middle adolescence. However, in this study, the two factors were highly correlated, indicating that adolescents with multiform EDS typically had both attitudinal and behavioral problems with eating and weight. Therefore, the two factors may be difficult to discern in practice.

The factor models of SCOFF tested in the present study were based on a previous Spanish research [279] where exploratory factor analyses provided a two-factor model for girls and a one-factor model for boys, indicating a gender-dependent factor structure of the Catalan version of SCOFF. A gender-free factor model, in turn, gained support in a large

Colombian study [278] of adolescents: the one factor model of the Spanish version of SCOFF was found acceptable for both genders. In comparison, another large study of adolescents in Mexico, resulted a one-factor model of the Spanish version of SCOFF for both genders based on an exploratory factor analysis after splitting the female and male samples randomly into halves. However, further analyses showed that the factor model for boys lacked stability: in the confirmatory factor analysis of the second half of the male sample, only the two-factor model showed good fit. [283.] Possible age effect on the factor structure was not tested in the studies described above. Taken together, both uni- and bi-dimensional factor models of SCOFF have gained support, but not consistently. Thus, no firm conclusions of the optimal factor structure of SCOFF in adolescent populations can be drawn.

To the knowledge of the author, this is also the first study assessing the usefulness of screening for eating pathology in adolescents within ordinary school health care. The findings of the efficiency of SCOFF, as compared to established health examination practice, in detecting EDS in adolescents support the use of SCOFF as a part of regular health examinations. About one fifth of adolescents self-reported EDS by means of SCOFF, whereas in health examinations, the risk for an eating disorder (ED) was detected in one out of 20 students, mostly on the basis of objective signs or health record information of eating pathology. As many as nine out of 10 adolescents who self-reported EDS were not detected in health examination. The results are in line with the previous findings of higher disclosure of disordered eating by means of a self-report questionnaire than in a face-to-face contact [272; 358]. However, screening with SCOFF did not reveal all cases. According to the school nurses' assessments, about one out of 30 adolescents without self-reported EDS in SCOFF had an ED risk. This reveals the limitation embedded in all self-report measures: the result may reflect an ideal rather than true status of the respondent. As previously pointed out, "*the test may serve, at best, only as a source of suggestions about individuals to be confirmed by other evidence*" [449], such evidence including, e.g., file review, interview, and using more detailed questionnaires, or multiple informants.

The findings of this study are in line with a recent study [296] assessing the screening value of SCOFF in clinical practice. Due to methodological differences between the French study and the present one, the prevalence estimates of eating pathology are not directly comparable. However, both studies support the previous findings [e.g., 5; 125] suggesting that, in primary health care, the majority of eating pathology cases tend to remain undetected. In clinical practice, eating pathology is difficult to detect without using a questionnaire specific to eating pathology [123].

6.2 Self-reported EDS and associated factors

The findings of this study show that, in mid-adolescence, girls report EDS more commonly than boys. At least one EDS was reported by every fifth adolescent (20%): one out of four girls (24%) girls and one out of six (16%) boys. Several EDS were found in one out of 20 adolescents (5%): in one out of 14 (7%) girls and one out of 36 (3%) of boys. Thus, girls outnumbered boys with a gender ratio of 3:2 in all EDS and of 2:1 in multiform EDS. The gender difference was based on the girl predominance in the attitudinal forms of EDS. The frequency of body dissatisfaction was for girls almost fourfold (10.9%) in comparison to boys (2.9%). Fear of losing control was twice as common in girls (14.3%) as in boys

(6.9%). In terms of behavioral forms of EDS, gender differences remained smaller and statistically nonsignificant.

The prevalence of self-reported EDS found in this study falls within the wide range of the prevalence of eating pathology reported in other studies (see section 2.4.4.). In Finland, the nationally representative School Health Promotion Study conducted in 2003 [350] reported on the loss of control over eating and self-induced vomiting among adolescents in the 8th and 9th grades at about similar rates as the present study. However, both lower and higher prevalence estimates are reported as well. For example, in 1995, the School Health Study among the pupils of the 8th and 9th grades detected fear of losing control over eating to a smaller extent [328] than the present study. In addition, in 1998, another school-based study [8] of the 7th and 9th graders revealed loss of control over eating at about the similar rate, but vomiting at a higher rate than the present study. Furthermore, in 2001/2002, a large international study of the Health Behavior in School-aged Children detected higher prevalence of self-induced vomiting for Finnish adolescents [9] than was found in the present study. Variation in findings across the previous surveys may partly be due to the differences in the definitions of "case" and the tools used to measure eating pathology. In the aforementioned studies, eating pathology was measured by means of self-made questions instead of an eating disorder questionnaire.

A comparison of the results of the present study with those obtained with the self-administered SCOFF in other studies shows that self-reported multiform EDS is far less common among Finnish adolescents (5%) than in other Western countries (from 13.5% to 46.0%). Further inspection of the individual items of SCOFF reveals that differences across countries in the separate forms of EDS vary from nonexistence to manifold rates (see Table 5). For example, the most commonly reported symptom in the present study, i.e., fear of losing control over eating, was nearly as common among boys in the USA [347], and girls in Spain [342]. In comparison, roughly two-fold prevalence was found among adolescents in Germany [343] and Austria [346], girls in the USA [347], and boys in Spain [342]. Intentional vomiting, which was the least reported symptom in this study, was much more common in Germany [343], Austria [346], USA [347], and especially in Spain [342] with 30- and 40-fold rates in girls and boys, respectively, as compared to the present study. Despite the discrepancies in the prevalence of eating pathology, which may partly be due to cultural differences and discordance between the translated versions of SCOFF [see e.g., 346], gender ratios of the separate forms of EDS showed less variation across the countries. In line with the present study, the gender ratios in the USA varied from about 1:1 in weight loss to the girl preponderance of 4:1 in body dissatisfaction [347]. In Germany, Austria and Spain, the gender differences were somewhat smaller, with the female:male ratios ranging from about 1:1 in weight loss to 2:1 [342; 343] and 3:1 [342; 346] in body dissatisfaction. To conclude, the gender difference between adolescent girls and boys presenting EDS is relatively small across the countries, which highlights the importance of screening for EDS in all adolescents without gender limitation.

Eating pathology typically co-occurs with other health-related problems. Some of these correlates may also be risk factors of eating disorders [16]. As such, they may partly explain the female preponderance in EDS had girls been exposed and susceptible to these factors more than boys. The results of this study show that girls are, indeed, more commonly than boys exposed to several health-related problems. Most of the gender differences occurred in psychological issues, such as dissatisfaction with appearance and weight, depressiveness, anxiety, and stress, which were about twice as common in girls as in boys. This is in line with the previous evidence that establishes problems with mental well-being [e.g., 8; 314; 331; 350; 450] and subjective health in general [331] to be more

common among adolescent girls than boys. However, according to this study, there is no gender difference in susceptibility to these factors in relation to self-reported EDS. Neither girls nor boys were more vulnerable to or better protected against the effect of psychological problems in relation to EDS. Hence, the higher rate of girls than boys exposed to these factors may partly explain why girls outnumber boys in the prevalence of self-reported EDS, on the condition that these factors are associated with EDS.

The results of the association between the health-related factors and self-reported EDS support the assumption of the higher number of girls than boys being exposed to factors associated with EDS. The multivariable analysis in this study showed that two factors more common among girls than boys, i.e., dissatisfaction with appearance and dissatisfaction with weight had an independent association with self-reported EDS. Adolescents who were dissatisfied with own appearance were almost three times as likely as the others to report co-occurring EDS. In addition, participants who reported weight dissatisfaction were about twice as likely as students satisfied with their weight to report EDS as well. The findings are in accordance with the evidence of the harmful effect of dissatisfaction with body and weight on eating behavior among youth [e.g., 339; 379; 382]. In fact, weight and shape concerns are rated as the most consistent predictors of the development of eating disturbances [16]. In theory, sociocultural factors, such as an ideal of a slender appearance of women in Western like cultures, set expectations and pressures on females. Especially during phases involving identity confusion, like adolescence, girls may experience discrepancy between the ideal feminine appearance and self, possibly leading to body dissatisfaction and the development of eating pathology [105; 106; 107]. Prospective studies conducted in non-Western populations support this explanation by showing that the invasion of Western culture leads to the occurrence of eating pathology in previously healthy females [e.g., 451]. Accordingly, in adolescence, female predominance in eating pathology may partly originate from the higher occurrence of appearance and weight dissatisfaction among girls than boys.

