# Eating Disorders in Middle Childhood

## Aspects of Prevalence and Influential Factors in the Development and Maintenance

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Dissertation zur Erlangung der Doktorwürde an der Philosophischen Fakultät der Universität Freiburg (Schweiz)

Genehmigt von der Philosophischen Fakultät auf Antrag der Professoren Simone Munsch (1. Gutachterin) und Jens Gaab (2. Gutachter)

Freiburg, den 23.06.2015

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**Erklärung der Selbstständigkeit**

Ich erkläre ehrenwörtlich, dass ich meine Dissertation selbstständig und ohne unzulässige fremde Hilfe verfasst habe und sie noch keiner anderen Fakultät vorgelegt habe.

Biel, März 2015

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Die vorliegende Dissertation umfasst folgende Publikationen:

Publikation 1: Dremmel, D., Wilhelm, P., Kurz, S., Fischer, S., Hilbert, A., & Munsch, S. (ready for submission). Delay of food-related gratification in children with BED, ADHD compared to healthy controls.

Publikation 2: Dremmel, D., Wilhelm, P., Kurz, S., De Albuquerque, J., Hilbert, A., & Munsch, S. (ready for submission). Emotional eating in children: The influence of parenting practices, expressed emotion and affective impulsiveness.

Publikation 3: van Dyck, Z., Bellwald, L., Kurz, S., Dremmel, D., Munsch, S., & Hilbert, A. (2013). Essprobleme im Kindesalter. *Zeitschrift für Gesundheitspsychologie, 21*(2), 91-100.

**Danksagung**

An erster Stelle bedanke ich mich bei meiner Doktormutter Frau Prof. Dr. Simone Munsch, ohne deren intensive, motivierende und kompetente Betreuung während aller Phasen des Doktorats diese Arbeit nie zustande gekommen wäre. Ein besonderer Dank gilt auch Herrn Dr. Peter Wilhelm, der mich intensiv und wohlwollend zu jeder Tageszeit durch die Methodik und Statistik dieser Arbeit begleitet hat.

Ein weiterer Dank gilt allen Personen des Studienteams und im Speziellen des Lehrstuhlteams, die nebst ihrem fachlichen Rat den Glauben an mich nicht aufgaben und mir mit unterstützenden Worten zur Seite standen.

Ebenso danken möchte ich meinem zweiten Gutachter, Herrn Prof. Dr. Jens Gaab sowie allen KoautorInnen meiner Publikationen, mitwirkenden StudentInnen, Schulzentren, StudienteilnehmerInnen und dem Schweizerischen Nationalfonds, welcher die Studie finanziell unterstützte.

Nicht zuletzt danke ich meiner Familie, meinen FreundInnen und MitbewohnerInnen sowie meinem Freund, die mir stets zur Seite standen.

Ich habe vieles gelernt und vor allem erkannt, dass eine Dissertation nebst Zeit viel innere Ruhe und Geduld benötigt. Dies zählt für mich zu einer der wichtigsten Erfahrungen.

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*Publikationen*

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Dremmel, D., Wilhelm, P., Kurz, S., Fischer, S., Hilbert, A., & Munsch, S. (ready for submission). Delay of food-related gratification in children with BED, ADHD compared to healthy controls.

Dremmel, D., Wilhelm, P., Kurz, S., De Albuquerque, J., Hilbert, A., & Munsch, S. (ready for submission). Emotional eating in children: The influence of parenting practices, expressed emotion and affective impulsiveness.

Fischer, S., Meyer, A. H., Dremmel, D., Schlup, B., & Munsch, S. (2014). Short-term cognitive-behavioral therapy for binge eating disorder: long-term efficacy and predictors of long-term treatment success. *Behaviour Research and Therapy*, *58*, 36-42. doi: 10.1016/j.brat.2014.04.007

Kurz, S., van Dyck, Z., Dremmel, D., Munsch, S., & Hilbert, A. (2014). Early-onset restrictive eating disturbances in primary school boys and girls. *European Child and Adolescent Psychiatry*, online 09 October. doi: 10.1007/s00787-014-0622-z

Hämmerli, K., Wyssen, A., Dremmel, D., Milos, G., Isenschmid, B., Trier, N., & Munsch, S. (2013). Leitlinien zur Diagnostik und Therapie der Essstörungen: eine Kommentierung. *Swiss Medical Forum*, *13*(43), 868-872.

van Dyck, Z., Bellwald, L., Kurz, S., Dremmel, D., Munsch, S., & Hilbert, A. (2013). Essprobleme im Kindesalter. *Zeitschrift für Gesundheitspsychologie, 21*(2), 91-100.

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## Contents

## Abstract

An experimental study and two questionnaire-based studies were conducted to examine the occurrence of problematic eating behaviours in community children and to identify the underlying mechanisms of maintenance, which are both essential for suggesting effective avenues for intervention.

The first study investigated the processes implicated in the delay of food-related gratification in children suffering from binge eating disorder (BED) compared to children with an attention deficit/hyperactivity disorder (ADHD) and healthy children. Children's behaviour during a delay of gratification task in terms of eating or waiting did not depend on psychopathology but was exclusively predicted by their body mass index (BMI). Self-reported experience during the task was independent of weight and strongly sensitive to psychopathology. Children with any disorder perceived an increased difficulty in waiting compared to healthy controls, whereas fears of losing control were most prominent in children suffering from BED.

The second study investigated whether parenting practices and parental expressed emotion influence children's emotional eating. Additionally it explored whether children's affective impulsiveness transmits the effects of parents' behaviour on children's emotional eating. Children's perceptions of emotional eating were strongly related to their self-rated affective impulsiveness, which in itself was significantly predicted by parental criticism. Parents' reports of elevated emotional eating in their children were associated with corporal punishment and authoritarian parenting.

The third study investigated the distribution of the newly defined eating disorder category avoidant/restrictive food intake disorder (ARFID) in a general school-based population of children aged 8-13 years. Avoidant or restrictive eating behaviours occurred in 29.8% of children when assessed by a brief self-report screening questionnaire. Underweight children reported more symptoms of the ARFID subtype "food avoidance emotional disorder".

Together, the findings suggest that problematic eating behaviours are common among children in middle childhood. Cases of a BED or emotional eating appear to be characterized by increased sensitivity to self-control difficulties in situations of food-temptation or intense affect and to co-occur with dysfunctional aspects of parental emotionality. Preventive or early treatment efforts should target self-regulation skills in children to cope with eating-related concerns and intense emotions but should also include specific interventions for parents.

# 1. Introduction

Eating disorders occur across almost all stages of life and have a severe and chronic natural course (Field et al., 2012; Kotler, Cohen, Davies, Pine, & Walsh, 2001). They are most prevalent in young female adults but can develop in children in middle childhood (Munsch & Hilbert, 2013). Research on the nosology and aetiology of eating disorders was for a long time dominated by studies on adolescent and adult populations. Therefore, specific middle childhood presentations of eating disorders remain under debate and have only recently been targeted by classification systems (Nicholls, Christie, Randall, & Lask, 2001).

The 5th revision of the Diagnostic and Statistical Manual of Mental Disorders (DSM-5; American Psychiatric Association [APA], 2013) marked an important step forward in the nosology of childhood eating disorders. A major target was to better acknowledge developmental variants in core features of anorexia nervosa (AN), bulimia nervosa (BN) and binge eating disorder (BED) (Ornstein et al., 2013) and to classify other childhood presentations of disordered eating that clearly diverge from AN, BN and BED, such as the newly defined diagnostic category of avoidant/restrictive food intake disorder (ARFID).

Despite significant progress in the classification of typical middle childhood eating disorders, data on their prevalence, course and prognosis remain scarce. Initial epidemiological studies on ARFID mainly focused on clinical populations of children, thereby leaving its distribution in the community largely unknown (Ornstein et al., 2013). Moreover, the maintenance processes specifically relating to eating disorders in middle childhood are not yet fully understood. Most existing studies focus on binge eating. While much emphasis has been given to negative affect and dietary restraint eating (Allen, Byrne, & McLean, 2012), recent evidence suggests that aspects related to a child's impulsivity and to family emotionality are of additional relevance and must be further examined in relation to binge eating in children (Pearson, Zapolski, & Smith, 2015; Reinblatt et al., 2014; Tetzlaff & Hilbert, 2014).

Middle childhood is a time of important developmental advances (Holodynski, 2013), with the consequences of eating disorders assumed to be particularly harmful (Nicholls, Wells, Singhal, & Stanhope 2002; Peebles, Wilson, & Lock et al., 2006). Thus, the characterization of concerned individuals and an empirical investigation of the maintenance factors are important, as this is directly useful to the development of age-adapted assessment and treatment methods (Combs, Pearson, & Smith, 2011; Stice, South, & Shaw 2012).

The present thesis contributes to filling the current gap in research on middle childhood eating disorders by means of three studies using experimental (Study 1) and questionnaire-based assessment methods (Study 2 and Study 3):

- To clarify the implications of impulsivity-related traits and correlates of family emotionality for the maintenance of BED or emotional eating behaviours in children (Study 1 and Study 2).
- To define the occurrence of ARFID symptoms in a general school-based population of children (Study 3).

Two introductive chapters precede the presentation of the studies. The first chapter offers an overview on current eating disorder research, including the symptomatology, epidemiology and aetiology of official diagnoses for AN, BN, BED and ARFID. The second chapter introduces the specificities regarding the classification and aetiological understanding of eating disorders in the age group of middle childhood. A final discussion takes up the central study results with regard to their clinical implications and significance for future research.

# 2. Overview of Eating Disorders

Eating disorders represent serious mental health conditions, implicating changes in all spheres of functioning, including cognition, behaviour, emotionality, and interpersonal relationships (Munsch & Hilbert, 2013). Eating disorders are more represented among adult women and adolescent girls but also occur in men, adolescent boys and children of both sexes (Muise, Stein, & Arbess, 2003; Pinhas, Morris, Crosby & Katzman, 2011). Although the clinical presentation of eating disorders may vary depending on a child's developmental stage, their core features remain constant across the life span.

## 2.1. Symptomatology

Eating disorders are characterized on a behavioural level by a restriction or avoidance of food, uncontrolled food intake or compensative behaviours to prevent weight gain. Frequent ruminations about weight, shape or eating represent a significant cognitive burden to concerned individuals and are related to the experience of negative emotions (Munsch & Hilbert, 2013). The currently released 5th revision of the DSM (APA, 2013) recognizes AN, BN, BED and ARFID as formal eating and feeding disorders. It further includes the section *feeding or eating disorder not elsewhere classified*, listing atypical or subthreshold presentations of AN, BN, and BED and other syndromes not formally included in the DSM-5, such as purging disorder and night eating disorder.

While the key feature of AN is clinically significant and persistent dieting despite a significantly low weight, the diagnosis of BN describes recurrent episodes of binge eating followed by inappropriate compensatory behaviour such as self-induced vomiting to prevent weight gain. BED is diagnosed when recurring episodes of binge eating occur but are not regularly followed by inappropriate compensatory behaviour to regulate weight such as in BN. Binge eating means consuming more food in a short period of time than most people would eat under similar circumstances, thereby experiencing a sense of a lack of control over eating. It is the perceived loss of control in association with additional features such as eating alone, eating without hunger or experiencing strong negative emotions following eating that distinguishes an episode of binge eating from overeating. ARFID captures restricted food-intake and malnutrition, which, unlike AN, is not associated with body image disturbance or fear of weight gain. The diagnosis ARFID replaces and extends the former DSM-IV category *feeding disorder of infancy or early childhood* (APA, 2000) by subdividing it into three presentations of food avoidance or limited interest in food based on (1) emotional reasons

(food avoidance emotional disorder), (2) sensory characteristics (selective eating) and (3) aversive experiences implicating a fear of swallowing, choking, or vomiting (functional dysphagia).

## 2.2. Epidemiology

### *2.2.1. Prevalence*

Epidemiological data on the risk group of young women indicate lifetime prevalence rates of 0.8% for AN, 2.6% for BN and 3% for BED (Stice, Marti, & Rohde, 2012). With a prevalence of 3.5% in women and 2% in men, BED is the most common eating disorder (Hudson, Hiripi, Pope, & Kessler, 2007), and unlike AN or BN, it is similarly distributed in women and men (Bulik et al., 2006; Hoek & van Hoeken, 2003; Hudson et al., 2007). Furthermore, BED has a later age of onset, frequently affecting individuals over age 35 (Harrison & Hefner, 2006). At the same time, a substantial number of individuals suffering from BED trace the onset of their binge eating problems to middle childhood (Abbott et al., 1998; Spurrell, Wilfley, Tanofsky, & Brownell, 1997).

Limited epidemiological data on ARFID are available. Among children and adolescents at eating disorder programs, 13.8% met ARFID criteria (Fisher et al., 2014). Similarly, when studying adolescent patients who sought medical services, Ornstein and colleagues (2013) identified ARFID in 14% of all cases. Finally, ARFID is estimated to make up approximately 40% of recorded cases of early-onset (< 13 years) eating disorders (2.6-3.01 cases per 100,000 person-years) according to national surveillance studies in Canada (Pinhas et al., 2011) and Great Britain (Nicholls, Lynn, & Viner, 2011).

### *2.2.2. Comorbidity*

Eating disorders have a variety of adverse outcomes (Mitchell et al., 2002). Concerned individuals experience significantly higher rates of comorbid psychopathology and medical problems (Field et al., 2012). Mental health comorbidity is most pronounced for affective and anxiety disorders (Brewerton et al., 1995; O'Brien & Vincent 2003; Wonderlich, Gordon, Mitchell, Crosby, & Engel, 2009). Individuals with BN and BED furthermore have a greater risk for substance dependency (Brewerton et al., 1995; Wietersheim et al., 2008). Rates of certain personality disorders are also higher in individuals with eating disorders, e.g., of the borderline type in BED and obsessive-compulsive type in AN (Cassin & von Ranson, 2005; Wietersheim et al., 2008). Medical complications from eating disorders appear mainly related

to the consequences of malnutrition. Starvation behaviours in AN may implicate severe metabolic alterations, especially neuroendocrine and neurotransmitter changes (Herpertz-Dahlmann, Seitz, & Konrad, 2011). BED in turn involves a continuous weight increase and is strongly associated with adiposity, which is an established risk factor of secondary medical disease (Herpertz & Munsch, 2008). In youth, full and subthreshold eating disorders predict greater functional impairment, distress, suicidality, mental health treatment, and an unhealthy body mass index later in life (Stice, Marti, & Rohde, 2012).

#### *2.2.3. Course and prognosis*

Research on the long-term course of eating disorders reveals an overall remission rate of approximately 50% for BN and AN (Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004). Data more specifically show that almost half of treated AN cases fully recovered after 5-6 years. Among the other half, 30% improved with only partial or residual features, while 20% remained chronically ill over the long term (Steinhausen, 2002). The mortality rate for AN has been estimated to be three to ten times higher than the normal population (Crow et al., 2009; Striegel-Moore, & Bulik, 2007). This applies in particular to individuals with a BMI < 13. Patients suffering from BN who followed cognitive-behavioural therapy presented a remission rate of approximately 50%, with two-thirds of individuals reaching substantial improvement of their symptoms (Tuschen-Caffier & Florin, 2012). A diagnostic crossover from BN to AN occurred in 5.7% of cases (Steinhausen & Weber, 2009). A reversed crossover from AN to BN appears even more frequently, in up to one-third of cases, but is also very unstable, with a majority of concerned individuals crossing back to AN (Eddy et al., 2008). BED takes a similarly chronic course to BN and AN but rarely progresses to another eating disorder (Striegel-Moore & Franko, 2008). Retrospective assessments indicate an average lifetime duration for BED of 14.4 years, which is longer than that of AN or BN (Pope et al., 2006).

### 2.3. Aetiology and Maintenance

The aetiology of eating disorders is widely thought to be multifactorial, involving a combination of biological, social and psychological risk factors (Stice, South, & Shaw, 2012). Studies on heritability estimates suggest a significant genetic contribution to eating disorders (Thornton, Mazzeo, & Bulik, 2011). To date the localisation of the genetic variants in neuronal or neuroendocrine systems remains largely unspecified, however (Pinheiro et al., 2010). There is also compelling evidence that an altered function of brain neuro-circuitry may

interfere with the development or maintenance of eating disorders. New research suggests that a hyper-responsive emotional/fear network to food and underactive ventral fronto-striatal circuits (cognitive flexibility) are implicated in severe AN, while impairments in inhibitory control networks appear related to BN (Friederich, Wu, Simon, & Herzog, 2013). Other factors with respect to the responsiveness of the reward system in the form of a hyper- or hypo-functioning somatosensory and/or gustatory cortex are currently being discussed with regard to overeating and binge eating in individuals with BN and BED (Friederich, et al., 2013).

### *2.3.1. Risk factor research*

Longitudinal risk factor research has revealed gender (being female), ethnicity (being of a non-Asian cultural affiliation), and non-genetic perinatal aspects common to other mental disorders, such as pregnancy complications, as risk factors for AN and BN (Jacobi & Neubert, 2005). Of central aetiological relevance to any eating disorder, with the exception of ARFID, are dysfunctional thoughts about the body, shape and weight (Jacobi et al., 2011; Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004; Killen et al. 1996; Stice, Marti, & Durant, 2011). Additional psychosocial variables evidenced as risk factors are the exposure to critical comments about eating from teachers, coaches or siblings before the age of 18, weight-related teasing by the family and temperamental traits such as negative emotionality, neuroticism, and perfectionism as well as a history of depression (Bulik et al., 2006; Jacobi et al., 2011; Leon, Fulkerson, Perry, & Early-Zald, 1995; Stice, 2002). New evidence suggests that the selection of a peer environment may represent a further risk factor for maladaptive weight control behaviours in adolescents (Eisenberg & Neumark-Sztainer, 2010; Keel & Forney, 2013; Keel, Forney, Brown, & Heatherton, 2013).

### *2.3.2. Integrative aetiological models of eating disorders*

Despite the manifest differences in the phenotypes, eating disorders share common pathogenic factors regarding their aetiology and maintenance (*transdiagnostic theory of eating disorders*, Fairburn, Cooper, & Shafran, 2003). Current integrative models basically explain the maintenance of eating disorders as an interaction of body- or eating-related concerns and negative affect with pathological eating behaviours (Stice et al., 2011). On the one hand, dysfunctional mechanisms implicated in the processing of eating- and body-related contents are considered to account for maladaptive eating behaviours in individuals with eating disorders. A negative view of food or eating can lead to dysfunctional body-related

thoughts in concerned individuals and trigger negative affect and/or maladaptive behaviours (Dittmar, 2009; Shafran & Robinson 2004). A second mechanism that has been implicated in disordered eating refers to an impaired ability to perceive and regulate emotions (Harrison, Sullivan, Tchanturia, & Treasure, 2010; Munsch, Meyer, Quartier, & Wilhelm, 2012). Hämmerli and colleagues (2013) have illustrated both of these pathways within a global aetiological framework (see figure 1) that summarizes the current conceptualizations of eating disorders, e.g., Stice et al. (2011) and Dittmar (2009).

Finally, it must be noted that based on findings to date, aetiological models of eating disorders do not account for ARFID, whose maintenance factors remain largely unexplored.

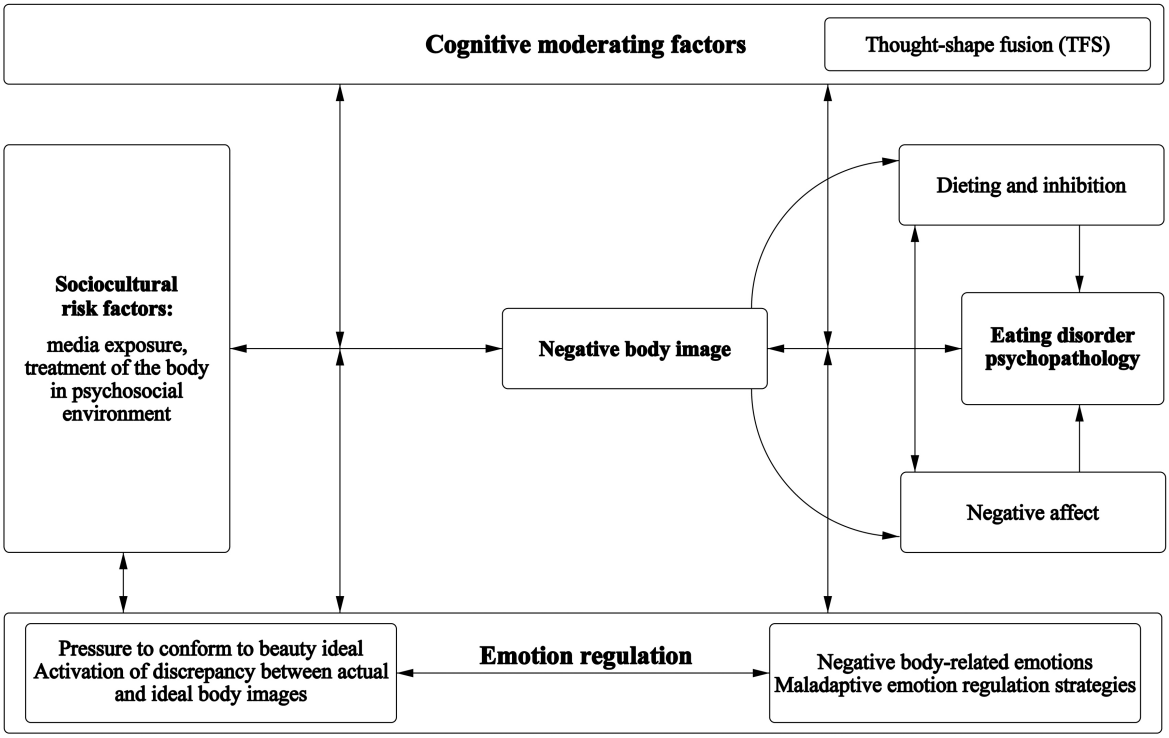


*Figure 1*. Integrative aetiological model of eating disorders (on the basis of Stice et al., 2011 and Dittmar, 2009) adapted from "Leitlinien zur Diagnostik und Therapie der Essstörungen: eine Kommentierung", by K. Hämmerli, A. Wyssen, D. Dremmel, G. Milos, B. Isenschmid, S. N. Trier, and S. Munsch, 2013. *Swiss Medical Forum*, 13 (*43*), p. 869. Copyright 2014 by the EMH Schweizerischer Ärzteverlag AG.

# 3. Eating Disorders in Middle Childhood

## 3.1. Classification

Clinical studies have revealed a wide range of eating difficulties encountered by children in middle childhood who consult health services (Bryant-Waugh & Lask, 2013). However, basing the classification of these difficulties on psychiatric nosologies such as the DSM (APA, 2000) has remained difficult for a long time due to the lower thresholds in the clinical presentation of symptoms and a lack of diagnostic categories to account for typical childhood disturbances that significantly diverge from AN, BN or BED (Bryant-Waugh & Lask, 2013). As a result, many clinically significant eating disturbances in children have often fallen into the category eating disorder not otherwise specified (EDNOS), which, in a broader sense, has hindered the development and validation of specific aetiological models for children.

Based on the recent 5th revision of the DSM (APA, 2013) classification system, research has been undertaken to better characterize the manifestations of eating disorders in childhood and to improve their diagnoses. On the one hand, new diagnostic criteria have been defined to account for eating disturbances, whose common feature is restriction or avoidance of food, without implying body image concerns or fear of weight gain. Past research has employed different terminologies, e.g., selective/picky eating, food avoidance emotional disorder, restrictive eating, food refusal, or specific fear/phobia leading to food avoidance (e.g., functional dysphagia) to describe such eating disturbances that are now captured by the diagnosis of ARFID (Fisher et al., 2014). On the other hand, the diagnostic criteria for AN, BN and BED have been adapted to better reflect the developmental variations in the clinical presentations of their core symptoms. The major changes include the reduction of the thresholds of symptom occurrence (e.g., binge eating in BN and BED) and less emphasis on the cognitive symptoms in favour of the behavioural manifestations (e.g., persistent behaviour that interferes with weight gain instead of fears of weight gain), making the diagnosis more appropriate for younger patients who do not yet have the capacity for abstract reasoning (Attia et al., 2013). Due to these innovations, a substantial decrease in EDNOS cases in children could be achieved (Ornstein et al., 2013).

### *3.1.1. Binge eating episodes in children*

The clinical utility of eating disorder diagnostic criteria has been particularly debated with respect to BED in childhood (Shomaker et al., 2010). Research on children has especially brought to question the criteria related to the objectification of the amount of consumed food

during an episode of binge eating. It has been considered challenging to define what constitutes a large amount of food for developing children with varying nutritional needs (Tanofsky-Kraff, Marcus, Yanovski, & Yanovski, 2008). Whereas a subgroup of children seems to present symptom profiles consistent with the adult definition of binge eating, a substantial part of children describe subjective feelings of loss of control without accompanying excessive food intake (subjective binge eating) (Tanofsky-Kraff et al., 2004). Importantly, the experience of a loss of control (LOC) irrespective of the quantity of consumed food has been found equally indicative of clinically significant eating disorder and general psychopathology as objective binge eating (including large amounts of foods) (Shomaker et al., 2010; Tanofsky-Kraff et al., 2011; Tanofsky-Kraff et al., 2004). Proposals have therefore been made to recognize the experience of a LOC itself, apart from the reported amount of food consumed during an episode of eating, as the hallmark of binge eating in children (Bravender et al., 2010; Shomaker et al., 2010). LOC eating is common in middle childhood, affecting 9.3% of 6 to 12-year-old normal and overweight children (Tanofsky-Kraff et al., 2004). It usually takes a moderately stable course and often presents in concert with disordered eating attitudes, emotional distress and being overweight (Hilbert et al., 2013; Tanofsky-Kraff et al., 2011). Affected children have been characterized by increased eating in the absence of hunger, as well as secretive and emotional eating (Hilbert & Munsch, 2005).

## 3.2. Maintenance Factors

Research on maintenance factors involved in childhood eating disorders has primarily focused on binge eating. Therefore, the interest has mainly remained on negative affect and dietary restraint eating, which are considered to be key maintenance factors of binge eating in aetiological models developed for adults (Fairburn et al., 2003; Stice, 1998; Stice et al., 2011) that have been adapted to children (Goldschmidt, Aspen, Sinton, Tanofsky-Kraff, & Wilfley, 2008). Other potentially relevant maintenance factors that are currently being researched refer to impulsivity-related traits and aspects of family emotionality.

### *3.2.1. Negative affect*

Emotional factors are considered to be central to the maintenance of any type of eating disorder (Aldao, Nolen-Hoeksema, & Schweizer 2010). A dysfunctional regulation of emotions, such as the avoidance of aversive mood states, has been found to be associated with binge eating and compensatory behaviours (self-induced vomiting, laxative abuse, over-

exercising) and is discussed in relation to excessive dieting (Corstorphine, Mountford, Tomlinson, Waller, & Meyer, 2007; Munsch & Hilbert, 2013; Polivy & Herman, 2002).

Different theoretical concepts account for the relationship between impaired emotion regulation and binge eating in BED or BN. Accordingly, binge eating is conceived of as a maladaptive coping strategy, serving to temporarily reduce a momentary negative affect (*affect regulation theory,* Arnow, Kenardy, & Agras, 1992) or to provide an escape from a negative self-awareness (*escape theory*, Heatherton & Baumeister, 1991). The expected hedonic value of food is thought to contribute to a momentary reduction of discomfort (*expectancy theory*, Hohlstein, Smith, & Atlas, 1998).

Empirical research on children has delivered cross-sectional and longitudinal evidence for the associations between negative affect or depressive symptoms and the occurrence of binge or LOC eating (Allen et al., 2012; Allen, Byrne, Puma, McLean, & Davis, 2008; Hilbert, Hartmann, Czaja, & Schoebi, 2013; Stice et al, 2011; Tanofsky-Kraff, Faden, Yanovski, Wilfley, & Yanovski, 2005). Furthermore, increases in states of negative affect could be identified as significant precursors of binge eating episodes in adults based on ecologically momentary assessment data (Haedt-Matt & Keel, 2011). Corresponding research in children has to date produced contrasting findings. Whereas overall levels of negative affect were higher on LOC days compared to non-LOC days, antecedent negative affect did not specifically precede episodes of LOC eating in children when it occurred at home (Hilbert, Rief, Tuschen-Caffier, de Zwaan, & Czaja, 2009) or during test-meals (Goldschmidt, Tanofsky-Kraff, & Wilfley, 2011; Hartmann, Rief, & Hilbert 2012; Hilbert, Tuschen-Caffier, & Czaja, 2010). In contrast, the experience of negative affect during earlier periods of the day and the exposure to parental shape- and weight-related critics during a test-meal significantly predicted a sense of LOC and increased food consumption in these children (Goldschmidt et al., 2011; Hartmann et al., 2012; Hilbert et al., 2010). These results have been interpreted to reflect a longer lag time between the onset of a negative mood and the compensatory loss of control eating in children. This in turn led some authors to emphasize the importance of studying the cognitive and emotional processes taking place during this interlude (Goldschmidt et al., 2011).

