The role of traumatic childhood experiences and life stresses before onset in the origins of eating disorders.

Schmidt, Ulrike Hermine

The copyright of this thesis rests with the author and no quotation from it or information derived from it may be published without proper acknowledgement.

END USER LICENCE AGREEMENT

Unless another licence is stated on the immediately following page this work is licensed under a Creative Commons Attribution-NonCommercial-NoDerivatives 4.0 International licence. https://creativecommons.org/licenses/by-nc-nd/4.0/

You are free to copy, distribute and transmit the work

Under the following conditions:

- Attribution: You must attribute the work in the manner specified by the author (but not in any way that suggests that they endorse you or your use of the work).
- Non Commercial: You may not use this work for commercial purposes.
- No Derivative Works - You may not alter, transform, or build upon this work.

Any of these conditions can be waived if you receive permission from the author. Your fair dealings and other rights are in no way affected by the above.

Take down policy

If you believe that this document breaches copyright please contact librarypure@kcl.ac.uk providing details, and we will remove access to the work immediately and investigate your claim.
The Role of Traumatic Childhood Experiences and Life Stresses Before Onset in the Origins of Eating Disorders

Ulrike H. Schmidt

Thesis submitted in partial fulfilment for the degree of Doctor Philosophiae

Institute of Psychiatry
London
To Dave, Lukas and my parents
Acknowledgements: Most of all I would like to thank my supervisor, Janet Treasure, whose wisdom, friendship and great sense of humour helped through all the ups and downs of the projects presented here. I would also like to thank Professor Gerald Russell for his support and encouragement all the way through. Tirril Harris trained me in the use of the CECA interview and the LEDS, was available for many stimulating discussions about the data and kindly provided control data for the life events study described in chapter 9 from the cohort described in Brown and Harris (1978). Bernice Andrews and Antonia Bifulco were involved in developing the pudicity ratings and Bernice provided control data for pudicity events from a cohort of young women in Islington (Andrews et al., 1990). A big thank you goes to Nick Troop for his generous help in the last stages of the thesis. Jane Tiller, Gaynor Slone, Matthew Hodes, Martin Blanchard, Kathryn Evans and Matthias Keilen helped to collect some of the data. Dr. Graham Dunn gave statistical advice.
Abstract
In a series of studies the role of traumatic childhood experiences in the aetiology of different eating disorders was explored. The links between childhood sexual abuse and psychosexual development were also studied in these patients. In an investigation into life stresses before onset, a new dimension of life events and difficulties (pudicity) was developed, to test the hypothesis that anorexia nervosa (AN) might be precipitated by sexual conflict.

Method: Eating disorder patients were assessed with the Childhood Experience of Care and Abuse Interview (Bifulco et al., 1994), the Life Events and Difficulties Schedule (Brown and Harris, 1978) and additional questionnaires.

Results: (1) Bulimia nervosa (BN) patients, especially those with a chronic disorder, had more childhood adversity than those with restricting anorexia nervosa (RAN).
(2) Obese binge eaters did not differ from normal weight bulimia nervosa patients in their childhood care, but a family history of obesity was more common in this group.
(3) In young onset BN parental lack of care occurred significantly more often than in a matched group of typical onset cases, with no other differences in childhood experiences. (4) Sexual milestones were delayed in patients with different eating disorders, and in RAN the delay may predate onset. Childhood sexual abuse did not account for this. (5) Eating disorder patients and community controls did not differ in the proportion of those with a severe life event in the year before onset, however, significantly more eating disorder patients than community controls had experienced a major difficulty. AN patients had significantly more pudicity events before onset than BN patients or controls.

Conclusions: AN and BN patients have different childhood antecedents. The findings presented here provide no support for a specific role of childhood sexual abuse in the onset of different eating disorders, but support the notion that RAN patients are sexually inhibited and in some cases the disorder is provoked by sexual conflict.
Statement on Authorship

Some of the work presented in this thesis has been published as peer reviewed articles in journals. An adapted form of the review of the literature in chapters 2 and 3 has been submitted for publication in the *European Eating Disorders Review* (Schmidt, U. et al. (1997) The role of general family environment and sexual and physical abuse in the origins of eating disorders).

The results of the study described in chapter 5 have been published in *Psychological Medicine* **23**, 663-672 (Schmidt et al. (1993c). Setting the scene for eating disorders: Childhood care, classification and course of illness).

The results of the study described in chapter 6 have been published in the *Journal of Nervous and Mental Disease* **181**, 202 - 204. (Schmidt et al. (1993a). Clinical symptomatology and aetiological factors in obese and normal weight bulimics. A retrospective case-control study).


The results of the study in chapter 8 have appeared in *Psychological Medicine* **25**, 413
- 417. (Schmidt et al. (1995a). Puberty, sexual milestones and childhood abuse: How are they related in eating disorders?).

The results of the study in chapter 9 are in press in Psychological Medicine. (Schmidt et al. (1997). Is there a specific trauma precipitating anorexia nervosa?). The author of this thesis designed the studies, performed the calculations and prepared the manuscripts.

I agree that this statement is correct.

Dr. Janet Treasure
TABLE OF CONTENTS

Acknowledgements 3
Abstract 4
Statement on Authorship 5
Table of Contents 7

PART I: Background 12
Chapter 1: General Introduction 13
1.1. Some Remarks on Classification 14
1.2. Some Considerations on Aetiology 15
1.3. Overview of the Thesis 18
1.4. Appendix 22

Chapter 2: The Role of the Family Environment and Childhood Care in the Origins of Eating Disorders 27
2.1. Introduction 27
2.2. Family Environment and Eating Disorders 28
2.3. Some Methodological Considerations about the Assessment of Childhood Family Functioning in Eating Disorders 29
2.4. Organization of the Literature Review 33
2.4.1. Does family functioning and childhood care of eating disordered individuals differ from that of normal controls? 34
2.4.2. Does family functioning or childhood care of eating disorder sufferers differ from that of psychiatric controls or that of other patient control populations? 37
2.4.3. Are there differences between different types of eating disorders in terms of family functioning or childhood care? 37
2.4.4. Is there any link between the severity of eating symptomatology and the severity of the family dysfunction? 38
2.4.5. Do the perceptions of other family members differ from the eating disordered subject’s view of family functioning and childhood care? 39
2.4.6. Are there any mediators between a dysfunctional family environment and later eating disorders? 40
2.4.7. Other studies on family functioning and childhood care. 41
2.5. Summary and Conclusions 41
2.6. Tables 43
Table 2.6.1. Studies of the family environment of individuals with eating disorders 43

Chapter 3: The Role of Sexual and Physical Abuse in the Origins of Eating Disorders 57
3.1. Introduction 57
3.2. Some Methodological Considerations 58
3.3. Organization of the Literature Review 59
3.4. Childhood Sexual Abuse 60
3.4.1. Do subjects with eating disorders differ from normal controls in terms of rates of sexual abuse reported? 60
3.4.2. Do eating disorder subjects differ from subjects with other psychiatric disorders in terms of rates of abuse reported? 61
3.4.3. Do women who have been abused have higher rates of eating disorders than those who have not been abused? 62
3.4.4. Are different types of eating disorders associated with different rates of childhood sexual abuse? 62
3.4.5. Is there any link between abuse characteristics and the severity of the eating symptomatology? 63
3.4.6. What - if any - contextual and psychological mediators exist between childhood abuse and later eating disorders? 64
3.5. Childhood Physical Abuse 66
3.6. Psychological Abuse 67
3.7. Summary and Conclusions 68
3.8. Tables 70
Table 3.8.1. Studies on sexual abuse and eating disorders 70
Table 3.8.2. Studies on physical abuse and eating disorders 84

Chapter 4: Stress and Eating Disorders 88
4.1. Introduction 88
4.2. The Biology of Stress and Eating 88
4.3. Measurement of Life Stress: Some Methodological Considerations 90
4.4. Stressful Life Events and Difficulties and the Onset of Eating Disorders 92
4.4.1. Anecdotal reports and case series 92
4.4.2. Controlled studies 94
4.4.3. Other studies 95
4.4.4. Shortcomings in existing studies on life events and difficulties in eating disorders 96
4.5. Summary and Conclusions 96

PART II: Experimental Section 98
Chapter 5: An Investigation into Childhood Adversity and Course of Illness in Different Types of Eating Disorders 99
5.1. Aims and Hypotheses 99
5.2. Introduction 99
5.3. Subjects and Method 100
5.3.1. Sample 100
5.3.2. Procedure 101
5.4. Results 106
5.4.1. Sample characteristics 106
5.4.2. Childhood care 106
5.5. Discussion 110
5.6. Summary and Conclusions 117
5.7. Tables 118
Table 5.7.1. Sample characteristics
Table 5.7.2. Number of family arrangements
Table 5.7.3. Reasons for change in family arrangements
Table 5.7.4. Indicators of childhood care based on behavioural evidence
Table 5.7.5. Reported ratings of childhood care
Table 5.7.6. Differences between recent onset (< 4 years) and chronic (> 4 years) cases.
Table 5.7.7. Indicators of childhood care in community studies using the same methodology as the present study

Chapter 6: Childhood Adversity and Other Familial Risk Factors in Obese Bulimic Patients: A Retrospective Case-Control Study
6.1. Aims and Hypotheses
6.2. Introduction
6.3. Subjects and Method
6.3.1. Sample
6.3.2. Procedure
6.4. Results
6.4.1. Sample characteristics
6.4.2. Clinical features
6.4.3. Associated psychopathology
6.4.4. Family factors
6.4.5. Childhood care and abuse
6.5. Discussion
6.6. Summary and Conclusions
6.7. Tables
Table 6.7.1. Clinical features and associated psychopathology
Table 6.7.2. Family factors

Chapter 7: Childhood Adversity and Other Familial Risk Factors In The Development of Early Onset Bulimia Nervosa: A Retrospective Case-Control Study
7.1. Aims and Hypotheses
7.2. Introduction
7.3. Subjects and Method
7.3.1. Sample
7.3.2. Procedure
7.4. Results
7.4.1. Sample characteristics
7.4.2. Clinical features
7.4.3. Associated psychopathology
7.4.4. Family factors
7.5. Discussion
Chapter 8: Puberty, Psychosexual Development and Childhood Sexual Abuse: Are They Related In Eating Disorder Patients?  
8.1. Aims and Hypotheses  152
8.2. Introduction  153
8.3. Subjects and Method  155
8.3.1. Sample  155
8.3.2. Procedure  156
8.4. Results  157
8.4.1. Sample characteristics  157
8.4.2. Pubertal development  157
8.4.3. Psychosexual development  158
8.4.4. Attitudes to sex, masturbation, marriage, children and pregnancy  159
8.4.5. Childhood sexual abuse and its relationship with puberty, sexual behaviour and attitudes  159
8.5. Discussion  160
8.6. Summary and Conclusions  165
8.7. Tables  166
Table 8.7.1. Pubertal and psychosexual development  166
Table 8.7.2. Median scores on visual analogue scales on attitudes to sex, masturbation, marriage, children and pregnancy of eating disorder patients and controls  167
Table 8.7.3. Childhood sexual abuse (CSA) in the eating disorder groups  168
Table 8.7.4. Attitudinal differences between abused and non-abused patients  169
8.8. Figures  170
Figure 8.8.1. Survival curve of breast development in the four eating disorder groups  170
Figure 8.8.2. Survival curve of menarche in the four eating disorder groups  170
Figure 8.8.3. Survival curve of age at first date in eating disorder and control groups  171
Figure 8.8.4. Survival curve of age at first kiss in eating disorder and control groups  171
Figure 8.8.5. Survival curve of age at first genital fondling in eating disorder and control groups  172
Figure 8.8.6. Survival curve of age at first sexual intercourse (coitarche) in eating disorder and control groups  172
Figure 8.8.7. Survival curve of age at first masturbation in eating disorder and control groups  173
Figure 8.8.8. Survival curve of age at first sexual intercourse
for those patients with coitarche before onset 173
8.9. Appendix 174

Chapter 9: The Role of Stressful Life Events and Difficulties Before Onset: Is There a Specific Trauma Precipitating Anorexia Nervosa?

9.1. Aims and Hypotheses 179
9.2. Introduction 179
9.3. Subjects and Method 180
9.3.1. Sample 180
9.3.2. Procedure 181
9.4. Results 183
9.4.1. Sample characteristics 183
9.4.2. Number of events and difficulties 184
9.4.3. Type of events and difficulties 185
9.4.4. Pudicity problems 186
9.5. Discussion 187
9.6. Summary and Conclusions 189
9.7. Tables 191
Table 9.7.1. Sociodemographic details 191
Table 9.7.2. Life events and difficulties 192
Table 9.7.3. Focus of relationship problems 193
Table 9.7.4. Pudicity problems 194
9.8. Appendix 195

Chapter 10: Discussion 197
10.1. Summarizing and Linking the Findings 197
10.1.1. Childhood care and abuse in eating disorders 197
10.1.2. Childhood sexual abuse and sexual development 201
10.1.3. Life events and difficulties as provoking agents 202
10.1.4. Bulimia nervosa: childhood adversity, life events before onset and potential mediators 204
10.1.5. A model of anorexia nervosa 207
10.2. Future Research 208
10.2.1. Childhood adversity 208
10.2.2. Attachment and eating disorders 209
10.2.3. Nature and nurture 210
10.2.4. Life events and difficulties 212
10.3. Implications for Clinical Practice 214

References 218
PART I: BACKGROUND
Chapter 1: General Introduction

Eating disorders are amongst the most common psychiatric disorders affecting young women in contemporary Western societies and have been thought to be culture-bound syndromes (Prince, 1985). Both anorexia nervosa and bulimia nervosa are now firmly enshrined in the American (DSM-III-R, DSM-IV; American Psychiatric Association, 1987 and 1994) and European classificatory systems (ICD-10; WHO, 1992). For definitions see appendix 1.4.

William Gull (1874) and Charles Lasègue (1873) are often credited with the first medical descriptions of anorexia nervosa and l'anorexie hysterique respectively. However, far earlier descriptions of examples of anorexia nervosa exist, including the case of St. Catherine of Siena recorded by her confessor Raymond of Capua (Rampling, 1985), Mr. Duke's daughter depicted by Richard Morton in 1689 (Silverman, 1983) and Louis-Victor Marcé's "delire hypochondriaque" (1860).

Bulimia nervosa was first described by Russell in (1979). However, long before its formal description examples of this disorder had been noticed by earlier investigators. For example, Janet (1903; quoted from Pope et al., 1985) and Wulff (1932; quoted from Stunkard, 1990) described cases in which the gorging of food associated with depression and the avoidance of food alternated. Within a few years of its "discovery", incidence figures for bulimia nervosa rapidly exceeded those of anorexia nervosa (Kendler et al., 1991; Soundy et al., 1995).
1.1. Some Remarks on Classification

A substantial proportion of cases of bulimia nervosa have had a history of anorexia nervosa. In younger samples almost 60% of cases of anorexia nervosa developed bulimic symptoms during some extended period between age 16 through to age 21 years (Rastam et al., 1995), whereas in older series of anorexia nervosa the rate lies between 7% and 40% (Herzog et al. 1992). This failure to account for the longitudinal course is a weakness in our current diagnostic criteria (Treasure and Holland, 1995).

DSM-IV has sub-categorized anorexia nervosa into cases with bingeing and purging and pure restrictors (see 1.4. appendix) as there are important differences between those two groups. Impulsive behaviours including stealing, drug abuse, suicide attempts, self-mutilations and mood lability are more prevalent in anorectic-bulimics compared with anorectic restrictors. The anorectic-bulimics also have a higher prevalence of premorbid obesity, familial obesity, debilitating personality traits and familial psychopathology (Casper et al., 1980; Eckert et al., 1987; Garfinkel et al., 1980; Strober et al., 1982; Yellowlees, 1985). There are also physiological differences between bulimics who lose large amounts of weight and meet criteria for anorexia nervosa and those who never lose weight (Halmi, 1995).

