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ScienceDirect
Appetite 49 (2007) 476–485
Appetite
www.elsevier.com/locate/appet

Research Report

Individual and family eating patterns during childhood and early adolescence: An analysis of associated eating disorder factors

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Received 27 April 2006; received in revised form 28 February 2007; accepted 12 March 2007

Abstract

To examine whether there is an association between individual and family eating patterns during childhood and the likelihood of developing an eating disorder (ED) later in life. The sample comprised 261 eating disorder patients [33.5% [$N = 88$] anorexia nervosa (AN), 47.2% [$N = 123$] with bulimia nervosa (BN) and 19.3% [$N = 50$] with Eating Disorders Not Otherwise Specified (EDNOS)] and 160 healthy controls from the Province of Catalonia, Spain, who were matched for age and education. All patients were consecutively admitted to our Psychiatry Department and were diagnosed according to DSM-IV criteria. Participants completed the Early Eating Environmental Subscale of the Cross-Cultural (Environmental) Questionnaire (CCQ), a retrospective measure of childhood eating attitudes and behaviours. In the control group, also the General Health Questionnaire-28 (GHQ-28) was used. During childhood and early adolescence, the following main factors were identified to be linked to eating disorders: eating excessive sweets and snacks and consuming food specially prepared for the respondent. Conversely, regular breakfast consumption was negatively associated with an eating disorder. Compared to healthy controls, eating disorder patients report unfavourable eating patterns early in life, which in conjunction with an excessive importance given to food by the individual and the family may increase the likelihood for developing a subsequent eating disorder.

Keywords: Eating disorders; Eating patterns; Anorexia nervosa; Bulimia nervosa; Eating disorders not otherwise specified

Introduction

A recent systematic review of the risk factor literature suggested that early eating and gastrointestinal difficulties may be developmental factors of relevance for eating disorders (Jacobi, Hayward, de Zwaan, Kraemer, & Agras, 2004). Early problematic eating patterns (e.g. struggle and conflict over eating) are associated with unhealthy eating behaviours later in the development (Kortegaard, Hoerder, Joergensen, Gillberg, & Kyvik, 2001). In a retrospective study comparing 51 teenage participants with anorexia nervosa (AN) and 51 controls, extreme feeding problems were more commonly reported by mothers of individuals with AN than of controls (Gillberg, Gillberg, Rastam, & Johansson, 1996).

Early childhood eating and problems for bulimia nervosa (BN) have been contradictory. While one study (Micali et al.[1]) of sister discordant for eating disorders found that cases of BN were more often overweight with

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[1]Micali, N., Holliday, J., Karwautz, A., Haidvogel, M., Wagner, G., Fernandez-Aranda, F., Badia, A., Gimenez, L., Solano, R., Breceli-Anderluh, M., Collier, D., & Treasure, J. L. (Accepted for publication). Developmental risk factors in eating disorders-childhood feeding and weight: A discordant sister-pairs comparison. *Psychotherapy and Psychosomatics*.

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doi:10.1016/j.appet.2007.03.004

less picky eating in childhood compared to their healthy siblings during childhood, Marchi and Cohen's (1990) study indicated that pica, early digestive difficulties and weight loss attempts were associated with subsequent bulimic behaviours. It should however be acknowledged that the research into early patterns of eating behaviour has been limited as the size of prospective longitudinal studies is rarely large enough to have sufficient power to be confident in the findings for eating disorders and the detail about the form of early eating risk may be insufficient.

Family eating patterns during childhood and early adolescence

Research has shown that children's attitudes toward food and children's evaluation of satiety are influenced by their parents and their family environment. Parents provide access to foods in the home, may operate as models and offer encouragement/discouragement for specific eating behaviours (Birch & Fisher, 1998; Birch et al., 2001). Accordingly, great difficulties over control issues between parents and children at mealtimes has often been linked to problematic food consumption in children, weight fluctuations and concerns about food, all of which could in turn result in an eating disorder later in the individual's development (Birch & Fisher, 1998). However research has not yet addressed whether distinct parental attitudes to food are associated to different eating disorder subtypes.