#### *3.2.2. Dietary restraint eating*

Dietary restraint eating is defined as a cognitive tendency towards restrictive eating, irrespective of the success of effective dietary restriction (Herman & Polivy, 1984). Because cognitive restraints imposed on eating challenge an individual's self-control capacities,

individuals engaging in dietary restraint eating are thought to become vulnerable to disinhibited eating when their cognitive controls over eating are disrupted, for example through negative affects. According to the *restraint theory* (Herman & Polivy, 1984), binge eating may either result from the caloric deprivation caused by effective dieting (dietary restriction) or interruptions in strict dietary control (dietary restraint eating). The risk of binge eating is considered especially high if dietary restraint eating occurs in combination with an accumulation of negative affect (Herman & Polivy, 1984; Wagner, Boswell, Kelley, & Heatherton, 2012).

The restraint theory has received support from empirical literature on adolescents whose dietary restraint eating has been validated as an onset predictor of binge eating (Field et al., 2003; Field et al., 2008; Neumark-Sztainer, Wall, Haines, Story, & Eisenberg 2007; Stice, Presnell, & Spangler, 2002). In children, its relative importance is suggested by a recent path analysis of prospective data (Allen et al., 2012) and by cross-sectional studies using structural equation modelling (Decaluwé & Braet, 2005; Goossens, Braet, & Bosmans, 2010), while a recent longitudinal study presented contrary results (Hilbert, Hartmann, et al., 2013). Existing studies have usually assessed efforts to restrict dietary intake (dietary restraint eating) rather than actual dietary restriction (dieting). There is some evidence from adult and adolescent studies to suggest that effective dieting might be more powerful for predicting binge eating than dietary restraint eating (Agras & Telch, 1998; Stice, Davis, Miller, & Marti, 2008). Finally, there is convincing evidence from a longitudinal study (Hilbert, Hartmann, et al., 2013) and a cross-sectional ecologically momentary assessment study (Hilbert, Rief, et al., 2009) that eating-disorder-specific thoughts about food/eating or the body also merit attention as maintaining factors of binge eating and may probably even represent stronger predictors of binge eating in children than dietary restraint eating.

#### *3.2.3. Temperament*

It is assumed that certain temperamental traits in combination with other risk factors increase a person's vulnerability to developing an eating disorder (Martin et al., 2000) and influence its prognosis (Segura-Garcia, Chiodo, Sinopoli, & De Fazio, 2013). As such, traits of neuroticism and perfectionism, especially high personal standards, have been associated with the development of AN and BN in adolescents and adults (Bulik et al., 2003; Castro et al., 2007; Sassaroli et al., 2008). Furthermore, individual differences in impulsivity have been implicated in the occurrence of binge eating in adults with BN or BED and in children with BED (Friederich et al., 2013; Steadman & Knouse, 2014). As a component of the

multifactorial concept of impulsivity, negative urgency accounting for an individual's disposition to act rashly under conditions of negative affect (Whiteside & Lynam, 2001) has been repeatedly associated with risk for binge eating onset in children and adolescents in longitudinal and cross-sectional research (Combs et al., 2011; Pearson et al., 2012; Pearson et al., 2015). Of additional relevance to the maintenance of BED are motivational aspects of impulsivity accounting for an individual's spontaneous reactions to food temptation (Friederich et al., 2013). Indeed, children with binge eating appear to be more likely to engage in pleasure-orientated eating than healthy children based on a laboratory test-meal study (Hilbert et al., 2010). This has been hypothesized to reflect a hyper-responsive reward system similar to what has been shown for obese individuals in studies on adolescents and adults using fMRI or eye tracking (Schag, Schönleber, et al., 2013; Schag, Teufel, et al., 2013; Schienle, Schäfer, Hermann, & Vaitl, 2009; Stice, Yokum, Burger, Epstein, & Small, 2011).

The ability to resist an immediately available gratification is usually approached through the concept of reward sensitivity (Gray & McNaughton, 2000). In today's obesogenic environment, which offers a variety of palatable and calorie-dense foods, an increased sensitivity to reward is thought to place an individual at risk for eating behaviours that are unrelated to hunger but related to hedonics or affective conditions (Stice, Spoor, Ng, & Zald, 2009). Empirically, an increased reward sensitivity measured through a computerized paradigm (door opening task) has been associated with overeating in community children when exposed to varied foods in the laboratory (Guerrieri, Nederkoorn, & Jansen, 2008). Initial research on obese, young adolescents with and without binge eating using the same computerized paradigm confirmed that there was an elevated reward sensitivity in obese young adolescents compared to normal weight adolescents that was even more elevated if they presented with regular binge eating (Nederkoorn, Braet, Van Eijs, Tanghe, & Jansen, 2006).

An alternative method for conceptualizing reward sensitivity is offered by behaviour observation methods using the delay of gratification task (DOG) (Mischel, Shoda, & Rodriguez, 1989). While the door opening task uses non-edible prizes handed out to study participants depending on the amount of points they earned in a gambling task (Nederkoorn et al., 2006), the DOG measures children's spontaneous reaction to an immediate food temptation. Previous studies integrating the DOG task have come to confirm cross and longitudinal relationships between an impaired ability to resist immediately available food rewards and risk for being overweight or weight-increase (Bonato & Boland, 1983; Francis &

Susman, 2009; Seeyave et al., 2009; Sobhany & Rogers, 1985). However, these studies did not integrate measures on concurrent binge eating. Additional research investigating DOG in community children with and without BED is required to establish whether and through which processes an increased sensitivity to food-rewards contributes to the maintenance of binge eating in middle childhood.

#### *3.2.4. Familial factors*

Research has for a long time focused on family influences when studying disordered eating in children. This is not surprising, considering that the family environment constitutes the principal socialization context for the development of eating habits during childhood (Tetzlaff & Hilbert, 2014). Familial characteristics are assumed to significantly influence eating disorders, attenuate or strengthen the influence of other genetic, sociocultural or temperamental factors and help children in coping with the latter (Reich, 2005).

Diverse familial factors have been assumed to be implicated in childhood eating disorders. A few have been evidenced as risk factors (parental critical comments about weight or shape, weight-related stigmatisation, few and irregular family meals), while many others (e.g., dysfunctional family relationships, high family control or parental disaccord) have been identified as correlates (Jacobi et al., 2011; Schuetzmann, Richter-Appelt, Schulte-Markwort, & Schimmelmann, 2008; Tetzlaff & Hilbert, 2014; Topham et al., 2011). Current research provides cross-sectional evidence for associations between specific aspects of parental behaviour such as aversive parenting practices (contradictory parental behaviour, parental rejection, non-reasoning punishment, high psychological control) and disordered eating, especially emotional eating in children and adolescents (Schuetzmann et al., 2008; Snoek, Engels, Janssens, & van Strien, 2007; Topham et al., 2011). Similar results have been found by a recent observation study on family functioning at mealtimes in home environments, revealing more dysfunctional interactive behaviour in families of children with LOC eating than in families of healthy control children (Czaja, Hartmann, Rief, & Hilbert, 2011). Together these findings provide initial indications for the possible role of a parent's own emotionality in the development or maintenance of BED. Family emotionality has been depicted by the construct of expressed emotion (EE) (Rutter & Brown, 1966), which is based on the amount of criticism and emotional overinvolvement expressed by parents when describing their relationship with their child. EE has come to be considered an important factor implicated in the course and treatment success of different mental disorders (Hooley, 2007), including eating disorders (Duclos, Vibert, Mattar, & Godart, 2012).

## 4. Aims of the Present Thesis

There is a growing interest in the current literature for studying impulsivity-related traits in conjunction with binge eating in children. Initial findings on increased levels of negative urgency (Pearson et al., 2015) and elevated reward sensitivity (Nederkoorn et al., 2006) associated with binge eating suggest that impulsivity may present as an essentially emotion-driven and motivational process in children suffering from BED. Nevertheless, there still remain uncertainties regarding the specific role of reward sensitivity in BED compared to related conditions such as obesity or other disorders of impulse-control such as attention deficit/hyperactivity disorder (ADHD). Furthermore, it is yet to be determined whether the initial findings of Nederkoorn et al. (2006) concerning increased reward sensitivity in hospitalized, obese young adolescents with binge eating will apply to community children with BED when reward sensitivity is approached within an ecologically valid laboratory situation using tempting foods as rewards.

The current literature also provides ample evidence for an implication of familial variables in the maintenance of eating disorders in children. Among the variety of familial variables that have been researched, aspects of family emotionality appear to be of particular significance because of the important role that has been attributed to negative affect in the manifestation of binge eating (Allen et al., 2012; Allen et al., 2008; Hilbert, Hartmann, et al., 2013). The tripartite model of familial influence accords a major role to parents' emotional attitudes and parenting practices in children's emotional development (Morris, Silk, Steinberg, Myers, & Robinson, 2007). Relatedly, LOC and binge eating may be viewed as a dysfunctional affect regulation strategy if children's development of emotion regulation is not supported by a favourable family atmosphere (Munsch & Hilbert, 2013). Yet only limited research has investigated the mechanisms through which the correlates of family emotionality impact children's eating behaviour. A child's disposition to rash action under strong emotional arousal, which is accounted by the construct of urgency, has come to be considered as an important predictor of disinhibited eating (Pearson et al., 2012; Pearson et al., 2015), and thus might constitute an important variable to consider in the study of parental influence.

Finally, certain eating disorders specific to middle childhood remain particularly understudied. Most significantly, there are still many open questions about the new ARFID diagnosis with respect to its prevalence, course and aetiological mechanism. To date, ARFID has primarily been described in clinical populations of children referred to medical health services (Nicholls & Bryant-Waugh, 2008). An important step forward is to specify its

occurrence and symptom profiles in the general population of middle childhood and to characterize the relevant demographical and clinical features of the affected children in the context of other eating disorders for this age span.

Altogether, additional epidemiological and aetiological studies are needed to clarify aspects of prevalence and influential factors in the development and maintenance of middle-childhood eating disorders, which is a condition sine qua non to the establishment of adequate evidence-based treatments for this age group (Combs et al., 2011). This thesis aimed to contribute to a better understanding of the role of impulsivity-related traits and familial factors in the maintenance of problematic eating behaviours in children by means of two different studies (Study 1 and Study 2), thereby setting the focus on BED and emotional eating. In a third study (Study 3), we intended to evaluate the occurrence of ARFID symptoms in a large community sample of children attending public school.

More specifically, Study 1 aimed to investigate the processes of food-related reward sensitivity in children at the age of middle childhood with BED, ADHD or both diagnoses (BED&ADHD) compared to healthy children. As there is a high co-occurrence of obesity in these clinical populations (Nazar et al., 2012), the influence of children's BMI was statistically controlled, which allowed us to specify the unique effects of the psychopathology. To enhance the ecologically validity of the measures, we used a slightly modified version of the DOG task (Mischel et al., 1989), approximating children's every day behaviour when confronted with tempting foods as closely as possible. We assessed children's individual taste preferences for a selected range of common snack-foods in advance, and used their preferred food-reward in the experiment to ensure its attractiveness for the child. Additionally, we aimed to evaluate whether self-report of experienced difficulties during the DOG task were in line with the observable behaviour of the child, which allowed us to further clarify the processes implicated in children's delaying behaviour. Finally, we also intended to investigate the influences of current negative emotions, depressiveness and impulsiveness on the ability to delay gratification in children of all groups. To increase the generalizability of findings, we examined a food-specific DOG in a sample of children from the general population and not in the obese or clinical population, as has often been done in earlier research (Nederkoorn et al., 2006).

Study 2 aimed to clarify the relationships between aspects of family emotionality and disordered eating behaviours in children. We chose to focus on emotional eating, which is defined as an individual's tendency to eat in response to negative emotional states (Evers,

Stok, & de Ridder, 2010), because it represents a clinically relevant eating behaviour in young children that commonly co-occurs with and precedes formal eating disorders, especially BED (Ricca et al., 2012; Stice, Presnell, et al., 2002; Wildermuth, Mesman, & Ward, 2013). To increase measurement reliability, frequencies of emotional eating behaviours in children were assessed based on children's and parents' self-reports. Moreover, we aimed to simultaneously study the influences due to parenting practices and to parental expressed emotion when accounting for emotional eating in children. To specify the unique contributions of the parental predictors, we controlled for important confounding variables e.g., parental psychopathology or BMI of the child and participating parent. An additional aim of this study was to explore the possible indirect impact of parental factors transmitted through a child's disposition to act out intense affects (urgency). Unlike previous research that has mostly studied urgency in relation to negative affect, this study aimed to consider intense positive and negative mood states as part of the broad construct of mood-based rash action (Cyders et al., 2007). Urgency was renamed *affective impulsiveness*, as this reflects the underlying processes of emotion-driven impulsivity. Finally, to assure sufficient variability in the criterion variable, the study sample was composed of parent-child dyads including healthy children but also children with a diagnosis of ADHD or BED who are known to be characterized by increased impulsivity and to be at risk for emotional eating (Stice, Presnell, et al., 2002; Farrow, 2012).

Study 3 aimed to provide initial data on the occurrence of avoidant or restrictive eating behaviours that are characteristic of ARFID disturbance in the general population of middle childhood. To increase the representativeness of the sample, we recruited children aged 8-13 years from different regular schools of various socio-economic backgrounds and geographical regions of Switzerland (cantons of Fribourg, Lausanne and Bern). We also aimed to investigate the psychometric properties of the self-developed Eating Disturbances in Childhood – Questionnaire (EDCh-Q), which represents a brief screening tool for ARFID symptoms based on self-report.

# 5. Methods

## 5.1. General Procedure

The data collection of the present thesis was part of the Swiss University Study in Nutrition (SUN, Hilbert & Munsch, 2010) supported by the Swiss National Science Foundation (SNSF) (grant number 100014132045/1). The studies were conducted at the Department of Clinical Psychology and Psychotherapy of the University of Fribourg and at the Department of Clinical Psychology of Children and Adolescents at the University of Lausanne. The study languages were German and French. Ethical approval for the SUN project was granted through the Ethics Committees of the Canton of Fribourg and the Universities of Fribourg and Lausanne.

### *5.1.1. Recruitment*

The recruitment of 8- to 13-year old children with and without BED and with ADHD occurred in regular schools (3rd to 6th grades) from different linguistic and geographical regions of Switzerland (cantons of Fribourg, Lausanne and Bern). Children from all socioeconomic backgrounds in regular schools were included in the study after having obtained informed consent from the cantonal board of education and the school board. Parents were asked to give permission for children to participate in a school screening conducted during school hours. The children completed several self-report questionnaires, including the Conner's ADHD Index Form (Conners 3AI, Conners, 2008; German version, Lidzba, in preparation; French version, Dremmel, De Albuquerque, & Munsch, in preparation), the EDCh-Q (van Dyck et al., 2013) and selected items of the Eating Disorder Examination-Questionnaire adapted for children (ChEDE-Q, TODAY Study Group, 2007; German version, Hilbert, Hartmann, & Czaja, 2008; French version, Dremmel, De Albuquerque, & Munsch, in preparation). The French versions were established using forward- and back-translations. In the event of positive scores on the item of the ChEDE-Q assessing LOC eating and on one of the core diagnostic items of the Conner's self-report scale, a telephone screening was conducted with the child and the parent to determine eligibility. Eligible children and their parents interested in participating in the study were invited to attend a diagnostic session. After obtaining the informed consent and approval of the parent and child on all study procedures, the clinical interviews Child Eating Disorder Examination (ChEDE, Bryant-Waugh, Cooper, Taylor, & Lask, 1996; German version, Hilbert, Buerger, Hartmann, Spenner, Czaja, & Warschburger, 2013; French version, Dremmel & Munsch, in preparation)

and Schedule for Affective Disorders and Schizophrenia for school-aged children (K-SADS, Kaufman et al., 1997; German version, Delmo, Weiffenbach, Gabriel, Stadler, & Poustka, 2001; French version, Rothen et al., 2009) were used to ascertain diagnostic status.

### *5.1.2. Inclusion and exclusion criteria*

We used the child-adapted diagnostic criteria to establish the diagnosis of BED (Hilbert & Czaja, 2009; Marcus & Kalarchian, 2003; Tanofsky-Kraff et al., 2008). Accordingly, the inclusion criteria for BED were at least one episode of LOC eating per month during the previous 3 months, having at least some degree of distress associated with the LOC episodes, meeting at least two or more of the five behavioural symptoms and exhibiting the absence of regular inappropriate compensatory behaviours (e.g., purging, fasting, and excessive exercise).

For the ADHD group, the children were required to fulfil the DSM-IV-TR (APA, 2000) criteria for ADHD. Children with the inattentive, hyperactive/impulsive or combined subtypes of ADHD were included in the study.

The inclusion criteria for children in the control group were the absence of past or present LOC eating, compensatory behaviours, an eating disorder and absence of more than three symptoms of hyperactivity/impulsivity and inattention according to the diagnostic interview to assess mental disorders (K-SADS, Delmo et al., 2001; Kaufman et al., 1997; Rothen et al., 2009). The control group was matched to the BED group based on age, gender and BMI.

Additional inclusion criteria for all of the groups were age 8-13 years and sufficient German or French language skills of the child and participating parent. The exclusion criteria were compensatory behaviours (> 1 episode/last three months), psychotic disorders in the child or parent, medical conditions or medication with an effect on eating behaviour or body weight, treatment for being overweight and special education. Finally, all of the children with co-morbid BED and ADHD were excluded from the BED and ADHD groups, although they were separately assessed.

## 5.2. Participants

The three studies in this thesis were conducted at different time points in the overall project, whereby they included various samples. The participants in Study 1 and Study 2 were recruited based on the aforementioned diagnostic procedure including three steps (school screenings, telephone screenings, and diagnostic interviews) for defining the groups (BED,

ADHD, BED&ADHD, control group). In contrast, in Study 3, only school screening data were considered. Therefore, the sample included all children who were not further screened with regard to eventual psychopathology. This procedure resulted in a total sample of 730 participants. The participant flow charts and final sample sizes of Study 1 and Study 2 are displayed in Figure 2. A detailed description of the sample characteristics is provided in the corresponding publications (see Appendix).

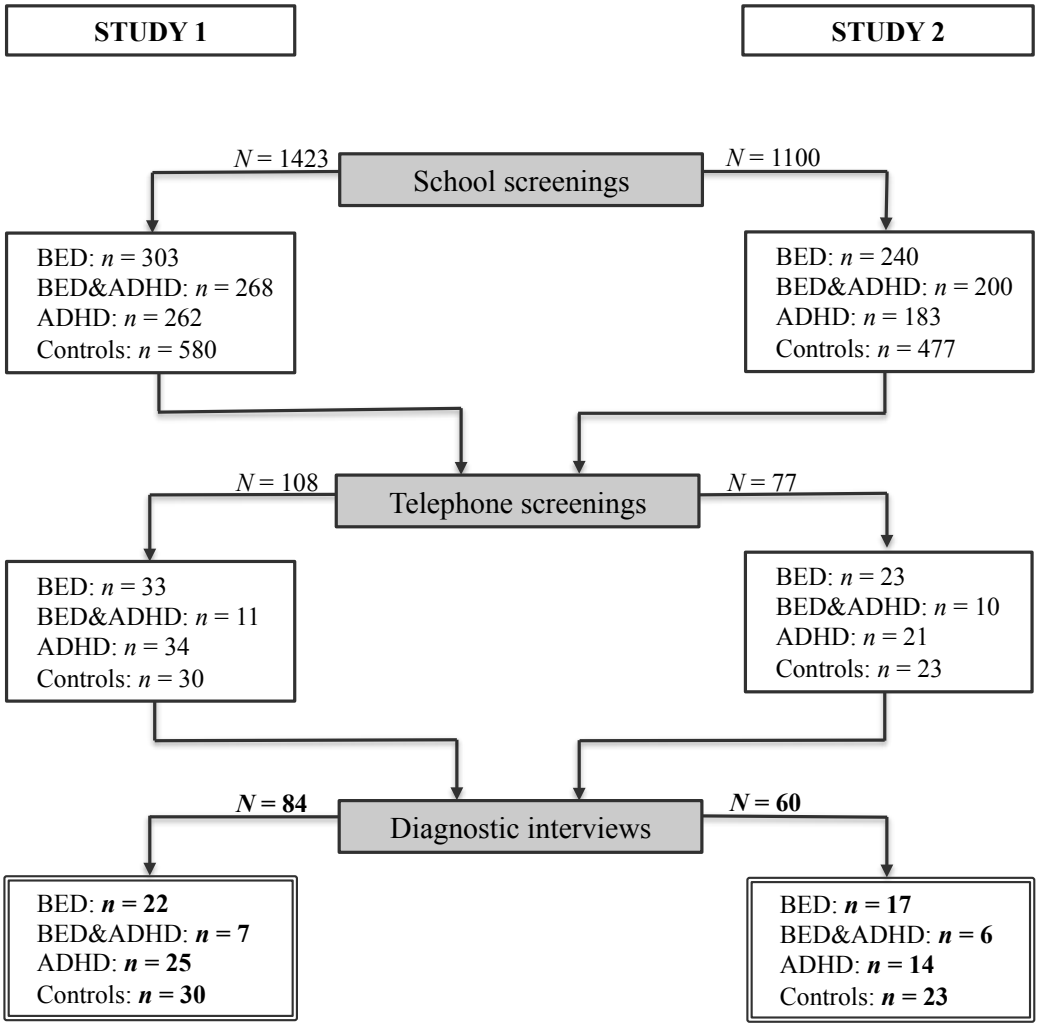


*Figure 2*. Flow of participants for Study 1 and Study 2.

## 5.3. Measures

We used a variety of measures to assess problematic eating behaviours and associated maintenance factors in children. These measures will be presented separately for each study. Study 1 and Study 2 additionally integrated the laboratory measures of children's and parents'

weight and height using a standardized balance and a stadiometer. The body mass index (BMI) was computed using the standard formula (weight in kg/height in metres$^2$).

### *5.3.1. Study 1*

Study 1 used a multi-method approach combining laboratory-based behavioural data with self-report data. The behavioural measures are considered to be valuable assessment tools for children because they are less susceptible to be influenced by factors that might bias a respondent's report (e.g., psychopathology or limited abstract reasoning) (Barkley, 1991; DuPaul, Anastopoulos, Shelton, Guevremont, & Metevia, 1992). Furthermore, the standardized laboratory measures of a child's behaviour have been found to be highly sensitive to early psychopathology (Marakovitz & Campbell, 1998; Barkley, 1991).

*Food-specific reward sensitivity* was assessed using a standardised behavioural observation procedure based on a slightly modified version of the DOG paradigm developed by Mischel et al. (1989). The DOG task is a widely accepted ecologically valid experiment for depicting an individual's ability to postpone immediate gratification (Gledhill & Petermann, 2013). Usually, a plate of treats is presented to the child, who is told that he or she will receive additional treats provided he or she waits until the researcher returns (e.g., two cookies or two marshmallows instead of one). In our study, this experiment was adapted in the following manner. First, we adjusted the type and quantity of food rewards according to a child's eating preferences. By means of an odour test conducted beforehand, the children indicated their preferred food among a range of common snacks (chocolate cookies, paprika chips and sweet-sour gummy candies) and how many packages (1, 2 or 3) they would like to eat if they were free to choose. Second, the DOG task was staged to a natural situation in that the children knew from the start that they would earn a reward after completing all of the studies. In the final DOG situation, the child then received 1 package of the preferred snack reduced to one-fifth of the total amount, thereby being told to receive more of that food (1, 2 or 3 entire packages depending on the answer in the odour test) if he or she postponed eating until the experimenter returned. The experimenter told the child that he or she needed to handle an urgent telephone call first and then would return with the promised food package/s. The experimenter stayed away for 15 minutes, and the children's delaying attempts were video-taped. At the end of the 15-minute wait, either the smaller quantity or the full quantity of the preferred food was given to the child. We assessed whether the children ate immediately or postponed eating.

*Children's self-reported experience during the DOG task* was captured using a self-developed self-report questionnaire consisting of 25 items. The children's perceived difficulty while waiting (e.g., "How long did you feel you had to wait?" Responses ranged from very short to very long) and their fears of losing control (e.g., "Did you fear you would lose control over eating while waiting?" 1 = not at all to 7 = very much) were considered.

*Current emotional state* of children was assessed at the beginning of the experiment and after the DOG task. The children's negative emotional state was assessed using the following items: "Do you feel … sad, embarrassed, anxious, guilty or angry?" The responses were rated using a 7-point scale (0, not at all, to 6, very much). As an indicator for each child's current negative emotional state, we documented the maximum value given in any of the five items. The children's mood was assessed using the following item: "Do you feel well?" (0, not at all, to 6, very much). To make the mood item compatible with the assessment of the negative emotional state, we reversed the coding of the item (0, very well, to 6, not well at all).

*Trait depressiveness* was assessed using the German Children's Depression Inventory (DIKJ 2nd revised edition, Stiensmeier-Pelster, Schürmann, & Duda 2000; French version, own translation). The DIKJ captures the presence and the severity of depressive symptoms in 8- to 17-year-old children and adolescents using 26 items (e.g., "I am sad all the time") on a three-point scale (0, not present; 1, moderately present; 2, strongly present). Stiensmeier-Pelster et al. reported Cronbach's alphas between .74 and .88. For the present sample, α was .87.

*Trait impulsivity* was measured using the child-adolescent version of the Barratt Impulsiveness Scale (BIS-11, German version, Hartmann, Rief, & Hilbert, 2011; French version, Michel & Coudret, in preparation). The BIS-11, which is a self-report instrument, consists of 30 items (e.g., "I do things without thinking") rated on a 4-point scale (1, rarely/never, to 4, almost always), including the factors attentional-, non-planning-, and motor impulsivity. Stanford et al. (2009) reported satisfying Cronbach's alphas for the attentional- and non-planning impulsivity (.74, and .72) but a low alpha for motor impulsivity (.59). Relying on the item composition of the English BIS-11 (Stanford et al., 2009), we obtained low alphas of .55, .65, and .58, respectively. The reliability of the total impulsivity was more satisfying; the alpha of the total score was .72.

#### *5.3.2. Study 2*

We used questionnaire-based assessments to investigate the influence of parenting practices and parental expressed emotion on the occurrence of emotional eating in children. To increase

the reliability of measures, we evaluated the children's emotional eating behaviour based on the child's self-report and parents' reports. We also introduced measures on potential confounding variables, e.g., parental psychopathology and parents' own emotional eating behaviours.

*Children's self-reported emotional eating behaviour.* Children completed the Emotional Eating subscale of the Dutch Eating Behaviour Questionnaire (DEBQ) for 7- to 13-year-old children (German version, Franzen & Florin, 1997; French version, own translation). This subscales consists of 10 items assessing the tendency to eat in response to negative emotions on a rating scale ranging from 1 = never to 4 = always (e.g., "I wish to eat when depressed or discouraged"). Grunert (1989) and Franzen and Florin (1997) reported Cronbach's α's between .84 and .94, which are similar to those obtained in the present sample (α French-language Sample (FS) = .84; α German-language Sample (GS) = .95).

*Children's self-reported affective impulsiveness.* A child-adapted version of the UPPS (Whiteside & Lynam, 2001) was developed to assess the tendency to rush into action when experiencing strong negative or positive effects, without considering negative consequences of this action (e.g., "When I am very angry or very sad, I do things without thinking that I might regret afterwards") (Zecca et al., in preparation; German version, own translation). The affective impulsiveness subscale includes 27 items combining the negative urgency scale with the highly correlated positive urgency scale. They were rated from 1 = "totally false" to 4 = "totally true." The internal consistency of this scale was high (α FS = .91, α GS = .94).