6.3   Self-reported EDS over 1-year follow-up

According to this study, a considerable number of Finnish mid-adolescents report EDS. With the 1-year follow-up period, about one out of ten adolescents reported EDS both in the 9th grade of secondary school and in the 1st year in upper secondary school. Almost one half (45%) of those who reported EDS at baseline were still symptomatic in the 1-year follow-up assessment. According to the results, one out of 20 respondents (5%) had recurring EDS. In addition, one out of 14 (7%) initially non-symptomatic participants developed EDS by the end of the follow-up period.

The findings are in line with the previous short-term prospective studies reporting a moderate continuity of disordered eating, a relatively low prevalence of prolonged eating pathology, and fluctuation between the groups of symptomatic and non-symptomatic individuals in adolescent populations. For example, studies in the USA have shown that more than every third of initially symptomatic adolescents are still symptomatic nine [119] to ten [198]) months later. Studies conducted among mid-adolescent schoolgirls in Poland [194] and England [193] have revealed somewhat similar rates for the maintenance of abnormal eating attitudes during the follow-up perios of 10 [194] and 12 [193] months. Researchers in Italy have found that over one half of the girls with symptoms at the outset remained symptomatic after a year [195]. Larger two-year follow-up studies performed in Norway [196] and Spain [199] have also evidenced corresponding rates for the

continuation of disordered eating. Generally, prolonged eating pathology has occurred in roughly 3% to 5% [193; 194; 195; 198; 199] of all adolescents. New cases, in turn, have been reported with a wide range; from less than 10% [193; 194; 195; 196; 378] to 40% [119] of initially non-symptomatic adolescents. Taken together, short-term follow-up studies of eating pathology provide an impression, that in many cases, the problem is temporary, suggesting that only a minority of adolescent population suffers from prolonged eating disturbance. However, taking into account its fluctuating nature, a follow-up period of a year or two is too short for drawing any conclusions of the evolution of eating pathology. Nevertheless, short-term studies are useful in shedding light on the proximal factors associated with the recurrence and new onsets of eating problems in adolescence.

6.3.1 Predictors of recurrent EDS

The findings of this study suggest that information gathered in school health care may benefit the identification of students with different courses of EDS. Individuals with recurring EDS have also other psychological problems more commonly than adolescents with temporary EDS. Of all the health-related factors measured at baseline, anxiety and dissatisfaction with appearance were the most distinctive features between the groups of adolescents with recurring EDS and those who showed remission at follow-up. In the multivariable analysis, baseline anxiety showed an independent association with the later recurrence of EDS whereas the predictive power of appearance dissatisfaction was totally explained by anxiety. Adolescents who reported both anxiety and EDS in the 9th grade were 19 times more likely to re-report EDS one year later than participants who had baseline EDS without anxiety.

The result is in line with the evidence of the negative affect being a risk factor for the onset of eating pathology [16]. However, no previous evidence of the predictive role of anxiety in the maintenance of eating pathology for, at least, a year was found. Thus, the finding of this study remains tentative until further evidence. The role of appearance dissatisfaction in the course of eating pathology is supported by the previous findings of Project EAT, showing an association between body dissatisfaction and the maintenance of unhealthy weight control behaviors [382]. However, the same project failed to find evidence of the effect of body dissatisfaction on the maintenance of binge eating. This may partly be due to cultural and methodological differences between the studies. Results of this study may not correspond to findings of the Project EAT, in which the definitions and measures of eating pathology and subjective ideas of appearance and physique differed from those used in the present study. It is also noteworthy, that unlike in Project EAT, the number of participants with eating pathology was relatively small in the present study. In addition, the attrition rate was higher among anxiety-free adolescents than in those with anxiety, which increases the risk of bias in results. It cannot be fully ruled out that adolescents who were free from anxiety and withdrew from the follow-up might have prolonged EDS. Furthermore, it is not possible to exclude the option that associations might be explained by factors that were omitted. Nevertheless, in theory, it is possible that anxiety promotes disordered eating by decreasing an individual's internal sensitivity to bodily cues and by provoking disturbed attitudes toward food [109; 110]. For some individuals, eating may provide a means to decrease anxiety. However, the relief of eating may turn into emotional distress due to weight increase. This may lead to compensatory behaviors which, in turn, provoke negative affect, thus creating a vicious circle of negative emotions and disturbed eating [98]. Still, more evidence is needed to be able to draw conclusions of the roles of anxiety and body dissatisfaction in the maintenance of eating pathology. Hence, there remains a need for replication to confirm the findings of this study.

6.3.2 Predictors of the onset of EDS

This study shows that adolescents who are at odds for developing EDS within a year differ from their EDS-free counterparts in relation to several health-related factors measured in school health care. Depressiveness, anxiety, dissatisfaction with appearance and weight, stress, and health complaints were more common in students who developed EDS during the follow-up period than in those who remained asymptomatic. In addition, the vast majority of individuals who developed EDS were girls and almost one half were overweight, whereas in the EDS-free group, the gender ratio was equal and overweight occurred to a lesser extent. The multivariable analyses showed that dissatisfaction with appearance and stress had independent associations with the later onset of EDS while the other associations were rendered nonsignificant. Adolescents who were dissatisfied with own appearance in the 9th grade were four times more likely to develop EDS within a year than those who reported no such dissatisfaction. Similarly, the odds of participants stressed at the outset for developing EDS by follow-up were three times that of initially stress-free adolescents.

The result is keeping with the previous findings of the association of body dissatisfaction and weight concern with the development of eating pathology. For example, among adolescents in the USA, initial body dissatisfaction increases the odds of the onset of DSM-5 eating disorders in girls [384] and unhealthy weight control behaviors in both genders [382], whereas increase in body satisfaction from middle to late adolescence reduces the odds of youth for the onset of binge eating in young adulthood [204]. In line with this, high concern with weight in adolescence predicts the onset of binge eating [339; 378] and purging within one [377; 378] or three [339] or seven years [379]. The probability of developing unhealthy weight control behaviors is higher in girls and boys who perceive weight concerns in adolescence [382].

The previous findings of the role of stress in the development of eating pathology are mainly based on cross-sectional and retrospective studies, the majority of which support an association between stress and disordered eating [for a review, see 411]. In theory, it is possible that body dissatisfaction promotes the negative affect and leads to dieting to control weight or to eating to alleviate negative emotions. Accordingly, body dissatisfaction may provoke the onset of disordered eating via dysfunctional attempts to achieve thinness or to cope with the negative affect. [89; 114.] As dieting exacerbates affective instability, these individuals may have significant vulnerability to any life stressor. As a consequence, increased stress may lead them to develop disordered eating behaviors. [112.]

6.4 Self-reported EDS over 4-year follow-up

This study provides evidence of a considerable rate of the recurrence and new onsets of eating pathology during the course of adolescence in females and with a lesser extent in males. Within the 4-year follow-up period, females reported EDS at about the similar rate in late adolescence as in mid-adolescence. Among males the number of individuals with EDS decreased from the outset to the follow-up. Of all the females with EDS at the outset, roughly one half were still symptomatic four years later. Among males, recurring EDS occurred in one out of six initially symptomatic. During the follow-up period, about a quarter of the initially asymptomatic girls developed EDS. In boys, new EDS were half as common as in girls. Overall, compared with males females were over five times as likely to have recurring EDS and more than twice as likely to develop EDS by late adolescence.