An integrative model for risk factors in eating disorders

Previously, risk factors for eating disorders have been proposed mainly from a particular theoretical viewpoint (e.g. biological, psychodynamic model, cognitive-behavioural), but not so commonly from an integrative standpoint (e.g. biopsychosocial model) (Jacobi et al., 2004). Yet, important developments in the level of understanding of the causes of complex disorders such as psychiatric problems is the recognition that simple unidimensional models of aetiology such as for instance genetic factors (Ribases et al., 2004, 2006) are insufficient and that rather complex models with interactions between a genetic predisposition and environmental precipitants may be necessary (Bulik, Sullivan, & Kendler, 2003; Karwautz et al., 2001). Interesting examples of research which have used such a paradigm have revealed that the short (inactive) form of the MAOI in combination with early adversity is associated with conduct disorder (Caspi et al., 2002) and that the short (inactive) form of the serotonin reuptake (5HTT) in combination with stressful life events leads to depression (Caspi et al., 2003). In order for this type of approach to be adopted in the field of eating disorders it is important to have tools, which clearly define the environmental risk factors of relevance. For this reason, effective prevention and treatment programmes require clear evidence-based models of aetiology.

Taken together the literature suggests that an unhealthy eating pattern early in life and parental influence upon children's eating styles can be part of the developmental trajectory into eating disorders. Nevertheless, it should be acknowledged that little previous research has addressed this topic, and that the few findings that have been reported have been conflicting. Furthermore preceding research has suffered from several fundamental shortcomings, including small sample sizes, lack of control groups, insufficient information on the methodology employed. Moreover, the reported risk factors are based on only a few studies mostly conducted with individuals from Anglo-Saxon populations and therefore their level of classification may vary with the appearance of more recent studies. For this reason, replication studies are required for the majority of the categorized risk factors.

Aims of the study

The aim of the present study therefore was to overcome the limitations of the previous studies by employing a large sample size of patients and healthy controls. Moreover, the present study aimed to develop an instrument, sensitive to the environmental factors associated with the development of eating disorders, which could be used in studies of gene environment interaction and to examine this instrument as a tool to measure related factors in individuals with eating disorders.

The specific objectives were to examine whether individual and family eating patterns and food choices during childhood and early adolescence were associated with disordered eating behaviours and weight (assessed through Body Mass Index [BMI]). We hypothesized that compared to the control sample, patients with eating disorders would exhibit more problematic eating behaviours early in life and that the family of these patients would exhibit more dysfunctional eating behaviours and attitudes towards food than the family members of the controls.

Method

Participants

The present study employed a case-control design. Entry into the study was between March 2001 and September 2002. The sample comprised 261 eating disorder patients (33.5% [$N = 88$] AN, 47.2% [$N = 123$] with BN and 19.3% [$N = 50$] with EDNOS) and 160 healthy controls. All clinical participants were diagnosed according to DSM-IV criteria (APA, 1994), using a semi-structured clinical interview [SCID-I] (First, Gibbon, Spitzer, & Williams, 1996), conducted by experienced psychologists and psychiatrists. All interviewers were trained in the administration of the SCID-I, although formal interrater reliability was not computed for this study. Clinical participants were consecutive referrals for assessment and treatment at the Department of Psychiatry of the University Hospital of Bellvitge in Barcelona.

The mean age of the total sample was 25.1 years (SD = 5.6). Age did not differ significantly between the

eating disordered patients and the controls (ED cohort: mean = 24.8, SD = 5.6; control group: 25.6, SD = 5.5; $p = 0.62$). However, the two groups differed on gender, with the eating disorder group comprising significantly more females than the control group (ED cohort: female = 93.89%; control group: female = 85.53%; $p = 0.02$). Furthermore in comparison to the control group, significantly more eating disorder patients were employed (ED cohort: employed = 62.45; control group: employed = 45.75; $p = 0.006$). The BMI of the patients differed between the groups, as would be expected with the AN group exhibiting the lowest BMI (mean = 16.8; SD = 1.95) and those with BN the highest BMI (mean = 23.3, SD = 4.49; $p = <0.0005$). Most of the participants had grown up in urban areas (75.3%).