At the time when the research studies that form the basis for this thesis were set up, DSM-IV had not come out yet, so with the exception of one study where ICD-10 criteria were used (see chapter 6 for further details) DSM-III-R diagnostic criteria were used. However, where appropriate, patients were subdivided into those with...
restricting anorexia nervosa, bulimic anorexia nervosa and bulimia nervosa with or without a history of anorexia nervosa.

1.2. Some Considerations on Aetiology

A recent review stated that: "eating disorders present a special kind of challenge to psychiatric approaches to theories of causality, particularly because they seem to implicate a strong degree of conscious choice." (Campbell, 1995). The author continues with the question: "Is the sufferer a deliberate agent or an afflicted victim?"

In the eyes of many experts this is probably a false dichotomy, as although the early stages of weight-loss may be the result of a conscious decision, once the disorder is more firmly established there is often nothing deliberate about it's maintenance.

Many researchers agree that eating disorders are multifactorial in causation (Halmi, 1995). Campbell (1995) cautions: "Too complacent an acceptance of a "multifactorial causation could direct attention away from the search for possibly identifiable singular and necessary causes. Or it may be that lurking behind claims of such "multifactorial" causation, there is often an unexpressed hope that..." "....new technology will one day reveal, if not the "anorexicoccus" and the "bulimichaete", at least one comparably definable physical entity or process which can do service as a necessary cause for the eating disorders".

In contrast, a more optimistic statement comes from one of the proponents of a multifactorial model of causation of eating disorders: "Eventually we should be able to decipher how much influence or what percentage of the variance is accounted for by
biological vulnerability, psychological predisposition, family characteristics and societal expectations in the development of AN and BN" (Halmi, 1995). However, in the recently published "Handbook of Eating Disorders" the editors in their introduction to the section on "aetiology" state that although they invited authors with different research interests to push each single perspective to its limits in order to gain a clearer picture of relative contributions: "Aetiological conclusions are modest. Most accounts seem almost silent on "necessary" causes, completely so on "necessary and sufficient" causes" (Szmukler et al., 1995).

Recently some progress has been made in the understanding of the biological origins of anorexia nervosa. It is now widely accepted that there is a genetic component underlying restricting anorexia nervosa (Treasure and Holland, 1989). It is thought that the vulnerability may be linked to abnormalities in central 5-HT pathways (Treasure and Campbell, 1994). In contrast, our understanding of the personal, familial and social causes is much more limited.

The study of the explanatory models employed by patients and health practitioners in a given cultural context, i.e. their accounts of a problem, its nature, origins, consequences and remedies, is an important tool in understanding the "personal and social meaning of the experience of sickness" (Kleinmann, 1980). Over the centuries lay and professional explanatory models of the disorder have changed dramatically (Schmidt and Treasure, 1993). Whilst in the middle ages control of appetite was linked to religious asceticism and self-mortification (Brumberg, 1988; Edelstein, 1989; Rampling, 1985), in the 17th century Morton's account was that of "nervous
consumption" resulting from "sadness" and "nervous cares" (Silverman, 1983). And in the second half of the 19th century abdominal discomfort was a common explanation used by anorexia nervosa patients, whilst their doctors thought of them as hysterical (Lasègue, 1873). Charcot was the first to observe morbid fear of fatness in a patient who he found wearing a tightly fastened rose-coloured ribbon round her waist and who told him "I prefer dying of hunger to becoming big as mamma" (Janet, 1903). And this has become the pre-dominant model in the late 20th century. Janet (1903) himself felt that the importance of weight concerns had been exaggerated by Charcot. He saw weight concerns as an aspect of more general worries about appearance, with an underlying fear of sexual maturity as the main cause of eating disorders, a theme that has been echoed in the psychoanalytical literature and in the writings of Crisp (1980). At the beginning of the twentieth century anorexia nervosa was thought to be the result of pituitary insufficiency (Simmonds, 1916), with a reappraisal as a psychogenic disorder in the 1930s (Ryle, 1936). Recently further psychological theories of the origins of eating disorders have been put forward, including family systems theory (Minuchin et al., 1978; Selvini-Palazzoli, 1974), feminist theory (Orbach, 1986), with sexual trauma being a recent addition (Esman, 1994).

There is an increasing recognition that the prevailing culture of thinness is not shared by all sufferers of eating disorders, and that for example male sufferers, those from ethnic minorities, those from non-Western cultures and those presenting in 'atypical' settings such as diabetic and thyroid clinic populations may have very different explanatory models (Schmidt and Treasure, 1993). With this in mind, Russell (1995), basing his argument on Birnbaum's (1923) idea that sociocultural pressures exert a
"pathoplastic influence" on the psychological contents and structure of an illness, writes: "A broader formulation of the psychopathology of anorexia nervosa may be called for. Thus it may suffice to establish that the patient avoids food and induces weight loss by virtue of a range of psychosocial conflicts whose resolution she perceives to be within her reach through the achievement of thinness and/or the avoidance of fatness. These conflicts will still include the dread of fatness, but may need to embrace the fear of sexuality and fertility, or the reluctance to acquire independence from the family, or some other as yet unpredictable issue." Thus psychosocial stress appears to be integral to the definition of the disorder.

The present thesis aims to examine some of the psychosocial hypotheses of the origins of eating disorders, i.e. those focusing on abnormalities of family life, childhood trauma and later traumatic life happenings before onset. Some emphasis will be put on the exploration of sexual trauma and its links with later psychosexual development and sexually traumatic events before the onset. However, although the main theme uniting the studies described in this thesis is that different aspects of the patients' sexuality are being explored, this is being done in the context of an exploration of broader aspects of childhood and adult environment before onset.

1.3. Overview of the Thesis

This thesis is divided into two parts. Part I presents the background literature which forms the starting point for the development of the questions and hypotheses examined in the experimental chapters presented in part II.
Part I: Overviews of the literature on different aspects of childhood family environment and childhood abuse as potential risk factors for a later eating disorder will be presented in chapters 2 to 3. In the first of these (chapter 2), the underlying theoretical assumptions and methodological problems found in many of these studies are evaluated. The literature on the general quality of family environment of eating disorder sufferers is examined, organized around a number of questions: Do eating disorder sufferers differ from normal controls in terms of family functioning or childhood care received? Do they differ from psychiatric controls or other patient populations? Do different types of eating disorders differ from each other? Are greater severity or chronicity of eating disorder symptomatology linked to greater family disturbance? Are there any mediators between a dysfunctional family environment and later eating disorders? Who are the best informants, parents, siblings or patients themselves?

Childhood sexual and physical abuse and their role as potential risk factors for eating disorders will be discussed in chapter 3. The reason for separating out the literature on abuse is purely pragmatic and reflects the fact that research into this area has been very much preoccupied with the issue of whether sexual abuse in particular might be a specific risk factor for eating disorders.

Chapter 4 examines the literature on the role of stress in the onset of eating disorders. It includes some discussion of the biology of stress and eating. This is followed by methodological considerations about the measurement of psychosocial stress. An overview of what is known about life stress in the onset and maintenance of eating
disorders concludes the chapter.

Part II: In part two a series of studies is presented in which the role of different traumatic childhood experiences in the aetiology of different eating disorders is explored. The links between pubertal and psychosexual development and childhood sexual abuse were also studied in these patients. As part of an investigation into traumatic events and difficulties before onset, a new dimension of life events and difficulties was developed (pudicity events) to test the hypothesis that anorexia nervosa might be precipitated by sexual conflict. For clarity, each of the five research studies starts with its own methodology section, rather than all the methods being incorporated into one chapter.

In chapter five a study is presented assessing the childhood antecedents in restricting anorexics, bulimic anorexics and bulimics with or without a history of anorexia nervosa using the Childhood Experience of Care and Abuse Interview (CECA; Bifulco et al., 1994). The impact of childhood experiences on the course of the illness was also examined.

In chapters six and seven, two case controls studies compare the childhood care of two specific subgroups (i.e. obese bulimics and that of young onset bulimia nervosa) with that of more typical cases of bulimia nervosa. These two subgroups represent groups of particular concern. Both of them have been thought to have experienced more childhood family disturbance and, in particular, more childhood sexual abuse than patients with a typical onset.
In chapter eight the links between childhood sexual abuse, pubertal development and psychosexual development are examined.

In chapter nine, using Brown and Harris' Life Events and Difficulties Schedule (LEDS; Brown and Harris, 1978; 1989) the stressful life happenings in the year before onset of anorexia nervosa and bulimia nervosa are examined. A new dimension of life events (pudicity) was defined following Janet's idea that sexual shame plays a role in the onset of the disorder.

In the final chapter (chapter 10) the links between the findings of the studies described in chapters 5 to 9 are discussed and directions for further research and clinical practice are outlined.
1.4. Appendix

DSM-III-R criteria (APA, 1987)

Anorexia nervosa

A. Refusal to maintain body weight over a minimal normal weight for age and height, (e.g. weight loss leading to maintenance of body weight 15% below that expected; or failure to make expected weight gain during period of growth, leading to body weight 15% below that expected.)

B. Intense fear of gaining weight or becoming fat, even though underweight.

C. Disturbance in the way in which one’s body weight, size or shape is experienced, e.g. the person claims to "feel fat" even when emaciated, believes that one area of the body is "too fat" even when obviously underweight.

D. In females, amenorrhoea, absence of at least three consecutive menstrual cycles when otherwise expected to occur (primary or secondary amenorrhoea). (A woman is considered to have amenorrhoea if her periods occur only following hormone, e.g. estrogen, administration).

Bulimia nervosa

A. Recurrent episodes of binge eating (rapid consumption of a large amount of food in a discrete period of time).

B. A feeling of lack of control over eating during the eating binges.

C. The person regularly engages in either self-induced vomiting, use of laxatives, strict dieting or fasting, or vigorous exercise in order to prevent weight gain.

D. A minimum average of two binge eating episodes a week for at least three months.

E. Persistent overconcern with body shape and weight.
DSM-IV criteria (APA, 1994)

Anorexia nervosa

A. Refusal to maintain body weight at or above a minimally normal weight for age and height, (e.g. weight loss leading to maintenance of body weight less than 85% of that expected; or failure to make expected weight gain during period of growth, leading to body weight less than 85% of that expected.)

B. Intense fear of gaining weight or becoming fat, even though underweight.

C. Disturbance in the way in which one’s body or shape is experienced, undue influence of body shape and weight on self-evaluation, or denial of the seriousness of current low body weight.

D. In post-menarchal females, amenorrhoea, i.e. the absence of at least three consecutive menstrual cycles. (A woman is considered to have amenorrhoea if her periods occur only following hormone, e.g. estrogen, administration).

Specify type:

Restricting Type: during the episode of anorexia nervosa, the person does not regularly engage in binge eating or purging behaviour (i.e. self-induced vomiting or the misuse of laxatives, diuretics or enemas).

Binge Eating/Purging Type: during the episode of anorexia nervosa, the person has regularly engaged in binge-eating or purging behaviour (i.e. self-induced vomiting or the misuse of laxatives, diuretics, or enemas).
Bulimia nervosa

A. Recurrent episodes of binge-eating. An episode of binge-eating is characterized by both of the following:

(1) eating, in a discrete period of time (e.g. in any two-hour period), an amount of food that is definitely larger than most people would eat in a similar period of time and under similar circumstances.

(2) a sense of lack of control over eating during the episodes (e.g. a feeling that one cannot stop eating or control what or how much one is eating).

B. Recurrent inappropriate compensatory behaviour in order to prevent weight gain, such as self-induced vomiting; misuse of laxatives, diuretics, enemas, or other medications; fasting; or excessive exercise.

C. The binge eating and inappropriate compensatory behaviours both occur on average, at least twice a week for 3 months.

D. Self-evaluation is unduly influenced by body shape and weight.

E. The disturbance does not occur exclusively during episodes of anorexia nervosa.

Specify type

Purging type: during the current episode of bulimia nervosa, the person has regularly engaged in self-induced vomiting or the abuse of laxatives, diuretics, or enemas.

Non-purging type: during the current episode of bulimia nervosa, the person has used other inappropriate compensatory behaviours, such as fasting or excessive exercise, but has not regularly engaged in self-induced vomiting or the misuse of laxatives, diuretics, or enemas.
ICD-10 (WHO, 1992)

Anorexia nervosa

For a definite diagnosis, all the following are required:

(a) Body weight is maintained at least 15% below that expected (either lost or never achieved), or Quetelet’s body-mass index\(^1\) is 17.5 or less. Prepubertal patients may show failure to make the expected weight gain during the period of growth.

(b) The weight loss is self-induced by avoidance of "fattening foods". One or more of the following may also be present: self-induced vomiting; self-induced purging; excessive exercise; use of appetite suppressants and/or diuretics.

(c) There is body-image distortion in the form of a specific psychopathology whereby a dread of fatness persists as an intrusive, overvalued idea and the patient imposes a low weight threshold on himself or herself.

(d) A widespread endocrine disorder involving the hypothalamic - pituitary - gonadal axis is manifest in women as amenorrhoea and in men as a loss of sexual interest and potency. (An apparent exception is the persistence of vaginal bleeds in anorexic women who are receiving replacement hormonal therapy, most commonly taken as a contraceptive pill.) There may also be elevated levels of growth hormone, raised levels of cortisol, changes in the peripheral metabolism of the thyroid hormone, and abnormalities of insulin secretion.

(e) If onset is prepubertal, the sequence of pubertal events is delayed or even arrested (growth ceases; in girls the breasts do not develop and there is a primary amenorrhoea; in boys the genitals remain juvenile). With recovery, puberty is often completed normally, but the menarche is late.

\(^1\): Quetelet’s body-mass index = weight (kg)/height (m\(^2\)) to be used for age 16 or
above.

Bulimia nervosa

For a definite diagnosis, all the following are required:

(a) There is a persistent preoccupation with eating, and an irresistible craving for food; the patient succumbs to episodes of overeating in which large amounts of food are consumed in short periods of time.

(b) The patient attempts to counteract the “fattening” effects of food by one or more of the following: self-induced vomiting; purgative abuse; alternating periods of starvation; use of drugs such as appetite suppressants, thyroid preparations or diuretics. When bulimia occurs in diabetic patients they may choose to neglect their insulin treatment.

(c) The psychopathology consists of a morbid dread of fatness and the patient sets herself or himself a sharply defined weight threshold, well below the premorbid weight that constitutes the optimum or healthy weight in the opinion of the physician. There is often, but not always, a history of an earlier episode of anorexia nervosa, the interval between the two disorders ranging from a few months to several years. This earlier episode may have been fully expressed, or may have assumed a minor cryptic form with a moderate loss of weight and/or a transient phase of amenorrhoea.
Chapter 2: The Role of Family Environment and Childhood Care in the Origins of Eating Disorders

2.1. Introduction

Childhood experiences have frequently been implicated as causal factors in the development of adult psychopathology. In the field of eating disorders the earliest descriptions mention the importance of family factors in their origins. In 1873, Lasègue emphasized that it is "wrong to confine ourselves to the examination of the patient alone", implying that the family needed to be involved in assessment and treatment, too. Ryle (1936) observed that in some cases of anorexia nervosa "unhappy or ill-conducted homes, sometimes with "spoiling" by foolish parents, were to blame" for the onset of the illness. Bruch (1973) argued that a central feature in the development of anorexia nervosa is a disturbed early mother-child relationship.