For females, the findings are in accordance with the previous long-term studies reporting fairly stable [200; 335] or somewhat increased [164; 197; 200; 335; 382] percentages of girls with eating pathology at each period of adolescence. The results also support the evidence of a considerable stability of eating pathology on the individual level during the course of adolescence. For example, in the USA, two thirds of subthreshold eating disorder symptoms [200] and two out of five eating disorders [197] continue from adolescence to young adulthood. In Germany, disordered eating persists 6–7 years in almost every third of the girls with EDS in early/middle adolescence [205]. In Finland, more than two out of five females with BN [5] and every third of those with AN [3] in middle adolescence still present eating disorder symptoms after five years. Somewhat lower rates of stability are found for partial syndromal eating disorders in Australia [161]. Although cultural and methodological differences complicate the comparison between the present and previous findings, these stability rates indicate that an impressive number of adolescent girls have prolonged eating problems.

The results of the present study are also keeping with the previous findings of a fair number of new cases of eating pathology in girls during the course of adolescence. For example, a five-year follow-up study conducted in the USA found roughly one third [200] of initially asymptomatic girls having subthreshold eating disorder symptoms at the follow-up assessment, and about two out of five individuals without unhealthy weight control behaviors at the outset being engaged in them five years later [333]. In studies investigating more strictly defined eating pathology, new cases are typically found to a lesser extent [e.g., 161; 200; 205; 379].

For males, the results of the present study are less consistent with existing literature. First, in previous studies, the relative number of boys with eating pathology is found to remain constant or slightly increase instead of decreasing during the years of adolescence. For example, in the USA, the percentage of boys with eating pathology is fairly similar or somewhat higher in middle/late adolescence [200; 382] and in young adulthood [335] as compared to early/middle adolescence. In Germany, the prevalence of disordered eating remains fairly stable from early/middle adolescence to late adolescence/early adulthood [205]. Second, in previous surveys, prolonged eating pathology is detected in boys at a higher rate than in the present study. For example, in the USA, roughly one half of boys engaging in unhealthy weight control behaviors at the outset [382], and about two out of five boys initially presenting subthreshold eating disorder symptoms [200] have those problems five years later as well. In Germany [205], the rates of prolonged disordered eating are also somewhat higher than in the present study. Third, according to previous findings, the relative number of boys developing eating pathology is higher than found in the present study. For example, in the USA, the new onsets of unhealthy weight control behaviors are detected in every fifth boy initially free from those behaviors [333], and more than a quarter of initially asymptomatic boys are found to develop subthreshold eating disorder symptoms within five years [200].

In sum, eating pathology rates for females found in this study are in line with existing knowledge, but for males the rates are lower than expected on the basis of previous studies. The inconsistency in results for males may partly be based on differences in the sample characteristics. In the present study, the attrition rate was higher among males and individuals with EDS than in females and non-symptomatic mid-adolescents. This may have biased the results. One cannot rule out the possibility that boys who had EDS in middle adolescence and withdrew from the follow-up might have prolonged EDS. Similarly, it is not possible to exclude the chance that initially EDS-free boys who withdrew from the follow-up might have developed EDS by late adolescence. Thus, the findings for males

should be interpreted with caution. However, taking into account that the gender ratio in eating pathology is typically more equal in samples of adolescents than adults (see section 2.4.2), one may expect an increase in the female predominance of eating pathology by late adolescence, which means a decrease in the number of symptomatic males when the number of symptomatic females remains stable as it did in this study.

Adapting the findings of the present study to population level suggests that a sizeable proportion of Finnish youth have EDS in late adolescence and many of them perceive disordered eating already in middle adolescence. Almost every third female and one out of eight males were estimated having at least one EDS in late adolescence. Accordingly, the female preponderance in EDS was estimated to increase from 1.5:1 found in mid-adolescence to 2.5:1 by late adolescence. In multiform EDS, the estimated gender ratio in late adolescence (11:1) was much higher than the one found in mid-adolescence (2:1). These estimates are supported by the previous findings of eating pathology in a national survey among university students in Finland: female preponderance among the participants aged 24 years or younger was roughly 3:1 in abnormal attitudes towards food, and 13–14:1 in disordered eating behaviors such as uncontrollable dieting [352].

Of the late-adolescent population with EDS, about every third female and every sixth male were estimated as having perceived eating pathology already in mid-adolescence. Prolonged EDS with at least one symptom was estimated to occur in roughly 10% of females and 2% of males, and with several symptoms in 6% of females and less than 1% of males. Thus, although the remission of EDS is common in youth, in a subset of adolescent population EDS becomes protracted. In participants with at least one prolonged EDS, the female:male ratio was 5:1, whereas in those with several prolonged EDS, the female preponderance increased to 9:1, thus approaching the gender ratio (10:1) often quoted in studies of adults with eating disorders. The deepening gender gap in the occurrence of eating pathology from adolescence towards adulthood, from a short-term issue to a long-duration problem and from single to multiform symptoms, might reflect the nature of eating pathology as being more persistent and complex in females than males. However, in light of previous studies reporting both lower [205; 382] and higher [203] gender ratios in adolescents' eating pathology than those found in the present study, the question of gender difference in the course of eating pathology remains to be answered. It is possible that cultural and methodological factors partly explain the mixed results. In addition, the aforementioned gender bias in the follow-up sample of this study may have misled to underestimate the number of males with EDS in late adolescence and overestimate the gender difference. Thus, the impression of the rarity of EDS in late-adolescent males in Finland should be taken with caution.

6.4.1 Predictors of recurrent EDS

The findings of this study suggest that some of the individual characteristics measured in mid-adolescence can be used to identify adolescents most in need for intervention to prevent prolonged EDS. Three health-related factors, i.e., depressiveness, anxiety, and dissatisfaction with appearance in mid-adolescence, were more typical of girls with recurring EDS than those showing remission. The detrimental influence of these problems was strongest when the problem continued from middle to late adolescence. The only exception was depressiveness, which affected the course of EDS on a temporary basis as well. Actually, depressiveness was the strongest predictor of prolonged EDS. Anxiety and appearance dissatisfaction did not contribute to the recurrence of EDS independently, but the association was attributed to depressiveness. Also parental factors measured in late

adolescence distinguished girls with recurring EDS and those showing remission. Females with experiences of average parental protection and paternal care, and those who recalled having received maternal care higher than average were more likely to show the remission of EDS by late adolescence than females with less favorable experiences. Of the parental factors, high maternal care was the strongest protector against prolonged EDS. However, when depressiveness was taken into account, the effect of maternal care diminished, suggesting that, in the maintenance of EDS, intra-individual factors may play a more important role than experiences of parental rearing style in childhood.

The detrimental effect of all types of depression (subsyndromal, brief depressive episode and depressive episode) on individuals' health is previously demonstrated among adults in the WHO's World Health Survey [452]. The findings of the present study extend the evidence to adolescents' health, revealing an adverse effect of depressiveness on the course of EDS from middle to late adolescence. Females with either episodic or prolonged depressiveness were more likely than those without depressiveness to have recurring EDS. The finding is in line with the previous review [366], which rated the negative affect as one of the maintenance factors for eating pathology. It is also in keeping with the recent results of Project EAT, suggesting that depressed mood in early/middle adolescence and changes therein during middle/late adolescence predict the persistence of unhealthy weight control behaviors [382]. Decrease in depression symptoms is found to be associated with the cessation of binge eating after late adolescence, but not in earlier adolescence. [204.] So, the predictive power of depressiveness on different types of eating pathology may vary during the course of development, which is in accordance with the previous notion of a possible age effect on the role of factors affecting eating pathology [e.g., 371]. Accordingly, it may be that in middle adolescence, depressiveness has detrimental effect on individuals' behaviors and attitudes towards eating and weight in general, whereas the association between depressiveness and more strictly defined eating pathology may occur later towards emerging adulthood. In theory, even short-term depressiveness co-occurring with EDS in mid-adolescence may contribute to the maintenance of eating pathology if the individual finds alleviation to the negative affect from disturbed eating behaviors. This could explain why these people no longer have depressiveness in late adolescence even if they still report EDS. Those who continue to report both depressiveness and EDS in late adolescence may have fallen into a vicious circle of dysfunctional mood modulatory behaviors which, when turning into habits, maintain disordered eating [e.g., 98; 116].