The mean duration of the eating disorder group was 6.37 years (SD = 4.77). The mean number of previous treatments was 0.82 (SD = 0.91). They reported a weekly average of 4.13 binges (SD = 5.62) and 6.58 vomiting episodes (SD = 8.84). The inclusion criteria for the study for the eating disorder sample were: (a) older than 18 years, (c) meeting DSMIV-TR criteria (APA, 2000a) for AN, BN or for EDNOS. Conversely, patients were excluded from the study if they met any of the following criteria: (a) missing values for any diagnostic items; (b) unable to complete the assessment because of cognitive impairment; or, (c) current psychotic disorder. For the present analysis, the following individuals had to be excluded from an initial sample of 283 patients: (a) patients ($N = 18$) with missing values for any diagnostic tools; (b) cognitive impairment ($N = 2$); (c) comorbid psychotic disorder ($N = 2$). Disposition decisions were made by psychologists or psychiatrists who completed the anamnesis together with the treatment team according to published treatment guidelines (APA, 2000b).

Healthy controls were recruited from individuals visiting the hospital for routine blood tests and were asked to volunteer in a study of environmental factors associated with the development of eating disorders. All controls were from the same catchment areas as index patients. The final control group included 160 volunteers who were matched for age and education. The exclusion criteria for the control group were: (a) younger than 18 years; (b) to have lifetime history of health or mental illnesses (including eating disorders), screened by the General Health Questionnaire-28 (Goldberg, 1981) and the DSM-IV criteria (APA, 1994). From an initial sample of 166 controls, 6 participants were excluded, who had had a lifetime eating disorder.

All participants provided informed consent and the study was approved by the Ethics committee of our University Hospital.

Assessment

The early Eating Environmental Subscale of the Cross-Cultural (Environmental) Questionnaire (CCQ)

This retrospective self-administered questionnaire entails a total of 51 items. It was developed by an expert group from various European countries in order to detect environmental factors associated with the development of eating disorders (childhood eating patterns, meaning and value of food, family style, independence, parenting, self-development and social ideals of thinness and fitness and activity). The CCQ was based on the major instruments in the field of eating disorders, which are the Oxford Risk Factor Interview (Fairburn et al., 1998; Fairburn, Welch, Doll, Davies, & O'Connor, 1997) and the McKnight Risk Factor Interview (Shisslak et al., 1999). We were particularly interested to include items of these domains that might be sensitive to differences within the European cultures. The questionnaire was initially piloted in several patients and amended in the interest of clarity and coherence. In the current study, only the 16 items of the food and eating family style domain were considered. These items were developed following focus groups of patients with eating disorders who were asked about their early eating behaviours (i.e. before age 12) from the UK and Spain and from a consensus meeting of expert clinician group from Spain, the UK, Italy, Austria, Finland, Slovenia and France (Healthy Eating Consortium) after a review of the literature and the major eating disorder risk factor instruments. The instrument is shown in the Appendix and a copy of the whole questionnaire can be requested from the first author.

Reliability of the CCQ

The internal consistency of the derived scales fluctuated between good and very good (Cronbach α coefficients between 0, 75 and 0, 88). The dimensional scores derived from the factorial analysis discriminated adequately between patients and controls ($p \leqslant 0.05$) and the global diagnostic capacity of the test was found to be satisfactory (area below the ROC curve $\geqslant 0.80$) (Bonillo, Granero, Krug, Anderluh, Bellodi, Collier, Karwautz, Nacmias, Treasure & Fernandez-Aranda)[2]. Test–retest reliability was measured through κ coefficients (for categorical items) and intraclass correlation coefficients (for quantitative items) for the domain assessed in this study. A very high agreement, with an average value of 0.92 (95% confidence interval: 0.89–0.93) was found for all the items.

General Health Questionnaire-28 [GHQ-28] (Goldberg, 1981)

The GHQ-28 is a self-report questionnaire that has been designed to detect and assess individuals with an enlarged probability of a present psychiatric disorder. The GHQ-28 comprises four subscales: Somatic Symptoms, Anxiety and Insomnia, Social Dysfunction, and Severe Depression. In the current study, the Likert scoring procedure (0–3) was

[2]Bonillo, A., Granero, R., Krug, I., Anderluh, M., Bellodi, L., Collier, D. A., et al. (2006). Psychometric Reliability and Validity of the Cross-Cultural (Environmental) Questionnaire (CCQ). In *Poster presented at the Hispano Latinoamericano Congress on eating disorders*, 6th June, Barcelona.

used. An SPSS computer code was utilized to score the GHQ-28, which generated new variables. A cut off score of 6/7 (6 = no case; 7 = case) was employed for the new total subscale variables in order to exclude individuals with an elevated likelihood of a present psychiatric disorder. In previous studies this cut off score has yielded a sensitivity of 76.9% and a specify of 90.2% (Lobo, Perez-Echeverria, & Artal, 1986; Molina et al., 2006).