*Parent's report of their children's emotional overeating.* Parents completed the emotional overeating (EOE) subscale of the Children's Eating Behaviour Questionnaire (CEBQ) (Wardle, Guthrie, Sanderson, & Rapoport, 2001; German and French versions, own translation). The EOE scale consists of four items that describe the tendency to eat more when being in a negative mood (e.g., "My child eats more when worried."). The items are rated from 1 = never to 5 = always. The original scale has a Cronbach's α between .74 and .91 and reasonable test-retest reliability (Wardle et al., 2001). For the present sample, Cronbach's α was high (α FS = .90; α GS = .88).

*Parent's self-reported emotional eating.* Parental emotional eating was assessed using the 10-items Emotional Eating subscale of the German adult-version of the Dutch Eating Behavior Questionnaire (DEBQ, van Strien, Frijters, Bergers, & Defares, 1986; German version FEV II: Grunert, 1989; French version, own translation). Cronbach's α was high for both the original and German version and was high in the present study (α FS = .95, α GS = .95).

*Parenting practices.* Parents completed the Alabama Parenting Questionnaire (Frick, 1991; German version, DEAPQ-EL-GS, Reichle & Franiek, 2009; French version, own translation). The extended German version contains descriptions of 40 different parenting practices rated from 1 = almost never to 5 = almost always. The German version additionally assesses the dimensions of responsible and authoritarian parenting. Functional parenting practices are assessed using the following: Involvement (e.g., "You ask your child about his/her day in school"; α DEAPQ > .60, α FS = .55, α GS = .46)[1], positive parenting behaviour (α DEAPQ = .84, α FS = .83, α GS = .72), and responsible parenting (α DEAPQ > .62; α FS = .33, α GS = .71). The other subscales assess dysfunctional parenting practices, such as the following: Inconsistent discipline (e.g., "It is difficult for you to be consistent in your education"; α DEAPQ = .72, α FS = .70, α GS = .60), poor monitoring (α DEAPQ > .65, α FS = .54, α GS = .25), corporal punishment (α DEAPQ > .55, α FS = .61, α GS = .63), and authoritarian parenting (α DEAPQ = .71, α FS = .74, α GS = .61).

*Expressed emotion (criticism and emotional overinvolvement).* Parental EE was measured using the Family Questionnaire (FQ) (Wiedemann, Rayki, Feinstein, & Hahlweg, 2002; French version, own translation). The FQ assesses attitudes and behavioural patterns of relatives towards the patients (or family members) using two subscales. Each subscale consists of 10 items rated from 1 = never/very rarely to 4 = very often. The subscale criticism (CC) includes critical attitudes and critical comments towards the child (e.g., "I have to try not to criticize him or her"). Wiedemann et al. (2002) reported Cronbach's α for CC to be .90 and .92. In the current sample, α was slightly lower (α FS = .89, α GS = .86). The subscale emotional overinvolvement (EOI) includes overconcern, overidentification and overprotectiveness (e.g., "I'm very worried about him or her"). Wiedemann et al. (2002) reported Cronbach's α between .79 and .82. In the current sample, α was .81 (FS) and .60 (GS).

*Global Severity Index (GSI).* The parents' general psychopathology was assessed using the Brief Symptom Inventory (BSI-53). Franke (2000) introduced a German version of the BSI-53, and Dreyfus and Guelfi (unpublished) used a French version. The BSI includes 53 items of clinically relevant psychological symptoms (e.g., "feeling easily annoyed or irritated") that are rated from 0 = not at all to 4 = very strong. Across the 53 items, the Global Severity Index

[1] Low alphas are likely due to skewed distributions of items and particularities of the rather small samples. In the original English version, low alphas (< .60) were reported for poor monitoring and corporal punishment (Dadds, Maujean, & Fraser, 2003).

(GSI) is computed as the average severity of symptoms experienced during the last seven days. The GSI has a high internal consistency (Cronbach's $\alpha > .90$) and stability (Derogatis, 1993; Franke, 2000) (in the present study, $\alpha$ FS = .87, GS = .97).

#### *5.3.3. Study 3*

For the purpose of Study 3, we developed a brief self-report screening questionnaire for assessing avoidant or restrictive food intake (Eating Disturbances in Childhood-Questionnaire (EDCh-Q) (van Dyck et al., 2013). The EDCh-Q assesses attitudes and behaviours that are characteristic of ARFID disturbance. The items were developed based on the proposed DSM-5 diagnostic criteria for ARFID, as well as the descriptions of problematic eating behaviours in children from the research literature and the GOS criteria (Watkins & Lask, 2009). The development of the questionnaire was based on a literature review and on an exchange with experts in the field of childhood eating disorders. The French formulation of items in the EDCh-Q was obtained by forward-translation and back-translation methods.

The scale consists of 14 items, while the last two questions concerning Pica and rumination disorder were not considered for this study. The remaining 12 items covered the three subtypes of restrictive or avoidant eating disturbances. Indeed, children are classified as selective eaters based on the items "I am a picky eater" and "I do not like to try food with a specific smell, taste, appearance, or a certain consistency (e.g., crispy or soft)." Avoidant eating behaviour due to lack of interest in food/emotional problems is assessed using the items "I do not eat when I'm sad, worried or anxious" and "Food does not interest me." Finally, the statements "I am afraid to swallow food" and "I'm afraid to choke or vomit while eating" classify fear-based food avoidance, such as functional dysphagia. Two additional items have been introduced to evaluate weight- or shape-related concerns, which represent an important exclusion criterion for the ARFID diagnosis (APA, 2013; Nicholls, Chater, & Lask, 2000). Each item is assessed using a seven-point Likert-scale ranging from "never true" (= 0) to "always true" (= 6).

### 5.4. Statistical Analysis

#### *5.4.1. Study 1*

We conducted a series of logistic regression (LR) and multiple linear regression (MLR) models to analyse the children's eating behaviours and self-reported experiences during the DOG task. Three criterion variables were used in the present study: children's self-reported

fears of losing control (strongly skewed) and children's eating behaviours were dichotomised and were analysed using LR (eating = 1 vs. not eating = 0; worries = 1 vs. no worries = 0), whereas self-reported difficulty while waiting was normally distributed and analysed using MLR. Each criterion variable was regressed on the same set of predictor variables. First, we entered the control variables: age (centred on approximately 11 years) and gender (male = 0) in the first step (model 1) and children's BMI (centred on approximately 20) in the second step (model 2). In the third step, we tested whether children with a mental disorder differed from healthy children by entering a dummy variable that was coded 0 for patients and 1 for healthy controls (model 3). In the fourth step, we added a dummy variable for children with ADHD and another for children with BED, to test whether children with a single diagnosis differed from children with a dual diagnosis (model 4). To show whether the diagnostic groups differed from healthy controls, we recalculated model 4 using the three diagnostic groups (BED, ADHD, and BED&ADHD) as the set of predictors (model 4r). Finally, we tested whether entering the different facets of impulsivity, depressive symptoms, or the emotional state before the DOG-task have an impact on children's behaviour and subjective experience during the DOG task, when age, gender, BMI, and type of disorder were already controlled (model 5). The continuous predictor variables were centred on the group mean.

In the Results section of Publication 1 (see Appendix), we report and interpret the change in $R^2$ for MLR and the change in Nagelkerke's $R^2$ ($R_N^2$) for LR as an estimate of the variance in the criterion variable that is explained by the predictor(s) added to the model at each step of the analysis. The corresponding *F*- or $\chi^2$ test indicates whether the additional variance explained is beyond chance. Power was sufficient (alpha = .05; power = .80) to detect a moderate increase in variance ($f^2$ = .096; $eta^2$ = .087, when one predictor was added, $f^2$ = .119; $eta^2$ = .106, when a set of two predictors was added). To test whether a single coefficient was different from zero, we used the corresponding *z*-test (alpha = .05). We used the one-tailed (o.t.) test to determine the coefficients of variables for which we had hypotheses.

### *5.4.2. Study 2*

In the first step, we used Pearson product moment correlations and Spearman rank correlations (when the variables were severely skewed) to investigate how parental variables (subcategories of EE and parenting practices) relate to children's affective impulsiveness and their eating behaviours. To obtain more stringent tests and to control for potential confounding variables, we computed multiple regression analyses for ordinal data in the second step. We used children's and parents' reports of children's emotional eating and

affective impulsiveness as criterion variables. Both measures of children's emotional eating were highly skewed (the minimum was the modal value). Therefore, the negative log-log link function was applied. Affective impulsiveness was less skewed and, thus, the logit link function was used. To obtain more robust estimates, we narrowed the range of the criterion variables by transforming the original values into an equidistant five-step scale. The predictor variables were centred on the group mean.

We considered gender, age, children's age-adjusted BMI, parents' BMI, parents' own emotional eating, and parents' general psychopathology as potential confounding variables and explored their unique effects on each of the three criterion variables (children's reported emotional eating, parents' reports of children's emotional eating, and affective impulsiveness). Except for gender, age, and parents' BMI, for which we did not have a prediction, we expected the potential effects of these variables on the criterion variables to be positive.

In the first step of the model building (model 1), every potential confounding variable that was at least marginally significant ($p < .10$, according to the $\chi^2$ test) was kept as a control variable. In the second step (model 2), we tested whether parenting practices, criticism and emotional overinvolvement still influenced emotional eating or affective impulsiveness when the control variables were already in the equation. In a final step, we added affective impulsiveness as a predictor of emotional eating (model 3). Comparing the results obtained in models 2 and 3 allowed us to explore whether affective impulsiveness transmits the effects of parenting practices, criticism, and emotional overinvolvement on emotional eating. When a predictor variable has a significant effect on children's emotional eating in model 2 that does not decrease substantially after affective impulsiveness has been added to the equation in model 3, the effect of this predictor variable is then demonstrated to be independent of the children's affective impulsiveness (and vice versa). However, when the predictor variable is related to affective impulsivity and its effect on emotional eating substantially decreases after affective impulsiveness has been added to the equation in model 3, and affective impulsiveness also has a significant effect on emotional eating, then the effect of this predictor variable is transmitted by affective impulsiveness.[2]

[2] If the relationships between variables are causally directed (predictor causes affective impulsiveness, which in turn causes emotional eating), then all of the necessary conditions will be satisfied to conclude that affective impulsiveness mediates the effect of the predictor variable on emotional eating (Baron & Kenny, 1986). Because

In the Results section of Publication 2 (see Appendix), we report and interpret Nagelkerke's $R^2$ ($R_N^2$) as an estimate of the variation in the criterion variable that is explained by the predictors in the model, together with the related $\chi^2$, which tests whether the variation explained by the model is indeed larger than zero. Because our sample size was small, many zero cells occurred. Additionally, the number of zero cells varied according to the combination of predictor variables. Consequently, the difference in the deviances between two nested models was not reliable and could not be used to test whether adding a new predictor substantially improves the model fit. We, therefore, report only the change in $R_N^2$ between two models to describe the impact of a newly added predictor.

With the program G*Power 3.1.7 (Faul, Erdfelder, Buchner, & Lang, 2009), we estimated the size of the correlation that could be detected with the current sample size of 60 dyads (children and parents). Fixing alpha to .05, two-tailed, the power was sufficient (.80) to detect medium to large effects ($\rho$ = 0.35). Regarding those variables for which we had hypotheses and expectations, we used the one-tailed test and achieved a power of .80 to detect medium effects ($\rho$ = 0.31). The power calculations for ordinal regression analyses are not possible in G*Power. We therefore calculated the multiple regression analyses as an approximation to the size of effects we could discover using the ordinal multiple regression analyses that we actually computed. Fixing alpha to .05 and assuming a sufficient power (.80), an increase in variance corresponding to nearly a medium effect size ($f^2$ = .14) could be discovered.

#### *5.4.3. Study 3*

To describe the frequency of the three-symptom profiles of ARFID, children with and without disordered eating were separated from each other. This approach was chosen based on previous studies that primarily focused on selective eating (Carruth, Ziegler, Gordon, & Barr, 2004; Jacobi, Schmitz, & Agras, 2008; Mascola, Bryson, & Agras, 2010). To meet the criteria of one of the three subtypes of avoiding/restrictive eating behaviours, the child had to report at least "often" the corresponding eating behaviour, which corresponds to a cut-off score of ≥ 4. Accordingly, the items were dichotomised by converting the categories "never true" to "sometimes true" into "no" and "often true" to "always true" into "yes." Unlike Carruth and colleagues (2004) and Jacobi and colleagues (2008), the value "sometimes true" has not been interpreted as a presence of problematic eating behaviours because self-report often

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we do not have sufficient evidence for the causal direction of the effects, we avoid the term "mediation" and use the term "transmission."

overestimates the psychopathology (Fairburn & Beglin, 1994). An important exclusion criterion for ARFID is the presence of weight or shape concerns, which is why only children obtaining a value of < 3 for this item ("sometimes true") using a 7-point Likert scale were included in the risk group of avoidant/restrictive eating disorders. Based on chi-squared tests of independence, it was determined whether the three subtypes differ significantly from each other in terms of gender, age, or BMI category. Because of a frequency value of < 5 obtained for overweight children regarding two subtypes, additional chi-squared tests of independence were performed (underweight, normal weight/overweight). All of the psychometric analyses were performed for the total sample as well as separately for both languages, thereby fixing alpha to .05, two-tailed.

Moreover, the factor structure of the self-developed questionnaire EDCh-Q was analysed using a principal components analysis with orthogonal varimax rotation. The extraction of factors was based on the eigenvalues considering the Kaiser-Guttmann criterion and the scree-test. The psychometric analysis addressed missing values, item difficulty index [$p_m$ = item-total/(N * maximum value of item)], adjusted item discrimination (corrected correlation between the single item and the scale) and homogeneity of subscales (average correlation between the items). Using the Kolmogorov-Smirnov test, we examined whether the distribution of items corresponded to a normal distribution. We determined the internal consistencies of scales that were identified using the principal component analysis and Cronbach's α.

# 6. Summary and Discussion of Results

## 6.1. Results of Study 1

The results indicated that neither mental health status, age, impulsiveness, nor depressiveness or affective state before the task were related to the children's ability to resist an immediate edible reward during the DOG task. Instead, the only important variable, accounting for 8% of the variance regarding the likelihood to eat immediately, was the children's BMI. A higher BMI was associated with a higher likelihood to prematurely eat during the task, which resulted in an increased likelihood to eat of more than .60 in obese children compared to normal-weight peers. In contrast, based on self-reported measurements of the children's experience during the DOG task, the difficulty while waiting or fear of losing control did not depend on BMI but was most prominent in children who suffered from a mental disorder. Although perceived difficulty during the wait period was explained by the presence of any mental disorder, the likelihood of experiencing fears of losing control was higher in children with a dual diagnosis (BED&ADHD) or a diagnosis of BED than in children with ADHD or in healthy controls.

Our results are consistent with data relating overweight to difficulties in postponing food rewards (Francis & Susman, 2009; Nederkoorn et al., 2006). However, to date, this study is the first to show that on a behavioural level, such as during the DOG task, "eating or waiting" in 8- to 13-year-old children is associated more with BMI than mental disorders, such as BED or ADHD, impulsivity, state or trait mood or age and gender. The exception was that, consistent with our expectations, children with a dual diagnosis of BED&ADHD compared to children suffering from BED only were more likely to immediately eat during the DOG task, which resulted in an increased likelihood to eat of almost .50. However, no effect was found for the presence of a mixed diagnosis in the prediction of children's DOG behaviour when the control group constituted the group of reference, perhaps because of the small sample size of the mixed group ($n$ = 7). Of these 7 children, 4 did not wait for the larger reward (see table 1 in Publication 1). Given that ADHD could exacerbate eating pathology and thus increase the difficulty to inhibit strong urges in children with BED (Cortese, Bernardina, & Mouren, 2007; Steadman & Knouse, 2014), additional research on this aspect in middle-aged children is warranted.

Our findings contrast with the data of Nederkoorn et al. (2006), who found that obese adolescents were significantly more sensitive to reward (gambled longer in the computerised

door opening task) if they presented with additional binge eating. However, the children in our sample were younger and significantly lower in weight compared to those studied by Nederkoorn et al., who all followed a treatment program due to severe degrees of overweight. Consequently, it may be assumed that the difficulty to delay immediate food-related rewards is primarily associated with increased levels of obesity, which often develop later as a consequence of binge eating (Tanofsky-Kraff et al., 2006).

Altogether, BED children in our study were characterised by an increased sensitivity towards own self-control difficulties and simultaneous high self-control if confronted with food in a laboratory situation. This situation may represent a specificity of the BED diagnosis that also reflects its mental illness status compared to obesity. In fact, our findings indicated that BMI, although related to eating prematurely in the laboratory, did not imply a subjective component of perceived self-control difficulties during the experimental task. Instead, increasing BMI levels appeared to be related to reduced self-awareness regarding food intake in our study.

The finding that fears of losing control evoked by food exposure distinguished children with BED from healthy children and children with ADHD moreover underlines the diagnostic relevance of eating- or food-related concerns for BED such as for eating disorders in general (Stice et al., 2011). Dysfunctional thoughts regarding food/eating are assumed to contribute to the maintenance of problematic eating behaviours in BED, such as binge eating or dietary restraint eating (Hilbert, Hartmann, et al., 2013; Hilbert, Rief, et al., 2009), and could also have accounted for more controlled reactions in situations where children feel observed, such as in the laboratory. The risk to act according to social desirability could be elevated in children with BED (Goldschmidt et al., 2011), who tend to (binge) eat in secret and experience increased shame/guilt when they eat in public compared to typically developing children (Tanofsky-Kraff et al., 2007). Future studies on the role of reward sensitivity in BED may therefore benefit from adding other forms of methodology, e.g., measures of children's psychophysiological activation during the waiting period or ecological momentary assessment methods.

### 6.2. Results of Study 2

When confounding variables such as parents' own emotional eating, parents' general psychopathology, or children's BMI were controlled for in regression analyses, corporal punishment was solely associated with a child's emotional eating based on both parents' and children's assessments. Although the effect was small (amount of variance: child's report,

4.2%; and parents' report, 2.9%), it remained significant when the children's affective impulsiveness was controlled. An authoritarian parenting practice and the EE subdimension criticism were also associated with children's emotional eating if confounding variables were controlled and accounted for 4.2% and 5.2%, respectively. The effect of criticism was transmitted by affective impulsiveness and was only related to emotional eating when assessed by the children themselves, whereas authoritarian parenting was only associated with emotional eating of the child when assessed by the child's parent.

Although we observed small effects, these findings are consistent with the results indicating an increased likelihood for obesity (Afifi, Mota, MacMillan, & Sareen, 2013) or bulimia nervosa (Rorty, Yager, & Rossotto, 1995) in adults retrospectively reporting corporal punishment in childhood. The finding that parents reporting an authoritarian parenting practice also reported increased levels of emotional eating in their children has also been shown in the work of Snoek et al. (2007). However, there is one contrasting result based on a cross-sectional study of Topham et al. (2011) that did not confirm an effect of corporal punishment on the occurrence of emotional eating in 6- to 8-year-old children from the general population. The divergent results might be due to the older age of our sample, the different measurement methods or the fact that behavioural and emotional consequences of corporal punishment become increasingly harmful with increasing age (Frick, Christian, & Wootton, 1999; Larzelere, 2000). Additionally, we investigated a smaller but mixed sample consisting of healthy children, as well as children suffering from BED, ADHD or both, where dysfunctional parenting practices are likely to be more pronounced than in a healthy population of, for example, Topham and colleagues.

To the best of our knowledge, this study is the first to show that in accordance with the tripartite model of familial influence (Morris et al., 2007), repeated criticism towards the child as a correlate of the parent's own emotional dysfunction is associated with the child's disposition to act rashly when emotionally aroused. In turn, affectively impulsive children had a high likelihood of engaging in emotional eating according to their self-reports, which is consistent with previous research that has demonstrated negative urgency as a longitudinal predictor of the onset of binge eating in children (Pearson et al., 2012; Pearson et al., 2015). However, in our study, affective impulsiveness was considered to be a tendency toward impulsive action relevant to intense positive and negative emotions. Future research on emotion-based impulsivity might benefit from specifying effects of singular positive and negative emotions in relation to the risk for uncontrolled eating behaviours. There are some

indications that LOC eating in children does not only occur under negative but also positive affective states (Tanofsky-Kraff et al., 2007).

Another important outcome is the divergent pattern of results depending on whether a child's self-report or the parents' rating perspective was considered. As in other studies (Braet et al., 2007; De Los Reyes & Kazdin, 2005), self-report and parental reports of the children's emotional eating were only moderately convergent. The parents' reports were highly influenced by their own level of emotional eating, their general psychopathology and the child's BMI, which accounted for more than half (54%) of the variance in their reports of children's emotional eating. In other words, the parents' ratings with respect to their children's emotional eating behaviour appeared to be guided, for the most part, by external factors, such as their own psychological health and eating problems as well as by their child's BMI. In turn, children's self-reports of emotional eating were unrelated to BMI and only marginally influenced by their parents' own tendency of emotional eating, but strongly accounted for by the internal experience of affective impulsiveness.

The interference of perceiver biases in parents due to psychopathology or a child's overweight is a well-known phenomenon in the assessment of behaviour in children (Barkley et al., 1991; Braet, Mervielde, & Vandereycken, 1997; DuPaul, et al., 1992). It has been assumed plausible that the presence of mental health symptoms in parents complicates a correct perception of children's inner feeling states. In this regard, a disconcerting result of our study is that parents with increased levels of psychopathology were likely to emphasise frequencies of emotional eating in their children, which in itself was related to maladaptive parenting behaviours.

Concerning children's weight-status, biases can likely affect both parents' and children's rating perspectives (Braet et al., 2007; Shomaker et al., 2010). On the one hand, it is plausible that parents may be better observers of eating in the absence of hunger than overweight children who rather underestimate their problematic eating behaviour. On the other hand, parents might likely tend to judge more severely the aberrant character of eating behaviours in children who already have weight problems (Braet et al., 2007; Hill, Draper, & Stack, 1994). Future longitudinal studies should therefore examine whether the increase in parenting control and punitive behaviours undermines a child's efforts to self-regulate eating behaviour or whether parental behaviour is influenced by a child's repeated problematic eating behaviour.

## 6.3. Results of Study 3

The results of Study 3 indicated that a significant part of community children (29.8%) are concerned with regard to avoidant or restrictive eating behaviour. The symptoms of selective eating (SE) were most frequently reported (20.3%), followed by food avoidance emotional disorder (FAED, 7.9%), whereas the symptoms of functional dysphagia (FD, 1.6%) were rare. Consistent with other studies (Jacobi et al., 2008; Mascola et al., 2010), the present study showed that selective eating, which frequently concerns young children, represents a common phenomenon in older children when assessed by self-report. For example, in the sample of Jacobi and colleagues (2008) including children of a similar age group, 18% of the boys and 19% of the girls were described as picky eaters by their parents. Similar prevalence rates have been reported in a study of 11-year-old children (Mascola et al., 2010). Together, these findings suggest that SE is not a temporary phase of development in many children. No data exist concerning the prevalence of FD and FAED in samples of the normal population. Compared to studies including clinical samples, symptoms of FAED were less common in the present study (Cooper, Watkins, Bryant-Waugh, & Lask, 2002; Nicholls et al., 2011), which can be explained by the seriousness of the later symptoms implying frequent co-morbid physical illness (Bryant-Waugh & Lask, 2013; Nicholls & Bryant-Waugh, 2008). Due to the significant burdens associated with FAED, concerned individuals more frequently seek professional help than is, for example, the case for individuals with SE, which explains its increased representation in clinical samples. In contrast, the risk of FD is also rarely observed in clinical samples (Cooper et al., 2002).

The EDCh-Q was not developed for diagnosing eating disorders but rather for identifying symptoms of problematic eating behaviour, which is why it can be assumed that the reported frequencies based on self-report are higher than those established on the basis of diagnostic interviews (Decaluwé & Braet, 2004; Fairburn & Beglin, 1994). Additional epidemiological studies, using a two-stage screening approach in accordance with structured diagnostic interviews, are required to establish the prevalence of restrictive/avoidant eating disorders in middle childhood.

Finally, no age differences were detected for any of the ARFID subtypes in our study. Consistently, Jacobi and colleagues found that SE occurs equally frequently in children aged 8 years to 12 years as in children of younger ages (Jacobi et al., 2008). Consistent with other studies on disordered eating behaviours in children (Kröller & Warschburger, 2011), these findings suggest that abnormal eating patterns are not uncommon in school-aged children and

often remain stable across age. We could not find any significant differences between girls and boys, which supports the assumptions from various studies that the gender ratio of eating disturbances in childhood is more balanced than in adulthood eating disorders (Bryant-Waugh & Lask, 1995; Jacobi et al., 2008; Lask & Bryant-Waugh, 1992; Peebles et al., 2006; Rosen, 2003). Overall, the findings revealed a strong link between self-reported underweight and the presence of FAED: Children with a low BMI were more likely to report symptoms of FAED. Consistent with this result, clinical practice has reported degrees of malnutrition and weight loss for individuals with FAED that are comparable to those found in patients with AN (Higgs, Goodyear, & Birch, 1989; Nicholls et al., 2002).

Concerning the psychometric proprieties of the EDCh-Q, our results revealed a very low and unproblematic proportion of missing values (Tabachnick & Fidel, 2007), which indicates a good applicability and acceptance of this assessment instrument. The exploratory factor analysis presented a 4-factor solution that constitutes a substantial part of total variance. All of the items could be clearly assigned to the scales that have been derived from the literature, thereby confirming the existence of the three clinical profiles of ARFID. The same four-dimensional structure could be found for both language versions of the questionnaire, which suggests that the factor structure of the EDCh-Q was little affected by linguistic differences. Overall, the difficulty of items were in the mid to high range with a low probability of elevated levels of eating disorder symptoms in the self-report ($.03 \leq p_m \leq .35$). The discrimination coefficients were in the mid to high range ($.30 \leq rit \leq .41$), with the exception of the item referring to the avoidance of food due to emotional reasons that presented a lower discriminating power ($rit = .24$). The scales' internal homogeneity turned out to be optimal ($.20 \leq r \leq .36$). Moreover, the high difficulty of items, as well as the left skewed and high peak distribution of items, suggests that the EDCh-Q accurately detects its concept of serious eating problems. It is therefore advisable to adjust the statistical analysis to the violation of normality assumption, either on the basis of larger samples or with the use of nonparametric tests (see Bortz & Döring, 2006). Finally, the dimensions identified by the exploratory factor analysis exhibited low internal consistencies, which probably reflect the small number of items per subscale. Thus, the consistency analysis may underestimate the reliability, especially in heterogeneous, multi-dimensional tests, which is not unusual for diagnostically oriented screening questionnaires. An improvement of the alpha value through item-exclusions was not likely; however, the average correlations between items of a scale were satisfactory.

# 7. General Discussion and Clinical Implications

The aims of the present thesis were to address specific questions regarding maintenance processes for maladaptive eating behaviours in middle childhood and to define the prevalence of specific middle childhood eating disturbances that to date have had little empirical investigation. Regarding the initial question on the role of food-related reward sensitivity in BED, we interestingly did not find an indication for greater deficits in the ability to postpone immediate food temptation either in children with BED or in children with ADHD or BED&ADHD compared to typically developing children. Consequently, it appears that what has been established as a sensitive indicator and long-term predictor of psychopathology in preschool children (Dalen et al., 2004; Gledhill & Petermann, 2013; Sonuga-Barke et al., 2003) does not apply to older children in our study.

In this respect, our results might reflect the age-dependent improvements in self-control usually occurring during middle childhood (Holodynski, 2013). Along with this progress, also increases a child's tendency to act in socially desirable terms, and thus, the urge needs to be more intense to lead to premature eating in the laboratory. Previous research has indeed indicated that effect sizes of delay aversion are largest when assessed in young children and that they become smaller in older children and adolescents (Bitsakou, Psychogiou, Thompson, & Sonuga-Barke 2009; Karalunas & Huang-Pollock, 2011; Marco et al., 2009). Interestingly, a constant exception seems to be overweight or obese children, who, in earlier studies (e.g., Bonato & Boland, 1983; Sobhany & Rogers, 1985), much like in our study, were more likely to encounter difficulties to delay food related rewards at various ages and up to 13 years of age, compared to normal-weight children. In a broader sense, this observation suggests that the conditioning processes that occur in repeated overeating might be important to understand the persisting difficulties of overweight or obese individuals to delay food related gratification similar to what has been shown for other addictive behaviours (e.g., substance abuse) (Volkow, Wang, Fowler, Tomasi, & Baler, 2012).