As the data collection of the present study began when the participants were 14–15-years-old, it is possible that prior to the outset of the study, some individuals had perceived depressiveness, which then had fostered the occurrence of EDS. In addition, due to the bidirectional relationship between eating pathology and depressiveness [453] one must not rule out the possibility that some of the participants had perceived EDS already in childhood, leading to depressive emotions and cognitions and thereby to prolonged EDS. Thus, it is possible that the episodic problems of depressiveness and EDS found in this study may be relapses with their origins in early age. Without data from earlier adolescence and childhood, the age of the onset and temporal order of these problems remain unknown. In addition, since several risk factors associated with eating problems are also related to mood disorders [454], the relation between depressiveness and recurring EDS may be explained by a 'third variable' not measured in this study. For example, nutritional deficits typical of those with eating pathology as well as depression may have had an effect on comorbidity and the course of these problems [191]. Accordingly, no conclusions about the causality of the association can be drawn.

In the present study, prolonged anxiety and appearance dissatisfaction were associated with recurring EDS in the bivariable, but not in the multivariable analysis where depressiveness and overweight were taken into account. The finding keeps with the previous review [366], suggesting that the influence of anxiety is less significant than that of negative affect on eating pathology, but on the other hand, it is in contrast to the conclusion of body dissatisfaction being one of the most robust maintenance factors for bulimic pathology [366]. The role of body dissatisfaction in the maintenance of eating pathology remains inconclusive also in light of the results of Project EAT, which suggest an association between body dissatisfaction and the persistence of unhealthy weight control behaviors [382], but not with the maintenance of binge eating [204].

Due to methodological differences between the studies, the comparison of the results is problematic. For example, in Project EAT, the analyses were not adjusted for depressed mood or depression symptoms. Therefore, a question remains whether the role of body dissatisfaction in the persistence of unhealthy weight control behaviors was independent or attributed to depressiveness. Thus, no firm conclusion can be drawn of the possible effect of an individual's dissatisfaction with appearance and body on protracted eating pathology. In addition, more evidence is needed of the possible role of anxiety in prolonged eating pathology. It is possible that neither anxiety nor appearance dissatisfaction independently affect prolonged eating pathology, as suggested by the results of this study, or their influence may occur in the short term and then fade. However, it is also possible that the association exists and has a long-term effect on the maintenance of eating pathology, but this study failed to detect it with its simple one-item measures.

To the knowledge of the author, this is the first study to investigate the possible effect of parental rearing style on adolescents' eating pathology from the viewpoint of average parenting, in other words, what is good enough parenting to enhance the remission and prevent the onset of EDS in youth after mid-adolescence. Previous studies have approached the question of parental role in the etiology of eating disorders by testing the assertion of the detrimental effect of dysfunctional parenting on individuals' eating psychopathology. Some of these studies using PBI as a continuous variable have found an association of low parental care and/or high overprotection [e.g., 419; 424; 455] with eating pathology, while others have established no evidence of such relations [e.g., 423; 456]. This study adds to the previous ones by showing an association between individuals' experiences of parental rearing style in childhood and the course of eating pathology in adolescence. The results show that an adolescent's perception of average parenting is generally sufficient to promote recovery from EDS – the only exception is maternal care. Accordingly, the odds of a girl for the remission of EDS were not significantly higher in those who had received high level of paternal care and low level of parental overprotection than in girls with experiences of average level of parenting. Maternal care was the only that needed to be higher than average to enhance the cessation of EDS. The result emphasizes the mother's role in a girl's development throughout childhood and adolescence, thus being in line with the findings of untreated recovery from eating disorders [457] by late adolescence, which state that the shortest duration of eating pathology and most complete spontaneous recovery occur in girls whose mother intervenes early and firmly in an empathic and supportive manner. Although youths' perceptions of the quality of family relationships generally decrease across adolescence, the relationship with mother remains closer than with father [458]. Mothers have a special task in mediating and moderating the appearance-oriented messages from the cultural

surroundings [see 459] which have an effect on individuals, especially during phases of identity confusion, such as adolescence [e.g.,105].

In the present study, the role of maternal care attenuated when depressiveness was taken into account. This might be due to recall bias caused by depressiveness, but it might also be due to the role of depressiveness operating as a part of causal chain between maternal care and the course of EDS. The first option is questionable in light of the evidence of the stability of responses concerning maternal care, irrespective of changes in the respondents' depression levels [430; 460], whereas the latter option is supported by the previous findings of an inverse association of maternal care with depression scores [461], depression, and dysthymia [462]. Accordingly, the protective effect of high maternal care against prolonged EDS may be based on the prevention of depressiveness. However, the retrospective assessment of parental behavior in this study does not permit causal inferences.

6.4.2 Predictors of the onset of EDS

The results of this study reveal several health-related differences between adolescents who develop EDS after mid-adolescence and those who remain EDS-free. However, significant differences did not appear until late adolescence, suggesting that individual health status in mid-adolescence does not predict who will have EDS in late adolescence and who will not. Initially non-symptomatic girls who developed EDS by late adolescence had depressiveness, anxiety, and dissatisfaction with appearance both in middle and late adolescence, or they experienced these problems or became overweight after mid-adolescence more commonly than girls who never reported EDS. In contrast, those who only reported these problems in mid-adolescence were not at odds for later onset of EDS. The strongest predictor of the initiation of EDS was dissatisfaction with appearance, which accounted for the association between the other factors and the onset of EDS. Development of EDS was also associated with the girl's experiences of the parenting style of her father, but not mother. Girls who recalled having received no more than average level of paternal protection or higher than average paternal care were better protected against EDS development than those with less benevolent experiences. In the multivariable analyses, the predictive power of paternal protection exceeded the effect of paternal care, but attenuated nonsignificant when dissatisfaction with appearance was taken into account. Thus, the result supports the idea of the higher importance of intra-individual characteristics than the parent-child relationship in the course of EDS.

Previously, the possible role of dissatisfaction with appearance has not attracted the researchers of the etiology of eating pathology. Instead, general interest has focused on parallel factors, such as body dissatisfaction, negative body image, and weight and shape concerns, which are well-established as risks for eating pathology in previous reviews [16; 366; 367]. Also more recent studies provide evidence of the association of body dissatisfaction [204; 382; 384] and weight concern [372; 379; 382] in adolescence with a later onset of eating pathology. However, none of these studies make distinction between youths who have temporary feelings of body/weight dissatisfaction/concern in mid-adolescence and those who still are or become dissatisfied/concerned with own body/weight in late adolescence. Thus, the findings are not quite comparable to the present study, which suggests that short-term dissatisfaction with appearance has no predictive power on later development of eating pathology, but the association occurs between concurrent problems. Unlike girls who were dissatisfied with own appearance in mid-adolescence only, those who failed to find satisfaction in or up to late adolescence

had increased odds for the onset of EDS. So, although appearance dissatisfaction in mid-adolescence was associated with the onset of EDS at one-year follow-up, the association diminished to nonsignificant during the four-year follow-up period, thus indicating a change in the role of appearance dissatisfaction from middle to late adolescence. However, it is also possible that the single-item measure that describes the respondents' subjective idea of appearance and physique in general missed some crucial aspects of the phenomenon and therefore failed to find the long-term effect.