In addition, the eating disorder group was assessed by a face-to-face structured clinical interview regarding their eating disorder and psychopathological symptoms and their family history of eating disorders (Fernandez-Aranda & Turon, 1998; First et al., 1996).

Procedure

Upon presentation at the eating disorders unit, experienced psychologists and psychiatrists conducted a 2 h structured diagnostic face to face interview to measure eating disorder symptoms and psychopathological traits. Eating-disorder diagnoses were based on this interview and were consensually derived among members of the clinical team who had participated in the assessment. Finally, participants completed the questionnaires individually in a room prior to starting the treatment. For the control group, screening for a current or lifetime eating disorder and/or general distress was measured by self-report with the GHQ-28 (Goldberg, 1981) and eating disorder DSM-IV criteria (APA, 1994). An information sheet at the start of the questionnaire informed the participants about the purpose of the study and assured confidentiality of the results. Furthermore, it was emphasized that participation in the study was completely voluntary and that participants were free to withdraw from the study at any time.

Statistical analysis

The statistical analysis was carried out with the SPSS program, version 12 for Windows. All significance tests were two-tailed. Primarily, various multiple logistic regression models were conducted. These models assessed the extent to which different eating patterns during childhood and early adolescence (independent variables) could predict the presence or not of a subsequent eating disorder (dependent variable). Moreover, differential analyses were carried out to distinguish between the different types of diagnoses, whereby each disorder was compared with the control group. Our aim was to choose the best model. This was an exploratory analysis and therefore a wide group of predictors was employed using automatic sequential procedures for the input (entry) and output (exit) of variables (backstep and forwardstep procedures). The selected models, which had been controlled for sex and age, included all the predictors that were significant ($p<0.05$) or marginally significant ($p<0.10$). The models' ability to discriminate between the groups was also assessed with the area under the receiver operator curve (ROC). The models' calibration was examined using the Hosmer and Lemeshow test. Finally, Nagelkerke R^2 was used to estimate how much variance was accounted for in the models. Secondly, multiple linear regression models were carried out to determine which factors of the food style (independent variables) could best predict the participants' BMI (dependent variable). These analyses also used automatic sequential procedures for the input–output of the variables and were also controlled for sex and age. Diverse models were obtained for the different DSM-IV diagnoses (APA, 1994) and the control group. The global predictive capacity of the selected models was valued with the adjusted R^2 coefficient.

Results

The effect of individual and family eating patterns on eating disorders

Table 1 demonstrates the results of the four models, which measure the effect of eating patterns during childhood and adolescence on the presence of an eating disorder later in life. The first model (dependent variable: controls versus cases), indicates that living with a sibling(s) increases the incidence of having an eating disorder (OR = 2.14, 95% CI: 1.01–4.52). This occurrence is augmented when food was specially prepared for the respondent (OR = 6.46) and when participants ate fatty/sugary snacks (OR = 1.99 if the frequency was for eating 2/6 times a week and OR = 3.85 for a daily frequency). On the other hand, having the first meal of the day before attending school lessons (not skipping breakfast) diminished the incidence of developing an eating disorder (OR = 1/0.298 = 3.36). The results of the second model (dependent variable: controls versus people with AN), indicate that the occurrence of AN is increased when grandparent(s) lived at home (OR = 2.09), when food was specially prepared for the respondent (OR = 7.52) and when the children ate a lot of fatty/sugary snacks (OR = 2.06 for eating moderately and OR = 4.01 for eating a lot). There was a trend for children with restricted access to salty/sugary snacks to have a lower incidence of presenting AN (OR = 1/0.464 = 2.16).