In general, two classes of pathological family experiences have been described as being implicated in the aetiology of eating disorders: (a) Those experiences leading to a failure to form stable and secure attachments to caregivers, i.e. the general quality of early social interactions and the inferences children draw about their acceptability. (b) Those related to physical and sexual abuse. Additionally, there is now a growing literature addressing specific problems in the realm of food, family meals or over appearance in the family of origin and their links to later eating disorders (Attie and Brooks-Gunn. 1989; Marchi and Cohen, 1990; Striegel-Moore and Kearney-Cooke, 1992 (quoted from Striegel-Moore, 1992)). The following review will limit itself to studies that have addressed the quality of family interactions. Studies of childhood physical and sexual abuse are described in chapter 3.
2.2. Family Environment and Eating Disorders

A substantial range of opinions exists regarding the association between patterns of family behaviour and the symptoms of anorexia nervosa (for review see Kog and Vandereycken, 1989a). Grigg et al. (1989) divided the theoretical postures of different experts in this area into three forms of explanation: uni-dimensional, bi-dimensional and multi-dimensional. The most prominent proponent of a uni-dimensional position is Minuchin, who stated that "certain transactional patterns seem to be characteristic of all anorexogenic families" (Minuchin et al., 1978), including overprotectiveness, conflict avoidance, rigidity and enmeshment. Selvini-Palazzoli's description (1974) is similar, with a need for a perfect and compliant child, marked family rigidity and the upholding of traditional family roles.

Strober and Yager (1985) identified two family patterns in anorexia nervosa. One pattern is characterized by a "centripetal process" dominated by themes of excessive cohesion, lack of permissiveness, reduced emotional expressivity and frequently impoverished extrafamilial contacts. The second family pattern is characterized by a "centrifugal process". These families lack cohesion and attachment and are highly conflicted before the onset of the anorexic symptoms. A variant of the bi-dimensional position is the so-called "binger-restricter dichotomy" which postulates that different family patterns are associated with bingeing or starving irrespective of the patient's weight.
Garfinkel and Garner (1982) and Grigg et al. (1989) are proponents of a multidimensional position. They see the families of anorexics as heterogeneous but suggest that there may be some risk factors related to the family predisposing a child to anorexia nervosa.

Descriptions of family pathology in bulimia nervosa have more or less assumed homogeneity of family pathology, describing these families as 'chaotic' with high levels of conflict, inadequate or inconsistent expression of emotions, low cohesiveness and with a lack of parental warmth and care. The exception are Root et al. (1986) who delineated three types (perfect, overprotective or chaotic) of bulimic families. These family types are all said to display problems with interpersonal boundaries, have a strong emphasis on weight and appearance and have extreme levels of paternal (as opposed to maternal) power. Differences between families include the nature of their conflicts and rules. In the "perfect family" which is often demanding and critical, bulimic symptoms may symbolise hostile strivings towards autonomy, whereas the same behaviour in the neglectful, abusive "chaotic family" may represent a form of self-abuse, or a search for nurturance, or dissociation from a problematic environment.

2.3. Some Methodological Considerations about the Assessment of Childhood Family Functioning in Eating Disorders

Despite general agreement between theoreticians of different orientations that early experience plays an important role in the development of adult psychopathology, there is widespread scepticism about the validity of retrospective reports about their
childhood obtained from subjects. A review of this topic was undertaken by Brewin et al. (1993). These authors conclude that although autobiographical memory is flawed to a certain extent, "the evidence supports the view that adults asked to recall salient factual details of their own childhoods are generally accurate, especially concerning experiences that fulfil the criteria of having been unique, consequential, and unexpected. This does not mean that adults necessarily recall a wealth of peripheral details associated with these experiences, but rather that their recollections of the central features of the event are accurate and reasonably stable over time."

More specifically there has been concern that a current psychiatric disorder, in particular depression, may lead to selective recall of negative experiences and an exaggeration or misrepresentation of the presence of childhood adversity. However, based on their review of the literature Brewin and coworkers conclude: "Both experimental and naturalistic studies reveal high stability in recall, even with changes in mood or clinical status. The test-retest reliability of accounts of early separations is similarly unrelated to the presence of psychiatric disorder. Furthermore, patients’ memories are in as much agreement with external criteria as are controls’, whether the criteria be siblings’ memories or independent records”.

Brewin et al. (1993) finally summarize that "claims that retrospective reports in general and those of psychiatric patients in particular are inherently unreliable are exaggerated. Nevertheless it is clear that retrospective reports are subject to various limitations and researchers and clinicians must take steps to enhance their reliability."

They advocate the use of two possible strategies to enhance reliability. i.e. to obtain accounts from other informants and to use structured investigative methods "that minimize unrealistic demands on subjects’ memory".
Recall by parents of their children tends to be much more positive than the report of the children themselves, their siblings or other independent observers. This is consistent with the existence of a self-serving bias in parents to minimize admissions of failure of their own or their children’s part. Parents’ reports are likely to be particularly unreliable if there has been serious maltreatment such as neglect or abuse of their children. Whilst sibling reports are probably the most valid compared with those of other family members, there may be problems, too. The siblings may not have been at home throughout an individual’s time at home. Especially if the siblings differ in sex and are very dissimilar in age it is likely that the parents would have behaved differently to them. (These non-shared family environmental factors are increasingly being recognized as important in determining personality and psychopathology (Reiss et al., 1991)).

Another method of assessing childhood care is to enhance subjects’ recall by the use of recognition cues in semi-structured interviews. Questionnaire-based self-reports are much more limited in their reliability and validity in the absence of clear referents and the requirement to sample among a potentially infinite number of relevant memories. Nonetheless, there are some well-validated questionnaires such as the Parental Bonding Instrument (Parker et al., 1979) which have yielded highly consistent results both across and within studies.

There is now a large number of studies assessing family functioning and childhood care in eating disorders (see table 2.6.1.; for review see Eisler, 1995: Kog and Vandereycken, 1989b). The research subjects in most of these studies are eating disordered patients, a few have looked at non-clinical (student populations) and only
three studies use population-based samples (Kendler et al., 1991; Rastam and Gillberg, 1991; Walters and Kendler, 1995).

The vast majority of studies use self-report measures of family interaction. The most commonly used questionnaires are: (1) The Family Environment Scale (FES) (Moos, 1974), which measures three aspects of family functioning: (a) interpersonal relationships (cohesion, expressiveness, conflict), (b) personal growth (independence, achievement orientation, intellectual-cultural orientation, active-recreational orientation, moral-religious emphasis and (c) basic organisational structure (organisation and control).

(2) The Family Assessment Device (FAD; Epstein et al., 1978), which assesses six areas of family functioning (a) task differentiation (roles); (b) concern and involvement between family members (affective involvement), (c) appropriate expression of emotions (affective responsiveness), (d) ability to resolve problems (problem solving), (e) clarity of communication (communication) and (f) clarity of "family rules" (behaviour control).

(3) The Family Cohesion and Adaptability Scales (FACES; Olson et al., 1979) which assesses cohesion and adaptability.

(4) The Parental Bonding Instrument (e.g. Parker et al., 1979), which unlike the others specifically assesses parental care and control below the age of 17.

Other studies are based on observation of family interaction, some on review of hospital records and only one study is based on a retrospective interview with the mother (Rastam and Gillberg, 1991). Most of these studies do not differentiate between current family functioning, childhood family functioning and pre-morbid
family functioning. Strictly speaking only the assessment of the latter allows aetiological inferences, yet all too often researchers extrapolate from current family functioning to premorbid family functioning. The parental bonding instrument assesses childhood care below the age of 17, however, a proportion of cases of eating disorders will have started earlier than that, thus abnormalities in parental care and protection may still be secondary to the onset of the eating disorder. Only the interview study by Rastam and Gillberg (1991) clearly separates out premorbid childhood problems from those that have arisen after the onset of the disorder.

2.4. Organization of the Literature Review

In the following, an overview will be given over studies addressing family functioning and childhood care in eating disorders. Studies were identified by a Medline search up to July 1996 (see table 2.6.1.). The aim was to answer a number of critical questions:

(1) Does family functioning and childhood care of eating disordered individuals differ from that of normal controls?

(2) Does it differ from that of psychiatric and other patient control populations?

(3) Are there differences between different types of eating disorders (anorexia nervosa vs bulimia nervosa or bingers vs restricters) in terms of family functioning and childhood care?

(4) Is there any link between the severity of the early family dysfunction and the severity of eating symptomatology?

(5) Do the perceptions of other family members differ from the eating disordered subject's view of family functioning and childhood care?
(6) Are there any mediators between a dysfunctional family environment and later eating disorders?

(7) Under this point other important studies not easily classifiable under the above headings are described.

2.4.1. Does family functioning and childhood care of eating disordered individuals differ from that of normal controls?

Most studies seem to agree that eating disorder patients show more family pathology than controls (Steiger et al., 1991a), including more conflict and disorganization (Kog et al., 1985; Kog et al., 1989ab), lower adaptability and cohesion (Waller et al., 1990a), low maternal and paternal care (Palmer et al., 1988) and high paternal overprotectiveness (Calam et al., 1990), less cohesion, less expressiveness, less orientation towards recreational activities, more conflict, and less emotional support (Shisslak et al., 1990). Kog et al. (1989ab) failed to find differences between eating disorder patients and their parents and control families on perceived cohesion. Using different methodology, the same authors (Kog and Vandereycken, 1989c) found that eating disordered families showed significantly fewer disagreements between parents and children (conflict avoidance) and more stability in their behavioural interaction in the family (rigidity), and saw this as support of Minuchin’s model of the psychosomatic family.

Looking specifically at the differences between anorexia nervosa sufferers and normal controls, several studies found that anorexics showed more family problems. Parents of anorexics gave a double message of nurturant affection combined with neglect of
their daughter's need to express herself (Humphrey, 1989). In another study major problems including deaths in first degree relatives were more common in anorexic families than in control families (Rastam and Gillberg, 1991). In a population-based study of over 2000 female twins high maternal overprotectiveness was significantly associated with anorexia nervosa (Walters and Kendler, 1995).

Compared with normal controls, bulimic anorexics rated their families as more isolated, nondisclosing, less involved, supportive and organised, with little intellectual emphasis (Humphrey, 1986b); as more negative, less positive and more contradictory in their communications (Humphrey et al., 1986), as more belittling, ignoring and less helping, trusting and nurturing (Humphrey, 1987) and as less cohesive and expressive and more conflicted (Stern et al., 1989).

In contrast, several other studies (Houben et al., 1989; Humphrey, 1986a; Leon et al., 1985; North et al., 1995; Russell et al., 1992; Steiger et al., 1991b) found no difference between the way in which anorexics and normal controls experienced their relationship with their parents. Blair et al. (1995) found little difference between anorexic and well families on self-report measures of family functioning, but interestingly, AN families showed more emotional overinvolvement than the well families on an interview measure of Expressed Emotion.

With the exception of the study by Rybicki et al. (1989), individuals with DSM-III bulimia or bulimia nervosa on the whole report more family problems than normal controls (Waller et al., 1990ab; Woodside et al., 1995a). This includes poorer general
family functioning (McNamara and Loveman, 1990) and specifically more conflict and less cohesion, expressiveness, independence and intellectual-cultural orientation (Blouin et al., 1990; Johnson and Flach, 1985; Ordman and Kirschenbaum, 1986; Steiger et al., 1991b; Stern et al., 1989), less nurturing and less empathy (Humphrey, 1986a), less maternal care (Pole et al., 1988) or warmth (Stuart et al., 1990), more hostile enmeshment with their mothers (Humphrey, 1989), more parental conflict, less parental attention and affection (Dolan et al., 1990), more parental rejection (Stuart et al., 1990), less paternal affection and more paternal control to them than towards their siblings (Wonderlich et al., 1994).

In a non-clinical sample, bulimics, subclinical bulimics and normal controls differed significantly in the percentage who perceived their families as chaotic with 67%, 57% and 36% falling into this category respectively (Hastings and Kern, 1994).

Kendler et al. (1991) in a population-based study of over 2000 female twins found that low paternal care was one of the risk factors for the development of bulimia nervosa.

One study of a non-clinical bulimic sample found no difference between bulimics and controls on the FES (Kent and Clopton, 1988). Two other studies (Blouin et al., 1990; Wonderlich and Swift, 1990a) noted that, if depression was controlled for, there was also no difference between bulimics and controls in their perception of family functioning.
2.4.2. Does family functioning or childhood care of eating disorder sufferers differ from that of psychiatric controls or that of other patient control populations?

Depending on what kind of control group is chosen, eating disorder patients show either less, comparable or greater amounts of family pathology. An early family observation study (Goldstein, 1981) found that the parents of pre-schizophrenic patients showed significantly more communication deviance and a more negative affective style than those of anorexic patients. In three other studies anorexia nervosa patients evaluated their families as less pathological than mixed psychiatric patients (Heron and Leheup, 1984; North et al., 1995; Russell et al., 1992). Two further studies (Stuart et al., 1990; Thienemann and Steiner, 1993) found no difference between different eating disorder subgroups and controls with depressive disorders. Blair et al. (1995), whilst finding little difference between AN families and families of cystic fibrosis sufferers on measures of self-report or on Expressed Emotion measures, found more households in the AN group to be enmeshed, over-protective and poor at problem-solving than in the cystic fibrosis group using family observation.

2.4.3. Are there differences between different types of eating disorders in terms of family functioning or childhood care?

Several studies provide evidence for a 'binger/restricter dichotomy', with bulimics and/or bulimic anorexics reporting more or different family pathology than restricting anorexics (Garner et al., 1985; Humphrey, 1989; Kog et al., 1989ab), in particular more conflict (Strober, 1981), more hostile enmeshment (Humphrey, 1989), more criticism (Dare et al., 1994; Szmukler et al., 1985), less cohesion (Dare et al., 1994;
Strober, 1981), less organization (Strober, 1981) and less nurturing (Humphrey, 1986a). In contrast, a small study by Kog and Vandereycken (1989c) found that families of anorexics, irrespective of whether the patient binged or restricted, tended to fit Minuchin’s psychosomatic family type, whereas the families of normal weight bulimics were more conflicted and disorganized. Compared with parents of bulimics and those of undergraduate controls, the parents of anorexics gave a "double message" of nurturant affection combined with neglect of their daughter’s need to express themselves (Humphrey, 1989). In a non-clinical sample of male and female students whilst there was no significant correlation between family variables and dieting behaviour, compulsive eating related to lack of cohesion among females and to rigidity and lack of cohesion among males (Kagan and Squires, 1985).

Several other studies failed to identify any difference between different types of eating disorders in terms of family functioning and childhood care (Calam et al., 1990; Leon et al., 1985; Shisslak et al., 1990; Steiger et al., 1989; Steiger et al., 1991a; Thienemann & Steiner, 1993; Waller et al., 1990b), or only found very minor differences between groups (Stern et al., 1989). In one study, differences between anorexic and bulimic families disappeared when levels of depression were controlled for (Wonderlich & Swift, 1990a).

2.4.4. Is there any link between the severity of eating symptomatology and the severity of the family dysfunction?

Four studies have addressed this question. Using a composite severity index, added up from a measure of binge frequency and a measure of the extent to which bulimic
behaviour impaired the patient's life, a study of bulimia nervosa patients and normal controls found that the greater the reported family disorganization the more severe the eating disorder (Johnson and Flach, 1985). In a non-clinical sample, inconsistent expression of affection by mother was the best predictor of severity of eating disturbance (Scalf-McIver and Thompson, 1989). In a third study, also carried out in a non-clinical population, the higher the score on the Eating Disorder Inventory the more dysfunctional the family background seemed to be (Kinzl et al., 1994). In a series of 81 bulimic women bingeing was more frequent when the family was perceived to have poor problem-solving skills, but less frequent when the women perceived their families as having a more cohesive style (Waller, 1994a).