In the previous reviews [16; 366], negative emotionality is considered a risk factor for eating pathology, whereas high BMI is not. In more recent studies, the findings are mixed. An association of the initial level of depressed mood [382] and increase in depression symptoms [381] with a later onset of eating pathology has been found in some studies, but not all [372]. In addition, in contrast to previous evidence, an association has been found between BMI in early adolescence and a later onset of eating disorders [372]. While waiting for more evidence of the possible effect of BMI and its change in adolescence on later development of eating pathology, one may consider elevated body mass as an operator in the origin of some other risk factors, e.g., body dissatisfaction [366], thus playing a part in the course leading to eating pathology.

As discussed in the previous section, the research of the effect of parental bonding on adolescents' behavior and attitudes towards eating and weight has provided mixed results. Some of the findings suggest an association of perceived paternal care and protection in childhood with eating pathology in young adulthood [97; 207], while others find paternal care but not protection [424], or protection but not care [455], or neither care nor protection [423; 456] being associated with eating pathology in adolescence. The present study found the effect of paternal parenting on the later onset of EDS in the univariate, but not in the multivariate analyses. The findings suggest that at least an average level of autonomy and independence, as well as, higher than the average level of emotional warmth, empathy, and closeness given by father protect girls from the onset of EDS after mid-adolescence.

Still, more important for the development of EDS was girls' feeling of personal appearance. Those who approached adulthood without finding satisfaction with appearance had more than 10 times higher odds for the onset of EDS than those who did not worry about their appearance. Usually, body dissatisfaction declines after puberty and turns to satisfaction, if the individual adolescent's experiences of the development are positive [463]. So, although paternal parenting may not have independent association with the onset of EDS, it may have an effect on the girls' appearance satisfaction. In fact, previous studies have found a negative correlation between paternal care and daughters' body dissatisfaction [423]. Thus, in addition to an average level of age-appropriate autonomy and independence, girls may need closer than an average relationship with their father to avoid the feelings of unattractiveness and subsequent development of EDS.

6.5 Methodological considerations

Following the procedure of etiological studies [see 362], the possible correlates of eating pathology were first explored using a questionnaire survey in a cross-sectional design, which was relatively efficient and economical to perform. Data of the independent variables were collected from participants' health records on the basis of the previous evidence of the factors associated with eating pathology. The cross-sectional findings guided the further stages of the study and facilitated the creation of efficient prospective

designs for the purpose of investigating the possible roles of the correlates in the course of eating pathology. In the following section, the strengths and weaknesses of this study are discussed in terms of the potential sources for bias and actions taken to avoid them stage by stage.

In stages I and II, the strengths of the study include firstly, homogeneity of the source populations. A commonly used sampling frame in the studies of mental health in adolescents consists of schools selected within a defined area [464]. In this study, the sampling frame consisted of the 8th and 9th grades in the Finnish-language secondary schools providing comprehensive education in the City of Turku during the school years 2003–2004 and 2004–2005. The source population consisted of students and school nurses. The students were at the age of mid-adolescence, when eating pathology typically emerges [340]. With a narrow age range, the confounding effect of students' age on results was reduced. The sampling method was non-random, and all students and school nurses were invited to participate in the study. The only exclusion criterion involved students with a handicap or a severe sensory deficit that would have disturbed independent responding to the questionnaire. Ability to complete the questionnaire without any assistance was important to reduce the potential for response bias, which is a particular problem in studies that investigate sensitive issues [465], such as disordered eating. Secretive reactions are less common when the individual is not met face to face [291].

Secondly, in the study, participants' health records were used as a source of information. This reduced the response burden and potential for non-response bias for the participants, or assessment bias originating from investigators.

Thirdly, the response rates were relatively high. The final sample consisted of 62% and 71% of the 8th and 9th grade students, and 100% of the school nurses invited in the study, thus resulting in a relatively large number of participants.

Finally, the data collection and analyses were performed to reduce the risk of bias and confounding effect as follows. School nurses were blinded to students' responses to SCOFF to avoid information bias. Gender-controlled logistic regression analyses were conducted to avoid the possible confounding effect of gender. In addition, multivariable analyses were performed to adjust for the effects of other factors measured at the baseline. Furthermore, school was used as a random factor due to possible intra-class correlation: individuals in the same school are more likely to have similar attitudes and behaviors as compared to randomly sampled people [466]. However, a possibility of the confounding effect of some other variable(s) not measured in this study could not be excluded. In addition, as the data was not complete in the whole sample and no imputations were done, missing values introduced potential for selection bias and a decrease in statistical power.

The main weaknesses of the study are found in external validity and the tools used in the data collection. First, the generalizability of the findings would have been better with a wider sampling frame representing mid-adolescents in the whole Finland. Although the sampling method used in this study was practical and the exclusion criteria were set to diminish bias, there remained potential for selection bias as some of the invited students elected not to participate. Generally, people with eating pathology are less willing to participate in studies concerning the topic than those without these problems [14]. However, potential for non-response bias is reduced when questionnaires are personally distributed [291], as they were in the present study. Still, despite the relatively high

response rates, a possibility remained that the rate of non-responses resulted in findings that underestimate the occurrence of EDS in mid-adolescent population. In addition, the non-response analysis showed that adolescents who did not respond to the survey differed from the respondents in gender; non-responding was more common among boys than girls. Accordingly, the results may be generalized to boys with caution, but not necessarily to non-Finnish-speaking students and those with a handicap or a severe sensory deficit. In addition, the results are not generalizable to ED patients in clinical settings or children and adult populations.

Secondly, the measurement of EDS and most of the health-related information was based on individuals' self-reports, thus being prone to response bias, particularly in questions concerning socially unacceptable and embarrassing issues. Only one half of the people with an eating disorder say that they would be truthful when answering questions about eating problems [129]. If these behaviors were under-reported in this study, the occurrence of EDS and the role of predictors would be underestimated. On the other hand, while the risk of EDS was estimated with odds ratio, the results may overstate the effect of predictors on EDS, since the occurrence of eating pathology was relatively high [see 467]. Using multiple informants, e.g., parents and teachers, might have provided a wider view on the topic. However, although adults may be an appropriate source of information when investigating children, they are less useful in questionnaire studies concerning adolescents: generally, adolescents are found to be the best informants regarding their own behavioral and emotional problems [246] which, in fact, may be concealed from others [241].

The use of interviews for data collection might have resulted in a somewhat different view of the topic. In psychiatric epidemiology, semi-structured interviews are regarded the 'gold standard'. As gold standard means 'error free' [468], clinical interviews are given great reliance. Nevertheless, the validity of clinical interviews has been questioned [67] since the agreement between the raters – even experienced clinicians – may vary a lot [e.g., 469], thus indicating interviewer bias. Especially in borderline cases typically found in community samples, the agreement may be low [470], because the standards for partial, sub-clinical and atypical eating disorders are not clear [225]. In addition, data collected by means of self-report questionnaires are known to better converge with data obtained with structured telephone interviews than with face-to-face interviews [358], and therefore, greater honesty of respondents may be attained when not met in person [302]. To date, there is no universally accepted assessment tool for the detection of disordered eating behaviors [343]. Therefore, no reference was used to test the accuracy of SCOFF and the occurrence of EDS because using an imperfect reference might have increased the potential for biased findings [see 468].

Originally, the cut-off point for SCOFF is two or more points to indicate a potential case of an eating disorder [271]. Using this threshold would have increased the comparability of the findings with previous studies. However, in this study, the threshold of one or more points in SCOFF was meaningful because the purpose of screening was to identify all adolescents with any EDS and not only those who already have an eating disorder.

Third, the validity of the results of the performance of SCOFF in comparison with an ordinary health examination was threatened by the possible variation in school nurses' assessments of adolescents' ED risk. Assessments were given by 14 school nurses and no comparisons were conducted between the responses of different nurses or different responses of each nurse.