Additional longitudinal studies in community children appear to be warranted to further differentiate the role of reward sensitivity in the aetiologies of BED and overweight/obesity. Existing studies on adults only provide limited insight into this question because they identified BED pathology as a predictor of increased reward sensitivity in individuals who were already overweight/obese (Schag, Schönleber, et al., 2013; Schag, Teufel, et al., 2013; Schienle, Schäfer, Hermann, & Vaitl, 2009). The same limitation applies to the Nederkoorn et al. study (2006) that examined reward sensitivity in a population of obese young adolescents.

Moreover, it appears likely that in middle-aged children suffering from a mental disorder, self-control in eating may interact with specific conditions, e.g., emotional factors. As shown elsewhere (Hartmann, Rief, & Hilbert, 2013), adolescents suffering from LOC eating seem to react impulsively especially in negative emotional situations. Concerned adolescents indeed presented with a greater increase of behavioural impulsivity (stop signal task) after negative mood induction in the laboratory than normally developing adolescents and adolescents with ADHD. The assumption that impulse-control deficits in BED may present as essentially an emotion-driven process is also suggested by our results of Study 2. Consistent with earlier studies on BED (Combs et al., 2011; Pearson et al., 2012; Pearson et al., 2015), emotional eating in children was highly associated with a general tendency to act out intense positive and negative affects based on children's self-reports. Therefore, a promising way to examine whether food-reward sensitivity is boosted by affective conditions in children with BED would be to apply experimental mood induction procedures to the DOG experiment. Natural levels of situational negative affect might in fact not be intense enough to cause disruptions in self-control and thus influence children's eating behaviours. Consistent with other studies (Hilbert, Rief, et al., 2009), we found low levels of negative affective states in most children who reported in general positive emotions before and after the DOG experiment.

Finally, as a major contribution, the findings of Study 2 suggest the necessity of considering child dispositional factors such as an impaired ability to regulate intense affects in the interrelationship with aspects of parental emotionality when accounting for the maintenance of disordered eating behaviours in children. More specifically, our results suggested that the parents' own psychological health, in addition to criticism and the use of punitive and authoritarian parenting practices, seems to amplify children's self-regulation difficulties with respect to food intake. From a developmental point of view (Bariola, Gullone, & Hughes, 2011), this means that children's self-regulation ability in general and with respect to eating might be undermined by a non-supportive family climate (Nelemans, Hale, Branje, Hawk, & Meeus, 2013), where parents use punitive authoritarian parenting practices or express repeated criticism. This perspective is consistent with earlier research postulating less adaptive emotion regulation processes in families of children engaging in LOC eating (Czaja et al., 2011). The particular use of corporal punishment was the most dysfunctional and distressing parenting practice that achieved a direct though small effect in the prediction of self- and parent-rated frequencies of a child's emotional eating. This finding further supports the relevance of the interpersonal model of binge eating (Elliott et al., 2010; Wilfley,

MacKenzie, Welch, Ayres, & Weissman, 2000) for children. Interpersonal threats as a major source of intense negative affects are attributed an important role in the maintenance of binge eating.

Based on the overall findings, the influences of child temperamental or parental factors on the occurrence of disordered eating in middle childhood seem to be principally attributable to self-regulation processes. In our study, children with either a BED or emotional eating not only self-reported an increased disposition towards affective impulsiveness but also experienced more concerns regarding self-control difficulties when exposed to palatable foods in the laboratory. The finding that exposure to tempting foods or intense emotions is associated with a risk of anticipated or effective self-control failures in concerned children is completely consistent with assumptions from current aetiological models of eating disorders developed for adults (Stice et al., 2011; Kostopoulou et al., 2013; Shafran & Robinson, 2004). A new element in our research refers to the demonstration of relationships between general self-regulation deficits in children, dysfunctional parental behaviours or critical attitudes and the occurrence of problematic eating behaviours in children. This element basically illustrates the risk in concerned children for a vicious circle engaging temperamental vulnerabilities and increases in parental control.

Against this background, early preventive or treatment efforts should not only act on maintenance factors that relate to children's own dispositions regarding the regulation of eating-related concerns or intense affect but also identify parents who will need specific interventions. A potential risk group is parents with increased levels of psychopathology or parents who have an overweight child and who in our study were found to emphasise the severity of eating difficulties in their children. This situation, in turn, was related to a more frequent use of authoritarian and punitive parenting practices. Corresponding interventions (educational programs, such as Triple P – Positive Parenting Program) should be directed at increasing the functional aspects and reducing the dysfunctional aspects of parenting behaviours. Indeed, current research offers several indications that positive parenting behaviours, such as warmth, constructive problem solving and feeding strategies that increase children's self-control over food but also monitor food intake, especially in children with high impulsivity, are associated with favourable eating- and weight-related outcomes in children (Decaluwé, Braet, Moens, & Van Vlierberghe 2006; Farrow, 2012; Kröller, Jahnke, & Warschburger 2013; Topham et al., 2011). Conversely, and consistent with our study, rigid parental control, disregard or non-reasoning punitive behaviours have been associated with

more dysfunctional eating behaviours in children (Schuetzmann et al., 2008; Topham et al., 2011; Van Strien, Snoek, Van der Zwaluw, & Engels, 2010).

Finally, interventions for children should consider differential treatment foci for overweight children without BED and normal-weight or overweight children with BED. The results of Study 1 offered an indication that food intake awareness might be decreased in overweight children, which appeared to be an important contrast to children with BED, who presented with high levels of sensitivity towards their own self-control difficulties when exposed to food temptation. Promoting strategies that increase self-awareness regarding food intake, therefore, appears to be particularly useful for overweight children (Braet et al., 2008). In turn, children with BED appear to lack coping skills regarding eating concerns and related feelings, which should be addressed by therapeutic interventions to prevent or stop binge eating, not in public while observed but later when alone.

The different questions raised by the present thesis regarding maintenance factors of middle childhood eating disorders do not apply to the recently defined diagnosis of ARFID. In contrast to BED where status has widely been established in classification research on adults and children, the clinical utility of the newly defined ARFID diagnosis remains to be further determined, especially in middle childhood, where data on its occurrence are scarce. Information on prevalence is essential to visualise how children in the general population are affected by eating problems and to derive corresponding preventative measures. With this aim, a second step of this thesis was to describe the occurrence of restrictive/avoidant eating behaviours exempt of body-image concerns in children in the normal population (Study 3).

The results of Study 3 confirmed that ARFID eating behaviours, especially of the SE and FAED subtypes, represent a significant phenomenon in middle childhood. Whereas many eating difficulties such as selective eating are considered to be developmentally normal for infants or preschool children (Bryant-Waugh & Lask, 2013; Watkins & Lask, 2002), children at the age considered in our study should already have outgrown these difficulties. This factor signals that ARFID, which has previously been far more considered to be a paediatric issue limited to infants or small children, also merits attention using preventive efforts directed at school-aged children. Carefully performed differential diagnosis to other eating disorders, especially AN, appears to be important because of the likely overlap in the external appearance regarding underweight. The important differences in cognitive features regarding body-image distortion between these disorders suggests fundamentally different treatment foci. Future prospective and longitudinal studies should, however, verify whether ARFID

presents a risk factor for other eating disorders with a later age of onset.

In contrast to AN and BN that have gained increasing attention in preventive campaigns, there is still less public awareness on ARFID. It is similar to BED, which has only recently benefited from increased research interest in children (Johnson, Rohan, & Kirk 2002; Munsch, Biedert, & Schlup, 2009). This observation is considerable because ARFID and BED symptoms seem to present with significantly higher occurrences in middle childhood than those of AN or BN (Bryant-Waugh & Lask, 2013; Tanofsky-Kraff et al., 2004). This finding highlights a need to target publicity work and public information on these disorders, including preventative programs in schools to enhance early detection of these types of disorders.

## 7.1. Strengths and Limitations of the Present Thesis

The limitations of this thesis are important. Only selected perspectives have been adopted on issues of prevalence and maintenance of middle childhood eating disorders. First, we evaluated the distribution of restrictive/avoidant eating behaviours but not of additional problematic eating behaviours, which would have been necessary to depict a more complete clinical scenario of how community children are concerned by eating disorder symptoms. Second, the aspects of maintenance have been exclusively addressed with respect to children's temperamental dispositions and family characteristics, whereas other relevant maintenance factors, such as dieting or satiety regulation, have not been considered. It is also important to note that we considered children's ability to regulate strong emotional impulses as a general trait of affective impulsiveness, without differentiating the effects of singular positive or negative emotions, which impose limits to the specificity of our results.

Furthermore, we chose separate study designs to investigate factors of maintenance without modelling them in a comprehensive model, e.g., through a structural equation modelling approach. Additionally, Study 1 and Study 2 focused exclusively on BED and related symptoms such as emotional eating, which means that our results cannot be generalised to other eating disorders in middle childhood. Moreover, the cross-sectional designs of these studies did not allow drawing causal conclusions regarding child dispositional and parental factors associated with disordered eating. It is noteworthy that our results cannot be generalised to the role of fathers because only seven fathers participated in Study 2. To investigate the differential effects of fathers' and mothers' characteristics on children's

development of eating behaviours, future research should emphasise the father's report (Freeman et al., 2012).

A primary concern also relates to the small sample size. For Study 2, this size implied that we could not consider interaction effects of, for example, the depressiveness or affective state and diagnostic groups when investigating the factors of an impaired DOG. Although behavioural observation procedures are highly valuable in children (Barkley, 1991; Marakovitz & Campbell, 1998), the laboratory setting implicates a risk for acting according to social desirability, which might have led the children not to eat, although they found it difficult to abstain. This situation may be especially salient when examining children with BED in which eating is a highly shame-related process (Tanofsky-Kraff et al., 2007).

Finally, future studies should consider longitudinal designs or ambulatory assessment methods (Mehl & Robbins, 2012) for depicting more precisely children's and parents' behaviours in everyday life and for decreasing limitations, such as retrospective assessment bias or social desirability, because of the laboratory context.

It should be positively noted that our studies (Study 1 and Study 2) are considered among the few that have studied community children with clinically significant eating disorders, thereby focusing on relevant maintenance mechanisms. A major part of the existing research is based on either clinical samples of treatment-seeking children or school children with symptoms of disordered eating rather than full syndrome disorders. Both of the later variants of sampling imply potential selection biases associated with treatment-seeking status or the absence of clinical significance of symptoms. In our studies (Study 1 and Study 2), we prevented both of the later selection biases using an extensive recruitment procedure based on initial school screenings followed by thorough diagnostic evaluations and validated eating disorder interviews. To guarantee further control of potential group differences, control children in our study were individually matched to children with BED according to age, gender and BMI. We did not achieve an optimal BMI matching for different reasons. On the one hand, the matching was based on parents' self-report of their child's weight and height at the time of the telephone screenings, which may have resulted in estimation errors. On the other hand, we encountered difficulties in recruiting healthy children with identical BMI levels as the children with a diagnosis of BED, who, for the most part, were overweight. Therefore, a statistical control of BMI was necessary. Finally, a major strength in this thesis refers to the multimodal assessment approach chosen in Study 1 combining behavioural and self-experience data, as well as the integration of both children's and parents' perspectives when

assessing children's emotional eating in Study 2. A major strength in Study 3 refers to the large population-based sample size, including both German- and French-speaking children from urban and rural regions of Switzerland speaking for a good generalisability of results.

## 7.2. Conclusion

An important part of this thesis was the examination of basic processes implicated in the maintenance of problematic eating behaviours in middle childhood. A primary focus relied on BED and emotional eating, which appeared to be underpinned by a general disposition to act out intense affects and by a negative expectancy regarding one's own self-control ability when confronted with tempting foods. Both of the latter aspects demonstrate the key implication of self-regulation processes in the maintenance of problematic eating. Directly associated with a child's self-regulation ability are aspects of the parent's own emotionality. In this respect, punitive, authoritarian parenting practices and parental critical attitudes were especially found to constitute significant interpersonal stressors that might interfere with children's dispositional self-control and the regulation of eating. As a second component of this thesis, we characterised the occurrence of avoidant or restrictive eating behaviours in middle childhood. Such eating behaviours were common among community children in our study. This factor, together with the severe psychosocial impairments associated with ARFID if present at a clinical level (Nicholls & Bryant-Waugh, 2008), highlights the need for early detection efforts. Finally, maintenance mechanisms of eating disorders evidenced in adults (Fairburn et al., 2003; Stice et al., 2011) regarding dysfunctional processing of emotions and food-related cognitions appear to be valuable for children and should be further investigated with respect to ARFID.

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# 9. Appendix

Publication 1:

Dremmel, D., Wilhelm, P., Kurz, S., Fischer, S., Hilbert, A., & Munsch, S. (ready for submission). Delay of food-related gratification in children with BED, ADHD compared to healthy controls.

Publication 2:

Dremmel, D., Wilhelm, P., Kurz, S., De Albuquerque, J., Hilbert, A., & Munsch, S. (ready for submission). Emotional eating in children: The influence of parenting practices, expressed emotion and affective impulsiveness.

Publication 3:

van Dyck, Z., Bellwald, L., Kurz, S., Dremmel, D., Munsch, S., & Hilbert, A. (2013). Essprobleme im Kindesalter. *Zeitschrift für Gesundheitspsychologie, 21*(2), 91-100.

# Delay of Food-Related Gratification in Children With BED, ADHD Compared to Healthy Controls

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This research was supported by the Swiss National Science Foundation (SNSF) (grant number 100014132045 /1) and by the German Federal Ministry of Education and Research (BMBF) (grant number 01EO1001).

## Abstract

This study investigated the behaviour and experience of children suffering from BED compared to children with ADHD or a dual diagnosis (BED&ADHD) and of healthy children during an adapted version of the classical delay of gratification task. In total, 84 children aged between 8 and 13 years participated in the study. It was observed whether children were able to delay a preferred edible reward, and whether the observable behavior was consistent with children's self-report of experienced difficulties during the task. Moreover, it was investigated whether self-reported trait impulsivity, depressiveness and emotional state prior to the DOG task influenced the behaviour and experience in all groups of children. Neither mental health status, age, impulsivity, nor depressiveness or the emotional state prior to the task were related to children's ability to resist an immediate edible reward during the DOG task. Premature eating during the task was best predicted by children's BMI. Experienced difficulties during the task were most prominent in children suffering from a mental disorder. A diagnosis of BED, and depressive symptoms and components of impulsivity in all children were associated with increased self-reported fears of losing control during the task.



**Keywords:** delay of gratification, food reward sensitivity, binge eating disorder, attention-deficit/hyperactivity disorder

## Introduction

The obesogenic environment in industrialized countries exposes children to a variety of palatable, calorie-dense food that likely promote food intake unrelated to hunger but related to hedonics or affective conditions (Stice, Spoor, Ng, & Zald, 2009). Uncontrolled eating accompanied by a sense of loss of control over eating (loss of control eating, LOC) in the absence of hunger until feeling uncomfortably full represents a core feature of binge eating disorder (BED) in DSM-5 (American Psychiatric Association [APA], 2013). LOC occurs in 6% of normal or overweight children aged six to 12 years old in the general population, and ample evidence has accumulated that regular binge eating is associated with clinically significant eating disorders and general psychopathology as well as with overweight and obesity (Goldschmidt et al., 2008; Smink, van Hoeken, Oldehinkel, & Hoek 2014). LOC and BED are increasingly prevalent during adolescence and are associated with continuous weight gain (Hilbert & Brauhardt, 2014). The maintenance of LOC in childhood is related to the interaction of impaired mood, dysfunctional satiety regulation and dieting behaviour with personality factors, such as an increased impulsivity (Tetzlaff & Hilbert, 2014). Nevertheless, data on specific etiological pathways to LOC in childhood remain scarce.

In a world full of tasty food temptations, reward sensitivity (or the ability to delay an immediate for the sake of a long-term reward) becomes increasingly important (van den Bos & de Ridder, 2006). According to the neuropsychological model by Gray (1982, 1987), reward sensitivity represents a key concept of the multifaceted construct of impulsivity. Individuals are considered to be impulsive if they prefer an immediate smaller amount of reward instead of a larger or more meaningful delayed reward. According to Gray's theory, the behavioural activation system (BAS) regulates appetitive and reward-seeking behaviour and increases motor output, whereas the behavioural inhibition system (BIS) promotes active avoidance of punishment and therefore decreases motor output. In sum, impulsive individuals

have a relatively stronger BAS than BIS. The general ability to delay develops gradually during childhood in interaction with the psychosocial environment and is influenced by trait impulsivity or depressiveness (Mischel et al., 2011; Rothbart, Ellis, & Posner, 2011). Delay aversion has been shown to be prospectively predictive of various impairments, such as poorer school and social functioning, behaviour problems and weight gain (Campbell & von Stauffenberg, 2009; Francis & Susman, 2009; Mischel et al., 2011).

There is evidence of high impulsivity and especially an impaired ability to resist food temptations in obese and binge eating individuals (Hartmann, Rief, & Hilbert, 2013; Schag, Schönleber, et al., 2013; Schag, Teufel, et al., 2013; Stice, Spoor, Bohon, Veldhuizen, & Small, 2008). Higher scores of impulsivity compared to normal weight controls have been confirmed in several questionnaire-based studies in obese children and adults (Nasser, Gluck, & Geliebter, 2004; Nederkoorn, Braet, Van Eijs, Tanghe, & Jansen, 2006; Steadman & Knouse, 2014). Behavioural observation studies showed that obese children at various ages (aged from 3.5 up to 13 years) preferred smaller food-related immediate rewards to postponing in favour of a larger food-related reward compared to normal-weight children (Bonato & Boland, 1983; Sobhany & Rogers, 1985). The aversion for a delay of food-related rewards in small children of the general population has further been shown to be predictive of weight gain at ages 11 and 12 (Francis & Susman, 2009; Seeyave et al., 2009). Similarly, increased reward sensitivity in treatment seeking obese children and adolescents has been found in applying computerized paradigms with non-food related presents as rewards (door opening task; Nederkoorn et al., 2006; Verbeken, Braet, Claus, Nederkoorn, & Oosterlaan, 2009). Reward sensitivity was most impaired in obese young adolescents seeking obesity treatment, especially if they also presented with regular binge eating (Nederkoorn et al., 2006). These findings are consistent with recent research in adult BED individuals who revealed elevated food-specific reward sensitivity and increased activation of reward

processing brain areas if confronted with visual or real food cues (eye tracking, fMRI) compared to weight-matched obese individuals and normal weight controls (Schag, Schönleber, et al., 2013; Schag, Teufel, et al., 2013; Schienle, Schäfer, Hermann, & Vaitl, 2009). Only the Nederkoorn et al. study has investigated delay aversion in obese binge eating and non-binge eating young adolescents, but it did not include food-related rewards. To clarify the differential role of reward sensitivity in obese and BED children, further data on the experiences and behaviour if food-related reward sensitivity is triggered is needed.

Increased impulsivity has further been proposed as a common pathogenetic pathway of attention-deficit/hyperactivity disorder (ADHD), overeating and BED (Agranat-Meged et al., 2005; Cortese, Bernardina, & Mouren, 2007; Davis, Levitan, Smith, Tweed, & Curtis 2006), which may also explain the increased co-occurrence of ADHD and obesity (Nazar et al., 2014; Pagoto et al., 2009; Reinblatt et al., 2014). ADHD is a common childhood mental disorder, which is estimated to affect more than 5% of school-aged children and is characterized by a persistent pattern of inattention, hyperactivity-impulsivity, or both (APA, 2013). Therefore, it is not astonishing that in children suffering from symptoms of attention deficit and/or hyperactivity, the ability to delay an immediate food-related (candy) or non-food related (toy) reward is severely impaired (Campbell & von Stauffenberg, 2009; Dalen, Sonuga-Barke, Hall, & Remington, 2004; Sonuga-Barke, Dalen, & Remington, 2003). Studies further indicate that compared to healthy children, this impairment, with regard to the delay in food-related rewards, persists up to the age of nine years (Marakovitz, & Campbell, 1998), whereas findings in older children with ADHD, including non-food related rewards (different prices or monetary reward), are more heterogeneous (Karalunas & Huang-Pollock, 2001; Sjöwall, Roth, Lindqvist, & Thorell, 2013; Solanto et al., 2001). Although data underline the common pathway of impulsivity linking ADHD and LOC, such as in BED, currently, there is no study that has investigated the specific role of food-related reward

sensitivity or food-related delay aversion in children suffering from BED compared to ADHD or children suffering from a dual diagnosis of BED and ADHD.

In sum, correlates of impulsivity seem to be associated with uncontrolled eating in obese, BED and ADHD children (Hartmann, Rief, & Hilbert, 2013; Nazar et al., 2012, 2014; Nederkoorn et al., 2006; Stice et al., 2008). To specify the role of food-related reward sensitivity in BED children compared to ADHD children, behavioural studies approximating children's routine behaviour if confronted with immediate food temptations are necessary. Existing studies on children's ability to delay a food-related reward have used uniform snack foods for all children, thereby not accounting for individual variations in taste preferences. Additionally, self-experiences in these situations have not yet been assessed but might shed additional light on delay processes. As the ability to delay is known to increase with age and to generally consolidate in middle childhood (Holodynski, 2013), preliminary evidence on delay aversion in BED and ADHD should be reassessed during middle childhood.

Accordingly, the aim of this study was to compare delay aversion in eight- to 13-year-old children with BED, ADHD, or a dual diagnosis (BED&ADHD) and with a weight and age matched healthy control group in terms of behaviour and experiences during an adapted delay of gratification task (DOG) according to Mischel, Shoda, and Rodriguez (1989). Children had the choice between an immediate small amount (a previously selected preferred palatable food) or a larger amount of the equal food-related reward. We assessed whether the children ate immediately or postponed eating and captured their self-reported experiences during the task, which allowed us to further specify similarities and differences between groups.

We expected that children with either BED or ADHD would rather eat than wait during the DOG-task compared to the weight and age matched children of the control group (hypothesis 1.1a). Because impulse-control deficits are expected to be more severe in children

with a dual diagnosis (Nazar et al., 2012; Meule, 2013), we assumed that children with BED and ADHD have a higher probability to eat during the task than children with a single diagnosis of either BED or ADHD (hypothesis 1.2a). Moreover, we assume self-reports of children's experiences during the DOG task, such as a fear of losing control and difficulty while waiting, correspond with their behaviour during the task. In other words, children suffering from BED, ADHD or both are expected to report more fears of losing control and more difficulty while waiting than healthy children (hypotheses 1.1b and 1.1c). We expected that children with a dual disorder would be the most affected (hypotheses 1.2b and 1.2c).

Additionally, based on the literature (Nazar et al., 2014; Meule, 2013), we examined whether questionnaire-based impulsivity and depressiveness, which are known to substantially impact binge eating (Nazar et al., 2014; Pearson, Zapolski, & Smith 2015), and the current emotional state prior to the DOG-task (DIKJ 2nd revised edition, Stiensmeier-Pelster, Schürmann, & Duda, 2000; self-developed visual analog scale, VAS) predict children' behaviour and experience during the task. We expected that independent of the diagnosis, children would a) have a higher probability to eat, b) report more fears of losing control over eating, and c) report more difficulty while waiting during the DOG-task if they were more impulsive (hypothesis 2.1), reported more depressive symptoms, (hypothesis 2.2), or experienced less positive emotional states before the DOG-task (hypothesis 2.3).

## Method

### Sample

The current study was part of the Swiss University Study of Nutrition[3] (SUN) that investigated the maintenance factors of BED. Recruitment occurred in rural and urban French and German speaking regions of Switzerland. Altogether, 28 primary schools (2nd to 6th

[3] This study was funded by the Swiss National Science Foundation (SNF, 100014_132045 / 1).

grade) of all socioeconomic backgrounds were included. Parents of 1423 children gave informed consent to complete questionnaires to screen for symptoms of ADHD (Conners 3 AI, Conners, 2008; German and French version – own translations) and eating disorder pathology (ChEDE-Q, TODAY Study Group, 2007; German version, Hilbert, Hartmann, & Czaja, 2008; French version, Dremmel, De Albuquerque, Hilbert, & Munsch, in preparation).

Children between 8 and 13 years of age reporting symptoms of either BED or ADHD were screened via a structured clinical telephone interview by trained interviewers. Exclusion criteria for study participation were vomiting or exercising behaviour (more than 1 episode during the past three months), psychotic symptoms, current treatment for overweight, medication or medical conditions influencing eating behaviours, and insufficient German or French language skills of the child or parent.

Based on the telephone interviews, 108 children met the study participation criteria and were invited with their parents to the laboratory, where they participated in a comprehensive structured clinical interview on eating disorders conducted by trained clinical psychologists (Eating Disorder Examination for Children, ChEDE, Bryant-Waugh, Cooper, Taylor, & Lask, 1996; German version, Hilbert et al., 2013; French version, Dremmel, De Albuquerque, Hilbert, & Munsch, in preparation). Additionally, the children and parents were interviewed separately to assess children's general psychopathology (Schedule of Affective Disorders and Schizophrenia for School-age Children – Present and Lifetime Version, K-SADS-PL; Kaufman et al., 1997; German version, Delmo, Weiffenbach, Gabriel, Stadler, & Poustka 2001; French version, Rothen et al., 2009).

Based on the clinical interviews, 22 children met the child-adapted DSM-IV-TR criteria for BED (Tanofsky-Kraff, Marcus, Yanovski, & Yanovski, 2008), 25 children met the criteria for ADHD, and seven children met the criteria for BED and ADHD. A total of 23 children, who initially reported symptoms (telephone interview), did not fulfil the required

diagnostic criteria for either BED or ADHD and had to be excluded; one child with a mixed diagnosis decided to interrupt study participation after the interview. Additionally, 30 healthy children, who participated in the initial school screening and did not present any mental diagnosis according to the diagnostic interviews, participated in the study as a control group and were matched to the BED group on age, gender, and BMI.

In total, 84 children between 8 and 13 years ($M$ = 11, $SD$ = 1.4) and at least one of their parents participated in the study. Forty-four spoke German, and 40 spoke French; 37 were male, and 47 were female. The characteristics of study participants are summarized in table 1.

**Experimental Procedure and Assessments**

First, children's current emotional state, depressiveness, impulsivity, and preferences of a selected range of common snack foods were assessed. The latter was performed by presenting and letting children smell chocolate cookies, paprika chips and sweet-sour gummy candies and asking them which of the snacks they would prefer and how many packages (1, 2 or 3) they would like to eat if they were free to choose. The children were then exposed to a slightly modified version of the DOG task (Mischel et al., 1989) and were presented their preferred snack as a reward, which was reduced to one-fifth of the portion presented initially. The child was told that he or she would receive either the preferred quantity of that food (1, 2 or 3 entire packages) if he or she waited to eat until the experimenter returned or that he or she would be allowed to eat the presented small quantity immediately but then would not receive the chosen quantity of food. The experimenter told the child that he or she needed to make an urgent telephone call first and would then return with the promised package/s of food. The experimenter stayed away for 15 minutes, and children's delaying attempts were video-taped. At the end of the 15 minute delay, either the smaller quantity or the full quantity

of the preferred food was given to the child. Finally, children self-reported their experiences during the DOG task.

Children's experiences during the DOG task were assessed via a self-developed self-report questionnaire consisting of 25 items. Children's perceived difficulty while waiting (e.g., "How long did you feel you had to wait?", very short – very long) and their fears of losing control over eating (e.g., "Did you fear losing control over eating while waiting?", 1 = not at all to 7 = very much) were considered.

Children's current negative emotional state and mood were assessed at the beginning of the experiment and after the DOG task. Children's negative emotional state was assessed with the following items: "Do you feel … sad, embarrassed, anxious, guilty or angry?" These items were rated on a 7-point scale (0 not at all – 6 very much). As an indicator for each child's current negative emotional state, we used the maximum value given to any of the five items. Children's mood was assessed with the item "Do you feel well?" (0 not at all – 6 very much). To make the mood item compatible with the assessment of the negative emotional state, we reversed the coding of the item (0 = very well to 6 = not well at all).