The third model (dependent variable: controls versus people with BN) found that the associated BN factors are: food prepared specially for the respondent (OR = 4.81), and eating a lot of fatty/sugary snacks (OR = 1.94 if the frequency was for eating 2/6 times a week and OR = 3.15 for a daily frequency). Having the first meal of the day before attending school lessons was however found to reduce this occurrence (OR = 1/0.247 = 4.05). Fathers appeared to have an impact on the increased incidence of BN. For example fathers with a greater value placed on food augmented the likelihood (OR = 2.30) for BN and there was a trend for fathers' attention to healthy eating to increase the occurrence.

The final model (dependent variable: controls versus EDNOS) shows that the incidence of presenting an

Table 1
Logistic regression (adjusted by sex and age): effect of food style on the disorder

Independent variables	*B*	*P*	e^B	95% CI	For e^B	Adjustment[a]
Model: controls versus cases ($N = 421$)						
Sibling(s) lived at home	0.760	0.046	2.138	1.01	4.52	AUC = 0.732
Grandparent(s) lived at home	0.489	0.086	1.631	0.93	2.85	H–L = 0.641
Had first meal of day before lessons	−1.212	0.001	0.298	0.15	0.59	$R^2 = 0.215$
Food prepared specially for respondent	1.865	0.004	6.455	1.83	22.73	
Father valued food more than others	0.567	0.028	1.763	1.06	2.92	
Freq. ate fatty/sugary snacks (1)[b]	0.689	0.006	1.992	1.22	3.26	
Freq. ate fatty/sugary snacks (2)[c]	1.349	0.001	3.853	1.89	7.86	
Model: controls versus anorexics ($N = 243$)						
Grandparent(s) lived at home	0.735	0.038	2.086	1.04	4.19	AUC = 0.736
Food prepared specially for respondent	2.017	0.004	7.518	1.87	30.17	H–L = 0.143
Freq. ate fatty/sugary snacks (1)	0.723	0.043	2.060	1.02	4.15	$R^2 = 0.223$
Freq. ate fatty/sugary snacks (2)	1.391	0.002	4.018	1.67	9.65	
Restricted access to salty/sugary snacks	−0.767	0.087	0.464	0.19	1.12	
Model: controls versus bulimics ($N = 277$)						
Had first meal of day before lessons	−1.397	0.001	0.247	0.12	0.53	AUC = 0.763
Food prepared specially for respondent	1.571	0.041	4.810	1.07	21.70	H–L = 0.235
Father valued food more than others	0.831	0.008	2.295	1.24	4.26	$R^2 = 0.248$
Father paid attention to healthy eating	1.583	0.081	4.871	0.83	28.76	
Freq. ate fatty/sugary snacks (1)[b]	0.664	0.040	1.943	1.03	3.67	
Freq. ate fatty/sugary snacks (2)[c]	1.148	0.009	3.152	1.34	7.42	
Model: controls versus EDNOS ($N = 208$)						
Ate meals together less than 3 times/day	0.803	0.042	2.231	1.03	4.83	AUC = 0.700
Had first meal of day before lessons	−1.367	0.004	0.255	0.10	0.65	H–L = 0.571
Restricted access to salty/sugary snacks	−1.124	0.050	0.325	0.11	1.00	$R^2 = 0.146$

[a]AUC = area under the ROC curve; H–L = Hosmer and Lemeshow test; R^2 = Nagelkerke R^2.
[b]Frequency ate fatty/sugary snacks (1): 2–6 times a week versus never or less than once a week.
[c]Frequency ate fatty/sugary snacks (2): Every day versus never or less than once a week.

EDNOS increases when participants consumed meals less than three times a day (OR = 2.23). Again the factors related to a reduced likelihood were eating breakfast before school lessons OR = 1/0.255 = 3.92 and having restricted access to salty/sugary snacks (OR = 1/0.325 = 3.08).

All the models included in Table 1 demonstrated a good ability to discriminate between the groups. This can be seen in the fact that the area under the receiver operator curve is over 0.70. Moreover, the adjustment was correct in all cases (values $p > 0.05$ in the Hosmer and Lemeshow tests). The total variance accounted for in the models was between 15% (controls versus EDNOS) and 25% (controls versus BN).

The effect of individual and family eating patterns on the BMI

Table 2 contains the linear regression models that evaluate the effect of eating pattern variables on the person's later BMI. We have obtained different regression equations for samples with diverse body compositions. In one group, the model could not be controlled for due to the lack of participants (there were only 14 non-purgative BN patients in the study).