Fourth, although the use of school health records reduced potential for non-response bias, its disadvantage was that only a limited number of potential predictors were recorded. In addition, only part of the health record information was based on standardized measures, such as RBDI [416]. Most of the factors were assessed with a single question, each designed for the practice of school health care. Thus, they may have missed some features of interest resulting in a limited picture of the issue. However, single questions are recommended for screening for symptoms in clinical practice as well as in research [471]. In addition, as the intention was to assess the usefulness of the health-related factors measured in school health care for the purpose of detecting EDS, it was warranted to use the adolescents' health records as such in data collection.

In stage III, the strength of the study is a prospective cohort design in a school-based sample of adolescents. Currently, eating pathology research is primarily conducted cross-sectionally among ED patients [373]. Here the study base consisted of students who participated in the baseline survey when in the 9th grade. The prospective design allowed for the focus on within-individual change, thus reducing the potential for alternative explanations of the results by excluding the possibility that any associations found might be due to some stable individual characteristics not measured in this study.

Unfortunately, a relatively high number of these students were lost at the 1-year follow-up, which is a weakness of the study and threatens the validity of the results. Attrition at the follow-up assessment increased potential for bias and may have led to underestimate the occurrence of EDS. Although attrition analyses showed no difference in the baseline EDS between participants who provided data at both assessments and those who were lost at the follow-up, it is possible that a higher response rate might have revealed higher 1-year stability of EDS. Besides the withdrawals from the follow-up assessment, the loss of participants was partly due to insufficient data collection procedure. The follow-up assessment was planned to be performed during the general health examination in the first year of upper secondary school or vocational institute that the students attended after the comprehenive secondary school. However, due to the school nurses' work load, a large number of students had no health check in the aforementioned year and therefore, they had no opportunity to participate in the follow-up survey. Had these students participated in the study, the findings might be different whichever direction.

Another weakness of the study concerns the data collection. As at the baseline, students' health records were used as a data source for putative predictors at the follow-up assessment. While part of the data were not comparable between the school grades, due to changes in measurement tools, some of the variables associated with EDS at baseline were excluded from the follow-up analyses. This may have increased potential for confounding, assuming that the excluded factors play a role in the course of EDS.

The most significant weakness of the study is that anxiety at baseline was less common in adolescents who were lost at the follow-up than in those who participated, thus indicating attrition bias. It is possible that the lost adolescents had not prolonged EDS to any larger extent than their counterparts who participated in the follow-up assessment. Still, it is also possible that anxiety-free adolescents who did not respond to the follow-up survey had prolonged EDS. In that case, the findings may overestimate the role of anxiety on the maintenance of EDS and underestimate the stability of EDS. Due to the small number of participants who reported both anxiety and EDS at the baseline, the 95% confidence interval for the estimate of the association between these factors was wide, indicating uncertainty around the estimate. Taken together, loss at the follow-up had a detrimental impact on the statistical inference of the role of anxiety in the maintenance of EDS.

In stage IV, the strengths of the study include firstly, a nested case-control design. In a nested case-control design, data are collected prospectively, thus avoiding potential recall bias. All individuals with EDS in mid-adolescence were invited to participate in the 4-year follow-up assessment. In addition, a group of late adolescents who were EDS-free in middle adolescence were recruited as controls. They were matched to symptomatic participants by the school, grade and gender, which were considered potential confounders for associations between the predictors and EDS. Thus, at the design phase, confounding was accounted for by matching cases and controls with regard to these three variables.

Secondly, in the analysis phase, confounding was accounted for by using multivariable analyses where the association between each factor and EDS was estimated by controlling for other factors. In addition, the analyses focused on within-individual change, thus reducing potential for alternative explanations of the findings by eliminating the effect of stable individual factors not measured in this study. Furthermore, while the course of EDS differed between girls and boys, as suggested by the time x gender interaction, the analyses were conducted separately for girls and boys. Missing data, which may decrease the statistical power of the analyses and compromise generalizability of the results, was addressed by imputing missing values as recommended [291; 416].

Thirdly, the response rate in the study was high (77%). Therefore, the potential for attrition bias remained relatively small. All data were self-reported, thus introducing potential for response bias. However, as the participants in the "case" group had revealed EDS already in mid-adolescence, they were supposed to feel no need to conceal the problem at follow-up assessment. In addition, since the data were collected via postal survey, the potential for dishonesty due to social desirability or feelings of shame was reduced.

Weaknesses of the study involve the attrition rate and incompleteness of the data. First, despite the large sample size, it is possible that attrition at the follow-up was non-random. In fact, the attrition analyses showed that those who declined to participate in the 4-year follow-up survey differed from the respondents in gender and the occurrence of EDS; attrition was higher among boys than girls as well as in adolescents with EDS than in non-symptomatic individuals. The attrition rate tended to be higher also in older and overweight people. Thus, the findings of the maintenance and new onset of EDS should be generalized to boys with caution. In addition, the results may underestimate the stability of EDS in population. Furthermore, as the assessments were conducted only twice, i.e., at the outset and the end of the follow-up period, it is possible that participants had experienced several transitions between asymptomatic and symptomatic statuses during the 4-year period. Thus, participants with prolonged EDS may have had EDS-free periods between the assessments, as well as those classified as asymptomatic may have developed EDS after the first assessment and then experienced remission before the final assessment.

The findings of the factors associated with the course of EDS were obtained among girls, wherefore the results should be generalized to boys with caution. In addition, since young females with eating concerns are found to overestimate their weight, while underestimation of weight is typical of those with weight concerns [472], self-reported height and weight at follow-up resulted in a possible error in the estimate of overweight. Thus, the possibility of misclassification of participants on the basis of self-reported height and weight cannot be fully excluded. Therefore, caution is recommended when interpreting the results of the association between overweight and the course of EDS.

Furthermore, imputation was not used for self-reported height, weight and anxiety, and in situations where the number of unanswered items compromised the estimation of missing values. Thus, potential for type II error and selection bias remains, since the number of participants with complete data in the subgroups of symptomatic individuals was small. In addition, due to the small size of the subgroups, the estimates of the associations between predictors and outcome obtained wide 95% confidence intervals, indicating uncertainty around the estimates.

When parental rearing styles are assessed solely on the basis of adolescents' retrospective reports, the reliability of the results is a concern. One may argue that recall bias and mood state at the time of responding may affect adolescents' responses, especially in individuals with depressiveness. However, in light of previous studies, there is substantial accuracy in early memories [473] and stability in the judgments of parenting in childhood irrespective of changes in respondents' depression levels [430; 474]. Furthermore, studies that have compared depressed individuals' reports of parental rearing styles to those of their relatives [461; 475], have shown that self-reported PBI succeeds in measuring actual parenting. What is more, early adults with AN in adolescence are found to recall experiences of parental bonding similar to those of their siblings [456]. In addition, retrospective assessment may cover a wider period of individual's life than prospective reporting. However, we cannot fully rule out the possibility that eating disturbance may have had an effect on the girls' recall of childhood experiences. Therefore, using multiple informants, e.g., siblings, might have increased the reliability of the results. Still, one should bear in mind that the way an adolescent perceives the relationship with parents is more important for the individual's eating behavior than others' perceptions concerning family relationships [476].