Depressiveness was assessed via the German Children's Depression Inventory (DIKJ 2nd revised edition, Stiensmeier-Pelster et al., 2000; French version, own translation). The DIKJ captures the presence and severity of depressive symptoms in 8-17 year old children and adolescents with 26 items (e.g., "I am sad all of the time") on a three-point scale (0 – not present, 1 – moderately present, 2 – strongly present). Stiensmeier-Pelster et al. (2000) reported Cronbach's alphas between .74 and .88. In the present sample, α was .87.

Impulsivity was measured via the child-adolescent version of the Barratt Impulsiveness Scale (BIS-11, original adult version, Patton, Stanford, & Barratt, 1995; German version, Hartmann, Rief, & Hilbert, 2011; French version, Michel & Coudret, in preparation). The BIS-11 is a self-report instrument. It consists of 30 items (e.g., "I do things

without thinking") rated on a 4-point scale (1 rarely/never – 4 almost always), including the factors attentional-, non-planning-, and motor impulsivity. Stanford et al. (2009) reported satisfactory Cronbach's alphas for the attentional- and non-planning impulsivity (.74 and .72, respectively) but a low alpha for motor impulsivity (.59). Relying on the item composition of the English BIS-11 (Stanford et al., 2009), we obtained low alphas of .55, .65, and .58. The reliability of the total impulsivity was more satisfying; the alpha of the total score was .72.

Body Mass Index of children was calculated by measuring the children's weight and height in the laboratory using a standardized balance and a stadiometer. Body Mass Index (BMI) was computed with the standard formula (weight in kg /height in meter$^2$).

----------Insert table 1 about here ----------

## Statistical Analysis

We conducted a series of logistic regression models (LR) to analyse children's eating behaviour during the DOG task (eating = 1 vs. not eating = 0). As most children reported not worrying about losing control, this item was strongly skewed and was dichotomized (worries = 1, no worries = 0) and analysed with LR as well. In contrast, children's report of difficulty while waiting was normally distributed and was analysed with multiple linear regression analysis (MLR).

Each criterion variable was regressed on the same set of predictor variables. First, we entered the following control variables: age (centred approximately 11 years) and gender (male = 0) in a first step (model 1) and children's BMI (centred approximately 20) in a second step (model 2). Then, in a third step, we tested whether children with a mental disorder differ from healthy children (hypotheses 1.1 a, b, c) by entering a dummy variable that was coded 0 for patients and 1 for healthy controls (model 3). In a fourth step, we further added a dummy variable for children with ADHD and another for children with BED to test whether children

with a single diagnosis differ from children with a dual diagnosis (hypotheses 1.2 a, b, c) (model 4). To show how diagnostic groups differ from healthy controls, we recalculated model 4 using the three diagnostic groups (BED, ADHD, BED&ADHD) as the set of predictors (model 4r). Finally, we tested whether entering different facets of impulsivity, depressive symptoms, or the emotional state prior to the DOG-task had an impact on children's behaviour and subjective experience during the DOG task if age, gender, BMI, and type of disorder were controlled (model 5; hypotheses 2.1 to 2.3). Note that continuous predictor variables were centred around the group mean.

In the result section, we report and interpret the change in $R^2$ for MLR and the change in Nagelkerke's $R^2$ ($R_N^2$) for LR as an estimate of the variance in the criterion variable that is explained by the predictor(s) added to the model at each step of the analysis. The corresponding *F*- or $\chi^2$ test indicates whether the additional variance explained is beyond chance. The power was sufficient (Alpha = .05; Power = .80) to detect a moderate increase in variance ($f^2$ = .096; eta$^2$ = .087, if one predictor was added, $f^2$ = .119; $eta^2$ = .106, if a set of two predictors was added). To test whether a single coefficient was different from zero, we used the corresponding *z*-test (Alpha = .05). Coefficients of variables for which we had hypotheses were tested one tailed (o.t.).

## Results

### Predicting Children's Probability to Eat During the DOG Task

The probability to eat during the DOG task was not related to children's age or gender (model 1: $R_N^2$change = .007, $\chi^2_{(2)}$ = 0.40, $p$ = .821) but was positively associated with children's BMI (model 2: $B$ = 0.145, $SE$ = 0.068, $z$ = 2.12, $p$ = .034), which explained eight percent of the variance ($R_N^2$change = .078, $\chi^2_{(1)}$ = 4.75, $p$ = .029). In contrast to hypothesis 1.1a, the probability to eat was not different if children with a mental disorder (all diagnostic

groups) were compared to healthy children (model 3: $B$ = -0.*30, SE* = 0.*55, z* = 0.54, $p$ = .591; $R_N^2$change = .012, $\chi^2_{(1)}$ = 0.29, $p$ = .588). However, there were substantial differences between the diagnostic groups that explained 10 percent of the variance (model 4: $R_N^2$change = .101, $\chi^2_{(2)}$ = 7.06, $p$ = .029). The coefficients of model 4 revealed that for children with BED, the probability to eat during the DOG task was significantly lower than for children with a dual diagnosis ($B$ = -2.22, $SE$ = 1.00, $z$ = 2.23, $p$ = .013 o.t.), which was predicted by hypothesis 1.2a. However, in contrast to hypothesis 1.2a, the probability to eat was not significantly lower for children with ADHD ($B$ = -0.55, $SE$ = 0.91, $z$ = 0.60, $p$ = .273 o.t.) compared to children with a dual diagnosis.

In table 2, the coefficients of model 4r are presented. Model 4r essentially contains the same predictors as model 4, except that healthy children are now the reference group to which each diagnostic group is compared. From the exponents of the coefficients, the odds ratios (OR) and the conditional probabilities can be calculated (Tabachnick & Fidell, 2007). The OR corresponding to the intercept (11 years old, male, healthy children, with a BMI of 20) was .23, and the conditional probability that a child in this group eats during the DOG task was .19. OR was .10 in children with BED, with everything else being equal (11 years old, male, with a BMI of 20); .52 in children with ADHD; and .89 in children with a dual diagnosis, which corresponds to a probability to eat during the task of .09, .34, and .47, respectively. Although probabilities to eat during the task appear to differ between groups, the differences were not statistically significant (see table 2), except for children with BED compared to children with a dual diagnosis, as reported above.

The probability to eat during the task increased significantly with children's weight (table 2). As outlined above, in 11-year-old healthy males, the probability to eat was .19 if they had a BMI of 20. Children with a lower BMI of 15 had a lower probability to eat of .075.

However, in children with overweight (BMI of 25) or obesity (BMI of 30), the probability to eat during the task was much higher (.39 and .64, respectively).

Contrary to hypotheses 2.1a, none of the trait measures of impulsivity, which were entered separately in a final model step, were substantially associated with the probability to eat during the DOG task ($R_N^2$change $\leq$ .021, $\chi^2_{(1)} \leq 1.42$, $p \geq .233$). Moreover, neither higher depressiveness ($R_N^2$change = .002, $\chi^2_{(1)} \leq 0.11$, $p \geq .233$) nor emotional state prior to the experimental task ($R_N^2$change $\leq$ .018, $\chi^2_{(1)} \leq 1.22$, $p \geq .740$) were associated with the probability to eat during the task (rejection of hypothesis 2.2a and 2.3a).

**Predicting Children's Probability to Fear Losing Control Over Eating During the DOG Task**

Children's fears of losing control over eating during the DOG task were not significantly related to their age and gender (model 1: $R_N^2$change = .081, $\chi^2_{(2)} = 4.12$, $p$ = .127) nor to their BMI (model 2: $R_N^2$change = .040, $\chi^2_{(1)} = 2.15$, $p$ = .143). As predicted by hypothesis 1.1b, healthy children reported a lower probability to worry during the task than children with a mental disorder (model 3: $B$ = -2.81, $SE$ =1.11, z = 2.54, p = .006 o.t.). The difference between healthy and disordered children was large and explained 20 percent of the variance ($R_N^2$change = .195, $\chi^2_{(1)} = 11.25$, $p$ = .001). Additionally, there were differences between diagnostic groups that explained an additional 12 percent of the variance (model 4: $R_N^2$change = .121, $\chi^2_{(2)} = 7.87$, $p$ = .020). Consistent with hypotheses 1.1b and 1.2b, coefficients of model 4 show that healthy children ($B$ = -3.73, $SE$ = 1.33, z = 2.79, $p$ = .003) and children with ADHD ($B$ = -2.56, $SE$ = 1.16, $z$ = 2.21, $p$ = .014 o.t.) had a significantly lower probability of fearing losing control over eating than children with a dual diagnosis, but not children with BED ($B$ = -0.36, $SE$ = 0.96, $z$ = 0.37, $p$ = 356 o.t). Coefficients of model 4r presented in table 2 further show that healthy children reported a lower probability of fear of

losing control than children with BED or BED and ADHD, but did not differ from children with a single diagnosis of ADHD.

The exponent of the intercept coefficient and the corresponding OR was small (0.03), indicating that in 11-year-old healthy male children with a BMI of 20, the probability that a child reported any worries of losing control over eating during the DOG task was 3 percent. In children with ADHD, OR was only slightly higher (.10). However, OR was .87 in children with BED and 1.24 in children with a double diagnosis, indicating that approximately every second child with BED or BED and ADHD reported worries of losing control (probabilities of .46 and .55, respectively).

Consistent with hypothesis 2.2.b, depressiveness was positively associated with the probability to worry during the task (see table 3) and explained a further 9 percent of the variance (model 5: $R_N^2$change = .090, $\chi^2_{(1)}$ = 6.38, $p$ = .012). Additionally, as predicted by hypothesis 2.1b, attentional and motor impulsivity were positively related to worries (table 3) and explained a marginally significant portion of variance ($R_N^2$change = .045, $\chi^2_{(1)}$ = 3.16, $p$ = .075; $R_N^2$change = .050, $\chi^2_{(1)}$ = 3.46, $p$ = .063). However, contrary to the hypotheses 2.1b and 2.3b, non-planning and total impulsivity did not explain additional variance ($R_N^2$change ≤ .033, $\chi^2_{(1)} \leq$.2.31, p ≥.129) nor did emotional state prior to the experimental task ($R_N^2$change ≤ .004, $\chi^2_{(1)} \leq$ 0.30, $p \geq$ .582).

----------Insert table 2 about here ----------

**Predicting Children's Ratings Regarding the Difficulty While Waiting**

Neither children's age, gender (model 1: $R^2$change = 048; $F_{(2, 81)}$ = 2.04, $p$ = .136) or BMI (model 2: $R^2$change = 008; $F_{(1, 80)}$ = 0.70, $p$ = .405) were significantly related to their ratings of the difficulty of the DOG task. As predicted by hypothesis 1.1c, healthy children rated the task as being less difficult than children with any psychopathology (model 3: $B$ = -

1.05, *SE* = 0.33, $\beta$ = -.34, *z* = -3.16, *p* = .001 o. t.). This difference was rather large and explained more than 10 percent of the variance (model 3: $R^2$change = 106; $F_{(1, 79)}$ = 9.98, *p* = .002). Contrary to prediction 1.2c, children with a single diagnosis did not significantly differ from those with a dual diagnosis (model 4: ADHD in contrast to BED&ADHD: *B* = -0.75, *SE* = 0.62, $\beta$ = -.23, *z* = -1.22, *p* = .114, o. t.; BED in contrast to BED&ADHD: *B* = -0.17, SE = .61, $\beta$ = -.05, *z* = -0.28, p = .390 o. t.). The differentiation of diagnostic groups did consequently not increase the explained variance ($R^2$change = .025; $F_{(2, 77)}$ = 1.17, *p* = .316). The coefficients of model 4r in table 2 show that children in all three diagnostic groups rated the DOG task as being more difficult than healthy children. Additionally, the coefficient of age became significant, indicating that the task difficulty increased with the age of the children.

Contrary to hypotheses 2.1a, b, c neither impulsivity scales ($R^2$change $\leq$ .015, $F_{(1, 75)} \leq$ 1.41, $p \geq$ .645), depressiveness ($R^2$change < .01, $F_{(1, 75)}$ < 0.01, *p* = .972), nor current emotional state prior to the task ($R^2$change $\leq$ .018, $F_{(1, 75)} \leq$ 1.68, $p \geq$ .199) significantly explained additional variance (see coefficients in table 3).

----------Insert table 3 about here ----------

## Discussion

The present study aimed to investigate factors contributing to impeded self-regulation in children suffering from BED, ADHD or a dual diagnosis (BED&ADHD) compared to healthy children in middle childhood. We used an adapted version of the classical DOG paradigm and observed whether a child was able to delay a preferred edible reward. Additionally, we applied questionnaires to assess the emotional states and impulsivity of the children and evaluated whether self-report of experienced difficulties during the DOG task were consistent with the observable behaviour of the child. We expected difficulties with the

delay of food-related rewards to be pronounced in children with mental disorders. We further assumed that trait impulsivity, depressiveness and a negative emotional state prior to the DOG task would decrease self-control in all groups and therefore influence the behaviour and self-reported experiences while waiting during the DOG task.

Interestingly, if the child's behaviour was considered, neither mental health status, age, impulsivity, nor depressiveness or negative emotional state prior to the task were related to the child's ability to resist an immediate edible reward during the DOG task. The only important variable, accounting for eight percent of the variance of the probability to eat immediately, was the children's BMI. Higher BMI was associated with a higher likelihood to prematurely eat during the task, which resulted in an increased probability to eat of more than .60 in obese children compared to normal-weight peers.

In contrast, based on self-reported measurements of the children's experience during the DOG task, the fear of losing control or difficulty while waiting did not depend on BMI but were most prominent in children who suffered from a mental disorder. Consistent with our assumptions, the likelihood of a fear of losing control over eating was higher in children with a dual diagnosis (BED&ADHD) or a diagnosis of BED than in children with ADHD or in healthy controls. The only finding, which did not corroborate our hypothesis, was that children with ADHD did not significantly differ from healthy controls. Additionally, depressive symptoms were associated with an increased fear of losing control over eating independently of children's diagnostic status. Similar results were found with respect to the perceived difficulty while waiting, which was explained by the presence of any mental disorder, whereas the presence of a dual diagnosis did not increase difficulty while waiting compared to children with either BED or ADHD. Surprisingly, contrary to our expectations, the likelihood of perceived difficulty while waiting increased with the age of the children but

was independent of children's current impulsivity, depressiveness and emotional state prior to the experimental task.

Our results are consistent with data relating overweight to difficulties in postponing food rewards (Francis & Susman, 2009; Nederkoorn et al., 2006), but we are the first to show that on a behavioural level, such as during the DOG task, "eating or waiting" in 8- to 13-year-old children is associated more with BMI than mental disorders such as BED or ADHD, impulsivity, state or trait mood or by age and gender. These findings contrast with data by Nederkoorn et al. (2006) who found binge eating to be more important than BMI in a hospitalized sample of obesity treatment seeking children aged 12 to 15 years using a computerized non-food related DOG paradigm. As BED children in the Nederkoorn study were both older (mean age = 13.7) and heavier (mean BMI = 26.6) compared to our sample, the difficulty to delay food related rewards may be primarily associated with increased levels of obesity, which often develop later as a consequence of binge eating (Tanofsky-Kraff et al., 2006).

Previously reported difficulties of young children (2.5-5.5 years) with ADHD in postponing immediate food-related rewards (Dalen et al., 2004; Sonuga-Barke et al., 2003) could not be replicated in our study in middle-age children using a food-related DOG paradigm. Therefore, our findings corroborate data on age-dependent improvements in the ability to delay gratification in children with ADHD (Karalunas & Huang-Pollock, 2011; Sjöwall et al., 2013). However, DOG experiences in middle age children with ADHD have only integrated non-edible rewards (various prices or monetary rewards) and have failed to approve effects (Karalunas & Huang-Pollock, 2011; Sjöwall et al., 2013).

A surprising result is the lack of effect of having an additional eating disorder because the behaviour of children in the mixed group (BED&ADHD) did not significantly differ from the behaviour of children in the control group. It has to be noted that only seven children had

a mixed diagnosis (BED&ADHD). Of these seven children, four did not wait for the larger reward. However, a larger sample size would have been required to approve an effect. Additionally, of the 84 children in our study, only 24 children did not wait for the larger reward (see table 1). In interpreting these results, it is important to consider that the effect of social desirability cannot be ruled out, as the children knew that their behaviour would be videotaped. The 24 children who ate included children with the lowest frequency of BED and the highest frequency of mixed diagnosis (see table 1). A study with a larger sample should determine whether BMI remains the most important factor in the ability to delay food-related gratification.

Another interesting result is that children's self-reported experience during the task, unlike their observable behaviour, was independent of weight and was strongly sensitive to psychopathology. This finding may reflect a methodological issue, as power was simply larger if relying on self-reports of all 84 children than if relying on observable eating in a much smaller subgroup including 24 children (see table 1). Nevertheless, self-report allows children to more accurately document their experiences and feelings. In contrast, trying to wait while being video-taped is likely to be influenced by social control, and therefore, the urge to eat should be much more intense, leading to premature eating in children. Our findings indicate that BMI, although related to eating prematurely in the laboratory, did not imply a subjective component of perceived self-control difficulties during the food-related DOG. In other words, children with elevated BMI were most likely to yield to immediate temptation of food, whereas children suffering from ADHD, BED, or both, resisted the temptation but worried much more while waiting.

BED children in our study are characterized by high levels of sensitivity towards their own self-control difficulties and high self-control if confronted with food in a laboratory, which might reflect the mental illness status of BED compared to obesity. In regard to

prevention and treatment efforts in BED and obesity, our findings have two major implications. First, they underline the need to increase self-awareness regarding food intake in overweight or obese children (Braet et al., 2008), and second, they suggest that BED children may benefit from training to cope with eating concerns and related feelings to prevent or stop binge eating, not in public while observed but later in secret.

Finally, we found that impulsivity, depressiveness and negative emotional state did not significantly affect the ability to delay a food-related reward in our study. Again, this picture was slightly different if subjective self-reports were considered. Interestingly, self-reported fears of losing control but not experienced difficulty while waiting were associated with depressiveness and partly with correlates of impulsivity, independent of the diagnostic group, age or BMI of the child. This finding is consistent with previous literature, indicating important associations between depressive symptoms and binge eating episodes in children and adults. Therefore, early identification and treatment of this clinical feature is important (Presnell, Stice, Seidel, & Madeley, 2009). Surprisingly, the older age of children was associated with increased perception of difficulty while waiting, which is contrary to the idea that children's self-control improves with age (Holodynski, 2013). This finding might be explained by the assessment modality because the children's reports of increasing difficulty while waiting might reflect the development of cognitive capacities to reflect inner states, whereas self-control of observable behaviour, such as delaying an edible reward, is already mostly developed in this age group.

In sum, this study was the first to investigate children's behaviour and self-reported experience during a standardized laboratory food-related DOG task including children from middle childhood suffering from ADHD, BED or both. The strengths of the study include the simultaneous inclusion of behaviour observations and the child's self-report of experiences during the task. Furthermore, the experimental situation was designed to approximate routine

situations of immediate food temptations, including food rewards adjusted to children's taste preferences.

The limitations of the present study include the tendency to act according to social desirability, which might have led the children to not eat, although they found it difficult to abstain. Moreover, to consider interaction effects of factors, such as depressiveness or the emotional state and diagnostic groups, a larger sample size is needed. Additionally, BMI-matching of control children with children of the BED group was not optimally achieved; therefore, a statistical control for the influence of BMI was required. A main strength of this study is that children's self-report was included concurrently with behaviour-oriented measures of DOG, which might become especially useful with growing age of children. Future studies may benefit from additional assessment of children's psychophysiological activation during the waiting period.

## Acknowledgements

This research was supported by the Swiss National Science Foundation (SNSF) (grant number 100014132045 /1) and by the German Federal Ministry of Education and Research (BMBF) (grant number 01EO1001).

## Conflicts of interest

None.

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Table 1

*Sample Description, Means and Standard Deviations of Measures*

| | Total sample ($N$ = 84) | BED *(n = 22)* | BED&ADHD *(n = 7)* | ADHD *(n = 25)* | Control *(n = 30)* |
|---|---|---|---|---|---|
| Boy/girl | 37/47 | 8/14 | 3/4 | 16/9 | 10/20 |
| German/French | 44/40 | 8/14 | 2/5 | 16/9 | 18/12 |
| BMI ($kg/m^2$) | 20.03 (3.90) | 21.50 (4.70) | 21.10 (2.82) | 18.55 (3.34) | 19.95 (3.59) |
| Age (years) | 11.07 (1.37) | 11.05 (1.29) | 11.29 (1.38) | 11.56 (1.53) | 11.45 (1.22) |
| Eating/waiting | 24/60 | 4/18 | 4/3 | 9/16 | 7/23 |
| Difficulty while waiting | 2.43 (1.50) | 3.02 (1.48) | 3.29 (1.91) | 2.4 (1.20) | 1.83 (1.46) |
| Fear of losing control | 0.21 (0.41) | 0.45 (0.51) | 0.57 (0.53) | 0.08 (0.28) | 0.04 (1.92) |

*Note.* Gender, language of study, BMI (mean and standard deviation), age (mean and standard deviation), eating or waiting during the DOG task, experienced difficulty while waiting during the DOG task (mean and standard deviation), experienced fear of losing control during the DOG task (mean and standard deviation).

Table 2

*Coefficients of Control Variables and Diagnostic Groups (Model 4r)*

| | Eating during the delay of gratification task (LR) | | | | | Fear of losing control (LR) | | | | | Difficulty while waiting (MLR) | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | *b* | *SE* | Exp(*b*) | *z*-value | | *b* | *SE* | Exp(*b*) | *z* | | *b* | *SE* | Beta | *z*-value | |
| Intercept | −1.48 | 0.60 | 0.23 | 2.45 | ** | −3.51 | 1.97 | 0.03 | 2.93 | ** | 1.98 | 0.34 | | 5.92 | *** |
| Gender (0 = male, 1 = female) | 0.49 | 0.59 | 1.63 | 0.83 | | −0.19 | 0.81 | 0.83 | 0.23 | | −0.42 | 0.35 | −.14 | −1.21 | |
| Age (age-11 years) | −0.35 | 0.23 | 0.71 | 1.50 | | 0.62 | 0.33 | 1.86 | 1.88 | | 0.28 | 0.13 | 0.25 | 2.12 | * |
| BMI (BMI-20) | 0.21 | 0.08 | 1.23 | 2.58 | ** | 0.04 | 0.90 | 1.04 | 0.47 | | 0.01 | 0.04 | 0.23 | 0.20 | |
| Diagnostic groups (0 = control group) | | | | | | | | | | | | | | | |
| - ADHD [c] | 0.82 | 0.67 | 2.26 | 1.21 | | 1.16 | 1.31 | 3.20 | 0.89 | | 0.70 | 0.40 | 0.21 | 1.72 | * |
| - BED [c] | −0.86 | 0.79 | 0.42 | 1.10 | | 3.37 | 1.16 | 29.07 | 2.91 | ** | 1.28 | 0.41 | 0.38 | 3.13 | *** |
| - BED and ADHD [c] | 1.36 | 0.90 | 3.92 | 1.52 | | 3.73 | 1.33 | 41.47 | 2.79 | ** | 1.45 | 0.59 | 0.27 | 2.44 | ** |

*Note.* LR = logistic regression analysis, MLR = multiple linear regression analysis.
[c] one tailed; * p < .05; ** p < .01; *** p < .001

Table 3

*Effects of Impulsivity, Depressiveness and Emotional State*

| | Eating during the delay of gratification task (LR) | | | | Fear of losing control (LR) | | | | | Difficulty while waiting (MLR) | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | *b* | *SE* | Exp(*b*) | *z-value* | *b* | *SE* | Exp(*b*) | *z-value* | | *b* | *SE* | Beta | *z-value* |
| M5a: BIS Attentional impulsivity | .10 | .09 | 1.11 | 1.17 | .19 | .11 | 1.21 | 1.68 | * | −.06 | .05 | −.14 | −1.19 |
| M5b: BIS Motor impulsivity | .01 | .07 | 1.01 | 0.19 | .18 | .10 | 1.20 | 1.73 | * | .04 | .04 | .11 | .92 |
| M5c: BIS Non-planning impulsivity | −.00 | .05 | 1.00 | 0.02 | .01 | .07 | 1.01 | 0.12 | | .03 | .03 | .09 | .81 |
| M5d: BIS Total score | .02 | .03 | 1.02 | 0.48 | .06 | .04 | 1.06 | 1.44 | | .01 | .02 | .06 | .46 |
| M5e: Child Depression Inventory | .02 | .05 | 1.02 | 0.33 | .16 | .08 | 1.18 | 2.08 | * | .00 | .03 | .00 | .04 |
| M5f: Negative mood before the task | .32 | .31 | 1.39 | 1.07 | .20 | .37 | 1.22 | 0.54 | | −.16 | .16 | −.11 | −1.04 |
| M5g: Negative emotions before the task | .16 | .59 | 1.17 | 0.27 | −.44 | .84 | .64 | 0.53 | | .45 | .35 | .15 | 1.30 |

*Note.* LR = logistic regression analysis, MLR = multiple linear regression analysis.* p < .05; ** p < .01; *** p < .001, one tailed.

Further coefficients of models with a significant effect on fear of losing control:

M5a: Intercept: B = -3.29 (1.25), z = 2.63**, Gender: B = 0.10 (0.87), z = 0.11, C_Age: B = 0.50 (0.35), z = 1.44, C_BMI: B = 0.07 (0.09), z = 0.72, ADHD: B = 0.40 (1.41), z = 0.28, BED: B = 2.54 (1.22), z = 2.08*, BED and ADHD: B = 2.92 (1.38), z = 2.12*

M5b: Intercept: B = -3.74 (1.28), z = 2.93**, Gender: B = -0.05 (0.86), z = 0.05, C_Age: B = 0.79 (0.37), z = 2.16*, C_BMI: B = 0.07 (0.10), z = 0.74, ADHD: B = 1.28 (1.36), z = 0.94, BED: B = 2.79 (1.19), z = 2.34*, BED and ADHD: B = 4.12 (1.39), z = 2.97*

M5e: Intercept: B = 2.58 (1.27), z = 2.04*, Gender: B = -0.62 (0.86), z = 0.72, C_Age: B = 0.75 (0.39), z = 1.93*, C_BMI: B = 0.03 (0.10), z = 0.34, ADHD: B = 0.01 (1.47), z = 0.01*, BED: B = 2.36 (1.23), z = 1.92*, BED and ADHD: B = 2.40 (1.45), z = 1.65

# Emotional Eating in Children: The Influence of Parenting Practices, Expressed Emotion and Affective Impulsiveness

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This research was supported by the Swiss National Science Foundation (SNSF) (grant number 100014132045 /1) and by the German Federal Ministry of Education and Research (BMBF) (grant number 01EO1001).

## Abstract

This study investigated the influence of parenting practices, parental expressed emotion and children's affective impulsiveness on emotional eating in children aged 8-13 years. Seventeen children with a binge eating disorder (BED), 14 children with an attention deficit hyperactivity disorder (ADHD), 6 children with a dual diagnosis (BED and ADHD), and 23 healthy children and their parents completed questionnaires. Children's perceptions of emotional eating were related to their self-rated affective impulsiveness. Children's affective impulsiveness was significantly predicted by parental criticism. Parents' reports of elevated emotional eating in their children were associated with corporal punishment and authoritarian parenting. Findings suggest that maladaptive parenting practices and critical attitudes may impede children's emotion regulation, thereby contributing to emotional eating behaviour in middle childhood.

**Keywords**: emotional eating, parenting practices, expressed emotion, affective impulsiveness

## Introduction

Emotional eating refers to eating in response to aversive emotional states. Emotional eating usually increases throughout childhood and persists into adolescence and adulthood. Additionally, emotional eating is related to increased general psychopathology in terms of levels of depression and anxiety (Goossens, Braet, Van Vlierberghe, & Mels, 2009) and to eating disorders such as bulimia or anorexia nervosa and binge eating disorder (BED) (Ricca et al., 2012; Stice, Presnell, & Spangler, 2002). Currently, there is growing interest in the role of psychosocial factors such as family environment (Blissett, Meyer, & Haycraft, 2010), which influences the development of emotional eating together with genetic and temperamental factors (Bekker, van de Meerendonk, & Mollerus, 2004). As emotional eating is related to impaired mental health already in middle childhood and as parental behaviour is one of the major sources of familial influences on eating behaviour (Blisset et al., 2010; Munsch et al., 2007), comprehensive assessments of the role of family factors in the development of emotional eating in childhood are needed.