The results of the models showed that only a few independent variables were statistically associated with the BMI. Since the adjusted R^2 coefficients vary between 6% and 16%, the eating pattern factors included in the study explain only a small part of the variance of this criterion. This result suggests that these models should be interpreted with caution. The EDNOS sample is the only exception, since in this case the R^2 value was found to be 71%. According to the results of this model, the EDNOS participants who lived together with sibling(s) had a lower BMI ($B = -1.83$, 95%; CI: −3.61 to −0.04).

For the rest of the models, the most notable results indicate that the BMI is bigger if: food was used as a reward, food was specially prepared for the grandparent, the father and the respondent, a high number of family members were present at meals and there was a shortage of luxury food in the family. Contrarily, the model shows that the BMI decreases when the access to food was restricted (punishment) and sibling(s) lived at home.

Discussion

The instrument that we have developed appears to be sensitive to the food-related environmental factors that might be associated with the development of an eating disorder. The significant sociodemografical findings (more females and more women employed in the clinical group than in the control group) could be a spurious finding. The

Table 2
Linear regression (adjusted by sex and age): effect of food style on the BMI

Independent variables	*B*	*P*	95% CI	For *B*
Model: cases and controls sample, $R^2 = 0.073$ ($N = 461$)				
Only few times meals were included in social events	1.105	0.034	0.09	2.13
Shortage of luxury foods in family	1.415	0.037	0.09	2.74
Model: case sample, $R^2 = 0.120$ ($N = 261$)				
Less than 3 times/day ate meals together after 12 years	2.288	0.006	0.67	3.91
Food prepared specially for grandparent	1.986	0.055	−0.05	4.02
Food prepared specially for father	3.215	0.020	0.52	5.91
Only few times meals were included in social events	1.350	0.082	−1.74	2.87
Model: anorexic sample, $R^2 = 0.098$ ($N = 83$)				
Less than 3 times/day ate meals together after 12 years	0.974	0.064	−0.06	2.01
Food was used as a reward	1.245	0.033	0.10	2.39
Model: restricted anorexic sample, $R^2 = 0.117$ ($N = 42$)				
Number of family members present at meals	0.810	0.040	0.04	1.58
Model: purgative anorexic sample, $R^2 = 0.073$ ($N = 41$)				
Restricted access to food (punishment)	−1.91	0.038	−3.70	−0.12
Model: bulimic sample, $R^2 = 0.164$ ($N = 117$)				
Less than 3 times/day ate meals together after 12 years	2.539	0.021	0.40	4.68
Food prepared specially for respondent	3.209	0.073	−0.31	6.73
Food was used as a reward	1.774	0.060	−0.08	3.62
Model: purgative bulimic sample, $R^2 = 0.104$ ($N = 103$)				
Food prepared specially for grandparent	2.173	0.085	−0.31	4.65
Model: non-purgative bulimic sample				
Small sample				
Model: EDNOS sample, $R^2 = 0.706$ ($N = 48$)				
Sibling(s) lived at home	−1.826	0.045	−3.61	−0.04
Model: purgative sample, $R^2 = 0.059$ ($N = 144$)				
Food prepared specially for grandparent	2.974	0.021	0.46	5.49
Model: non anorexic sample				
No variables were significant				
Model: control sample				
No variables were significant				

R^2 = adjusted R^2 coefficient.

likelihood of developing any form of eating disorder was diminished by eating breakfast and increased by eating snacks and having food specially prepared for the respondent, but also by the fathers' value on food. No significant differences between eating disorder subcategories appeared in our study on these variables.

Skipping breakfast and excessive ingestion of sweets and snacks

In the present study, the two specific eating patterns, that might be related to a later eating disorder, were: skipping breakfast and consuming excessively sweets and snacks before the age of 12. Skipping breakfast is often employed as a weight reduction method on nutritional intake among young women (Barker, Robinson, Wilman, & Barker, 2000; Belderson et al., 2003; Lattimore & Halford, 2003). Lattimore and Halford (2003) for instance found that women who were dieting to lose weight were three times more likely to skip breakfast than females who were not dieting. Breakfast skipping has also often been documented to be related to other adverse lifestyle habits such as smoking, alcohol use, low exercise and obesity (Keski-Rahkonen, Kaprio, Rissanen, Virkkunen, & Rose, 2003). The excessive ingestion of sweets and snacks may be linked to the conflict and difficulties around meals or problematic eating that have been found in various longitudinal risk factor studies (Kortegaard et al., 2001; Kotler, Cohen, Davies, Pine, & Walsh, 2001).