6.6 Implications

In practice, by adapting the population prevalence estimates of EDS in this study on the secondary schools in Finland, one can assume that, in a school of 100 mid-adolescents, 10–11 girls and 6 boys report at least one EDS in SCOFF. Without early detection and intervention, 5–6 girls and 1 boy are estimated to still have self-reported EDS in late adolescence. Furthermore, roughly one half of these individuals, i.e., 3 girls and 0–1 boy would have several self-reported EDS in late adolescence, indicating a suspicion of a full-blown eating disorder. Accordingly, every second girl and one out of six boys reporting EDS in mid-adolescence need an intervention to arrest the development of prolonged eating pathology. However, if no screening for eating pathology is performed, the vast majority of these students remain undetected. Therefore, using SCOFF as a routine part of adolescents' health examinations is justified in order to increase the rate of identified EDS. Since eating pathology is a problem for both genders, universal screening of all adolescents is warranted. In addition, universally performed screening for EDS increases both equality and the quality of health care; the detection of EDS no longer depends on the expertise of a single school nurse or doctor. Furthermore, as eating pathology has a fluctuating nature, it is important to perform screening annually throughout adolescence. While the benefit of the bi-dimensional model of the Finnish version of SCOFF remains questionable so far, it is preferable to use the simple one-factor model of SCOFF until sufficient evidence of the possible utility of the two-factor model is presented.

To identify those at the highest risk for prolonged eating pathology, adolescents' health records provide a useful source of information. In females, the maintenance of eating

pathology is tied to the negative affect, whereas dissatisfaction with appearance is typical of those with the initiation of EDS. The same may be presumed in males on the basis of previous evidence (see section 2.5.2). Accordingly, adolescents who report both EDS and negative affect, and those who fail to find "feeling of being at home in own body" [463], are the main target for clinical attention.

In the Current Care Guideline of Eating Disorders [15], the active role of parents is underlined in the treatment of youth with eating disorders. Undoubtedly, parents may benefit early detection and prevention of eating pathology as well. They may notice changes in adolescent's behavior and realize its abnormality for this person sooner than the school nurse who probably sees the student only once a year. They may also engage in preventive interventions, thus improving the outcome [for a review, see 210]. Today, extended health examinations provide a good opportunity for conversation with parents and thereby a wider view into the youth's possible need of intervention [477].

In future, methods for the deeper examination of the screen-positive adolescents should be explored for the possibility that some of the positive responses to the items of SCOFF reflect something else than true EDS (e.g., a misleading response given intentionally). In addition, it is important to assess the extent to which a more detailed secondary screen of eating pathology might benefit school nurses in the assessment of adolescent's status and possible need for the treatment. Targets for such tests could be, for example EDI-3-RF [269] and EDE-Q [252]. In addition, for external validation, a replication of this study is needed in a nationally representative sample of adolescents.

To enhance the detection of the high-risk individuals and to find the potential targets for preventive actions, more evidence is needed of the factors affecting the maintenance as well as the onset of eating pathology. However, as noted by Jacobi and co-writers [16], it is difficult to say whether any of the factors proximal to the onset of eating disorders are risk factors or merely early symptoms of eating disorders in evolution. Therefore, longer follow-up periods starting already in early childhood, are warranted to reveal the actual role of these factors in the course of eating pathology. Accordingly, additional longitudinal prospective population-based studies with repeated measurements with shorter intervals are needed to gain further clarity about the factors that are involved in the course of eating pathology, thus being potential signs of the high risk group, objects for experimental tests, and targets for preventive efforts. Identifying also the antecedents and correlates of these factors would provide deeper understanding about the causal chains leading to prolonged eating pathology. For example, anxiety as a typical precursor of depression [478], body-image dissatisfaction as a strong predictor of chronic depression [479], and youths' experiences of perceived parenting deserve further inspection. An ideal design would be a prospective study to observe these factors over time across age levels, developmental periods, and the course of eating pathology.

7   CONCLUSIONS

Eating disturbances have attracted researchers' interest for many decades. Even so, there is still no universal definition of eating pathologies apart from categorical eating disorders. Despite an increased evidence of the factors associated with eating pathology, there is still no comprehensive, universally sound theory of the etiology of eating disturbances either. However, the detrimental consequences of prolonged eating pathology and the importance of early detection of disturbed eating are well acknowledged. Accordingly, researchers have developed a wide variety of definitions and measurement tools to investigate eating pathology. Thus, the existing findings across epidemiological studies are not comparable, and much of the literature is cross-sectional and conducted in samples of patients. Up to date, prospective studies of the factors affecting the onset and maintenance of eating pathology in population are few.

This study extends the existing literature first, by providing novel evidence of the feasibility and importance of screening for eating pathology in Finnish school health care. SCOFF, a simple 5-item questionnaire of eating disorder symptoms (EDS), was found to be useful in the primary screening of adolescents. With the self-administered SCOFF, the detection of disturbed eating behaviors and attitudes increased by 300%, that is from 5% to 21%, as compared to ordinary health examinations where no screening for eating pathology was performed. Second, the findings lend support for a universal screening of all adolescents without gender limitation. Although self-reported EDS was more common in girls than boys, a significant number of boys were found screen-positives. Given this evidence, it can be seen that universal screening reveals eating pathology better than a health check without a screening procedure, or targeted screening where the potential targets are selected on the basis of school nurses' risk assessment.

Third, this study shows that adolescents' health records provide useful information to identify those of the screen-positive individuals who have the highest risk for prolonged eating pathology. Anxiety was characteristic of adolescents who remained screen-positive after a 1-year follow-up period. Those, who still had EDS after a 4-year follow-up had reported depressiveness in mid-adolescence more commonly than individuals who showed remission of EDS in late adolescence. Health records may also help finding those of the screen-negative individuals who are at risk for the onset of eating problems within a year. Typically, these adolescents were dissatisfied with own appearance. However, the association between appearance dissatisfaction and the onset of EDS diminished nonsignificant during the four-year follow-up, suggesting that one's dissatisfaction with personal looks may have a role as a proximal, but not as a distal risk for eating pathology. Accordingly, the evidence seems to indicate that health status of individuals with EDS in mid-adolescence is predictive of their EDS status in late adolescence, whereas the predictive power of the health information of EDS-free mid-adolescents is lost by late adolescence.

Fourth, this study adds to the previous evidence by investigating the protective effect of average parenting on the course of EDS, which has not been addressed in previous studies. The findings show that adolescents' experiences of their parents' rearing styles have a smaller impact on the development and course of EDS than their negative mood has. In general, average parenting in childhood proved to be good enough for youth to avoid EDS or to show it only temporarily. Still, the beneficial role of a mother on her daughter's recovery from EDS as well as the role of a father on his daughter's EDS-free

development should be recognized as a potential resource in preventive interventions; it appears that the parents may improve the outcome significantly.

Finally, this study extends the previous evidence by indicating that, in Finnish population, 6%–9% of youth have protracted EDS from middle to late adolescence. What is more, one half of them have multiform EDS, suggesting that 3%–5% of late adolescent population in Finland, i.e., 9 000–15 000 individuals aged 18–22 years, is suspected of having an eating disorder. This means a great challenge to the health care system in terms of ability to provide treatment for all those in need. On this basis, it may be concluded that screening for EDS to enhance early detection is warranted to enable preventive interventions against the development of prolonged eating pathology.