2.4.5. Do the perceptions of other family members differ from the eating disordered subject's view of family functioning and childhood care?

This question looks at differences between mothers and fathers (separately or together) and the patient. No study to date has compared eating disordered subjects with their siblings in this respect. Observational studies where whole families are assessed or studies where parents only are assessed are discussed under questions 1, 2, and 3 as appropriate. Two small studies show good agreement between bulimic-anorexic patients and their parents in their perception of family functioning, which both patients and parents saw as more disturbed than normal controls (Humphrey 1986b; 1987). In all other studies addressing this point eating disordered subjects rated their families as more disturbed than their parents (Dare et al., 1994; Stern et al., 1989; Waller et al., 1990b; Woodside et al., 1995b). This difference between generations occurs regardless of eating disorder diagnosis (Stern et al., 1989) with mothers' ratings being
more similar to their eating disordered daughters than the ratings of fathers (Waller et al., 1990b).

2.4.6. Are there any mediators between a dysfunctional family environment and later eating disorders?

(Studies in this section are not included in table 2.6.1). There is some suggestion that early childhood experiences have more of an effect on Axis II than on Axis I disorders. Johnson et al. (1990) divided a consecutive series of anorexic and bulimic patients into those with or without borderline personality features along a number of dimensions including family environment. Borderline patients reported feeling enmeshed in a disengaged, but controlling family, with high levels of conflict and little opportunity to express their feelings. In contrast, non-borderline patients were very similar in their perception of their families to normal controls. A further study found that eating disorder subjects with a borderline personality disorder perceived their relationships with their mothers as mutually attacking and hostilely withdrawn, whereas their relationship with their fathers distinguished them much less from the other eating disordered groups (Wonderlich and Swift, 1990b). Another study (Steiger et al., 1991a) found greater personality dysfunction to be linked to reports of the family's inability to respond empathetically. In a study by Head and Williamson (1990) particular types of personality were associated with specific family environments, however, there were no links between family patterns and eating symptomatology.
2.4.7. Other studies on family functioning and childhood care

Two interesting studies fall outside the questions asked above (not included in the table). In a comparison of 14 and 15 year old Asian and Caucasian schoolgirls Ahmad et al. (1994) found that Asian girls rated both their parents as more overprotective and their mothers as lower in care than Caucasians. In the Asian girls, greater eating pathology was correlated with perceptions of low maternal care and high maternal control. These findings were replicated by McCourt and Waller (1995) in a cohort of Asian and Caucasian schoolgirls aged 12 to 16.

2.5. Summary and Conclusions

Bearing in mind the differences in the populations and control groups assessed and the limitations in the assessment methods used in most of the above studies it is difficult to reach any firm conclusions about the quality of family functioning before onset of eating disorders.

(1) There is evidence of more family pathology in eating disorder families than in normal control families, although this may be mainly due to more family pathology in bulimic or bulimic anorexic families. Depression may be a confounding factor here.

(2) Different eating disorder subgroups show more or less family pathology than psychiatric or other medical controls depending on the nature of the comparison group.

(3) There is some evidence for a difference between anorexia nervosa and bulimia nervosa sufferers and for a binger/restricter dichotomy.

(4) There is some evidence that high reported family disturbance is associated with greater severity of bulimic symptomatology.
(5) Parents of eating disordered subjects on the whole rate their families as less disturbed than their children.

(6) There is some suggestion that family functioning may have more of an effect on Axis II than on Axis I disorders.

None of the studies described so far use the strategies outlined by Brewin et al. (1993) to help avoid unrealistic demands on individuals memories and to increase reliability of retrospective reports about childhood family functioning, i.e. either accounts from siblings close in age and gender or semistructured interviews using recognition cues.
### 2.6. Tables

**Table 2.6.1. Studies of the family environment of individuals with eating disorders (in order of appearance).**

<table>
<thead>
<tr>
<th>Authors</th>
<th>n</th>
<th>Comparison Groups</th>
<th>Subjects</th>
<th>Measures</th>
<th>Research Question</th>
<th>Past, Present or Premorbid Family Environment Assessed</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Goldstein, 1981</td>
<td>11</td>
<td>AN psychiatric controls</td>
<td>parents</td>
<td>Thematic Apperception Test Interview of parents</td>
<td>2</td>
<td>current</td>
<td>Parents of pre-schizophrenic patients showed significantly more communication deviance and negative affective style.</td>
</tr>
<tr>
<td></td>
<td>52</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Strober, 1981</td>
<td>22</td>
<td>RAN BAN</td>
<td>parents</td>
<td>FES</td>
<td>3</td>
<td>current</td>
<td>Parents of bulimic anorexics rated their families as higher on conflict and lower on cohesiveness &amp; organization than did parents of restrictors</td>
</tr>
<tr>
<td></td>
<td>22</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Garfinkel et al., 1983</td>
<td>23</td>
<td>AN controls</td>
<td>patients + parents</td>
<td>FAD</td>
<td>1</td>
<td>current</td>
<td>Fathers scored higher on conscientiousness, mother and daughters scored more family problems.</td>
</tr>
<tr>
<td></td>
<td>12</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Heron &amp; Leheup, 1984</td>
<td>16</td>
<td>AN psychiatric controls</td>
<td>patients</td>
<td>Case-note review</td>
<td>2</td>
<td>current and past</td>
<td>Families of AN patients were closer, more exclusive and happier than those of controls and had less external stress.</td>
</tr>
<tr>
<td></td>
<td>40</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Study</td>
<td>Sample Size</td>
<td>Group 1</td>
<td>Group 2</td>
<td>Measure</td>
<td>Mean</td>
<td>Time</td>
<td>Findings</td>
</tr>
<tr>
<td>---------------------</td>
<td>-------------</td>
<td>---------</td>
<td>---------</td>
<td>---------</td>
<td>------</td>
<td>------</td>
<td>----------</td>
</tr>
<tr>
<td>Garner et al., 1985</td>
<td>59</td>
<td>RAN</td>
<td>patients</td>
<td>FAM</td>
<td>3</td>
<td>current</td>
<td>Bulimic anorexics and bulimics scored significantly more family pathology than restricters.</td>
</tr>
<tr>
<td></td>
<td>59</td>
<td>BAN</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>59</td>
<td>bulimia</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Johnson &amp; Flach, 1985</td>
<td>105</td>
<td>bulimia</td>
<td>patients</td>
<td>FES</td>
<td>1.4</td>
<td>current</td>
<td>BN patients rated families as higher on conflict and lower on cohesion, expressiveness, independence and intellectual-cultural orientation than controls. Family disorganization associated with severity of symptoms.</td>
</tr>
<tr>
<td></td>
<td>86</td>
<td>controls</td>
<td>students</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kagan &amp; Squires, 1985</td>
<td>105</td>
<td>male</td>
<td>students who tended to overeat or restrict diet</td>
<td>FACES II</td>
<td>3</td>
<td>current</td>
<td>No significant correlation between cohesion or adaptability and dieting behaviour among males or females. Compulsive eating related to lack of cohesion among females and to rigidity and lack of cohesion among males.</td>
</tr>
<tr>
<td></td>
<td>195</td>
<td>female</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kog et al., 1985</td>
<td>50</td>
<td>ED</td>
<td>patients + families</td>
<td>LFQ</td>
<td>1</td>
<td>current</td>
<td>All eating disorder groups scored significantly more conflict and more disorganization.</td>
</tr>
<tr>
<td></td>
<td>210</td>
<td>controls</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Study</td>
<td>N</td>
<td>Group</td>
<td>Condition</td>
<td>Measure</td>
<td>Effect Size</td>
<td>Note</td>
<td></td>
</tr>
<tr>
<td>-------------------</td>
<td>----</td>
<td>--------</td>
<td>--------------------</td>
<td>---------</td>
<td>-------------</td>
<td>-------------------------------------------</td>
<td></td>
</tr>
<tr>
<td>Leon et al., 1985</td>
<td></td>
<td>RAN</td>
<td>patients + families</td>
<td>FES</td>
<td>1.3</td>
<td>No differences in youngsters' perception</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>BAN</td>
<td>families</td>
<td></td>
<td></td>
<td>Normal parents scored significantly higher on cohesion and expressiveness. Normal mothers, compared to AN mothers, scored higher on the independence scale.</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>control</td>
<td>students + families</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Szmukler et al., 1985</td>
<td>34</td>
<td>AN</td>
<td>patients + parents</td>
<td>CFI</td>
<td>3</td>
<td>Parents of bulimics more critical than those of anorexics.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>17</td>
<td>BN</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Humphrey, 1986a</td>
<td>20</td>
<td>RAN</td>
<td>patients</td>
<td>SASB</td>
<td>1.3</td>
<td>The two bulimic subgroups perceived their parents as less nurturing and less empathic than controls, whereas classical anorexics did not. However, only the deficit in parental nurturance was also significantly more severe in bulimics than in restricting anorexia nervosa.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>20</td>
<td>Bulimia</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>20</td>
<td>BAN</td>
<td>students</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>20</td>
<td>controls</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Humphrey, 1986b</td>
<td>16</td>
<td>BAN</td>
<td>patients + parents</td>
<td>FES,</td>
<td>1.5</td>
<td>Patients and parents rated their families as more isolated, nondisclosing, less involved, supportive and organised with little intellectual emphasis.</td>
<td></td>
</tr>
<tr>
<td></td>
<td>24</td>
<td>controls</td>
<td>patients + parents</td>
<td>FACES</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Study</td>
<td>Sample Size</td>
<td>Intervention</td>
<td>Comparison</td>
<td>Duration</td>
<td>Findings</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-----------------------------------------</td>
<td>-------------</td>
<td>--------------</td>
<td>------------</td>
<td>----------</td>
<td>----------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Humphrey et al., 1986</td>
<td>16, 24</td>
<td>BAN controls</td>
<td>patients + parents</td>
<td>1</td>
<td>Both systems concurred in describing families with a bulimic anorexic daughter as more disturbed (more negative, less positive and more contradictory in their communications) than normal controls.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ordman &amp; Kirschenbaum, 1986</td>
<td>25, 36</td>
<td>Bulimia controls</td>
<td>patients + students</td>
<td>1</td>
<td>Bulimia patients reported less cohesion, expressiveness and active recreational orientation and more conflict than normal controls.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Humphrey, 1987</td>
<td>16, 24</td>
<td>BAN controls</td>
<td>patients + parents</td>
<td>1.5</td>
<td>Questionnaires: Families of patients showed more Belittling and Appeasing, Ignoring and Waiting Off and were less Helping and Trusting and Nurturing and Approaching than were their nondisturbed counterparts. Observation: Parents of patients use more &quot;double-binding&quot; towards their daughters.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kent &amp; Clopton, 1988</td>
<td>27, 21, 35</td>
<td>Bulimic subclinical bulimics</td>
<td>female students</td>
<td>1</td>
<td>No difference between bulimics and controls on family variables.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Palmer et al., 1988</td>
<td>35, 37</td>
<td>AN bulimia</td>
<td>patients</td>
<td>1</td>
<td>Compared with normative data patients reported low parental care, but normal protectiveness.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Study</td>
<td>N</td>
<td>Groups</td>
<td>Participants Description</td>
<td>Instrument</td>
<td>Score</td>
<td>Time Period</td>
<td>Summary</td>
</tr>
<tr>
<td>-----------------------</td>
<td>---</td>
<td>---------------------</td>
<td>-----------------------------------------------</td>
<td>-------------</td>
<td>-------</td>
<td>-------------</td>
<td>-------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Pole et al., 1988</td>
<td>56</td>
<td>30</td>
<td>bulimia controls</td>
<td>patients</td>
<td>PBI</td>
<td>past</td>
<td>Bulimics perceive their parents, especially their mothers, as less caring</td>
</tr>
<tr>
<td></td>
<td></td>
<td>students or</td>
<td></td>
<td>professionals</td>
<td></td>
<td></td>
<td>than controls</td>
</tr>
<tr>
<td></td>
<td></td>
<td>professionals</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Houben et al. 1989</td>
<td>43</td>
<td>AN</td>
<td>controls</td>
<td>patients +</td>
<td>BLRI</td>
<td>1</td>
<td>current</td>
</tr>
<tr>
<td></td>
<td>18</td>
<td>parents</td>
<td>patients + parents</td>
<td>students +</td>
<td></td>
<td></td>
<td>AN patients experienced their relationship with their parents as similar</td>
</tr>
<tr>
<td></td>
<td></td>
<td>parents</td>
<td>patients + parents</td>
<td>parents</td>
<td></td>
<td></td>
<td>to normal daughters</td>
</tr>
<tr>
<td>Humphrey, 1989</td>
<td>16</td>
<td>AN</td>
<td>controls</td>
<td>patients +</td>
<td></td>
<td>1.3.5</td>
<td>current</td>
</tr>
<tr>
<td></td>
<td>18</td>
<td>bulimia</td>
<td>patients + parents</td>
<td>patients +</td>
<td></td>
<td></td>
<td>Compared to controls and other eating disorders bulimics and their</td>
</tr>
<tr>
<td></td>
<td>16</td>
<td></td>
<td>patients + parents</td>
<td>parents</td>
<td></td>
<td></td>
<td>mothers showed hostile enforcement; parents of anorexics differed from</td>
</tr>
<tr>
<td></td>
<td>24</td>
<td>controls</td>
<td>patients + parents</td>
<td>students +</td>
<td></td>
<td></td>
<td>parents of other eating disorder patients and controls in giving a</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>patients + parents</td>
<td>parents</td>
<td></td>
<td></td>
<td>double message of nurturant affection combined with neglect of their</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>patients + parents</td>
<td></td>
<td></td>
<td></td>
<td>daughter's needs to express themselves</td>
</tr>
<tr>
<td>Kog et al., 1989ab</td>
<td>31</td>
<td>RAN</td>
<td>controls</td>
<td>patients +</td>
<td>LFQ</td>
<td>1.3.5</td>
<td>current</td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>BAN</td>
<td>families</td>
<td>families</td>
<td></td>
<td></td>
<td>Patients and their parents report significantly more conflict &amp;</td>
</tr>
<tr>
<td></td>
<td>9</td>
<td>bulimia</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>disorganization but do not differ from control families on perceived</td>
</tr>
<tr>
<td></td>
<td>5</td>
<td>EDNOS</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>cohesion; BN and BAN patients show more family disturbance than</td>
</tr>
<tr>
<td></td>
<td>220</td>
<td>controls</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>RAN patients</td>
</tr>
<tr>
<td>Study</td>
<td>N1</td>
<td>N2</td>
<td>Group 1</td>
<td>Group 2</td>
<td>Measure</td>
<td>Effect Size</td>
<td>Year</td>
</tr>
<tr>
<td>-------------------------------</td>
<td>----</td>
<td>----</td>
<td>---------</td>
<td>---------</td>
<td>---------</td>
<td>-------------</td>
<td>------</td>
</tr>
<tr>
<td>Kog &amp; Vanderreycken, 1989c</td>
<td>25</td>
<td>5</td>
<td>patients + parents</td>
<td>controls</td>
<td>LFQ + behavioural tasks</td>
<td>1.3</td>
<td>current</td>
</tr>
<tr>
<td>Rybicki et al., 1989</td>
<td>38</td>
<td>20</td>
<td>DSM-III bulimia normal controls</td>
<td>patients normal controls</td>
<td>FES</td>
<td>1</td>
<td>current</td>
</tr>
<tr>
<td>Szlavit-McLer &amp; Thompson, 1989</td>
<td>175</td>
<td></td>
<td>female students</td>
<td>--</td>
<td>FES, PILS</td>
<td>4</td>
<td>current and past</td>
</tr>
<tr>
<td>Steiger et al., 1989</td>
<td>15</td>
<td>9</td>
<td>RAN, BAN, BN, BN/HistAN controls</td>
<td>patients</td>
<td>PBI</td>
<td>1.3</td>
<td>past</td>
</tr>
<tr>
<td>Study</td>
<td>Sample Size</td>
<td>Group Description</td>
<td>Measure</td>
<td>Year</td>
<td>Comparison</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-----------------------</td>
<td>-------------</td>
<td>------------------------------------------------</td>
<td>---------</td>
<td>------</td>
<td>---------------------------------------------------------------------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stern et al., 1990</td>
<td>20</td>
<td>AN, AN + BN, bulimia, controls</td>
<td>patients + parents</td>
<td>FES</td>
<td>1, 3, 5, current</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>13</td>
<td>AN, AN + BN</td>
<td>normal controls from different sources</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>24</td>
<td>controls</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>57</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Comparison between eating disorder groups: parents of bulimic patients rated their families higher on achievement orientation than parents of patients with bulimic AN. Compared to controls: subjects with bulimia and bulimic anorexia rated families as low in cohesion, all patients rated families as low in expressiveness, parents of subjects with bulimic AN rated their families as high in conflict, subjects with bulimia rated family as high in achievement orientation.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blouin et al., 1990</td>
<td>99</td>
<td>bulimia, controls</td>
<td>patients</td>
<td>FES</td>
<td>1, current</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>37</td>
<td></td>
<td>students</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Depressed bulimics perceived their families as less cohesive, less independent, more achievement oriented, less expressive and less involved in recreational pursuits than controls. Non-depressed bulimics differed little from controls.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Calams et al., 1990</td>
<td>31</td>
<td>AN, BN/HistAN, BN, controls</td>
<td>patients</td>
<td>PBI</td>
<td>1.3, past</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>34</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>33</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>242</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>Patients recalled mothers as low in caring, and fathers as overprotective and low in care. Diagnostic groups were not well differentiated by the PBI.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Study</td>
<td>Sample 1</td>
<td>Sample 2</td>
<td>Method</td>
<td>Sample 3</td>
<td>Sample 4</td>
<td>Sample 5</td>
<td>Sample 6</td>
</tr>
<tr>
<td>------------------------------</td>
<td>----------</td>
<td>-------------------</td>
<td>-----------------</td>
<td>----------</td>
<td>----------</td>
<td>----------</td>
<td>----------</td>
</tr>
<tr>
<td>Dolan et al., 1996</td>
<td>50</td>
<td>bulimia controls</td>
<td>patients</td>
<td>POP +</td>
<td>1</td>
<td>current</td>
<td></td>
</tr>
<tr>
<td></td>
<td>40</td>
<td>repeat-dieters</td>
<td>GP-attenders</td>
<td>additional questions</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>McNamara &amp; Loveman, 1990</td>
<td>31</td>
<td>bulimia</td>
<td>students</td>
<td>FAD</td>
<td>1</td>
<td>current</td>
<td></td>
</tr>
<tr>
<td></td>
<td>61</td>
<td>repeat-dieters</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>59</td>
<td>non-dieters</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Shisslak et al., 1990</td>
<td>13</td>
<td>BAN</td>
<td>patients</td>
<td>FES,</td>
<td>1.3</td>
<td>current</td>
<td></td>
</tr>
<tr>
<td></td>
<td>24</td>
<td>bulimia controls</td>
<td></td>
<td>FDS</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>41</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stuart et al., 1990</td>
<td>30</td>
<td>bulimia</td>
<td>patients</td>
<td>EMBU</td>
<td>1.2</td>
<td>past</td>
<td></td>
</tr>
<tr>
<td></td>
<td>15</td>
<td>depression</td>
<td>patients</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>100</td>
<td>controls</td>
<td>normals</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Waller et al., 1990a</td>
<td>41</td>
<td>eating disorder</td>
<td>patients</td>
<td>FACES II</td>
<td>1</td>
<td>current</td>
<td></td>
</tr>
<tr>
<td></td>
<td>27</td>
<td>controls</td>
<td>normals recruited from different sources</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Waller et al., 1996b</td>
<td>14</td>
<td>AN</td>
<td>patients + parents + normals + parents (recruited from different sources)</td>
<td>FAD</td>
<td>1.3,5</td>
<td>current</td>
<td>No difference between AN and BN; patients raised their families as more unhealthy on all subscales of the questionnaire, mothers of ED patients rated their families as less healthy on 2 subscales and fathers did not differ from control fathers; sufferers themselves had most realistic perceptions of their families' interactional styles.</td>
</tr>
<tr>
<td>---------------------</td>
<td>-----</td>
<td>-------</td>
<td>--------------------------------------------------------------------------------</td>
<td>-----</td>
<td>-------</td>
<td>---------</td>
<td>-------------------------------------------------</td>
</tr>
<tr>
<td></td>
<td>34</td>
<td>BN</td>
<td>controls</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>30</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Wonderlich &amp; Swift, 1996a</td>
<td>11</td>
<td>AN</td>
<td>patients</td>
<td>SASB</td>
<td>1.3</td>
<td>current</td>
<td>When mood was controlled for, there were no significant differences between the different groups.</td>
</tr>
<tr>
<td></td>
<td>11</td>
<td>BAN</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>26</td>
<td>BN</td>
<td>students</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kendler et al., 1991</td>
<td>2163</td>
<td>female twins</td>
<td>population-based</td>
<td>PBI</td>
<td>1</td>
<td>past</td>
<td>Low parental care was a risk factor for development of BN.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rasmussen &amp; Gilberg, 1991</td>
<td>51</td>
<td>AN</td>
<td>parents of population-based cases and patients</td>
<td>FACES; interview with mother</td>
<td>1</td>
<td>current, past and premorbid</td>
<td>No support for typical anorexic family, however there were more major problems in the anorexia group including a high prevalence of death in first degree relatives.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>51</td>
<td>controls</td>
<td>parents of normal students</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Study</td>
<td>Sample Size</td>
<td>Conditions</td>
<td>Comparison</td>
<td>Results</td>
<td>Notes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>-------------------------------</td>
<td>-------------</td>
<td>------------</td>
<td>------------</td>
<td>---------</td>
<td>----------------------------------------------------------------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Steiger et al., 1991a</td>
<td>22</td>
<td>RAN</td>
<td>patients</td>
<td>FAD 1.3</td>
<td>No difference between different eating disorder types; eating disorder patients showed more family pathology than controls.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>12</td>
<td>BAN</td>
<td></td>
<td>FAD</td>
<td>current</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>14</td>
<td>BN/HistAN</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>20</td>
<td>BN</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>25</td>
<td>controls</td>
<td>students</td>
<td>FAD</td>
<td>current</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Steiger et al., 1991b</td>
<td>14</td>
<td>restricters</td>
<td>students</td>
<td>FAD 1</td>
<td>Restricters not different from asymptomatic controls, bingers more family pathology than normals.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>10</td>
<td>underweight</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>20</td>
<td>bingers</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>51</td>
<td>normal-weight</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>410</td>
<td>bingers</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>atypical eating disorders</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>asymptomatic</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Russell et al., 1992</td>
<td>54</td>
<td>RAN</td>
<td>adolescent patients</td>
<td>PBI 1.2</td>
<td>Anorexia's report of childhood experiences more like that of normal controls than that of psychiatric controls.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>54</td>
<td>psychiatric controls</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>54</td>
<td>normal adolescents</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>adolescent patients</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>adolescent students</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Thienemann &amp; Steiner, 1993</td>
<td>118</td>
<td>AN</td>
<td>patients + parents</td>
<td>FES 1.2,3</td>
<td>No difference on FES between diagnostic groups, no difference from a normative population, no difference between parental FES response of patients and parental response from normative sample, however, when subjects were grouped by self-reported level of depression those with high levels of depression reported family environment as different independent of diagnosis.</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>BAN</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>bulimia</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>major depression</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Study</td>
<td>Sample Size</td>
<td>Gender</td>
<td>Age</td>
<td>Diagnosis</td>
<td>Measure</td>
<td>Score</td>
<td>Time Frame</td>
</tr>
<tr>
<td>------------------------</td>
<td>-------------</td>
<td>--------</td>
<td>-----</td>
<td>-------------------</td>
<td>---------</td>
<td>-------</td>
<td>------------</td>
</tr>
<tr>
<td>Dare et al., 1994</td>
<td>18</td>
<td>AN</td>
<td>8</td>
<td>AN and BN</td>
<td>patients</td>
<td></td>
<td>current</td>
</tr>
<tr>
<td>Hastings &amp; Kern, 1994</td>
<td>30</td>
<td>female</td>
<td>37</td>
<td>bulimic</td>
<td>FES</td>
<td>1</td>
<td>current</td>
</tr>
<tr>
<td>Kinai et al., 1994</td>
<td>202</td>
<td>female</td>
<td>N/A</td>
<td>University</td>
<td>BIDBD</td>
<td>4</td>
<td>past</td>
</tr>
<tr>
<td>Study</td>
<td>FES</td>
<td>BN controls</td>
<td>BN cases</td>
<td>AN cystic fibrosis</td>
<td>AN normal</td>
<td>Side effects</td>
<td>FAM, EFS, CHI</td>
</tr>
<tr>
<td>-------</td>
<td>-----</td>
<td>-------------</td>
<td>----------</td>
<td>-------------------</td>
<td>-----------</td>
<td>--------------</td>
<td>--------------</td>
</tr>
<tr>
<td>Wunderlich et al. 1994</td>
<td>27</td>
<td>29</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Blair et al. 1995</td>
<td>27</td>
<td>29</td>
<td>31</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Study</td>
<td>Sample Size</td>
<td>Sample Description</td>
<td>Measure</td>
<td>Score</td>
<td>Status</td>
<td>Notes</td>
<td></td>
</tr>
<tr>
<td>------------------------------</td>
<td>-------------</td>
<td>--------------------</td>
<td>---------</td>
<td>-------</td>
<td>----------</td>
<td>-----------------------------------------------------------------------</td>
<td></td>
</tr>
<tr>
<td>North et al., 1995</td>
<td>35 35 35</td>
<td>AN psychiatric controls community controls patients + mothers</td>
<td>FAD, MCSIFF</td>
<td>1.2</td>
<td>present</td>
<td>AN patients and community controls did not differ on FAD scores, with psychiatric controls rating worse family functioning. On the interview measure which was given to anorexic families only, the median ratings from this group fall in the clinical range.</td>
<td></td>
</tr>
<tr>
<td>Walters &amp; Kendler, 1995</td>
<td>2163</td>
<td>female twins population-based</td>
<td>PBI</td>
<td>1</td>
<td>past</td>
<td>Maternal overprotectiveness associated with anorexia nervosa.</td>
<td></td>
</tr>
<tr>
<td>Woodside et al., 1995</td>
<td>93</td>
<td>BN patients</td>
<td>FAM</td>
<td>1.5</td>
<td>present</td>
<td>BN patients reported more family disturbance compared to population norms. Patients reported more family disturbance than their parents.</td>
<td></td>
</tr>
</tbody>
</table>
Table 2.6.1. Studies of the family environment of individuals with eating disorders.