Fathers' attitudes to food

Furthermore, the fathers' attitudes towards food may be associated with the development of a later eating disorder (namelly BN) in their daughters. Studies in support of our findings have for instance indicated that eating disordered

females recounted a poor father–daughter relationship (Botta & Dumlao, 2002), lower paternal care (Palmer & Treasure, 1999), less paternal empathy (Steiger, Fraenkel, & Leichner, 1989) and overprotection (Berger et al., 1995; Lavik, Clausen, & Pedersen, 1991; Waller, Slade, & Calam, 1990). Furthermore, in a longitudinal risk factor study maladaptive paternal attitudes (not related to food and weight) were linked to the risk of developing an eating disorder (Johnson, Cohen, Kasen, & Brook, 2002). On the other hand, other studies revealed that eating disorder patients reported feeling closer to their fathers than to their mothers (Kent & Clopton, 1992). Accordingly, further studies have indicated that while mothers' and daughters' perceptions of family functioning differed significantly (Dancyger, Fornari, & Sunday, 2006), they did not differ significantly between fathers and daughters (Dancyger, Fornari, Scionti, Wisotsky, & Sunday, 2005).

Coming from a large family and living with grandparents at home

Two other social factors that were found in this study and might be related to an increased occurrence of eating disorders were coming from a large family and living with grandparents at home. These factors might be country specific, as they have not been previously mentioned in other studies. Meal patterns are related to socio-economic structure, work schedules, life course, living conditions and feeding styles, as shown in previous studies (Birch & Fisher, 1998; Cullen, Baranowski, Rittenberry, & Olvera, 2000; Neumark-Sztainer, Story, Perry, & Casey, 1999). Thus parents increasingly have to rely on the grandparents' help to bring up their children. The present findings could have resulted from inadequate feeding styles (permissive vs. not flexible) employed by grandparents. Research has shown that authoritarian feeding was related to lower ingestion of fruit, juices, and vegetables (Birch & Fisher, 1998; Cullen et al., 2000). However, it should be noted that eating disorders have multifactorial causes and that this might only be one out of many contributing factors (Bulik et al., 2003). Further validation of the relationship between living with other family members and the development of a subsequent eating disorder is therefore required.

Eating patterns and BMI

The last but not least finding of our study was that some eating patterns during childhood and early adolescence might be associated with an increased BMI, especially not having regular meal patterns and using food as a reward. This result is in concordance with previous eating disorder studies (Killen et al., 1996; Patton, Selzer, Coffey, Carlin, & Wolfe, 1999). Similar findings have also been observed in obesity, where a positive relationship between skipping breakfast and lifetime obesity in children has been revealed (Berkey, Rockett, Gillman, Field, & Colditz, 2003; Pastore, Fisher, & Friedman, 1996; Summerbell, Moody, Shanks, Stock, & Geissler, 1996).

This study has some limitations that do not permit a direct generalization of the present results. First, the retrospective and self-report data collection procedures may have limited the validity and the reliability of our findings, which are subject to unreliability of individual recall and potential memory bias. Set against this are the high values obtained on the test–retest reliability of the procedure. Furthermore, given the retrospective design of the study, it could also be that other factors, which have not been considered in this study might be related to early childhood feeding problems and the development of a subsequent eating disorder. Second, the cross-sectional design does not allow us to determine causality of the variables assessed, since we do not know the direction of the association—for e.g. it could be that those who develop an eating disorder remember their breakfast or weight or other details in a different way than those who do not have an eating disorder.

Future research could expand these results employing longitudinal designs addressing the potential mediating role of individual and family eating patterns in the etiological factors and clinical course of eating disorders. Furthermore, forthcoming research could examine whether the people with a particular genotype and common psychosocial factors (as those studied here), are more vulnerable for developing an eating disorder.