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APPENDICES

A list of items and response options of the RBDI questionnaire [416] that had been modified for the use in school health care

Number of item (response option)	Original RBDI	Modified version
1 (5)	Olen niin masentunut ja alavireinen, etten kestä enää	Olen alakuloinen jatkuvasti ja alavireinen, etten kestä enää
2 (5)	Tulevaisuus tuntuu minusta toivottomalta, enkä jaksa uskoa, että asiat muuttuisivat parempaan päin	Tulevaisuus tuntuu minusta toivottomalta, enkä jaksa uskoa, että asiat muuttuisivat
7 (2)	En ajattele vahingoittaa itseäni	En ajattele enkä halua vahingoittaa itseäni
7 (4)	Minulla on vakaa aikomus tehdä itsemurha	Minulla on tarkat suunnitelmat itsemurhasta
8	Miten suhtaudutte toisten ihmisten tapaamiseen?	Miten suhtaudut vieraitten ihmisten tapaamiseen?
9 (3)	Yritän lykätä päätösten tekemistä	Varmuuteni on vähentynyt ja yritän lykätä päätösten tekoa
10 (2)	En mielestäni näytä sen huonommalta kuin ennenkään	Ulkonäössäni ei ole minua haittaavia piirteitä
10 (3)	Olen huolissani, että näytän vanhalta tai epämiellyttävältä	Olen huolissani siitä, että näytän epämiellyttävältä
10 (4)	Minusta tuntuu, että ulkonäköni on muuttunut pysyvästi ja että näytän nyt epämiellyttävältä	Minusta tuntuu, että näytän rumalta
10 (5)	Olen varma, että näytän rumalta ja epämiellyttävältä	Olen varma, että näytän rumalta ja vastenmieliseltä
13 (4)	Ruokahaluni on nyt paljon huonompi kuin ennen	Ruokahaluni on paljon huonompi kuin ennen
13 (5)	Minulla ei ole enää lainkaan ruokahalua	Minulla ei ole lainkaan ruokahalua

Calculations of the estimates of the late-adolescent population at risk for any EDS, multiform EDS, prolonged EDS with at least one symptom, and prolonged EDS with multiform symptoms, and 95% confidence intervals (CI) for these estimates

Participants' points (p) in SCOFF (n, %) from middle to late adolescence

Mid-adolescence (n = 1891) ⟶	Late adolescence (n = 722)
0 p (n = 1569, 83%)	0 p (n = 402, 78%) 1 p (n = 66, 13%) 2–5 p (n = 46, 9%)
1 p (n = 232, 12%)	0 p (n = 97, 63%) 1 p (n = 29, 19%) 2–5 p (n = 28, 18%)
2–5 p (n = 90, 5%)	0 p (n = 20, 37%) 1 p (n = 13, 24%) 2–5 p (n = 21, 39%)

Late-adolescent population at risk for any EDS:

0.13 x 0.83 + 0.09 x 0.83 + 0.19 x 0.12 + 0.18 x 0.12 + 0.24 x 0.05 + 0.39 x 0.05 = 0.2585 ≈ 26%

95% CI: 0.2585 ± 1.96 x √ [0.2585 x (1-0.2585):1891] => 95%CI: 0.2388 – 0.2782 ≈ 24–28%

Late-adolescent population at risk for multiform EDS:

0.09 x 0.83 + 0.18 x 0.12 + 0.39 x 0.05 = 0.1158 ≈ 12%

95% CI: 0.1158 ± 1.96 x √ [0.1158 x (1-0.1158):1891] => 95%CI: 0.1014 – 0.1302 ≈ 10–13%

Late-adolescent population at risk for prolonged EDS with at least one symptom:

0.19 x 0.12 + 0.18 x 0.12 + 0.24 x 0.05 + 0.39 x 0.05 = 0.0759 ≈ 8%

95% CI: 0.0759 ± 1.96 x √ [0.0759 x (1-0.0759):1891] => 95%CI: 0.0640 – 0.0878 ≈ 6–9%

Late-adolescent population at risk for multiform prolonged EDS:

0.18 x 0.12 + 0.39 x 0.05 = 0.0411 ≈ 4%

95% CI: 0.0411 ± 1.96 x √ [0.0411 x (1-0.0411):1891] => 95%CI: 0.0322 – 0.0500 ≈ 3–5%

Calculations of the estimates of the late-adolescent female population at risk for any EDS, multiform EDS, prolonged EDS with at least one symptom, and prolonged EDS with multiform symptoms, and 95% confidence intervals (CI) for these estimates

Participants' points (p) in SCOFF (n, %) from middle to late adolescence

Mid-adolescence (n = 1035) ⟶ Late adolescence (n = 535)	
0 p (n = 815, 79%)	0 p (n = 279, 74%) 1 p (n = 51, 14%) 2–5 p (n = 45, 12%)
1 p (n = 148, 14%)	0 p (n = 63, 55%) 1 p (n = 25, 22%) 2–5 p (n = 27, 23%)
2–5 p (n = 72, 7%)	0 p (n = 14, 31%) 1 p (n = 12, 27%) 2–5 p (n = 19, 42%)

Late-adolescent female population at risk for any EDS:

0.14 x 0.79 + 0.12 x 0.79 + 0.22 x 0.14 + 0.23 x 0.14 + 0.27 x 0.07 + 0.42 x 0.07 = 0.3167 ≈ 32%

95% CI: 0.3167 ± 1.96 x √ [0.3167 x (1-0.3167):1035] => 95%CI: 0.2884 – 0.3450 ≈ 29–35%

Late-adolescent female population at risk for multiform EDS:

0.12 x 0.79 + 0.23 x 0.14 + 0.42 x 0.07 = 0.1564 ≈ 16%

95% CI: 0.1564 ± 1.96 x √ [0.1564 x (1-0.1564):1035] => 95%CI: 0.1343 – 0.1785 ≈ 13–18%

Late-adolescent female population at risk for prolonged EDS with at least one symptom:

0.22 x 0.14 + 0.23 x 0.14 + 0.27 x 0.07 + 0.42 x 0.07 = 0.1113 ≈ 11%

95% CI: 0.1113 ± 1.96 x √ [0.1113 x (1-0.1113):1035] => 95%CI: 0.0921 – 0.1305 ≈ 9–13%

Late-adolescent female population at risk for multiform prolonged EDS:

0.23 x 0.14 + 0.42 x 0.07 = 0.0616 ≈ 6%

95% CI: 0.0616 ± 1.96 x √ [0.0616 x (1-0.0616):1035] => 95%CI: 0.0470 – 0.0762 ≈ 5–8%

Calculations of the estimates of the late-adolescent male population at risk for any EDS, multiform EDS, prolonged EDS with at least one symptom, and prolonged EDS with multiform symptoms, and 95% confidence intervals (CI) for these estimates

Participants' points (p) in SCOFF (n, %) from middle to late adolescence

Mid-adolescence (n = 856) $\longrightarrow$	Late adolescence (n = 187)
0 p (n = 754, 88%)	0 p (n = 123, 88%) 1 p (n = 15, 11%) 2–5 p (n = 1, 0.7%)
1 p (n = 84, 10%)	0 p (n = 34, 87%) 1 p (n = 4, 10%) 2–5 p (n = 1, 3%)
2–5 p (n = 18, 2%)	0 p (n = 6, 67%) 1 p (n = 1, 11%) 2–5 p (n = 2, 22%)

Late-adolescent male population at risk for any EDS:

0.11 x 0.88 + 0.007 x 0.88 + 0.10 x 0.10 + 0.03 x 0.10 + 0.11 x 0.02 + 0.22 x 0.02 = 0.1226 ≈ 12%

95% CI: 0.1226 ± 1.96 x √ [0.1226 x (1-0.1226):856] => 95%CI: 0.1006 – 0.1446 ≈ 10–14%

Late-adolescent male population at risk for multiform EDS:

0.007 x 0.88 + 0.03 x 0.10 + 0.22 x 0.02 = 0.0136 ≈ 1%

95% CI: 0.0136 ± 1.96 x √ [0.0136 x (1-0.0136):856] => 95%CI: 0.0058 – 0.0214 ≈ 0.6–2%

Late-adolescent male population at risk for prolonged EDS with at least one symptom:

0.10 x 0.10 + 0.03 x 0.10 + 0.11 x 0.02 + 0.22 x 0.02 = 0.0196 ≈ 2%

95% CI: 0.0196 ± 1.96 x √ [0.0196 x (1-0.0196):856] => 95%CI: 0.0103 – 0.0289 ≈ 1–3%

Late-adolescent male population at risk for multiform prolonged EDS:

0.03 x 0.10 + 0.22 x 0.02 = 0.0074 ≈ 0.7%

95% CI: 0.0074 ± 1.96 x √ [0.0074 x (1-0.0074):856] => 95%CI: 0.0017 – 0.0131 ≈ 0.2–1%

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