Comparison groups: AN: anorexia nervosa, BN = bulimia nervosa, BN/HisAN: bulimia nervosa with a history of anorexia nervosa; RAN: restricting anorexia nervosa; BAN: bulimic anorexia nervosa; ED: eating disorder; EDNOS: eating disorder not otherwise specified.

Measures:

Semistructured Interviews
EE = Expressed Emotion (Brown and Rutter, 1966)
CFI = Camberwell Family Interview (Vaughn & Leff, 1976)
SCFI = Standardized Clinical Family Interview (Kinston & Loader, 1984)

Family Interaction Coding Systems
MICS = Marital Interaction Coding System (Robin & Weiss, 1980)
MCSIFF = McMaster Structured Interview of Family Functioning (Bishop et al., 1987; quoted from North et al., 1995)
SASB = Structured Analysis of Social Behaviour (Benjamin, 1983)

Self-Report Questionnaires
BIDBD = Biographic Inventory for Diagnosis of Behavioral Disturbances (Jaeger et al., 1976). This instrument is similar to the PBI.
BLRI = Barrett-Lennard Relationship Inventory (Barrett-Lennard, 1962)
EFS = Edinburgh Family Scale (Blair et al., 1995).
EMBU (Perris et al., 1986).
FACES = Family Adaptability and Cohesion Evaluation Scale (Olson et al., 1979)
FACES II (Olson et al., 1982)
FAD = Family Assessment Device (Epstein et al., 1978)
FAM = Family Assessment Measure (Skinner et al., 1983)
FES = Family Environment Scale (Moos, 1974)
FDS = Family Dynamics Survey (Berren & Shisslak, 1980)
LFQ = Leuven Family Questionnaire (Kog et al., 1985)
PBI = Parental Bonding Instrument (Parker, 1979)
PILS = Parental Inconsistency of Love Scale (Schwarz & Zuroff, 1979)
POP = Perception of Parents Questionnaire (Schutz, 1966)
SIDE = Sibling Inventory of Differential Experiences (Daniels & Plomin, 1985)
SASB = Structured Analysis of Social Behaviour (Benjamin, 1983)

Research Questions:
1: Differences between eating disorders and normal controls
2: Differences between eating disorders and other patient populations (psychiatric and non-psychiatric)
3: Differences between different types of eating disorders
4: Associations between severity of family disturbance and severity of eating disorder
5: Differences between eating disordered subjects and other family members in perception of family problems
Chapter 3: The Role of Sexual and Physical Abuse in the Origins of Eating Disorders

3.1. Introduction

Approximately one hundred years after Freud posited the experience of "sexual seduction" as the universal cause for psychoneuroses "... we appear once again to be confronted with a unitary concept of pathogenesis. Childhood sexual abuse is increasingly invoked as the causative agent for a broad spectrum of disorders, especially in women, ranging from depression through eating disorders to such complex personality configurations as the borderline conditions and multiple personality disorder. More and more troubled people are "remembering" sexual violations, often under the supportive, encouraging, even coercive influence of therapists who are certain that the evocation and abreaction of such memories is the sine qua non of therapeutic success. .....Such unitary etiological concepts are, of course, nothing new; diabolical influences, "hereditary degeneration", exposure to the "primal scene" - each has had its day, has enjoyed its vogue, and has either passed into the dustbin of history or assumed its appropriate place in the etiological spectrum." (Esman, 1994).

The literature on eating disorders is replete with suggestions that sexual conflict during adolescence is causally related to anorexia nervosa. It has often been alleged that those with the illness are psychosexually immature and it has been speculated that anorexia nervosa is a rejection of adult sexuality (for review see Scott, 1987). In early psychoanalytic thinking, loss of appetite was equated with "in sexual terms, loss of libido" (Freud, 1902) and later a view developed of anorexia nervosa as a defense
against oral impregnation fantasies (Waller et al., 1940). Sexual abuse thus has by some been seen as a missing link explaining the anorexic's refusal of sexuality. Others, however, have suggested that the apparent association of sexual abuse and eating disorders may be coincidental rather than causal, due to the high prevalence of each of the two phenomena (Finn et al., 1986; Pope and Hudson, 1992). Given this controversy, it is not surprising that a large number of studies have appeared researching the association between childhood sexual abuse (CSA) and eating disorders (see table 3.8.1.).

In contrast to the marked interest in childhood sexual abuse, very little has been written about the role of physical abuse in the childhoods of patients with eating disorders (see table 3.8.2.), although a link between physical abuse and adult psychiatric disorder has been documented (Brown and Anderson, 1991).