During early to middle childhood, parents are the primary role models and influence children's eating habits through their own eating attitudes and parenting practices (Munsch et al., 2007; Savage, Fisher, & Birch, 2007). Prior questionnaire studies in an obese sample and in samples from the general population have shown an association between positive parenting behaviour such as warmth, supportive control, constructive problem solving or monitoring of food-intake especially in children with high impulsivity and favourable eating- and weight related outcomes in children aged from six up to 16 years (Decaluwé, Braet, Moens, & Van Vlierberghe, 2006; Farrow, 2012; Topham et al., 2011). In contrast, rigid parental control, disregard and non-reasoning punitive behaviours are known to increase emotional eating in 6- to 13-year-old children (Topham et al., 2011; Schuetzmann, Richter-Appelt, Schulte-Markwort, & Schimmelmann, 2008; van Strien, Snoek, van der Zwaluw, & Engels, 2010).

Even though there is increasing interest in parental factors influencing emotional eating during childhood and adolescence, most studies assess global parental styles and do not specify concrete parental practices and only a few focus on middle childhood, known to be an important development phase for the consolidation of self-regulatory competences (Holodynski, 2013).

Moreover, the way parents express and regulate their emotions might further influence children's tendency to eat in response to emotional distress. Parents' emotional expressions towards their child have been conceptualized as Expressed Emotion (EE). The influence of EE on psychological well-being and on the course and relapse of mental disorders has been highlighted repeatedly (Hooley, 2007). Nevertheless, to our best knowledge, the influence of parental EE in terms of general criticism and emotional overinvolvement has not yet been studied in relation to emotional eating.

Finally, a child's tendency to act impulsively in response to intense mood states, defined as urgency (Whiteside & Lynam, 2011), has received increasing attention in recent years and has further been found to be associated with the manifestation of disinhibited eating (Pearson et al., 2012; Pearson et al., 2015).

Altogether, based on prior research, the present study aims at investigating the combined influence of parenting practices and of parental EE on children's emotional eating in middle childhood children, while taking into account children's affective impulsiveness. Therefore we investigated a sample of parent-child dyads including healthy children, children with a diagnosis of attention deficit/hyperactivity disorder (ADHD) or BED, known to be characterized by impulsivity and to be at risk for emotional eating (Stice et al., 2002; Farrow, 2012). Based on literature, we expected dysfunctional parenting practices and higher levels of parental EE to be positively related to children's emotional eating (hypothesis 1) and to children's affective impulsiveness (hypothesis 2). We further assumed higher levels of

children's affective impulsiveness to be associated with higher levels of emotional eating (hypothesis 3). Finally, we explored whether the influence of parenting practices and parental EE on children's emotional eating was transmitted by children's affective impulsiveness.

## Method

### Recruitment Procedure and Study Sample

Children with BED, ADHD and healthy control children together with their families participated in the Swiss University Study of Nutrition (SUN). Recruitment took place in 25 regular primary schools (2nd to 6th grade) in French and German speaking parts of Switzerland (Lausanne, Fribourg and Bern). Initially, 1100 children completed screening questionnaires to assess symptoms of ADHD (Conners 3 AI, Conners, 2008)[1] and eating disorder psychopathology (ChEDE-Q, Hilbert, Hartmann, & Czaja, 2008; TODAY Study Group, 2007).

Children aged between 8 and 13 years with symptoms of either ADHD or BED were further interviewed by telephone. Exclusion criteria for study participation were compensatory behaviours (more than one episode during the past three months), current treatment for overweight, medication with effect on eating behaviour, serious medical conditions influencing eating behaviour or impeding the participation in the experimental study as well as insufficient language skills. Seventy-seven children and their parents were invited to the laboratory and participated in a semi-structured diagnostic interview to assess mental disorders (K-SADS PL from Kaufman et al., 1997; Delmo, Weiffenbach, Gabriel, Stadler, Poustka, 2001; Rothen et al., 2009) and eating disorder psychopathology (ChEDE, Bryant-Waugh, Cooper, Taylor, & Lask, 1996; Hilbert et al., 2013). Interviews were

[1] If there was no German or French version of an instrument, a three-step back-translation procedure was applied. First step included initial translation, second step a back-translation in the original language by a native speaker of the target language and third step a final adaption (Questionnaires are available by the authors).

conducted by trained clinical psychologists. Seventeen children met criteria for BED, 14 children were diagnosed with ADHD, six were diagnosed with both BED and ADHD. Seventeen children didn't fulfil the required diagnostic criteria for either BED or ADHD and were therefore excluded from the sample. In addition, 23 healthy children from the initial school screening population participated in the study.

In total 60 children completed questionnaires and took part in different experimental tasks, not further considered in this article. Twenty-eight children spoke German, 32 spoke French; 27 were male and 33 were female. The mean age of children was 11 years (SD=1.2). Fifty-three mothers and seven fathers completed questionnaires. The mean age of parents was 43 (SD=5.6) years. Based on their profession, 16% of the parents were categorized as belonging to a low socio-economic status (SES) group, 68% were categorized as middle SES, and 16% as high SES. The local ethical committee approved the study and all participants gave their informed consent prior to study participation. Full study participation was compensated with 250 CHF.

**Measures**

*Children's self-reported emotional eating behaviour*

Children completed the Emotional Eating subscale of the Dutch Eating Behavior Questionnaire (DEBQ) for 7- to 13-year-old children (German version, Franzen & Florin, 1997; French version, own translation). This subscale consists of 10 items assessing the tendency to eat in response to negative emotions on a rating scale from 1 = never to 4 = always (e. g. "I wish to eat when depressed or discouraged"). Grunert (1989) and Franzen and Florin (1997) reported Cronbach's α's between .84 and .94, which are similar to those obtained in the present sample (α French-language Sample (FS) = .84, α German-language Sample (GS) = .95).

*Children's self-reported affective impulsiveness*

A child-adapted version of the UPPS (Whiteside & Lynam, 2001) was developed to assess the tendency to rash into action when experiencing strong negative or positive affects, without considering negative consequences of this action (e. g. "When I am very angry or very sad I do things without thinking that I regret afterwards") (Zecca et al., in preparation; German version, own translation). The affective impulsiveness subscale includes 27 items combining the negative urgency scale with the highly correlated positive urgency scale. They were rated from 1 = "totally false" to 4 = "totally true". The internal consistency of this scale was high (α FS = .91, α GS = .94).

*Parent's report of their children's emotional overeating*

Parents completed the emotional overeating (EOE) subscale of the Children's Eating Behavior Questionnaire (CEBQ; Wardle, Guthrie, Sanderson, & Rapoport, 2001; German and French version, own translation). The EOE scale consists of four items that describe the tendency to eat more when being in a negative mood. ("My child eats more when worried "). Items are rated from 1 = never to 5 = always. The original scale has a Cronbach's α between.74-.91 and reasonable test-retest reliability (Wardle et al., 2001). For the present sample Cronbach's α was high (α FS = .90, α GS = .88).

*Parent's self-reported emotional eating*

Parental emotional eating was assessed via the 10-items emotional eating subscale of the German DEBQ adult-version (DEBQ, van Strien, Frijters, Bergers, & Defares, 1986; German version FEV II, Grunert, 1989, French version, own translation). Cronbach's α was high for both the original and German version and was high in the present study, too (α FS = .95, α GS = .95).

*Parenting practices*

Parents completed the Alabama Parenting Questionnaire (Frick, 1991; German version, DEAPQ-EL-GS, Reichle & Franiek, 2009; French version, own translation). The extended German version contains descriptions of 40 different parenting practices rated from 1 = almost never to 5 = almost always. The German version additionally assesses the dimensions of responsible and authoritarian parenting. Functional parenting practices are assessed with: Involvement (e.g. "You ask your child about his/her day in school"; α DEAPQ > .60, α FS = .55, α GS = .46)[2], positive parenting behaviour (α DEAPQ = .84, α FS = .83, α GS = .72), and responsible parenting (α DEAPQ > .62, α FS = .33, α GS = .71). The other subscales assess dysfunctional parenting practices: Inconsistent discipline (e.g. "It is difficult for you to be consequent in your education"; α DEAPQ = .72, α FS = .70, α GS = .60), poor monitoring (α DEAPQ > .65, α FS = .54, α GS = .25), corporal punishment (α DEAPQ > .55, α FS = .61, α GS = .63), and authoritarian parenting (α DEAPQ = .71, α FS = .74, α GS = .61).

*Expressed emotion (criticism* and *emotional overinvolvement)*

Parental expressed emotion (EE) was measured via the Family Questionnaire (FQ, Wiedemann, Rayki, Feinstein, & Hahlweg, 2002; French version, own translation). The FQ assesses attitudes and behavioural patterns of relatives towards patients (or family members) on two subscales. Each subscale consists of 10 items rated from 1 = never/very rarely to 4 very often. The subscale criticism (CC) includes critical attitudes and critical comments towards the child (e. g. "I have to try not to criticize him or her"). Wiedemann et al. (2002) reported Cronbach's α for criticism to be .90 and .92. In the current sample α was slightly lower (α FS = .89, α GS = .86). The subscale emotional overinvolvement (EOI) includes

[2] Low alphas are probably due to skewed distributions of items and particularities of the rather small samples. In the original English version low alphas (< .60) were reported for poor monitoring and corporal punishment (Dadds, Maujean, & Fraser, 2003).

overconcern, overidentification and overprotectiveness (e. g. "I'm very worried about him or her"). Wiedemann et al. (2002) reported Cronbach's α between .79 and .82. In the current sample α was .81 (FS) and .60 (GS).

*Global severity index (GSI)*

Parents' general psychopathology was assessed via the Brief Symptom Inventory (BSI-53). Franke (2000) introduced a German version of the BSI-53 and Dreyfus and Guelfi (unpublished) a French version. The BSI includes 53 items of clinically relevant psychological symptoms (e. g. "feeling easily annoyed or irritated") that are rated from 0 = not at all to 4 = very strong. Across the 53 items the Global Severity Index (GSI) is computed as the average severity of symptoms experienced during the last seven days. The GSI has a high internal consistency (Cronbach's α > .90) and stability (Derogatis, 1993; Franke, 2000), (in the present study: α FS = .87, GS = .97).

*Body mass index (BMI) of parents and children*

Weight and height were measured using a standardized balance and a stadiometer. For parents BMI was computed (weight in kg/height in meter$^2$). For children an age and gender adjusted BMI was computed based on the following formula: [actual BMI/Percentile 50 of BMI for age and gender] * 100 (Decaluwé et al., 2006). The 50th percentile was taken from German normative data (Kromeyer-Hauschild et al., 2001).

## Statistical analysis

We computed Pearson product moment correlations and Spearman rank correlations, when variables were severely skewed, providing an initial test for hypotheses 1 to 3. To obtain more stringent tests and to control for potential confounding variables, we computed multiple regression analyses for ordinal data, using children's and parents' reports of children's emotional eating, and affective impulsiveness as criterion variables. Both measures

of children's emotional eating were highly skewed (the minimum was the modal value). Therefore, the negative log-log link function was applied. Affective impulsiveness was less skewed and thus the logit link function was used. In order to obtain more robust estimates, we narrowed the range of the criterion variables by transforming the original values into an equidistant five step scale. Predictor variables were centred around the group mean.

We considered gender, age, children's age adjusted BMI, parents' BMI, parents' own emotional eating, and parents' general psychopathology as potential confounding variables and explored their unique effects on each of the three criterion variables (child reported emotional eating, parents' reports of children's emotional eating, affective impulsiveness). Except for gender, age, and parents' BMI, for which we did not have a prediction, we expected the potential effects of these variables on the criterion variables to be positive.

In the first step of the model building (model 1), every potential confounding variable that was at least marginally significant ($p < .10$, according to the $\chi^2$-test) was kept as a control variable. In the second step (model 2), we tested, whether parenting practices, criticism and emotional overinvolvement still had an effect on emotional eating or affective impulsiveness when the control variables were already in the equation (test of hypotheses 1 and 2). In a final step we added affective impulsiveness as a further predictor of emotional eating (model 3). The comparison of results obtained in model 2 and model 3 allows us to explore whether affective impulsiveness transmits the effects of parenting practices, criticism, and emotional overinvolvement on emotional eating. When a predictor variable has a significant effect on children's emotional eating in model 2 that does not decrease substantially after affective impulsiveness has been added to the equation in model 3, the effect of this predictor variable is then proofed to be independent of children's affective impulsiveness (and vice versa). However, when the predictor variable is related to affective impulsivity (hypothesis 2), and its effect on emotional eating substantially decreases after affective impulsiveness has been

added to the equation in model 3, and affective impulsiveness also has a significant effect on emotional eating (hypothesis 3), then the effect of this predictor variable is transmitted by affective impulsiveness (hypothesis 4). [3]

In the result section we report and interpret Nagelkerke's $R^2$ ($R_N^2$) as an estimate of the variation in the criterion variable that is explained by the predictors in the model, together with the related $\chi^2$ which tests whether the variation explained by the model is indeed larger than zero. Because our sample size was relatively small, many zero cells occurred. In addition, the number of zero cells varied according to the combination of predictor variables. As a consequence, the difference in the deviances between two nested models was not reliable and could not be used to test whether adding a new predictor substantially improves the model fit. We therefore, only report the change in $R_N^2$ between two models to describe the impact of a newly added predictor.

With the program G*Power 3.1.7 (Faul, Erdfelder, Buchner, & Lang, 2009) we estimated the size of the correlation that could be detected with the current sample size of 60 dyads (children and parents). Fixing Alpha to .05, two tailed, the power was sufficient (.80) to detect medium to large effects ($\rho$ = 0.35). For those variables for which we had hypotheses and expectations, we tested one tailed, and achieved then a power of .80 to detect medium effects ($\rho$ = 0.31). Power calculations for ordinal regression analyses are not possible in G*Power. We therefore calculated multiple regression analyses as an approximation to the size of effects we could discover with the ordinal multiple regression analyses that we actually computed. Fixing Alpha to .05, and assuming a sufficient power (.80) an increase in variance corresponding to almost a medium effect size ($f^2$ = .14) could be discovered.

[3] If in addition, the relationships between variables is causally directed (predictor causes affective impulsiveness, which in turn causes emotional eating), then all the necessary conditions will be satisfied to conclude that affective impulsiveness mediates the effect of the predictor variable on emotional eating (Baron & Kenny, 1986). Because we do not have sufficient evidence for the causal direction of the effects we avoid the term mediation and use the term transmission instead.

# Results

## Descriptive Results

Table 1 shows a correlation matrix for all variables used in the study as well as the means, and standard deviations for each variable.

----------Insert table 1 about here ----------

*Emotional eating, BMI, and GSI:* As expected, parents' reports of their children's emotional eating are moderately correlated with children's self-reported emotional eating. However, the correlation between parents' ratings of their own emotional eating and their ratings of their children's emotional eating was much higher. In addition, parents' self-reported emotional eating was correlated with poor monitoring and corporal punishment. Children's BMI correlated with parents' BMI, parents' ratings of children's emotional eating, and parents' ratings of their own emotional eating. Interestingly, children's own ratings of emotional eating were less strongly related to their BMI, than those of their parents. Parent's global severity index correlated positively with parents' reports of their children's emotional eating, but neither with children's nor with parents' self-reported emotional eating.

*Children's' emotional eating, parenting practices and EE*: As predicted by hypothesis 1, children's self-reported emotional eating correlated positively with emotional overinvolvement, criticism, and two dysfunctional parenting practices, namely with corporal punishment and authoritarian parenting. However, there was no correlation with inconsistent discipline, poor monitoring, or functional parenting practices. In addition parents' reports of their children's emotional eating correlated positively with criticism, emotional overinvolvement, and authoritarian parenting, which was in line with hypothesis 1. However, other parenting practices did not correlate with parents' reports of their children's emotional eating.

*Children's emotional eating and affective impulsiveness:* As predicted by hypothesis 3 children's self-reported emotional eating as well their parents' reports of children's emotional eating correlated positively with affective impulsiveness.

*Affective impulsiveness, parenting practices and EE:* Children's self-reported affective impulsiveness positively correlated with authoritarian parenting and expressed emotion subscales, which was predicted by hypothesis 2. However, neither other dysfunctional nor functional parenting practices were associated with affective impulsiveness.

**Predicting Children's Affective Impulsiveness While Controlling for Children's BMI**

*Model 1:* The exploration of potential control variables (children's gender, age, BMI, parents' BMI, parents' own emotional eating, and parents' general psychopathology) revealed that only children's BMI had a marginally significant positive association with affective impulsiveness ($R_N^2$ = .054; $\chi^2_{(1)}$ = 3.15; $p$ = .076; $b$ = .019, SE = .011, $p$ = .048; one tailed, like all tests reported in this paragraph; $R_N^2$ for the other variables was $\leq$ .025; $\chi^2_{(1)} \leq 1.41$; $p \geq$ .235).

*Model 2:* In a second set of analyses, effects of parenting practices and EE on affective impulsiveness were tested, controlling for children's BMI. Results of these analyses are presented in table 2. They, show that the two subscales of EE, emotional overinvolvement and criticism were positively related to affective impulsiveness and led to models that explained a significant portion of variance. Emotional overinvolvement which had the largest effect increased $R_N^2$ about .120, whereas criticism added .095 to the $R_N^2$. In addition, authoritarian parenting was positively related to affective impulsiveness and added .069 to the $R_N^2$ (see table 2). This was in line with the predictions of hypothesis 2. However, no other parenting practice was significantly associated with affective impulsivity.

----------Insert table 2 about here ----------

**Predicting Children's Self-Reported Emotional Eating While Controlling for Parents' Emotional Eating**

*Model 1:* The only control variable that was significantly associated with children's self-reported emotional eating was parental self-reported emotional eating ($R_N^2 = .074$; $\chi^2_{(1)} = 4.25$; $p = .039$; $b = .035$, SE = .017, $p = .021$; one tailed, like all tests reported in this paragraph; $R_N^2$ for the other variables was $\leq .036$; $\chi^2_{(1)} \leq 2.05$; $p \geq .153$).

*Model 2:* Adding variables of parental practices led to several models that explained a significant amount of variance (see table 3). The increase in $R_N^2$ was largest for corporal punishment (.042), and criticism (.054). Coefficients of both variables were significant. They indicate that children's emotional eating tended to be higher the more parents' relied on corporal punishment or criticized their children which was predicted by hypothesis 1 (see table 3, model 2).[4]

*Model 3:* Adding affective impulsiveness as the third predictor, increased $R_N^2$ to a large extend (between .216 and .275), with the consequence that the proportion of explained variance became significant in each model. Coefficients of affective impulsiveness did not vary much between the models and were between $b = .602$, SE = .178, $p < .001$, and $b = .673$, SE = .184, $p < .001$). This indicates, as predicted by hypothesis 3, that children who reported high levels of affective impulsiveness also reported high levels of emotional eating. When affective impulsiveness was in the model and therefore controlled, the former significant effect of criticism disappeared (see table 3, model 3). This, together with the result that criticism predicted affective impulsiveness, suggests that affective impulsiveness transmits the effect of criticism on children's emotional eating. In contrast, the effect of corporal

[4] It should be noted, that the assumption of parallel lines did not hold for every model and that the just given description of a positive linear association between corporal punishment and children's emotional eating might be too simple.

punishment on children's eating behaviour did not decrease, when affective impulsiveness was put into the equation. Thus, the effect of corporal punishment on children's emotional eating behaviour cannot be explained by children's affective impulsivity.

----------Insert table 3 about here ----------

**Predicting Parents' Ratings of Children's Emotional Eating While Controlling for Children's BMI, Parents' Emotional Eating, and Parent's GSI**

*Model 1:* Among the explored control variables, children's BMI ($R_N^2$ = .226; $\chi^2_{(1)}$ = 14.27; $p < .001$), parental emotional eating ($R_N^2$ = .322; $\chi^2_{(1)}$ = 21.24; $p < .001$), and parental GSI ($R_N^2$ = .069; $\chi^2_{(1)}$ = 4.04; $p$ = .044) were significantly associated with children's emotional eating rated by parents. Together these variables explained more than half of the variation ($R_N^2$ = .542; $\chi^2_{(3)}$ = 41.94; $p < .001$). The corresponding coefficients were all positive and significantly indicating that each of the three variables had a substantial influence on parents' rating of children's emotional eating, that was independent of the other variables in the equation (parental emotional eating: $b$ = .735, SE = .169, $p < .001$, one tailed, like all tests reported in this paragraph; children's age adjusted BMI: $b$ = .029, SE = .009, $p < .001$; and parental GSI: $b$ = 1.627, SE = .473, $p < .001$).

*Model 2:* Adding parenting practices, or EE into the equation did not substantially increase the explained variance (increase in $R_N^2 \leq .018$). However, there were two exceptions. When authoritarian parenting and corporal punishment were added, $R_N^2$ increased about .042 and .029 respectively, and coefficients of these variables became significant (see table 4). These coefficients indicate that parents who reported authoritarian parenting behaviour or corporal punishment, perceived their child to overeat more frequently in negative emotional situations.

*Model 3:* Entering affective impulsiveness in the last step did not improve the models (increase in $R_N^2 \leq .009$), and across all models, none of the coefficients of affective impulsiveness was different from zero (coefficients ranged between $b = .085$, $SE = .197$, $p = .667$ and $b = .166$, $SE = .185$, $p = .370$). Thus, hypothesis 3 had to be rejected for parents' ratings of children's emotional eating, when other variables were controlled for.

----------Insert table 4 about here ----------

## Discussion

This study investigated whether parenting practices and parental expressed emotions influenced children's emotional eating assessed by child- and parent-based questionnaires. Additionally it was explored, whether children's affective impulsiveness transmitted the effects of parents' behaviour on children's emotional eating.

We found both dimensions of EE and three out of four dysfunctional parenting practices to be positively associated with higher levels of children's emotional eating. Additionally, emotional eating was more likely in affectively impulsive children. When confounding variables such as parents' own emotional eating, parents' general psychopathology, or children's BMI were controlled for in regression analyses, solely corporal punishment turned out to be associated with emotional eating in children based on both children's and parents' assessment of emotional eating. The effect was rather small (amount of variance: child report: 4.2%; and parent report: 2.9%) but remained when children's affective impulsiveness was controlled. Even though we revealed a small effect, these findings are in line with results indicating increased probabilities of obesity (Afifi, Mota, MacMillan, & Sareen 2013) or bulimia nervosa (Rorty, Yager, & Rossotto, 1995) in adults retrospectively reporting corporal punishment in childhood. In contrast, another cross-sectional study investigating the effect of corporal punishment on emotional eating in 6- to 8-

year-old children from the general population did not find any relationship (Topham et al., 2011). The divergent results might be due to the older age of our sample, different measurement methods or to the fact that behavioural and emotional consequences of corporal punishment become increasingly harmful with increasing age (Frick, Christian, & Wootton, 1999; Larzelere, 2000). Additionally, we investigated a smaller but mixed sample consisting of healthy children, as well as children suffering from BED, ADHD or both, where dysfunctional parenting practices are likely to be more pronounced than in a healthy population of e.g. Topham and colleagues.

Authoritarian parenting practices and the EE subdimension criticism were also associated with children's emotional eating if confounding variables were controlled and accounted for 4.2% and 5.2%, respectively. The effect of criticism was transmitted by affective impulsiveness and was only related to emotional eating when assessed by the children themselves, whereas authoritarian parenting was only associated to emotional eating of the child when assessed by parents. From a developmental point of view (Bariola, Gullone, & Hughes 2011), these findings suggest that children's self-regulation ability with respect to eating might be undermined by a non-supportive family climate, where parents express repeated criticism (Nelemans, Hale, Branje, Hawk, & Meeus 2012). The finding that parents reporting authoritarian parenting practices also report increased levels of emotional eating in their children has also been shown in Snoek, Engels, Janssens, & van Strien (2007). Future, longitudinal studies should examine, whether the increase in parenting control undermines a child's efforts to self-regulate eating behaviour or whether parental behaviour is rather influenced by a child's repeated eating in the absence of hunger.

In contrast to previous research, we found no positive effects of positive parenting practices on emotional eating. This might be due to different sample characteristics. In our sample more than half of the children suffered from externalizing behaviour problems and/or

binge eating. As a consequence, functional parenting practices are probably rare and not as efficacious as in a healthy sample from the general population (Topham et al., 2011) or in a sample of obese youngsters (Decaluwé et al., 2006).

As in other studies (Reyes & Kazdin, 2005; Braet et al., 2007), children's and parents' reports of the child's emotional eating behaviour were only moderately convergent. Parents' reports were highly influenced by their own level of emotional eating, their general psychopathology and the child's BMI, which accounted for more than half of the variance (54%) of their reports of children's emotional eating. Children's self-reports of emotional eating were only marginally and exclusively influenced by parents' own emotional eating; accounting for 7% of the child's emotional eating. Children's ratings of affective impulsiveness had a much stronger effect on their self-reported emotional eating than on parents' respective assessments. Parents' ratings of their children's emotional eating behaviour seem to be guided mostly by their own psychological health and eating problems as well as by their child's BMI; whereas in children, emotional eating is in line with their affective impulsivity. Accordingly, in order to reduce the risk of an assessment bias, future studies should use a multi-informant assessment approach (Kraemer et al., 2003).

Some of the study limits are especially important. First, sample size was small. As a consequence, the statistical models and the estimated parameters are less robust and reliable. Moreover, only moderate to large effects could be detected and we decided not to perform alpha level adjustments for multiple testing. Therefore our findings are preliminary and need replication with a larger sample. Second, at the time of the data assessment there was no available child-version of the CEBQ. Consequently, parents filled in the CEBQ and were asked to evaluate the frequency of their child's tendency to *overeat* in response to negative affect, while the children were asked to report about their own emotional eating behaviour but without specifying the amount of food consumed according to the DEBQ. Although

children's questionnaire did not include the amount of food intake, the literature reveals that this aspect is less salient in the evaluation of problematic eating and related distress in children than their experience of loss of control eating over food (Marcus & Kalarchian, 2003; Tanofsky-Kraff et al., 2008). Future studies should consider ambulatory assessment methods (Mehl & Robbins, 2012) in order to depict families' behaviour in everyday life more precisely and to decrease limitations such as retrospective assessment bias. Third, the results cannot be generalized to the role of fathers in children's emotional eating, as only seven fathers participated in the study. In order to investigate the differential effect of fathers and mothers characteristics on children's development of eating styles, future research should emphasize father's reports (Freeman et al., 2012). Finally, the cross-sectional study design does not allow drawing causal conclusions regarding parent and child factors associated with emotional eating.

Nevertheless, if replicated, the findings suggest an important association between parents' general psychopathology, their own emotional eating and children's emotional eating in a sample of eight to 13 years old healthy children and children suffering from BED, ADHD or both. Parents were especially likely to emphasize the severity of eating difficulties in children with an increased BMI, which in turn was related to a more frequent use of punitive, authoritarian parenting practices. Especially in children with an increased BMI, early preventive or treatment efforts should not only aim at improving healthy eating habits and physical activity but also at identifying parents in need for specific interventions, to reduce maladaptive parenting practices and dysfunctional criticism.

## Summary

Taken together, parents own psychological health along with criticism and a punitive, authoritarian parenting practice seems to amplify children's self-regulation difficulties in terms emotional eating and energy intake. Corporal punishment was directly related to

emotional eating based on both children's and parents' rating perspective. Furthermore, critical attitudes of parents about their child indirectly affected children's self-reported emotional eating by influencing the child's affective impulsiveness. Additionally, the present results underline the need to consider both children's and parents' perspective when assessing children's emotional eating (Braet et al., 2007). Based on the findings of this study, parental trainings should be integrated in preventive and early therapeutic interventions.

## Acknowledgements

This research was supported by the Swiss National Science Foundation (SNSF) (grant number 100014132045 /1) and by the German Federal Ministry of Education and Research (BMBF) (grant number 01EO1001).

## Conflicts of interest

None.