Our findings suggest that if programs to improve childhood and adolescent eating patterns are to be successful, they need to focus on a wide range of environmental factors. In particular, the family should be informed about the importance of structuring meal times with shared meals particularly breakfast and increasing the accessibility and promotion of snacks. Furthermore, maintaining structured family meals might encourage healthier diets in children and adolescents and could also allow the family to gain a better understanding of the child's food choices. Finally, the social interaction at meal times may promote children to experience eating constructively and could therefore help in creating positive attitudes towards food, which could persist into later years.

In conclusion, the findings from the present study agree with the growing body of research indicating that a variety of environmental and social factors are associated with unhealthy individual and family eating patterns during childhood and early adolescence and which if not detected early could result in the development of a subsequent eating disorder.

Acknowledgements

Financial support was received from the European Union (Framework—V Multicenter Research Grant, QCK1-1999-916), Fondo de Investigación Sanitario (CIBER; CB06/03/0034), Generalitat de Catalunya (2005SGR00322) and FI (2005 FI 00425). This work is part of the dissertation of IK at the University of Barcelona.

Appendix

Section 3: Individual and family eating patterns

(5) During your childhood, which family members, and how many family memebers were living in your home? (tick appropriate boxes)?

		Lived at home		Number
		Yes (1)	No (0)	
a	Father			
b	Mother			
c	Sibling(s)			
d	Step-mother/father			
e	Step/half-sibling(s)			
f	Aunt/Uncle			
g	Grandparent(s)			
h	Other			

(6) During your childhood, how many family members would be present at most meals?_____

(7) How often did you eat meals together as a family (children plus at least one parent/care-giver) *before* you were 12 and *after* you were 12 when you were living at home (*please tick*)?

		3 times a day (0)	1-2 times a day (1)	2-7 times a week (2)	Once a week or less (3)
a	Before you were 12				
b	After you were 12 and living at home				

(8) Before you were 12, did you usually eat meals at regular set times of the day (*tick one*)?
☐ Yes ☐ No

(9) Before you were 12, did you usually have your first meal of the day before school/lessons started?
☐ Yes ☐ No

(10) Before you were 12, how often would you eat meals an fast food franchised restaurants, McDonalds etc. (*tick one*)?

Never	
Once a month or less	
1-4 times a month	
At least once a week	

(11) During your childhood, did anyone in your family have food prepared specially for them on a regular basis (e.g. due to a physical illness, different taste)?

		Yes (1)	No (0)
a	Grandparent		
b	Mother		
c	Father		
d	Sibling		
e	Yourself		
f	Other household member/care-giver		

(12) During your childhood, how much value was put on food by your mother and/or your father (*tick as applicable*)?

		An average amount or less value than most people (0)	More value than most people (1)	A lot more value than most people (2)
a	Mother			
b	Father			

(13) During your childhood, did anyone in the family pay specific attention to healthy eating, such that it had a profound effect on the food that was eaten in your family (*tick as appropriate*)?

		Yes (1)	No (0)
a	Mother		
b	Father		
c	Sibling		
d	You		
e	Other household member/caregiver		

(14) During your childhood, how often were meals included as an important part of social events in your extended family or among your family's friends and how often were you included in these meals?

		Once a week or more (0)	1-4 times a month (1)	5-8 times a year (2)	2-4 times a year (3)	Less than twice a year (4)
a	Frequency of 'social' meals					
b	Frequency of inclusion in 'social' meals					

(15) Compared to your friends, did your parents have strict rules about the times of food you ate as a child?
☐ Yes ☐ No

(16) If yes, how often did you follow these rules (*tick one*)?

Never	
Occasionally	
Frequently	
Always	

(17) How often did you eat fatty or sugary snacks (e.g. crisps, chocolate, sweets, sugary fizzy drinks) as a child?

Never	
Less than once a week	
2-6 times a week	
Every day (less than 3 times)	
Every day (3 or more times)	

(18) Was your access to fatty or sugary snacks restricted, compared to your friends?
☐ Yes ☐ No

(19) How often did your family use food as a reward (e.g. for good behaviour) and how often did your family restrict your access to food as a form of punishment (*tick one*)?

		Never (0)	Occasionally (1)	Frequently (2)
a	Food used as a reward			
b	Food access restricted as a form of punishment			

(20) During your childhood, was there ever a shortage of food in your family (*if yes, please indicate type of food*)?

		Yes (1)	No (0)
a	Shortage of basic foods		
b	Shortage of luxury foods		

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