3.2. Some Methodological Considerations

Reported rates of sexual abuse in subjects with eating disorders have varied greatly between studies, depending on populations studied (clinical or non-clinical), the method used to ascertain the abuse (e.g. one-off screening question, questionnaire, structured or clinical interview, therapy disclosure), response rates, the definition of abuse (whether non-contact abuse was included; whether there was an age gap between victim and perpetrator) and the age cut-off for inclusion as childhood abuse. Some studies have included abuse that occurred after onset, when purportedly being interested in factors leading to onset. Differences between eating disorder subjects and control subjects may sometimes appear exaggerated due to the fact that control groups were recruited from health workers, who may have been uncertain about the
confidentiality of the information they gave. Other methodological problems may arise through repression of abuse experiences or abuse confabulation (false memory syndrome), although some investigators and clinicians do not believe that the former exists (Andrews et al., 1995a; Pope and Hudson, 1995). Individuals who are at the point of study being sexually abused may be less likely to disclose than those who were abused in the past. For a full discussion of methodological problems in the study of sexual abuse see Briere (1992).

3.3. Organization of the Literature Review

The research literature in the area of sexual and physical abuse was screened using Medline up to July 1996. Studies were included if they addressed the questions outlined below, which are along similar lines to the ones used to describe the literature on family functioning in chapter two (see also tables 3.8.1 and 3.8.2.).

Research questions asked included:

1) Do subjects with eating disorders differ from normal controls in terms of rates of sexual or physical abuse?

2) Do eating disorder subjects differ from subjects with other psychiatric disorders in terms of rates of abuse reported?

3) Do women who have been abused have higher rates of eating disorders than those who have not been abused?

4) Are different types of eating disorders associated with different rates of childhood sexual and physical abuse?

5) Is there any link between abuse characteristics and the severity of the eating symptomatology?
(6) What - if any - psychological and contextual mediators exist between childhood abuse and later eating disorders?

For completeness, I have also included case series of eating disorder patients in the table, where no comparison was attempted. Under the table heading 'research question' these are designated as case series.

The question of whether there is agreement between different family members on the issue of abuse was not asked as there is no study comparing siblings and clearly there would be considerable ethical problems asking the parents of abuse victims.

3.4. Childhood Sexual Abuse

3.4.1. Do subjects with eating disorders differ from normal controls in terms of rates of sexual abuse reported?

Four of eleven studies failed to detect differences between eating disorder subjects and normal controls in terms of their rates of sexual abuse (Abramson and Lucido, 1991; Beckman and Burns, 1990; Rorty et al., 1994; Stuart et al., 1990). In contrast, Steiger and Zanko (1990) found a much higher rate of sexual abuse in bulimic anorexics and bulimic patients compared to their normal control group. However, the questionnaire was not anonymous and controls included hospital staff who may have been reluctant to reveal any sexual abuse. Two non-clinical studies (Hastings and Kern, 1994; Miller et al., 1993) also found higher rates for bulimics than controls. Vize and Cooper (1995) who interviewed eating disorder patients and non-eating disordered controls using part of the CECA (Bifulco et al., 1994) found a significantly
higher rate of childhood sexual abuse in their anorexic and bulimic patients. In a community study women with a life-time history of disordered eating had higher levels of intrafamilial sexual abuse than women selected at random (Bushnell et al., 1992). Likewise, another community study (Garfinkel et al., 1995) found bulimic women to have almost three times higher rates of childhood sexual abuse than non-bulimic women. The best study to date, a carefully conducted community-based case-control study (Welch and Fairburn, 1994) found the rates of childhood sexual abuse in bulimia nervosa in the community to be higher than in the general population, with no difference between those bulimic subjects who were or were not in treatment.

3.4.2. Do eating disorder subjects differ from subjects with other psychiatric disorders in terms of rates of abuse reported?

A number of studies failed to find differences between eating disorder patients and psychiatric controls [panic disorder (Ross et al., 1989); schizophrenia (Ross et al., 1989); mixed psychiatric disorders (Folsom et al., 1993; Köpp, 1994; Steiger and Zanko, 1990; Tice et al., 1989); depression (Stuart et al., 1990; Vize and Cooper, 1995); depression and anxiety disorders (Welch and Fairburn, 1994)]. In one of these studies (Tice et al., 1989) the results were reported as a higher rate of contact sexual abuse in eating disorder patients than in controls. However, when adult and post-onset abuse were excluded and gender differences between the study groups were corrected, the difference between groups was no longer significant (Pope and Hudson, 1992). In contrast, Palmer and Oppenheimer (1992) found the rate of CSA to be lower in eating disorder patients than in a mixed group of psychiatric patients and Ross et al. (1989) lower than in multiple personality disorder.
3.4.3. Do women who have been abused have higher rates of eating disorders than those who have not been abused?

A different way of approaching the question of whether there is an association between sexual abuse and eating disorders is to compare the rates of eating disorders in abused populations with non-abused populations. One study (Finn et al., 1986) failed to find any difference between sexually abused and non-abused women in the prevalence of bulimia nervosa, whereas three others found higher rates of eating disorders in sufferers of abuse (Mullen et al., 1993; Pribor and Dinwiddie, 1992; Shearer et al., 1990), one of them a large methodologically sound community study (Mullen et al., 1993). A study of female psychiatric in-patients found higher EDI subscores in those who had been sexually abused compared to those who had not been abused (Zlotnick et al., 1996). In three studies of non-clinical samples one did (Smolak et al., 1990) and two did not (Kinzl et al., 1994; Schaaf and McCanne, 1994) find any significant differences in eating disorder related variables between abused and non-abused students. It is difficult to draw any conclusions from this.

3.4.4. Are different types of eating disorders associated with different rates of childhood sexual abuse?

Most studies fail to find a difference between different eating disorder subtypes in rates of childhood sexual abuse (DeGroot, et al., 1992; Folsom et al., 1993; Hall et al., 1989; Köpp, 1994; McClelland et al., 1991; Oppenheimer et al., 1985; Palmer et al., 1990; 1992; Piran et al., 1988; Tice et al., 1989; Vize and Cooper, 1995). Three studies compared restricting anorexics with other eating disorder subtypes (Steiger and Zanko, 1990; Waller, 1991; Waller et al., 1993a) and found lower rates of sexual
abuse in restricters than in the other groups. Tobin and Griffing (1996) found significantly higher rates of sexual abuse in eating disorder patients who used compensatory behaviours (55%) than in those who did not (17%). However, all these studies used lenient definitions of sexual abuse (often including non-contact abuse, post-onset abuse, no minimum age-difference between victim and perpetrator specified and adult abuse) and in Waller’s study (1991) if stricter criteria were applied to his data, the differences between restricters and other patient groups become much less marked or may even disappear altogether. In one further study of over 700 eating disordered patients rates of contact sexual abuse below the age of 18 were higher among patients with bulimia nervosa (35%) and atypical eating disorders (31%) than in those with restricting (12%) or bulimic anorexia nervosa (11%) (Fullerton et al., 1995), however, it is unclear whether and to what degree these differences may have been inflated by post-onset abuse.

3.4.5. Is there any link between abuse characteristics and the severity of the eating symptomatology?

There is some evidence that the nature and severity of sexual abuse may also be associated with degree of eating pathology. In a clinical series of 40 bulimic women, drawn from Waller’s (1991) series, a reported history of sexual abuse was associated with more frequent bingeing (Waller, 1992a). These symptoms were more marked when the abuse was intrafamilial, involved force, or occurred before the victim was 14 years old. Women were also more likely to vomit more frequently if the reported abuse was intrafamilial (Waller, 1992b). Similarly, in a non-clinical study, Calam and Slade (1989) found that sexual abuse was associated with higher scores on the Eating
Attitudes Test, particularly in those cases with multiple abuse and abuse using force. Greater eating pathology was positively correlated with a greater number of childhood sexual experiences and with greater severity of the abuse (Abramson and Lucido, 1991).

In contrast, nine other studies (Bailey and Gibbons, 1989; Bushnell et al., 1992; Folsom et al., 1993; Fullerton et al., 1995; McCarthy et al., 1994; Pope et al., 1994; Smolak et al., 1990; Sullivan et al., 1995; Vize and Cooper, 1995) found no link between severity of eating symptomatology and abuse characteristics. Instead, Folsom et al. (1993) reported that sexually abused eating disorder patients had more psychological disturbance of obsessive and phobic nature than non-abused subjects and Sullivan et al. (1995) found greater depressive symptoms in bulimic patients with CSA compared to those without.

3.4.6. What - if any - contextual and psychological mediators exist between childhood abuse and later eating disorders?

Browne and Finkelhor (1986) propose that three interrelated types of factors influence the outcome of childhood sexual abuse: abuse characteristics, the child’s personality and contextual influences. The former was discussed above under point 3.4.5., the latter will be discussed in this section. One important contextual influence is the family’s ability to support the abused child. Smolak et al. (1990) found that for abused women there was a direct relationship between parental unreliability and higher EDI scores.

The victim’s negative response (fear and/or shock) at the time of the abuse and a negative evaluation in retrospect of the childhood experience were positively
correlated with eating pathology (Abramson and Lucido, 1991). In a case series of 117 eating-disordered women a negative response to disclosure was associated with significantly higher levels of vomiting and borderline personality symptoms and no response to disclosure was associated with a greater level of borderline personality symptoms (Waller and Ruddock, 1992). The same group (Everill and Waller, 1995b) examining a non-clinical sample, found that those with a perceived adverse response to disclosure showed greater levels of psychopathology, particularly oral control, dissociation and self-denigration than those who had not been abused or had had a supportive response.

In a study using strict criteria of CSA, eating disorder patients with a history of CSA were more likely to have an associated personality disorder than those without CSA (McClelland et al., 1991). In three other papers, based on 100 (Waller, 1993a) and 115 patients (Waller, 1993b; Waller, 1994b), Waller describes an association between reported sexual abuse and borderline personality disorder, but also that the links between reported sexual abuse and borderline personality disorder vary according to the nature of the eating disorder (Waller, 1993b). In particular, these two phenomena were associated in bulimic anorexia, but not in bulimia nervosa or in restrictive anorexia. Two other studies of patients with eating disorders failed to find any relationship between childhood sexual abuse and borderline personality disorder (Sullivan et al., 1995; Vize and Cooper, 1995).

Body-image distortion does not seem to mediate between childhood sexual abuse and eating disorders (Waller et al., 1993b; Schaaf and McCanne, 1994).
It has been suggested that levels of bulimic behaviour are greater among eating disordered women who have been abused, because the women have elaborate schemata relevant to the abuse and they may use bulimic behaviours to 'block out' the heightened awareness of self-denigratory cognitions. In support of this, one study found an association between vomiting and self-denigratory cognitions and emotions in abused bulimics (Pitts and Waller, 1993). In this study, general self-esteem was not significantly correlated with frequency of bingeing or vomiting in these patients.

Dissociative mechanisms have also been thought to have a mediating role between traumatic childhood experiences and later eating symptomatology (for review see Everill and Waller, 1995ac; Herzog et al., 1993; Miller et al., 1993; Vanderlinden et al., 1993), however, not all studies have found such a link (Berger et al., 1994; McCarthy et al., 1994). Moreover, it is not clear, whether bulimic behaviours result from dissociative phenomena or vice versa (McManus, 1995).

3.5. Childhood Physical Abuse

Only a handful of studies to date have focused on childhood physical abuse in eating disorder patients. Physical abuse may be easier to admit than sexual abuse, given the shame associated with incest (McCarthy et al., 1994). Studies of physical abuse have the same shortcomings as studies of sexual abuse. A number of studies do not give information on the perpetrator and his/her age difference to the victim, several studies do not specify an age cut-off for the abuse, none of the studies make an attempt to separate out abuse that occurred before onset of the eating disorder and several studies give no information on the severity of the abuse included. Those studies that do, seem
to include moderate to severe physical abuse. Not surprisingly, rates of physical abuse vary hugely across studies ranging from 3% in a mixed group of eating disorder patients (Vanderlinden et al., 1993) to 57% in a group of bulimic patients (McCarthy et al., 1994). Bulimic patients had significantly higher rates of physical abuse than normal controls without an eating disorder (Rorty et al., 1994). Eating disorder patients can have lower (Ross et al., 1989) or similar rates of physical abuse to other psychiatric patients (Folsom et al., 1993) depending on the type of psychiatric disorder in the control patients. In a non-clinical study those who had experienced childhood physical abuse did not differ from non-abused controls in terms of a number of eating disorder related variables (Schaaf and McCane, 1994). Fullerton et al. (1995) found that those with bulimic anorexia and bulimia nervosa reported physical abuse more frequently (33% and 30%) than subjects with restricting anorexia nervosa (11%) and subjects with atypical eating disorders (22%). Piran et al. (1988) identified a similar trend in a comparison of restricting anorexics with DSM-III bulimics. One study found a significant relationship between physical abuse and the severity of bulimia (Bailey and Gibbons, 1989), whereas two others found no correlation between a history of physical abuse and the severity of bingeing (Fullerton et al., 1995; McCarthy et al., 1994). Dissociation may be a mediator between physical abuse and eating disorders (Berger et al., 1994; McCarthy et al., 1994).

3.6. Psychological Abuse

Only one study to date has examined parental psychological abuse using a 7-item scale which required the subject to rate the extent to which her mother and her father separately had subjected her to "yelling, insults, criticism, guilt-inspiring statements,"
ridicule, embarrassment in front of others and attempts to make the child feel like a bad person" (Rorty et al., 1994). Bulimia nervosa subjects reported significantly higher rates of maternal and paternal psychological abuse than non-eating disordered control subjects, with 76.3% of bulimics reporting psychological maltreatment by at least one parent compared to 37.5% of controls.

3.7. Summary and Conclusions

Whilst a number of studies of different methodological standards report no difference between eating disorder subjects and normal controls, the best studies to date in this area suggest that the rates of sexual abuse in anorexia nervosa (Vize and Cooper, 1995) and bulimia nervosa (Vize and Cooper, 1995; Welch and Fairburn, 1994) are higher than in the general population. Depending on which psychiatric control groups are chosen, eating disorder patients may have similar, higher or lower rates of childhood sexual abuse. The majority of studies fail to find differences between different eating disorder subtypes in terms of rates of sexual abuse. The evidence on whether there is any link between abuse characteristics and the severity of eating symptomatology is inconclusive. On balance it seems likely that childhood sexual abuse is not a specific risk factor for either bulimia nervosa or other eating disorders (Connors and Morse, 1993; Pope and Hudson, 1992). Nonetheless, in clinical practice we are faced with the need to decide whether reported abuse is relevant in formulating the individual eating disorder and in planning treatment (Waller et al., 1994).