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Table 1

*Correlation Coefficients, Means and Standard Deviations of Variables*

| | | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 | 17 | 18 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| **1** | Self-reported child's emotional eating [a] | | | | | | | | | | | | | | | | | | |
| **2** | Parent reported child's emotional eating [a] | .34** [b] | | | | | | | | | | | | | | | | | |
| **3** | Affective impulsiveness | .45** [b] | .25* [b] | | | | | | | | | | | | | | | | |
| **4** | Gender | .06 | −.01 | −.08 | | | | | | | | | | | | | | | |
| **5** | Age | .10 | .01 | −.09 | .36** | | | | | | | | | | | | | | |
| **6** | BMI child age-adjusted | .26* [b] | .51** [b] | .16 [b] | −.23 | .05 | | | | | | | | | | | | | |
| **7** | BMI parent | −.03 | −.02 | −.01 | .10 | .24 | .31* | | | | | | | | | | | | |
| **8** | General parental psychopathology [a] | .09 [b] | .33** [b] | .14 [b] | .03 | −.21 | −.06 | −.24 | | | | | | | | | | | |
| **9** | Self-reported parental emotional eating [a] | .30* [b] | .57** [b] | .11 [b] | −.04 | .09 | .32* | .23 | .11 | | | | | | | | | | |
| **10** | Inconsistent discipline | .12 [b] | .25* [b] | .01 [b] | .12 | .04 | .22 | .22 | .10 | .21 | | | | | | | | | |
| **11** | Poor monitoring [a] | .18 [b] | .16 [b] | .10 [b] | .08 | .19 | .23 | .01 | .11 | .27* | .25 | | | | | | | | |
| **12** | Authoritarian parenting | .33** [b] | .40** [b] | .23* [b] | −.02 | .05 | .13 | .08 | .14 | .12 | −.08 | .08 | | | | | | | |
| **13** | Corporal punishment [a] | .38** [b] | .17 [b] | .21 [b] | .05 | .13 | .01 | .07 | .08 | .28* | .32* | .28* | .35** | | | | | | |
| **14** | Criticism | .29* [b] | .27* [b] | .32** [b] | −.03 | −.10 | .10 | .03 | .36** | .23 | .06 | .35** | .37** | .28* | | | | | |
| **15** | Emotional overinvolvement | .25* [b] | .28* [b] | .36** [b] | −.13 | −.06 | .16 | .10 | .25 | .21 | .11 | .39** | .11 | .29* | .68** | | | | |
| **16** | Positive parenting | −.05 [b] | −.08 [b] | −.14 [b] | −.16 | −.09 | .08 | −.10 | −.40** | −.10 | −.20 | −.25 | −.02 | −.03 | −.50** | −.39** | | | |
| **17** | Responsible parenting | .04 [b] | .20 [b] | .15 [b] | −.27* | −.04 | .25 | −.11 | .22 | .19 | .15 | .02 | .15 | .20 | −.00 | −.08 | .09 | | |
| **18** | Involvement | −.21 [b] | −.07 [b] | −.07 [b] | −.32* | −.33* | −.01 | −.25 | −.16 | −.08 | −.24 | −.24 | .05 | −.07 | −.21 | −.10 | .56** | .26* | |
| | *M* | 16.72 | 2.28 | 59.11 | 1.55 | 11.28 | 116.03 | 25.80 | .28 | 12.72 | 2.51 | 1.37 | 3.36 | 1.62 | 20.00 | 20.66 | 4.25 | 3.58 | 3.78 |
| | *SD* | 6.60 | 1.01 | 15.47 | .50 | 1.18 | 21.13 | 4.66 | .35 | 10.28 | .54 | .37 | .55 | .56 | 5.09 | 3.82 | .49 | .53 | .54 |

*Note.* [a] Variables were severely skewed, therefore Spearman correlation coefficients are reported. [b] Hypotheses and expectations were directed and therefore significance tests were one tailed. * $p < .05$; ** $p < .01$; *** $p < .001$.

Table 2

*Ordinal Regression Analyses: Predictors of Affective Impulsiveness*

| | Model 2: (children's BMI was controlled) | | | | | | |
|---|---|---|---|---|---|---|---|
| | Parameter estimates | | | | Variance explained | | |
| | *b* | *SE* | *z*-value [#] | | Nagelkerke's $R^2$ | $\chi^2$(2 df) | |
| Positive parenting | −0.77 | 0.49 | −1.57 | | .094 | 5.62 | |
| Responsible parenting | 0.45 | 0.46 | 0.97 | | .068 | 4.00 | |
| Involvement | −0.20 | 0.44 | −0.46 | | .058 | 3.37 | |
| Inconsistent discipline | −0.12 | 0.44 | −0.28 | | .055 | 3.23 | |
| Poor monitoring | 0.43 | 0.65 | 0.66 | | .061 | 3.55 | |
| Authoritarian parenting | 0.92 | 0.45 | 2.07 | * | .124 | 7.47 | * |
| Corporal punishment | 0.39 | 0.43 | 0.92 | | .068 | 4.02 | |
| Criticism | 0.12 | 0.05 | 2.37 | ** | .149 | 9.16 | * |
| Emotional overinvolvement | 0.17 | 0.07 | 2.57 | ** | .174 | 10.78 | ** |

*Note*. The Logit was chosen as the link function. [#] one tailed test. * p < .05; ** p < .01; *** p < .001.
Model 1: Age adjusted BMI of the child was the only variable in the equation ($b$ = 0.019, $SE$ = .011, $p$ < .05 o.t.); Nagelkerke's $R^2$ = .054 ($\chi^2_{(1)}$ = 3.15, $p$ = .076).
Model 2: The minimum and maximum coefficients across the 9 analyses for age adjusted BMI of the child are: $b$ = 0.015, SE = .011, $p$ =.097 o.t.; $b$ = 0.022, SE = .012, $p$ < .05 o.t..

Table 3

*Ordinal Regression Analyses: Predictors of Children's Self-Reported Emotional Eating*

| | Model 2: (parents' self-reported emotional eating controlled) [a] | | | | | | | Model 3: (parents' self-reported emotional eating and affective impulsiveness controlled) [b] | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Parameter estimates | | | | Variance explained | | | Parameter estimates | | | | Variance explained | | |
| | *b* | *SE* | *z*-value [#] | | Nagelkerke's $R^2$ | $\chi^2$(2 df) | | *b* | *SE* | *z*- value [#] | | Nagelkerke's $R^2$ | $\chi^2$(3 df) | |
| Positive parenting | .02 | 0.37 | 0.00 | | .074 | 4.25 | | .27 | 0.36 | 0.77 | | .296 | 19.04 | *** |
| Responsible parenting | −.16 | 0.35 | −0.47 | | .078 | 4.45 | | −.46 | 0.36 | 1.30 | | .314 | 20.44 | *** |
| Involvement | −.56 | 0.35 | −1.62 | | .115 | 6.67 | * | −.44 | 0.34 | 1.31 | | .310 | 20.11 | *** |
| Inconsistent discipline | .23 | 0.35 | 0.66 | | .082 | 4.68§ | | .49 | 0.36 | 1.34 | | .310 | 20.11 | *** |
| Poor monitoring | .62 | 0.47 | 1.31 | | .104 | 6.02 | * | .26 | 0.47 | 0.56 | | .294 | 18.93 | *** |
| Authoritarian parenting | .43 | 0.33 | 1.29 | | .104 | 6.03 | * | .37 | 0.37 | 1.01 | | .303 | 19.57 | *** |
| Corporal punishment | .59 | 0.35 | 1.71 | * | .116 | 6.73§ | * | .67 | 0.35 | 1.89 | * | .325 | 21.34 | *** |
| Criticism | .07 | 0.04 | 1.92 | * | .128 | 7.50 | * | .02 | 0.04 | 0.40 | | .291 | 18.71 | *** |
| Emotional overinvolvement | .06 | 0.05 | 1.29 | | .105 | 6.11§ | * | .01 | 0.05 | 0.14 | | .290 | 18.59 | *** |

*Note*. The Negative log-log was chosen as the link function. [#] one tailed test. * p < .05; ** p < .01; *** p < .001. § The $\chi^2$ test of parallel lines was significant ($p < .01$). Model 1: Parent's self-reported emotional eating was the only variable in the equation and explained 7.4 % of the variance ($\chi^2_{(1)} = 4.25$, $p = .039$).

[a] Model 2: The effect of parents' self-reported emotional eating was controlled. Coefficients of this variable ranged between $b = .026$, SE = .018, $p = .078$ and $b = .037$, SE = .018, $p = .021$ (one tailed). [b] Model 3: The effects of parent's self-reported emotional eating and child rated affective impulsiveness were controlled. Coefficients of parent's self-reported emotional eating were between $b = .016$, SE = .018, $p = .192$ and $b = .032$, SE = .018, $p = .038$ (one tailed), and coefficients of affective impulsivity were between $b = .602$, SE = .178, $p < .001$, and $b = .673$, SE = .184, $p < .001$ (one tailed).

Table 4

*Ordinal Regression Analyses: Predictors of Parents' Reports of Children's Emotional Eating*

| | Model 2: (parents' self-reported emotional eating, parents' GSI, and children's BMI were controlled) [a] | | | | | | | Model 3: (parents' self-reported emotional eating, parents' GSI, children's BMI and children's affective impulsiveness were controlled) [b] | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Parameter estimates | | | | Variance explained | | | Parameter estimates | | | | Variance explained | | |
| | *b* | *SE* | *z*-value [#] | | Nagelkerke's $R^2$ | $\chi^2$(4 df) | | *b* | *SE* | *z*-value [#] | | Nagelkerke's $R^2$ | $\chi^2$(5 df) | |
| Positive parenting | −.54 | 0.41 | 1.32 | | .554 | 43.25 | *** | −.45 | 0.41 | 1.10 | | .558 | 43.80 | *** |
| Responsible parenting | .75 | 0.43 | 1.75 | | .563 | 44.41 | *** | .50 | 0.39 | 1.27 | | .566 | 44.74 | *** |
| Involvement | −.40 | 0.35 | 1.15 | | .552 | 43.10 | *** | −.37 | 0.35 | 1.08 | | .559 | 43.94 | *** |
| Inconsistent discipline | .01 | 0.38 | 0.02 | | .542 | 41.94 | *** | .08 | 0.38 | 0.21 | | .551 | 42.94 | *** |
| Poor monitoring | .29 | 0.51 | 0.56 | | .544 | 42.20 | *** | .26 | 0.50 | 0.52 | | .552 | 43.12 | *** |
| Authoritarian parenting | .70 | 0.35 | 1.98 | * | .584 | 46.91 | *** | .68 | 0.36 | 1.87 | * | .586 | 47.15 | *** |
| Corporal punishment | .78 | 0.38 | 2.08 | * | .571 | 45.27§ | *** | .72 | 0.38 | 1.90 | * | .573 | 45.51 | *** |
| Criticism | .05 | 0.04 | 1.12 | | .554 | 43.30 | *** | .04 | 0.05 | 0.81 | | .556 | 43.48 | *** |
| Emotional overinvolvement | .08 | 0.05 | 1.49 | | .560 | 43.97§ | *** | .04 | 0.05 | 1.22 | | .562 | 44.24 | *** |

*Note*. The Negative log-log was chosen as the link function. § The $\chi^2$ test of parallel lines was significant ($p < .01$). GSI = global severity index; BMI = body mass index. [#] one tailed test. * p < .05; ** p < .01; *** p < .001.

Model 1: Variables in the equation were parents' self-reported emotional eating ($b = 0.735$, $SE = .169$, $p < .001$ o.t.); parents' GSI ($b = 1.627$, $SE = .473$, $p < .001$ o.t.), and children's age-adjusted BMI ($b = 0.029$, $SE = .009$, $p = .001$ o.t.); Nagelkerke's $R^2 = .542$ ($\chi^2_{(3)} = 41.94$, $p < .001$).

[a] Model 2: For each variable in the equation minimum and maximum of the corresponding coefficients across the 9 analyses are reported in brackets: parents' self-reported emotional eating ($b = 0.684$, SE = .175, $p < .001$ o.t.; $b = 0.849$, SE = .179, $p < .001$ o.t.), parents' GSI ($b = 1.449$, SE = .508, $p = .002$ o.t.; $b = 1.752$, SE = .486, $p < .001$ o.t.), children's BMI ($b = 0.026$, SE = .009, $p = .003$ o.t.; $b = 0.031$, SE = .001, $p = .001$ o.t.).

[b] Model 3: For each variable in the equation minimum and maximum of the corresponding coefficients across the 9 analyses are reported in brackets : parents' self-reported emotional eating ($b = .685$, SE = .175, $p < .001$ o.t.; $b = .838$, SE = .178, $p < .001$ o.t.), parents' GSI ($b = 1.542$, SE = .493, $p = .001$ o.t.; $b = 1.782$, SE = .489, $p < .001$ o.t.); children's BMI ($b = .026$, SE = .009, $p = .003$ o.t.; $b = .031$, SE = .009, $p = .001$ o.t.); children's affective impulsiveness ($b = .085$, SE = .197, $p = .334$ o.t.; $b = .166$, SE = .185, $p = .185$ o.t.).

21. Jahrgang / Heft 2 / 2013

# Zeitschrift für Gesundheitspsychologie

2/13

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## Themenheft

## Ernährung bei Kindern und Jugendlichen

Petra Warschburger (Hrsg.)



HOGREFE

# Zeitschrift für Gesundheits-psychologie



HOGREFE

Zeitschrift für Gesundheitspsychologie, 21 (2), 91 – 100 © Hogrefe Verlag, Göttingen 2013

# Essprobleme im Kindesalter

## Screening in der allgemeinen Bevölkerung

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**Zusammenfassung.** Essstörungen im Kindes- und Jugendalter werden immer häufiger und eine frühzeitige Erkennung ist von großer Bedeutung. Neben den „klassischen" Essstörungen besteht eine Anzahl von Essproblemen im Kindesalter, die sich durch vermeidende oder restriktive Nahrungsaufnahme kennzeichnen und zurzeit nicht im DSM-IV Klassifikationssystem aufgeführt werden. Ziel der Untersuchung war es, das Vorkommen dieser Essprobleme in einer allgemeinen, schulbasierten Stichprobe in der Schweiz zu untersuchen und die psychometrischen Kennwerte eines kurzen Screeningfragebogens zur Erfassung vermeidend oder restriktiver Nahrungsaufnahme im Selbstbericht zu ermitteln. Es beantworteten 730 Kinder im Alter von 8 – 13 Jahren den Eating Disturbances in Childhood – Questionnaire (EDCh-Q). 29.8 % der Kinder gaben an, vermeidendes oder restriktives Essverhalten aufzuzeigen. Der EDCh-Q zeigte insgesamt gute Itemcharakteristika. Die vierfaktorielle Struktur konnte bestätigt werden, allerdings mit geringen internen Konsistenzen der Subskalen. Untergewichtige Kinder gaben häufiger an, Symptome einer Nahrungsvermeidung mit emotionaler Störung aufzuzeigen. Vermeidendes oder restriktives Essverhalten ist nicht unüblich bei Kindern im Schulalter. Der EDCh-Q ist ein diagnostisch orientierter Screeningfragebogen zur Identifizierung dieser Essprobleme in der mittleren Kindheit. Weitere Forschung ist wünschenswert zur Validierung des EDCh-Q in allgemeinen und klinischen Stichproben.
Schlüsselwörter: Vermeidend/restriktive Ernährungsstörung, Kinder, Psychometrie, Epidemiologie, Selbstbeurteilungsfragebogen, DSM-5

Eating disorders in childhood and adolescence

**Abstract.** Since eating disorders in childhood and adolescence have increased significantly, their early identification is of importance. Besides the "typical" eating disorders, there are a number of eating disturbances that typically present in middle childhood, characterized by avoidance or restriction of intake, that are not currently classified in the DSM-IV system. The purpose of the present study is to investigate the occurrence of these disturbances in a general, school-based population, and to report psychometric properties of a brief self-report screening questionnaire assessing avoidant or restrictive food intake. A total of 730 children aged 8 – 13 years were assessed with the Eating Disturbances in Childhood – Questionnaire (EDCh-Q). Altogether, 29.8 % of the children reported avoidant or restrictive eating behavior. Item characteristics were overall favourable. The four-factorial solution was replicated, however, with poor internal consistencies of the subscales. Underweight children reported more symptoms of food avoidance emotional disorder (FAED). Avoidant or restrictive eating behavior is common in school-aged children. The EDCh-Q is a diagnostically orientated screening questionnaire to identify these eating disturbances in middle childhood. A larger study is warranted to validate the EDCh-Q in community and clinical samples.
Key words: Avoidant/restrictive food intake disorder, children, psychometrics, epidemiology, self-report questionnaire, DSM-5

Epidemiologische Studien zeigen, dass die Inzidenz von Essstörungen über die letzten Jahre relativ stabil geblieben ist (Currin, Schmidt, Treasure & Jick, 2005; Rosen, 2003; Smink, van Hoeken & Hoek, 2012), jedoch das Alter des erstmaligen Auftretens immer niedriger wird (Favaro, Caregaro, Tenconi, Bosello & Santonastaso, 2009; van Son, van Hoeken, Bartelds, van Furth & Hoek, 2006). Eine frühere Erstmanifestation von Essproblemen stellt ein erhöhtes Risiko für Essstörungen im Jugend- und Erwachsenenalter dar (Kotler, Cohen, Davies, Pine & Walsh, 2001; Marchi & Cohen, 1990; Russell, 1992) und sagt schlechtere Erfolgsaussichten einer Behandlung voraus (Castro, Gila, Puig, Rodriguez & Toro, 2004; Rosen, 2003; Steinhausen, Grigoroiu-Serbanescu, Boyadjieva, Neumärker & Winkler-Metzke, 2008; Wichstrøm, 2000),

Diese Arbeit wurde unterstützt durch Drittmittelprojekt 100014 132045 /1 des Schweizerischen Nationalfonds und durch Projekt 01EO1001 des deutschen Bundesministeriums für Bildung und Forschung.

DOI: 10.1026/0943-8149/a000091

weshalb eine frühzeitige Erkennung von großer Bedeutung ist.

Essprobleme bei Kindern im Schulalter wurden in der Literatur oft vernachlässigt, da sie sich diagnostisch im Bereich zwischen Fütter- und Essstörungen befinden (Nicholls, Christie, Randall & Lask, 2001). Es gibt bislang nur wenig epidemiologische Untersuchungen zu den Symptombildern, die man in dieser Altersklasse vorfindet (Nicholls & Bryant-Waugh, 2008; Watkins & Lask, 2002), und aktuelle Kenntnisse stammen weitgehend aus klinischen Beobachtungen, mit meist kleinen Stichproben (Cooper, Watkins, Bryant-Waugh & Lask, 2002; Fosson, Knibbs, Bryant-Waugh & Lask, 1987; Gowers, Crisp, Joughin & Bhat, 1991; Peebles, Wilson & Lock, 2006).

Forschungsarbeiten in diesem Bereich werden zusätzlich dadurch erschwert, dass bisher kein allgemein anerkanntes Klassifikationssystem kindlicher Essstörungen existiert. Die Anwendung der aktuellen Kriterien des Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR; American Psychiatric Association, 2000) führt zur Diagnose einer unspezifischen, nicht näher bezeichneten Essstörung (EDNOS) für einen Großteil der Kinder (Birgegård, Norring & Clinton, 2012; Bravender et al., 2010; Nicholls, Chater, & Lask, 2000; Peebles et al., 2006; Pinhas, Morris, Crosby & Katzman, 2011). In Anbetracht dieser Tatsache wurden altersadaptierte Kriterien entwickelt. Hierzu zählen die von Bryant-Waugh und Lask (1995) vorgeschlagenen Great Ormond Street (GOS) Kriterien, oder auch die Empfehlungen der Workgroup for Classification of Eating Disorders in Children and Adolescents (WCEDCA; Bravender et al., 2010; WCEDCA, 2007). Die DSM-5 Arbeitsgruppe für Essstörungen kommt diesen Entwicklungen durch den Vorschlag der vermeidend/restriktiven Ernährungsstörung (Avoidant/restrictive food intake disorder; ARFID) nach, die alle Altersklassen umfassen soll (American Psychiatric Association, 2012).

Diesen klassifikatorischen Ansätzen ist gemein, dass sie sich auf Essprobleme konzentrieren die sich durch Nahrungsvermeidung oder eingeschränkte Nahrungsaufnahme kennzeichnen, ungeachtet von Überessen oder medizinischen Problemen (Bryant-Waugh & Nicholls, 2011). Die kindliche Ernährung ist unangemessen in Bezug auf den Verzehr unterschiedlicher Nahrungsmittel und/oder die Menge im Sinne der Kalorienzufuhr (Bryant-Waugh, Markham, Kreipe & Walsh, 2010; Watkins & Lask, 2002). Das problematische Essverhalten kann mit einem Gewichtsverlust, unzureichender Gewichtszunahme oder Wachstumsstörungen einhergehen, dies ist aber nicht zwingend der Fall (Kreipe & Palomaki, 2012; Nicholls, Wells, Singhal & Stanhope, 2002). In der Literatur werden diese restriktiven/vermeidenden Essprobleme in drei Subtypen unterteilt, welche sich von den „typischen“ Essstörungen unterscheiden und nicht mit Gewichts- oder Figursorgen einhergehen (Bryant-Waugh et al., 2010). Es handelt sich entweder um ein problematisches Essverhalten basierend auf einer allgemein eingeschränkten Nahrungsaufnahme, dem Umfang an akzeptierten Nahrungsmitteln, oder der Vermeidung aufgrund einer spezifischen Angst (Bryant-Waugh et al., 2010).

In der Kategorie der allgemein unangemessenen Nahrungsaufnahme wird im mittleren Kindesalter oft die Nahrungsvermeidung mit emotionaler Störung genannt (Food avoidance emotional disorder; FAED; Higgs, Goodyer & Birch, 1989). Higgs und Kollegen beschrieben mit diesem Begriff eine Gruppe von Kindern, die eine unangemessene Nahrungsaufnahme und emotionale Probleme aufzeigen (Higgs et al., 1989). Es gibt bisher kaum Arbeiten zur Epidemiologie von FAED (Watkins & Lask, 2002). Cooper und Kollegen (2002) untersuchten 126 Patienten einer pädiatrischen Essstörungsstation, von denen 43 % die Diagnose Anorexia Nervosa (AN) bekamen, 29 % FAED, 19 % selektives Essverhalten (selective eating; SE) und 9 % unter einer anderen Essstörungen litten. Andere Untersuchungen haben gezeigt, dass 20–30 % der unter 13-jährigen Symptome einer FAED aufweisen (Madden, Morris, Zurynski, Kohn, & Elliot, 2009; Nicholls, Lynn & Viner, 2011). Gemäß Christie, Bryant-Waugh, Lask und Gordon (1998) sind in erster Linie Mädchen betroffen, wobei Lask (2000) von einem ausgeglichenen Geschlechterverhältnis berichtet.

Ein Erscheinungsbild, welches sich durch die eingeschränkte Akzeptanz an Nahrungsmitteln kennzeichnet, ist das selektive Essen (Bryant-Waugh, 2000). Es handelt sich um ein verbreitetes Essverhalten mit Beginn häufig im frühen Kindesalter, das zwischen 8 % und 50 % der Kinder in verschiedenen Stichproben betrifft (Carruth, Ziegler, Gordon & Barr, 2004; Chatoor, 2002; Nicholls et al., 2000). SE ist gekennzeichnet durch ein extrem eingeschränktes Nahrungsspektrum, starke Essensvorlieben und eine Weigerung, neue Speisen auszuprobieren (Nahrungsmittel-Neophobie), was oft zu erheblichen sozialen Problemen führt (Bryant-Waugh & Lask, 2007; Nicholls et al., 2001). Diese Kinder essen typischerweise nur bis zu zehn verschiedene Nahrungsmittel oder -gruppen, nehmen jedoch eine normale Menge an Kalorien zu sich (Bryant-Waugh & Lask, 2007). Die Vermeidung von Essen beruht dabei oft auf sensorischen Merkmalen (Chatoor, 2002; Kreipe & Palomaki, 2012; Smith, Roux, Naidoo, & Venter, 2005). Hinsichtlich der Geschlechterverteilung deuten einige Studien darauf hin dass mehr Jungen als Mädchen betroffen sind (Bryant-Waugh & Lask, 2007; Nicholls et al., 2001), wohingegen andere Untersuchungen keine Unterschiede feststellen konnten (Jacobi, Schmitz & Agras, 2008; Mascola, Bryson & Agras, 2010).

Nahrungsvermeidung aufgrund spezifischer Ängste findet sich in der funktionellen Dysphagie (functional dysphagia; FD) wieder, welche sich durch eine Meidung von Nahrungsmitteln aus Angst sich zu verschlucken, zu

würgen oder zu erbrechen kennzeichnet (Bryant-Waugh, 2000). Diese Störung tritt oft nach einem aversiven Erlebnis im orofazialen oder gastrointestinalen Bereich auf. Die Gültigkeit der funktionellen Dysphagie als diagnostische Kategorie muss noch geklärt werden, da man sie als Symptom auch bei anderen Störungsbildern vorfindet (Nicholls et al., 2000). Es gibt bisher nur wenige Informationen zu diesem Symptombild und es sind keine gesicherten Angaben zur Prävalenz und Inzidenz bekannt (Watkins & Lask, 2002). Ersten Untersuchungen nach sind Jungen und Mädchen gleich häufig betroffen (Lask, 2000).

Reliable, entwicklungsorientierte Screeninginstrumente sind wichtig für die Früherkennung von Essstörungen (Fairburn & Beglin, 1994). In Anbetracht des Mangels an Untersuchungen zu restriktiv/vermeidenden Essproblemen bei älteren Kindern, sowie des Bedarfs an psychometrisch validen Instrumenten zu deren einheitlichen Erfassung, wurde der Eating Disturbances in Childhood-Questionnaire (EDCh-Q) entwickelt. Das Instrument enthält 14 Items und deckt relevante Symptome der drei aus der klinischen Praxis hergeleiteten und im DSM-5 beschriebenen Untergruppen ab.

Ziel der vorliegenden Studie ist es, die psychometrischen Kennwerte des EDCh-Q für den deutschen Sprachraum zu ermitteln und die Prävalenz der diagnostischen Untergruppen in einer allgemeinen, schulbasierten Stichprobe in der Schweiz einzuschätzen.

# Methode

## Rekrutierung und Stichprobe

Kinder im Alter von 8 – 13 Jahren wurden im Rahmen eines populationsbasierten Screenings über Schulen (3.–6. Klassen) in der Schweiz (Städte Fribourg, Bern, Lausanne sowie umliegende Gebiete) rekrutiert. Die Erhebung fand im Rahmen der vom Schweizerischen Nationalfonds (SNF) geförderten Studie Swiss University Study of Nutrition (SUN) statt, die von der Ethikkommission des Departements für Psychologie der Universität Freiburg in der Schweiz positiv begutachtet wurde. Ein übergeordnetes Ziel des Forschungsprojekts SUN war es, die Prävalenz kindlicher Essprobleme in der Schweiz einzuschätzen, weshalb die hier vorgestellten Ergebnisse sich ausschließlich auf eine schweizerische Stichprobe beziehen.

Die Studiensprachen waren Deutsch und Französisch. Es wurden Schulen aller sozioökonomischen Hintergründe eingeschlossen und deren Einverständnis wurde eingeholt (kantonale Erziehungsdirektion, Schuldirektionen). Für das schulbasierte Screening wurden die reliablen und validen Selbstbeurteilungsfragebögen Eating Disorder Examination-Questionnaire für Kinder (ChEDE-Q; deutsche Fassung: Hilbert, Hartmann & Czaja, 2008; französische Fassung: Dremmel, De Albuquerque & Munsch, in Vorb.), die Conners 3 ADHD – Index – Selbstbeurteilungsskala (Conners 3AI, Conners, 2008; deutsche Fassung: Lidzba, in Vorbereitung; französische Fassung: Dremmel, De Albuquerque & Munsch, in Vorb.), sowie der durch die Autoren entwickelte Eating Disturbances in Childhood-Questionnaire (EDCh-Q) angewandt. Die deutschen Versionen der Fragebögen wurden durch zwei fließend Deutsch und Französisch sprechende Psychologinnen ins Französische übersetzt und von Experten mittels Rückübersetzung überprüft. Vor dem Beginn der Untersuchung wurde das Einverständnis der Eltern eingeholt, das den Kindern die Erlaubnis erteilt, die Screeningfragebögen zu bearbeiten.