In the area of physical abuse the following conclusions can be drawn: Bulimics experience physical abuse more often than normal controls and there also seems to be clear evidence for a restricter binger dichotomy, with bulimics experiencing childhood
physical abuse more often than anorexics. However, compared to other psychiatric populations eating disorder patients experience less or similar amounts of physical abuse. Dissociation may be a mediator between physical abuse and the development of an eating disorder.
### 3.8. Tables

#### Table 3.8.1: Studies on sexual abuse and eating disorders

<table>
<thead>
<tr>
<th>Authors</th>
<th>Sample</th>
<th>Methods</th>
<th>Perpetrator</th>
<th>Age Cut-Off for Abuse</th>
<th>Abuse before Onset</th>
<th>Info on Severity of Abuse</th>
<th>Research Question</th>
<th>Findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Oppenheimer et al., 1985</td>
<td>75 female eating disorder patients; 36 AN; 33 BN; 9 both; DSM-III, outpatients</td>
<td>SLEQ</td>
<td>at least 3 to 5 years older</td>
<td>age 16 or below</td>
<td>no info</td>
<td>non-contact included</td>
<td>4</td>
<td>33% of AN, 24% of BN, 33% of mixed group reported CSA. When age difference criterion eliminated: 64% of AN and 66% of BN reported sexual abuse.</td>
</tr>
<tr>
<td>Finn et al., 1996</td>
<td>87 women attending psychotherapy groups</td>
<td>checklist followed by structured interview</td>
<td>intrafamilial abuse by adult or extrafamilial abuse</td>
<td>no age cut off</td>
<td>no info</td>
<td>contact abuse only</td>
<td>3</td>
<td>No difference between abused and non-abused women in the prevalence of bulimia nervosa.</td>
</tr>
<tr>
<td>Piran et al., 1988</td>
<td>30 RAN and 38 BAN patients</td>
<td>CEQ</td>
<td>no info</td>
<td>no info</td>
<td>no info</td>
<td>no info</td>
<td>4</td>
<td>CSA was reported by 8% of BAN patients and none of the RAN patients. This difference was not significant.</td>
</tr>
<tr>
<td>Root &amp; Fallon, 1988</td>
<td>172 DSM-III bulimia (outpatients)</td>
<td>Questionnaire</td>
<td>no info</td>
<td>no info</td>
<td>no info</td>
<td>no info</td>
<td>case series</td>
<td>28.5% of women had been sexually abused as children.</td>
</tr>
<tr>
<td>Study</td>
<td>Sample Description</td>
<td>Methodology</td>
<td>Data</td>
<td>N</td>
<td>Findings</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-------------------------------</td>
<td>----------------------------------------------------------</td>
<td>----------------------------------------------</td>
<td>-------------------------------------------</td>
<td>-----</td>
<td>---------------------------------------------------------------------------------------------------------------------------------------------</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bailey &amp; Gibbons, 1989</td>
<td>294 female college students</td>
<td>Questionnaire; response rate 99%</td>
<td>no info</td>
<td>5</td>
<td>13% reported sexual molestation, 11% rape, 8% battery, 6% childhood physical abuse. Unlike physical abuse, sexual abuse did not have a significant relationship with the diagnosis or severity of bulimia.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bulik et al., 1989</td>
<td>35 patients with DSM-III bulimia, of these 29 fulfilled DSM-III-R criteria</td>
<td>Semi-structured interview</td>
<td>no info</td>
<td>case series</td>
<td>29% had CSA.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Calam &amp; Slade, 1989</td>
<td>130 female undergraduates</td>
<td>SEQ; response rate 65 to 75%</td>
<td>divided into intra- and extrafamilial</td>
<td>5</td>
<td>58% of subjects reported some form of unwanted sexual experience. 31% of women had CSA before age 14; 20% reported intrafamilial abuse. Sexual abuse was associated with higher scores on the EAT, with those reporting the higher number of unwanted sexual events showing the highest scores. The experience of one or more sexual events involving force was also associated with higher EAT scores.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Study</td>
<td>Participants</td>
<td>Method</td>
<td>Details</td>
<td>Case Series</td>
<td>Finding</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>----------------</td>
<td>--------------</td>
<td>-------------------------</td>
<td>-------------------------------------------------------------------------</td>
<td>-------------</td>
<td>-------------------------------------------------------------------------</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ross et al., 1989</td>
<td>20 patients with AN or BN, 20 with anxiety disorder, 20 with schizophrenia, 20 with multiple personality disorder</td>
<td>Clinical interview</td>
<td>no info; no detailed information is given, yet it is unclear whether sexual activities by peers are included</td>
<td>no info; no post-onset abuse included; 4 different categories of abuse described</td>
<td>2; 20% of eating disorder patients reported CSA vs 10% of patients with anxiety disorder or schizophrenia and 80% of multiple personality patients.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hall et al., 1989; Tice et al., 1989</td>
<td>56 BN, 16 AN patients; controls: (n=86) a mixed group of patients with morbid obesity, depression, thought disorder or physical conditions</td>
<td>Questionnaire (based on Finkelhor, 1979)</td>
<td>peer experiences included; divided into below age 12 or above; no non-contact abuse included</td>
<td>no contact only</td>
<td>2.4; 50% of AN or BN group had CSA vs 28% of comparison group (p &lt; 0.01); however, sample includes cases with adult abuse (+post-onset) and comparison group not matched for sex (more males). When comparison group corrected to be sex matched, difference between groups on CSA no longer significant.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Beckman &amp; Burns, 1990</td>
<td>340 female undergraduates; of these 44 were classified as bulimic on the basis of a BULIT (Smith &amp; Thelen, 1984) cut-off score of 88.</td>
<td>Clinical Interview</td>
<td>parents, grandparents; at least 5 years older; below age 15; no info</td>
<td>contact only</td>
<td>1; No differences between groups on sexual experiences with adult below age 12 (7.5% in bulimics vs 10.8% in non-bulimics).</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lacey, 1990</td>
<td>112 BN patients</td>
<td>Clinical Interview</td>
<td>parents, grandparents</td>
<td>no contact only</td>
<td>7% reported CSA.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Shearer et al., 1991</td>
<td>40 female in-patients with borderline personality disorder</td>
<td>Clinical Interview</td>
<td>no info; at least 5 years older; below age 15; no info</td>
<td>contact only</td>
<td>3; Borderline patients with abuse were significantly more likely than non-abuse patients to have a concurrent eating disorder.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Study</td>
<td>Sample Description</td>
<td>Questionnaire Details</td>
<td>Scores Details</td>
<td>23% of sample reported CSA</td>
<td>Note</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>--------------------</td>
<td>-------------------------------------------------------------------------------------</td>
<td>-------------------------------------------</td>
<td>--------------------------------------------------------------------------------</td>
<td>-----------------------------</td>
<td>----------------------------------------------------------------------</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Smolak et al., 1990</td>
<td>298 female undergraduates (based on Finkelhor (1979); response rate 39%)</td>
<td>at least 5 years older</td>
<td>below age 16, not applicable, non-contact included, but abuse stratified into different degrees of severity</td>
<td>3.5, b</td>
<td>23% of sample reported CSA. The abused group had significantly higher total EDI scores than non-abused group with no sub-scale differences. Severity of abuse was unrelated to EDI scales.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Steiger &amp; Zanko, 1990</td>
<td>73 eating disorder out-patients (16 RAN, 12 BAN, 20BN/HistAN, 25 BN) vs 21 women with mixed psychiatric disorders vs 24 normal controls</td>
<td>Self-report questionnaire Divided into intrafamilial (sibling, parent) or extrafamilial divided into age bands up to age 20, no info various degrees of severity</td>
<td>1.2, 4</td>
<td>6% of RAN patients, 42% of BAN patients, 25% of BN/HistAN, 46% of BN patients, 33% of psychiatric controls and 9% of normal controls reported CSA; differences less pronounced when only incest included.</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stuart et al., 1990</td>
<td>30 BN patients vs 15 depressed patients vs 100 &quot;supernormal&quot; comparison women</td>
<td>Victimization Inventory no information</td>
<td>below age 18, no info, no info no info</td>
<td>1.2</td>
<td>50% of bulimics, 40% of depressed women and 28% of controls reported sexual mistreatment during childhood. This difference was not significant.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Abramson &amp; Lucido, 1991</td>
<td>63 females recruited from variety of sources: undergraduate psychology classes, in-patient eating disorder programs, private practitioners, bulimia support groups; of these 16 were bulimic on the BULIT; response rate 50%</td>
<td>SLEQ no info</td>
<td>age 12 or below, no info, no info</td>
<td>1.5, 6</td>
<td>69% of bulimics and 70% of non-bulimics reported CSA; bulimics reported significantly more incestuous relationships; BULIT scores correlated with greater number and higher severity of the abuse.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Study</td>
<td>Sample Size</td>
<td>Methodology</td>
<td>Sexual History</td>
<td>Age at Onset</td>
<td>Age Cut-off</td>
<td>Contact Exclusion</td>
<td>Results</td>
<td></td>
</tr>
<tr>
<td>-------</td>
<td>-------------</td>
<td>-------------</td>
<td>----------------</td>
<td>--------------</td>
<td>-------------</td>
<td>------------------</td>
<td>---------</td>
<td></td>
</tr>
<tr>
<td>McClelland et al., 1991</td>
<td>50 ED patients (28 RAN, 12 BAN, 10 BN)</td>
<td>Sexual Life Events Inventory (after Oppenheimer et al., 1985)</td>
<td>&gt; 5 years older</td>
<td>age 16 or below</td>
<td>14 out of 15 cases before onset</td>
<td>non-contact excluded, 4.6</td>
<td>25% of RAN, 55% of BAN and 20% of BN patients reported CSA.</td>
<td></td>
</tr>
<tr>
<td>Walter, 1991</td>
<td>67 AN and BN patients</td>
<td>SEQ: Clinical interview</td>
<td>no age cut-off</td>
<td>no age cut-off</td>
<td>no info</td>
<td>including non-contact</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Bushnell et al., 1992</td>
<td>Community sample of 301 women</td>
<td>Structured interview</td>
<td>Intrafamilial</td>
<td>no info</td>
<td>no info</td>
<td>Some</td>
<td>1.5</td>
<td></td>
</tr>
<tr>
<td>DeGroot et al., 1992</td>
<td>184 eating disorder outpatients (15 AN; 146 BN, 23 with both)</td>
<td>DSED</td>
<td>No info</td>
<td>No info</td>
<td>No info</td>
<td>Forceful abuse</td>
<td>4</td>
<td></td>
</tr>
<tr>
<td>Palmer &amp; Oppenheimer, 1992</td>
<td>80 AN patients 78 BN patients 115 non-eating disordered psychiatric patients</td>
<td>Self-report questionnaire</td>
<td>At least 4 years older</td>
<td>Below age 16</td>
<td>No info</td>
<td>Non-contact included</td>
<td>2.4</td>
<td></td>
</tr>
</tbody>
</table>

Women with a lifetime history of disordered eating had higher rates of intrafamilial sexual abuse than women selected at random from the sample. No relationship between sexual abuse and severity of eating symptoms.

Rates of abuse did not differ between different eating disorder subgroups (AN: 26.5%; BN: 22.8%; AN + BN: 21.7%). Sexual abuse was associated with greater psychological disturbance on the EDI and EAI.

Significantly fewer eating disorder patients than control patients (31% vs 49.6%; p < 0.01) had experienced CSA.
<table>
<thead>
<tr>
<th>Study</th>
<th>Sample Description</th>
<th>Methodology</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pribor &amp; Dinwiddie, 1992</td>
<td>52 incest victims compared with general population</td>
<td>not applicable, relatives too close to marry, excluding mutual exploration between siblings</td>
<td>no info, no info, non-contact excluded, 3, Rate of bulimia higher than expected in incest victims (23%).</td>
</tr>
<tr>
<td>Waller, 1992a</td>
<td>40 bulimic women with BAN, BN/HistAN or BN (extension of Waller’s 1991 sample)</td>
<td>as in Waller, 1991, as in Waller, 1991, as in Waller, 1991, as in Waller, 1991</td>
<td>5, Sexual abuse associated with more frequent bingeing, eating disorder symptoms more severe in those with severe abuse</td>
</tr>
<tr>
<td>Waller, 1992b</td>
<td>54 bulimic women with BAN, BN/HistAN or BN (extension of Waller’s 1991 sample)</td>
<td>as in Waller, 1991, as in Waller, 1991, as in Waller, 1991, as in Waller, 1991</td>
<td>5, Vomiting more frequent in cases of intrafamilial abuse</td>
</tr>
<tr>
<td>Folsom et al., 1993</td>
<td>102 eating disorder patients (57 BN, 17 AN, 19 BAN, 9 EDNOS) vs 49 general psychiatric patients</td>
<td>SLEQ, &gt; 5 years older, except non-consenting sex between peers &gt; age 12, various cut-offs, no info, various degrees of severity</td>
<td>2.4, 69% of eating disorder patients and 80% of general psychiatric patients reported a history of sexual abuse (not significant). No differences between different eating disorder subtypes.</td>
</tr>
<tr>
<td>Herzog et al., 1993</td>
<td>20 eating disorder subjects</td>
<td>TAI, DES, no info, &lt; age 18, 92% had abuse before onset, coercive, case series 6</td>
<td>65% had CSA, CSA subjects had increased rates of comorbidity, Abused subjects had higher dissociation scores.</td>
</tr>
<tr>
<td>Study</td>
<td>Methodology</td>
<td>Measurement</td>
<td>Cut-offs</td>
</tr>
<tr>
<td>-------------------</td>
<td>------------------------------------------------------------------------------</td>
<td>----------------------</td>
<td>-------------------</td>
</tr>
<tr>
<td>Miller et al., 1993</td>
<td>144 female undergraduates, of these 72 identified as having BN on BITE (Henderson &amp; Freeman, 1987) and 72 without eating disorder</td>
<td>SLEQ</td>
<td>various cut-offs in- or excluding peers</td>
</tr>
<tr>
<td>Mullen et al., 1993</td>
<td>large community study: at stage two 248 women with CSA and 244 women without CSA were interviewed</td>
<td>detailed interview</td>
<td>excluding non-coercive sexual experimentation with peers</td>
</tr>
<tr>
<td>Pitts &amp; Waller, 1993</td>
<td>41 patients with BN or BAN</td>
<td>as in Waller (1991)</td>
<td>as in Waller (1991)</td>
</tr>
<tr>
<td>Waller, 1993b</td>
<td>100 eating disorder patients with RAN, BAN, BN/HOSAN or BN (extension of Waller's 1991 series)</td>
<td>as in Waller, 1991</td>
<td>as in Waller, 1991</td>
</tr>
<tr>
<td>Waller, 1993b</td>
<td>115 eating disorder patients with RAN, BAN, BN/HystAN or BN ('extension of Waller's 1991 series)</td>
<td>as in Waller, 1991</td>
<td>as in Waller, 1991</td>
</tr>
<tr>
<td>Waller et al., 1993a</td>
<td>100 AN patients (36 restrictors, 36 restrict + purge; 21 restrict + purge + binge; 7 restrict + binge); 2 case series from London and Manchester</td>
<td>Manchester: as in Waller (1991) London: clinical interview</td>
<td>Manchester, London: no age cut-off</td>
</tr>
<tr>
<td>Waller et al., 1993b</td>
<td>53 eating disorder patients</td>
<td>as in Waller, 1991</td>
<td>as in Waller, 1991</td>
</tr>
<tr>
<td>Waller &amp; Ruddock, 1993</td>
<td>117 eating disordered patients (48 AN, 69 BN)</td>
<td>as in Waller (1991)</td>
<td>no age cut-off</td>
</tr>
<tr>
<td>Authors</td>
<td>Sample Description</td>
<td>Instrument/Methodology</td>
<td>CSAQ Details</td>
</tr>
<tr>
<td>------------------</td>
<td>-------------------------------------------------------------------------------------</td>
<td>----------------------------------------------------------------------------------------</td>
<td>-------------------------------------------------------------------------------</td>
</tr>
<tr>
<td>Berger et al., 1994</td>
<td>44 Japanese eating disorder patients</td>
<td>DES</td>
<td>no info</td>
</tr>
<tr>
<td></td>
<td></td>
<td>1-item screening question</td>
<td>no info</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Gleave &amp; Eberenz, 1994</td>
<td>464 DSM-III-R BN patients</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hastings &amp; Kern, 1994</td>
<td>786 university students divided into 30 bulimics, 37 subclinical bulimics and 719 normal subjects on the BULIT-R (Thelen et al., 1991)</td>
<td>CSAQ</td>
<td>at least 5 years older</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Kinzi et al., 1994</td>
<td>202 female University students (response rate 58%)</td>
<td>SLEQ</td>
<td>consensual sexual experimentation with peers excluded</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Köpp, 1994</td>
<td>Patients with BN (n=18), AN (n=22), obesity (n=14) and other psychosomatic problems (n=73)</td>
<td>Case note review</td>
<td>patients divided into different age bands; most patients below age 15 when abuse started</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>McCarthy et al., 1994</td>
<td>30 DSM-III-R BN patients</td>
<td>LEQ</td>
<td>no info</td>
</tr>
<tr>
<td>----------------------</td>
<td>---------------------------</td>
<td>-----</td>
<td>---------</td>
</tr>
<tr>
<td>Pope et al., 1994</td>
<td>33 Austrian students with BN, 33 American students with BN and 25 Brazilian women with BN (all volunteers)</td>
<td>screening questions followed by clinical interview</td>
<td>at least 5 years older</td>
</tr>
<tr>
<td>Rorty et al., 1994</td>
<td>40 currently bulimic volunteers (DSM-III-R criteria), 40 recovered bulimics and 40 women without a life-time history of eating disorder</td>
<td>SAEQ (Ryan et al., 1992)</td>
<td>at least 5 years older</td>
</tr>
<tr>
<td>Schaaf &amp; McCauley, 1994</td>
<td>670 female students</td>
<td>CHQ</td>
<td>no info</td>
</tr>
</tbody>
</table>

37% reported CSA. Dissociation did not differentiate between abused and non-abused women. Narrowly defined pre-onset sexual abuse occurred in 15%, 21% and 32% of women in USA, Austria and Brazil respectively. No association between abuse and severity of eating disorder symptomatology. No significant difference between sexual abuse in bulimic group vs non-bulimic group (28.8% vs 20.0%).