Wie aus Tabelle 1 ersichtlich, wurden insgesamt 730 Kinder rekrutiert, wobei 362 deutschsprachige und 368 französischsprachige Versionen des Fragebogens ausgefüllt wurden. Die Gesamtstichprobe umfasste 408 Mädchen und 322 Jungen mit einem durchschnittlichen Alter von 10.96 Jahren ($SD = 1.08$). Der aufgrund subjektiver Einschätzung von Größe und Gewicht durch die Kinder anhand von Referenzwerten (Kromeyer-Hauschild et al., 2001) bestimmte Body-Mass-Index (BMI; kg/m$^2$) betrug 17.37 kg/m$^2$ ($SD = 2.49$), und der mittlere BMI-Standard-Deviation Score (BMI-SDS) -0.23 ($SD = 0.04$). Gemäß der Leitlinien der Arbeitsgemeinschaft Adipositas im Kindes- und Jugendalter (AGA) waren 12.7 % der Kinder dieser Stichprobe untergewichtig (91/730; BMI < 10. BMI-Perzentil), 79.5 % waren normalgewichtig (580/730; 10. – 90. BMI-Perzentil) und 5.3 % waren übergewichtig oder adipös (39/730; BMI > 90. BMI-Perzentil). Diese Prävalenzschätzungen entsprechen somit den Angaben zur Häufigkeit gestörten Essverhaltens sowie von Übergewicht und Adipositas bei Schweizer Kindern (Narring et al., 2004; Olds et al., 2011). Für 2.7 % der Kinder (20/730) konnte der BMI aufgrund fehlender Werte für Größe und/oder Gewicht nicht berechnet werden.

## Eating Disturbances in Childhood-Questionnaire

Die Selbstbeurteilungsskala EDCh-Q wurde von Z. van Dyck und A. Hilbert entwickelt, zur Erfassung von Einstellungen und Verhalten, die für die ARFID kennzeichnend sind. Die Items wurden aufgrund der für das DSM-5 vorgeschlagenen diagnostischen Kriterien für ARFID, sowie der Beschreibungen problematischen Essverhaltens im Kindesalter aus der Forschungsliteratur und der GOS-Kriterien entwickelt. Die Entwicklung des Fragebogens beruhte auf einer Literaturübersicht und auf dem Austausch mit Experten im Bereich kindlicher Essstörungen. Die französischen Itemformulierungen des EDCh-Q wurden durch Übersetzung-Rückübersetzungsverfahren gewonnen.

*Tabelle 1.* Beschreibung der Stichprobe ($N = 730$)

| | % | *N* | Mittelwert | *SD* | Range |
|---|---|---|---|---|---|
| Geschlecht | | | | | |
| Jungen | 44.1 | 322 | | | |
| Mädchen | 55.9 | 408 | | | |
| Alter (Jahre) | | | 10.96 | 1.08 | 8 – 13 |
| Alter Gruppen | | | | | |
| 8 – 10 | 24.2 | 177 | | | |
| 11 – 13 | 75.8 | 553 | | | |
| Sprachregion | | | | | |
| Deutsch | 49.6 | 362 | | | |
| Französisch | 50.4 | 368 | | | |
| BMI ($kg/m^2$) | | | 17.37 | 2.5 | 12.3 – 29.9 |
| BMI-SDS | | | -0.23 | 0.04 | -3.30 – 2.40 |
| BMI-Status | | | | | |
| Untergewicht | 12.7 | 93 | | | |
| Normalgewicht | 79.5 | 580 | | | |
| Übergewicht/Adipositas | 5.3 | 39 | | | |

Für den EDCh-Q wurden 14 Items entwickelt, wobei die beiden letzten Fragen zu Pica und Ruminationsstörung für diese Studie nicht betrachtet wurden. Die verbleibenden 12 Items decken die drei Untergruppen von restriktiven oder vermeidenden Essproblemen ab. Die Kinder werden aufgrund der Items „Ich bin ein wählerischer Esser" und „Essen mit einem bestimmten Geruch, Geschmack, Aussehen, oder einer bestimmten Konsistenz (z. B. knusprig oder weich), mag ich nicht probieren" als selektive Esser eingestuft. Vermeidendes Essverhalten wegen mangelndem Interesse am Essen/ emotionaler Probleme, wird anhand der Items „Ich esse nicht, wenn ich traurig, besorgt oder ängstlich bin" und „Essen interessiert mich nicht", erfasst. Angstbasierte Nahrungsvermeidung wie die funktionelle Dysphagie wird durch die Aussagen „Ich habe Angst, Essen herunterzuschlucken" und „Ich habe Angst, beim Essen zu ersticken oder zu erbrechen" klassifiziert. Zusätzlich wurden 2 Items zur Erfassung von Gewichts- oder Figursorgen entwickelt, da es sich dabei um ein wichtiges Ausschlusskriterium der vermeidend/ restriktiven Ernährungsstörung handelt (American Psychiatric Association, 2012; Nicholls et al., 2000). Jedes Item wird auf einer sieben-stufigen Likert-Skala von „stimmt nie" (= 0) bis „stimmt immer" (= 6) eingeschätzt.

## Auswertung

Die faktorielle Struktur der Items wurde mittels einer Hauptkomponentenanalyse mit orthogonaler Varimax Rotation analysiert. Als Extraktionskriterien wurden die Eigenwerte unter Berücksichtigung des Kaiser-Guttmann-Kriteriums und der Scree-Test herangezogen. Für die psychometrischen Analysen wurde der Anteil fehlender Werte, Itemschwierigkeiten [$p_m$ = Summe der Itemwerte/(N * maximaler Itemwert)], korrigierte Itemtrennschärfen $r_{it}$ (korrigierte Korrelation zwischen dem einzelnen Item und der Skala) und Homogenitäten der Subskalen (durchschnittliche Korrelation zwischen Items) bestimmt. Anhand eines Kolmogoroff-Smirnov-Tests wurde untersucht, ob die Verteilung der Items einer Normalverteilung entspricht. Die internen Konsistenzen der durch die Hauptkomponentenanalyse identifizierten Skalen wurden anhand von Cronbach's $\alpha$ bestimmt.

Zur deskriptiven Untersuchung der Häufigkeit der Symptombilder wurden Kinder mit und ohne Vorkommen von Essstörungssymptomen voneinander getrennt. Dieses Vorgehen wurde aufgrund vorheriger Studien ausgewählt (Carruth et al., 2004; Jacobi et al., 2008; Mascola et al., 2010), die sich vorwiegend mit selektivem Essverhalten beschäftigten. Um die Kriterien eines der drei Subtypen des vermeidend/restriktiven Essverhaltens zu erfüllen, musste das Kind angeben zumindest „oft" das entsprechende Essverhalten aufzuzeigen, was einem cut-off Wert von $\geq 4$ entspricht. Demzufolge wurden die Items dichotomisiert, indem die Kategorien „stimmt nie" bis „stimmt manchmal" in „nein" und „stimmt oft" bis „stimmt immer" in „ja" umkodiert wurden. Anders als bei Carruth und Kollegen (2004) und Jacobi und Kollegen (2008), wurde der Wert „stimmt manchmal" nicht als Vorhandensein problematischen Essverhaltens interpretiert, da die Psychopathologie im Selbstbericht oft überschätzt wird (Fairburn & Beglin, 1994). Ein wichtiges Ausschlusskriterium für diese Störungsgruppe ist das Vorhandensein von Gewichts- oder Figursorgen, weshalb nur Kinder in die Risikogruppe einer vermeidend/restriktiven Ernährungsstörung eingeschlossen wurden, die für dieses Item einen Wert $< 3$ („stimmt manchmal") auf der 7-stufigen Likert Skala angaben.

*Tabelle 2.* Hauptkomponentenanalyse der EDCh-Q Items ($N = 730$)

| | ARFID Subtyp | Faktoren I | II | III | IV | $h^2$ |
|---|---|---|---|---|---|---|
| *Unrotierte Faktorenlösung* | | | | | | |
| Eigenwerte | | 2.25 | 1.25 | 1.19 | 1.17 | |
| % Varianz | | 22.54 | 12.54 | 11.93 | 11.69 | |
| *Rotierte Faktorenlösung* | | | | | | |
| Eigenwerte | | 1.59 | 1.55 | 1.37 | 1.36 | |
| % Varianz | | 15.91 | 15.54 | 13.66 | 13.58 | |
| *Faktorenladungen nach Rotation* | | | | | | |
| Nahrungsvermeidung | | | **.787** | | .184 | .66 |
| Interesse am Essen | | .152 | **.739** | | | .57 |
| Emotionsbedingte Essensvermeidung | | .146 | **.534** | .151 | | .33 |
| Untergewicht | | | .252 | **.766** | | .66 |
| Wunsch zuzunehmen | | | | **.847** | | .73 |
| Selektives Essverhalten | | **.611** | .131 | | | .40 |
| Verweigerung neuer Nahrungsmittel | | **.821** | | | | .69 |
| Angst vor dem Schlucken | | | | | **.815** | .62 |
| Angst vor dem Ersticken/Erbrechen | | .109 | | | **.771** | .67 |
| Sensorische Nahrungsvermeidung | | **.690** | | .151 | .213 | .55 |

*Anmerkungen:* Hauptkomponentenanalyse mit orthogonaler Varimax-Rotation. Kaiser-Normalisierung. Es werden nur Ladungen >.10 angegeben. Die höchste Ladung pro Item ist fett gedruckt.

Anhand von Chi-Quadrat-Tests auf Unabhängigkeit wurde ermittelt, ob sich die drei Subtypen hinsichtlich Geschlecht, Alter oder BMI-Kategorie signifikant voneinander unterscheiden. Wegen einem Häufigkeitswert $< 5$ für übergewichtige Kinder bei zwei Subtypen, wurden zusätzliche Chi-Quadrat-Tests auf Unabhängigkeit mit zwei BMI-Kategorien durchgeführt (Untergewicht, Normalgewicht/Übergewicht).

Alle psychometrischen Analysen wurden sowohl für die Gesamtstichprobe, als auch separat für beide Sprachen durchgeführt. Ein zweiseitiges $\alpha$-Niveau von .05 wurde allen statistischen Analysen zugrunde gelegt. Alle Analysen wurden mit SPSS 20.0.0 durchgeführt (SPSS Inc., Chicago, Illinois, USA).

## Ergebnisse

*Faktorielle Struktur.* Zur Bestimmung der faktoriellen Struktur der Items wurde eine Hauptkomponentenanalyse durchgeführt (siehe Tab. 2). Der Kaiser-Meyer-Olkin-Koeffizient ergab einen Wert von .66, was die Durchführung der exploratorischen Faktorenanalyse rechtfertigte. Bei einer Stichprobengröße von $n = 730$ ergab sich als Grenzwert für signifikante Faktorladungen .21 (Bühner, 2004). Die Hauptkomponentenanalyse mit Eigenwerten >1 (Extraktionskriterium) zeigte, dass sich die Items zu 4 Faktoren aggregieren ließen, die 58.7 % der Gesamtvarianz erklärten. Nach einer Varimax-Rotation erklärte der erste Faktor 15.9 %, der zweite 15.5 %, der dritte 13.7 % und der vierte 13.6 % der Varianz der Items. Faktor I setzte sich aus drei Items zusammen, die sich auf eine (emotionale) Nahrungsvermeidung beziehen. Faktor II beinhaltete drei Items des Subtypen selektiven Essverhaltens. Faktor III umfasste zwei Items zu Untergewicht. Faktor IV setzte sich aus den beiden Items einer Nahrungsvermeidung aufgrund von Angst zusammen. Zusätzlich wurden getrennte Hauptkomponentenanalysen für die deutsche und französische Version des EDCh-Q durchgeführt, wobei die gleiche vierdimensionale Struktur des EDCh-Q für beide Subgruppen gefunden wurde.

*Itemanalysen.* Da sich für die Itemcharakteristika keine bedeutsamen Unterschiede zwischen der französischsprachigen und der deutschsprachigen Version gefunden haben, werden im Folgenden nur die Ergebnisse für die Gesamtstichprobe angeführt. Der Anteil fehlender Werte auf Itemebene war mit 0.0 % – 0.8 % ($n = 33$) an nicht beantworteten Items gering. Insgesamt wurden 58.1 % der Items mit 0 („stimmt nie“) beantwortet, wobei dies zwischen den Items variierte (28.4 % – 91.4 %). Selektives Essverhalten wurde am wenigsten mit „stimmt nie“ beantwortet und die stärksten Bodeneffekte fanden sich für die Items, die sich auf eine Nahrungsvermeidung aufgrund spezifischer Ängste beziehen. Die Verteilungen der Items wichen alle signifikant von einer Normalverteilung ab (alle $p < .001$; Tabelle 3) und signifikante Schiefewerte zeigten linkssteile Verteilungen an, was sich auch in hohen Itemschwierigkeiten widerspiegelte. Auch

*Tabelle 3.* Verteilung der Items ($N = 730$)

| EDCh-Q Item | Kolmogorov-Smirnoff | | | Schiefe | Kurtosis |
|---|---|---|---|---|---|
| | D | *Df* | *p* | z-Wert | z-Wert |
| *Skala unangemessene Nahrungsaufnahme* | | | | | |
| Nahrungsvermeidung | .36 | 727 | .00 | 19.89*** | 16.38*** |
| Interesse am Essen | .32 | 729 | .00 | 15.43*** | 7.03*** |
| Emotionsbedingte Essensvermeidung | .24 | 724 | .00 | 10.33*** | -2.86** |
| *Skala selektives Essen* | | | | | |
| Selektives Essverhalten | .20 | 728 | .00 | 7.61*** | -3.95*** |
| Verweigerung neuer Nahrungsmittel | .23 | 728 | .00 | 9.67*** | -2.54* |
| Sensorische Nahrungsvermeidung | .21 | 729 | .00 | 9.13*** | -2.05* |
| *Skala funktionelle Dysphagie* | | | | | |
| Angst vor dem Ersticken/Erbrechen | .45 | 724 | .00 | 35.87*** | 59.57*** |
| Angst vor dem Schlucken | .51 | 727 | .00 | 61.52*** | 189.95*** |
| *Skala Gewicht* | | | | | |
| Untergewicht | .35 | 727 | .00 | 16.96*** | 6.32*** |
| Wunsch zuzunehmen | .45 | 728 | .00 | 28.70*** | 32.54*** |

*Anmerkungen:* $*p < .05$, $**p < .01$, $***p < .001$

*Tabelle 4.* Itemschwierigkeiten und Trennschärfen des EDCh-Q ($N = 730$)

| EDCh-Q Items | *M* | *SD* | $p_m$ | $r_{it}$ |
|---|---|---|---|---|
| *Skala unangemessene Nahrungsaufnahme* | | | | |
| Nahrungsvermeidung | 0.78 | 1.28 | .13 | .36 |
| Interesse am Essen | 1.08 | 1.53 | .18 | .32 |
| Emotionsbedingte Essensvermeidung | 1.75 | 2.13 | .29 | .24 |
| *Skala selektives Essen* | | | | |
| Selektives Essverhalten | 2.10 | 2.00 | .35 | .30 |
| Verweigerung neuer Nahrungsmittel | 1.74 | 1.99 | .29 | .41 |
| Sensorische Nahrungsvermeidung | 1.74 | 1.84 | .29 | .37 |
| *Skala funktionelle Dysphagie* | | | | |
| Angst vor dem Ersticken/Erbrechen | 0.45 | 1.19 | .08 | .31 |
| Angst vor dem Schlucken | 0.18 | 0.78 | .03 | .31 |
| *Skala Gewicht* | | | | |
| Untergewicht | 1.13 | 1.83 | .19 | .36 |
| Wunsch zuzunehmen | 0.61 | 1.48 | .10 | .36 |

*Anmerkungen: M:* Mittelwert, *SD:* Standardabweichung, $p_m$: Itemschwierigkeit, $r_{it}$: Trennschärfe.

im Hinblick auf den Exzess fielen die Itemverteilungen weitgehend signifikant hochgipflig aus (siehe Tab. 3).

Die Ergebnisse der Itemschwierigkeiten und Trennschärfen befinden sich in Tabelle 4. Insgesamt lagen die Schwierigkeiten der Items im mittleren bis hohen Bereich (vgl. Bortz & Döring, 2006), mit einer eher geringen Wahrscheinlichkeit für hohe Ausprägungen der Essstörungssymptome im Selbstbericht ($.03 \leq p_m \leq .35$). Die Trennschärfekoeffizienten lagen im mittleren bis hohen Bereich ($.30 \leq r_{it} \leq .41$), mit Ausnahme des Items das sich auf eine emotionsbedingte Nahrungsvermeidung bezieht und eine niedrigere Trennschärfe aufzeigt ($r_{it} = .24$). Die Homogenität der einer Skala zugeordneten Items stellte sich als optimal heraus ($.20 \leq r \leq .36$).

*Reliabilität.* Die internen Konsistenzen der durch die Hauptkomponentenanalyse bestätigten Skalenstruktur, fielen mit Werten von $.45 \leq \alpha \leq .55$ gering aus. Alle Interkorrelationen zwischen den vier Faktoren waren signifikant, mit einer durchschnittlichen Korrelation zwischen Subskalen von $r = .17$ ($r = .11 - .25$). Es wurden vergleichbare Werte in beiden Sprachräumen ermittelt.

*Prävalenz.* Am häufigsten wurden Symptome von SE beobachtet (20.3 %; $n = 148$), gefolgt von FAED (7.9 %;

$n = 58$). Am seltensten wurden Symptome einer FD festgestellt (1.6 %; $n = 12$). Einige Kinder wiesen komorbid Symptome mehrerer Subtypen auf (3.3 %; $n = 24$), wobei es sich hauptsächlich um die Kombination zwischen FAED und SE handelt (2.5 %; $n = 18$). Ein Kind erfüllte die Bedingungen aller drei Screeningkategorien. Insgesamt gaben 29.8 % ($n = 218$) der Kinder an, mindestens eines der drei Essprobleme regelmäßig aufzuzeigen.

Für keinen der drei Subtypen bestand ein signifikanter Unterschied zwischen Jungen und Mädchen (FAED: $\chi^2(1, 730) = 1.60$, $p = .206$; SE: ($\chi^2(1, 730) = 0.06$, $p = .812$); FD: ($\chi^2(1, 730) = 2.52$, $p = .113$)), sowie Altersklassen (FAED: $\chi^2(1, 730) = 3.59$, $p = .058$; SE: ($\chi^2(1, 730) = 0.70$, $p = .404$); FD: ($\chi^2(1, 730) = 0.01$, $p = .951$)). Es zeigten 20.9 % der untergewichtigen und 6 % der normal- und übergewichtigen Kinder FAED auf (BMI: $\chi^2(1, 730) = 24.25$, $p \leq .001$). Weder für SE, noch für FD zeigten sich signifikante Unterschiede zwischen den BMI-Kategorien.

## Diskussion

Der EDCh-Q ist ein kurzer Screeningfragebogen zur Identifikation von Einstellungen und Verhaltensweisen, die für Essprobleme in der mittleren Kindheit charakteristisch sind, und sich durch ein vermeidend/restriktives Essverhalten kennzeichnen. Die vorliegende Studie bietet erste psychometrische Daten des EDCh-Q anhand einer allgemeinen, schulbasierten Stichprobe mit 8 – 13 jährigen Jungen und Mädchen aus der Schweiz. Der Anteil fehlender Werte war sehr gering und kann als unproblematisch interpretiert werden (Tabachnick & Fidell, 2007), was für eine gute Anwendbarkeit und Akzeptanz des Messinstruments spricht. Die exploratorische Faktorenanalyse legte eine 4-Faktoren-Lösung mit einer substantiellen Aufklärung an Gesamtvarianz nahe. Alle Items konnten eindeutig den anhand der Literatur entwickelten Skalen zugeordnet werden, was die Existenz der drei klinischen Profile einer vermeidend/restriktiven Ernährungsstörung bestätigt. Die gleiche vierdimensionale Struktur wurde für beide Sprachversionen gefunden, was darauf hindeutet dass die faktorielle Struktur des EDCh-Q wenig beeinflusst wurde von den sprachlichen Unterschieden. Aufgrund der geografischen Nähe zwischen dem deutsch- und französischsprachigen Rekrutierungsraum, kann man davon ausgehen dass keine bedeutsamen kulturellen Unterschiede vorlagen.

Als Haupttestmerkmale nach der klassischen Testtheorie wurden Schwierigkeit, Trennschärfe und Reliabilität des EDCh-Q berechnet. Die hohen Itemschwierigkeiten, sowie die linkssteilen und hochgipfligen Verteilungen der Items, deuten darauf hin dass der EDCh-Q konzeptgemäß schwerwiegende Essprobleme erfasst. Es empfiehlt sich also, die statistische Auswertung auf die Verletzung der Normalverteilungsvoraussetzung abzustimmen, entweder anhand größerer Stichproben oder mit der Anwendung nichtparametrischer Tests (vgl. Bortz & Döring, 2006).

Die mittels einer exploratorischen Faktorenanalyse bestätigten Dimensionen wiesen insgesamt geringe interne Konsistenzen auf. Alpha-Werte unter .6 können als Hinweis darauf interpretiert werden, dass die betreffende Skala entweder Items enthält die nicht zu den übrigen passen, oder aber faktisch aus mehreren Dimensionen besteht (Bortz & Döring, 2006). Eine Verbesserung des Alpha-Wertes durch Itemausschlüsse war nicht möglich und insgesamt wurden zufriedenstellende durchschnittliche Korrelationen zwischen Items einer Skala gefunden. Die geringen internen Konsistenzen spiegeln vermutlich die geringe Anzahl an Items pro Subskala wider, da die Konsistenzanalyse insbesondere bei heterogenen, mehrdimensionalen Tests die Reliabilität unterschätzen kann, was für einen diagnostisch orientierten Screeningfragebogen nicht unüblich ist.

In Übereinstimmung mit anderen Studien hat die vorliegende Untersuchung gezeigt, dass SE auch bei älteren Kindern und im Selbstbericht ein relativ häufiges Phänomen darstellt. So fanden zum Beispiel Jacobi und Kollegen (2008) in einer gleichaltrigen Stichprobe, dass 18 % der Jungen und 19 % der Mädchen von ihren Eltern als wählerische Esser bezeichnet wurden. Ähnliche Prävalenzraten wurden in einer Untersuchung 11-jähriger Kinder berichtet (Mascola et al., 2010). Diese Befunde deuten darauf hin, dass SE bei vielen Kindern keine vorübergehende Entwicklungsphase darstellt. Es gibt bisher noch keine Angaben zur Prävalenz von FAED oder FD in nicht-klinischen Stichproben. Im Vergleich zu Untersuchungen an Patientenstichproben, waren Symptome von FAED in der vorliegenden Studie weniger verbreitet (Cooper et al., 2002; Nicholls et al., 2011). Dieser Befund kann dadurch erklärt werden, dass es sich um schwerwiegende Symptome handelt, mit häufiger komorbider Erscheinung körperlicher Erkrankungen (Bryant-Waugh & Lask, 2007; Nicholls & Bryant-Waugh, 2008). Aufgrund der hohen Belastung suchen Betroffene häufiger professionelle Hilfe auf als dies beispielsweise für SE der Fall ist, was die stärkere Vertretung des Symptombildes in klinischen Stichproben erklärt. Das Risiko einer FD wird auch in klinischen Stichproben sehr selten beobachtet (Cooper et al., 2002).

Der EDCh-Q wurde nicht entwickelt um Essstörungsdiagnosen zu vergeben, sondern um Symptome problematischen Essverhaltens zu identifizieren, weshalb man davon ausgehen kann dass die hier berichteten Häufigkeiten im Selbstbericht höher sind als solche, die anhand diagnostischer Interviews festgestellt werden (Decaluwé & Braet, 2004; Fairburn & Beglin, 1994). Weitere, zweistufige epidemiologische Studien mit nachfolgenden strukturierten diagnostischen Interviews sind geplant, um die Prävalenz restriktiver/vermeidender Ernährungsstörungen im Kindesalter zu überprüfen.

Es wurden für keinen der Subtypen Altersunterschiede nachgewiesen. Übereinstimmend fanden Jacobi und Kollegen, dass wählerisches Essen bei 8 – 12-jährigen gleichermaßen vorhanden ist als bei präadoleszenten Kindern (Jacobi et al., 2008). Im Einklang mit vorhergehenden Untersuchungen (Kröller & Warschburger, 2011) deuten diese Befunde darauf hin, dass auffällige Essverhaltensmuster bei Kindern im Schulalter nicht selten vorhanden sind und oftmals über das Alter hinweg stabil bleiben. Es konnten keine signifikanten Unterschiede zwischen Mädchen und Jungen gefunden werden, was die Annahme diverser Studien unterstützt, dass das Geschlechterverhältnis von Essproblematiken im Kindesalter ausgeglichener ist als bei Essstörungen im Erwachsenenalter (Bryant-Waugh & Lask, 1995; Jacobi et al., 2008; Lask & Bryant-Waugh, 1992; Peebles et al., 2006; Rosen, 2003). Insgesamt ergab sich ein ausgeprägter Zusammenhang zwischen selbstberichtetem Untergewicht und dem Vorhandensein von FAED: Kinder mit einem niedrigen BMI gaben häufiger an, Symptome einer FAED aufzuzeigen. Im Einklang mit diesem Ergebnis wird in der klinischen Praxis für FAED ein Ausmaß an Mangelernährung und Gewichtsverlust berichtet, das vergleichbar ist mit dem das man bei AN Patienten vorfindet (Higgs et al., 1989; Nicholls et al., 2002).

Die Interpretation der Ergebnisse sollte im Kontext einer Berücksichtigung der Stärken und Schwächen der Untersuchung erfolgen. Zu den Begrenzungen zählt die Tatsache, dass Körpergewicht und Körpergröße ausschließlich im Selbstbericht erhoben wurden; subjektiv gewonnene Daten unterschätzen den objektiv gemessenen Wert oftmals (Glaesmer & Brähler, 2002). Dies kann dazu geführt haben, dass die Effekte von BMI auf die Essstörungssymptomatik unterschätzt wurden.

Weiterhin zu beachten ist dass Essstörungssymptome im Selbstbericht erfasst wurden. Es wurde gezeigt, dass Kinder im Schulalter (insbesondere > 7 Jahre) in Fragebogenerhebungen reliable Angaben zu ihrer eigenen Gesundheit machen können (Riley, 2004). Dennoch wäre es für weitere Forschung wünschenswert, Essprobleme zusätzlich im Elternbericht und auch in objektiven, laborbasierten Settings zu erheben. Dies wird vor allem vor dem Hintergrund deutlich, dass geringe Übereinstimmungen zwischen selbst- und fremdberichtetem Essverhalten beobachtet wurden wenn tatsächlich problematisches Essverhalten vorlag (Steinberg et al., 2004). Anzumerken ist auch die Tatsache, dass die Kinder, die im EDCh-Q mit auffälligen Scores abgeschnitten haben bisher noch nicht nachuntersucht wurden, man also noch nicht weiß wie viele dieser Kinder tatsächlich mit Essproblemen belastet sind und einer Behandlung zugeführt werden sollten.

Positiv anzumerken ist die große und breit angelegte, schulbasierte Stichprobe dieser Untersuchung, die Kinder aus unterschiedlichen Sprachregionen (französisch- und deutschsprachige Kantone der Schweiz) umfasst. Nichtsdestotrotz ist es nicht auszuschließen, dass eher Kinder ohne schwerwiegende Ess- oder Gewichtsprobleme und deren Eltern einwilligten, an einer Fragebogenuntersuchung zum Thema Essverhalten teilzunehmen.

Zusammenfassend deutet die vorliegende Untersuchung darauf hin, dass Symptome von vermeidendem und restriktivem Essverhalten ohne Gewichts- oder Figursorgen in der mittleren Kindheit nicht selten vorhanden sind und dass sich die drei in der Literatur beschriebenen Symptombilder unterscheiden. Die Entwicklung und Etablierung eines einfach einsetzbaren Instrumentes ist relevant um Kinder mit erhöhten Risikofaktoren zu identifizieren und wenn nötig einer adäquaten Behandlung zuzuführen, da leichtere oder früh diagnostizierte Störungen einfacher behandelt werden können. Die Entwicklung des EDCh-Q ist ein erster Schritt um diese Lücke zu schließen. Weitere Untersuchungen zur Ermittlung der konvergenten und diskriminanten Validität des Fragebogens sind in Planung.

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