No significant differences between abused and non-abused groups in terms of a number of eating disorder related variables.
<table>
<thead>
<tr>
<th>Study Source</th>
<th>Participants</th>
<th>Research Design</th>
<th>Methodology</th>
<th>Age Gap</th>
<th>Age Cut-off</th>
<th>Abuse Before Onset</th>
<th>Contact Excluded</th>
<th>Notes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Welch &amp; Fairburn, 1994</td>
<td>50 community based subjects with BN vs 50 BN patients vs 50 community based comparison subjects with other psychiatric disorder and 100 community comparison subjects without an eating disorder</td>
<td>Investigator-based interview</td>
<td>no age-gap</td>
<td>below and above age 12</td>
<td>only abuse before onset</td>
<td>non-contact excluded</td>
<td>1,2,4</td>
<td>Any sexual abuse involving physical contact: Community based BN 26%. BN-patients 16%. Psychiatric comparison subjects 24% and comparisons without an eating disorder 10%.</td>
</tr>
<tr>
<td>Everill &amp; Waller, 1995b</td>
<td>69 female undergraduates</td>
<td>SEQ</td>
<td>no info</td>
<td>no age cut-off</td>
<td>not applic.</td>
<td>no info</td>
<td>6</td>
<td>A perceived adverse reaction to disclosure was associated with greater levels of psychopathology in particular oral control, dissociation and self-denigration.</td>
</tr>
<tr>
<td>Fullerton et al., 1995</td>
<td>98 AN, 243 BN, 18 BAN, 353 EDNOS</td>
<td>Clinical interview</td>
<td>no info</td>
<td>below age 18</td>
<td>no info</td>
<td>non-contact excluded</td>
<td>4,5</td>
<td>Rates of sexual abuse were higher among patients with BN (35%) and EDNOS (31%) than in those with AN (12%) or BAN (11%).</td>
</tr>
<tr>
<td>Garfinkel et al., 1995</td>
<td>community survey of 8116 female and male subjects (of these 62 had full-syndrome BN and 28 had partial-syndrome BN)</td>
<td>Semistructured interviews</td>
<td>no info</td>
<td>childhood</td>
<td>no info</td>
<td>serious i.e. contact and/or force</td>
<td>1</td>
<td>Childhood sexual abuse was experienced by female subjects with full-and partial-syndrome bulimia nervosa in 32.7% and 36.4% respectively which was significantly higher than for a female non-bulimic comparison group (13.9%).</td>
</tr>
<tr>
<td>Study</td>
<td>Sample Characteristics</td>
<td>Methodology</td>
<td>Age Gap</td>
<td>Contact</td>
<td>Contact Inclusion</td>
<td>Case Series</td>
<td>Notes</td>
<td></td>
</tr>
<tr>
<td>-----------------------------</td>
<td>------------------------</td>
<td>----------------------</td>
<td>---------</td>
<td>---------</td>
<td>-------------------</td>
<td>-------------</td>
<td>----------------------------------------------------------------------</td>
<td></td>
</tr>
<tr>
<td>Sullivan et al., 1995</td>
<td>90 BN</td>
<td>standard interview</td>
<td>no age gap defined</td>
<td>16</td>
<td>no info</td>
<td>case series, 5,6</td>
<td>44% had CSA; CSA did not correlate with bulimic symptoms (behavioural or cognitive); women with CSA had more severe depressive symptoms and had significantly earlier ages of onset of BN.</td>
<td></td>
</tr>
<tr>
<td>Vise &amp; Cooper, 1995</td>
<td>98 AN, 60 BN, 40 depression, 40 controls</td>
<td>structured interview, CECA</td>
<td>sexual contact with peers excluded</td>
<td>below age 17</td>
<td>pre-onset abuse excluded in purified subsample</td>
<td>non-contact included</td>
<td>1,2,4,5,6</td>
<td>An equally high rate of abuse was found in all three clinical samples. The rate of CSA in the clinical groups was significantly higher than in the normal controls. There was no link between the severity of eating symptomatology and the rate, duration or nature of the abuse.</td>
</tr>
<tr>
<td>Tobin &amp; Griffing, 1996</td>
<td>15 binge eating disorder, 12 non-purging BN, 32 purging BN, 13 RAN, 10 BAN, 8 obese EDNOS, 13 purging EDNOS</td>
<td>clinical interview + case notes</td>
<td>≥ 5 years age gap in those 46% who gave info on age of perpetrator</td>
<td>no cut-off</td>
<td>no contact only</td>
<td>4</td>
<td>Overall rate of sexual abuse 40%, however, with marked differences between subgroups ranging from 12% in compulsive overeaters to 75% in normal-weight EDNOS patients who used compensatory behaviours.</td>
<td></td>
</tr>
<tr>
<td>Zionick et al., 1996</td>
<td>134 psychiatric in-patients</td>
<td>self-report questionnaire</td>
<td>no info</td>
<td>&lt; age 16</td>
<td>no</td>
<td>contact abuse only</td>
<td>3</td>
<td>69% of women reported sexual abuse. A complex of EDI subscales was significantly related to a history of sexual abuse and correctly classified 82% of subjects according to their abuse status.</td>
</tr>
</tbody>
</table>
Table 3.8.1.: Studies on sexual abuse and eating disorders (in order of appearance)

**Comparison Groups:** AN: anorexia nervosa, BN: bulimia nervosa, BN/HistAN: bulimia nervosa with a history of anorexia nervosa; RAN: restricting anorexia nervosa; BAN: bulimic anorexia nervosa; ED: eating disorder; EDNOS: eating disorder not otherwise specified.

**Measures:**
- CEQ = Childhood Events Questionnaire (Barnes and Trachtenberg, 1985)
- CECA = Childhood Experiences of Care and Abuse (Bifulco et al., 1994)
- CHQ = Childhood History Questionnaire (Milner et al., 1990)
- CSAQ = Childhood Sexual Abuse Questionnaire (Walters et al., 1987)
- DDIS = Dissociative Disorders Interview Schedule (Heber et al., 1987; quoted from Ross et al., 1989)
- DES = Dissociative Experiences Scale (Bernstein & Putnam, 1986)
- DSED = Diagnostic Schedule for Eating Disorders (Johnson, 1985)
- LEQ = Life Experiences Questionnaire (Bryer et al., 1987)
- SEQ = Sexual Events Questionnaire (Russell, 1983)
- SAEQ = Sexual Abuse Exposure Questionnaire (Ryan et al., 1992)
- SLEQ = Sexual Life Events Questionnaire (Finkelhor, 1979)
- Victimization Inventory (Kilpatrick, 1985)
- TAI = Traumatic Antecedents Interview (Herman et al., 1989)

**Research Questions:**
1: Differences between eating disorders and normal controls
2: Differences between eating disorders and other patient populations (psychiatric and non-psychiatric)
3: Differences in rates of eating disorders between abused and non-abused women
4: Differences between different types of eating disorders
5: Associations between abuse characteristics and severity of eating disorder
6: Mediators between CSA and later eating disorders
Table 3.8.2.: Studies on physical abuse and eating disorders

<table>
<thead>
<tr>
<th>Authors</th>
<th>Sample</th>
<th>Methods</th>
<th>Perpetrator</th>
<th>Age cut-off for Abuse</th>
<th>Abuse Before Onset</th>
<th>Info on Severity of Abuse</th>
<th>Research Question</th>
<th>Rate of Physical Abuse</th>
</tr>
</thead>
<tbody>
<tr>
<td>Piran et al., 1988</td>
<td>30 AN patients, 38 bulimia patients</td>
<td>CEQ</td>
<td>no info</td>
<td>no info</td>
<td>no info</td>
<td>no info</td>
<td>4</td>
<td>16.7% of restrictors reported physical abuse and 28.9% of bulimics. This difference was not significant</td>
</tr>
<tr>
<td>Root &amp; Fallon, 1988</td>
<td>172 consecutive female applicants to bulimia treatment program</td>
<td>Questionnaire and interview</td>
<td>no info</td>
<td>no info</td>
<td>no info</td>
<td>no info</td>
<td>case series</td>
<td>29.1% of women had been physically abused as children</td>
</tr>
<tr>
<td>Bailey &amp; Gibbons, 1989</td>
<td>294 female college students</td>
<td>Questionnaire</td>
<td>no info</td>
<td>no info</td>
<td>no info</td>
<td>no info</td>
<td>3.5</td>
<td>6% reported childhood physical abuse. Physical abuse had a significant relationship with the diagnosis and severity of bulimia</td>
</tr>
<tr>
<td>Folsom et al., 1993</td>
<td>102 eating disorder patients vs 49 general psychiatric patients</td>
<td>Questionnaire</td>
<td>primary care giver</td>
<td>early adolescence</td>
<td>no info</td>
<td>yes</td>
<td>2</td>
<td>44% of eating disorder patients vs 53% of general psychiatric patients</td>
</tr>
<tr>
<td>Study</td>
<td>Type of Patients</td>
<td>Assessment Method(s)</td>
<td>Age At Assessment</td>
<td>Abuse Information</td>
<td>Comments</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>-------------------------------</td>
<td>-----------------------------------------------------------</td>
<td>-------------------------------</td>
<td>-------------------</td>
<td>--------------------</td>
<td>--------------------------------------------------------------------------</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ross et al., 1989</td>
<td>20 patients with AN or BN, 20 with anxiety disorder, 20 with schizophrenia, 20 with multiple personality disorder</td>
<td>DDIS</td>
<td>no info</td>
<td>no info</td>
<td>25% in ED patients; 5% in panic disorders; 25% in schizophrenia; 75% in multiple personality disorder.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vanderlinden et al., 1993</td>
<td>98 eating disorder patients: 34 RAN, 24 BAN, 28 BN and 12 EDNOS</td>
<td>questionnaire (Lange, 1990); clinical interview</td>
<td>no info</td>
<td>no info</td>
<td>yes 3% of total sample.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Berger et al. (1994)</td>
<td>44 Japanese eating disorder patients</td>
<td>DES</td>
<td>no info</td>
<td>no info</td>
<td>Physical abuse was associated with high dissociation.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>McCarthy et al., 1994</td>
<td>30 DSM-III-R BN patients</td>
<td>LEQ, DES</td>
<td>family member</td>
<td>below age 16</td>
<td>57% no correlation between history of abuse and severity of bingeing.</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Rorty et al., 1994</td>
<td>40 bulimic volunteers, 40 recovered bulimics and 40 women without lifetime eating disorder</td>
<td>Assessing Environments III-Form SD (Rausch &amp; Knutson, 1991)</td>
<td>parent</td>
<td>age 17 or below</td>
<td>yes Significantly more BN patients had been physically abused than non-bulimic comparisons (17.5% vs 2.5%).</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Study</td>
<td>Sample Description</td>
<td>Method</td>
<td>Age Group</td>
<td>Notes</td>
<td>Score</td>
<td>Additional Notes</td>
<td></td>
<td></td>
</tr>
<tr>
<td>------------------</td>
<td>--------------------</td>
<td>-----------------</td>
<td>-----------</td>
<td>----------------</td>
<td>-------</td>
<td>------------------</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Schaaf &amp; McCane, 1994</td>
<td>670 female college students</td>
<td>CHQ, carers</td>
<td>below age 13</td>
<td>no info, marked as severe</td>
<td>3.6</td>
<td>No difference between abused and non-abused women on a number of eating disorder related variables.</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fullerton et al., 1995</td>
<td>98 AN, 243 BN, 18 BAN, 353 EDNOS</td>
<td>Clinical interview, no info</td>
<td>below age 18</td>
<td>no info, no</td>
<td>3.4</td>
<td>Subjects with BAN and BN reported physical abuse more frequently (33% and 30%) than subjects with anorexia nervosa (11%) and subjects with EDNOS (22%).</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Table 3.8.2.: Studies on physical abuse and eating disorders (in order of appearance)

**Comparison Groups:**
AN: anorexia nervosa, BN: bulimia nervosa, BN/HistAN: bulimia nervosa with a history of anorexia nervosa; RAN: restricting anorexia nervosa; BAN: bulimic anorexia nervosa; ED: eating disorder; EDNOS: eating disorder not otherwise specified.

**Measures:**
CEQ = Childhood Events Questionnaire (Barnes and Trachtenberg, 1985)
CHQ = Childhood History Questionnaire (Milner et al., 1990)
DES = Dissociative Experiences Scale (Bernstein and Putnam, 1986)
DDIS = Dissociative Disorders Interview Schedule (Heber et al., 1987; quoted from Ross et al., 1989)
LEQ = Life Experiences Questionnaire (Bryer et al., 1987)

**Research Questions:**
1. Differences between eating disorders and normal controls
2. Differences between eating disorders and other patient populations (psychiatric and non-psychiatric)
3. Differences in rates of eating disorders between abused and non-abused women
4. Differences between different types of eating disorders
5. Associations between abuse characteristics and severity of eating disorder
6. Mediators between abuse and later eating disorders
Chapter 4: Stress and Eating Disorders

4.1. Introduction

Stressful life events and difficulties have been implicated in the onset and maintenance of a number of psychological and physical disorders. Not surprisingly, stress has also been thought to be of importance in the onset and course of both anorexia nervosa and bulimia nervosa (Gomez and Dally, 1980; Mitchell et al., 1985; Sohlberg et al., 1992). Anorexia and bulimia nervosa patients (Greenberg, 1986; Soukoup et al. 1990) report more stress than controls. However, Troop (1996) points out that to experience more stress is not the same as experiencing more stressors as other factors such as appraisal, coping, and support affect such a perception. Likewise, Cattanach and Rodin (1988) emphasised that stress should be viewed as "a process that includes not only stimulus and response, but also appraisal, coping processes, control, social supports, personality factors, and other intervening variables predisposing one to experience more stressors or to be more reactive to potential stressors".

In the following, after some brief considerations of the biology of stress and eating, the literature on what is known about stressful events and difficulties in the onset of eating disorders will be reviewed.

4.2. The Biology of Stress and Eating

Eating and stress are intimately linked. A variety of stressors are capable of producing stress-induced eating or starvation in different animals (for review see Morley et al., 1986; Treasure, 1995). Pigs and other farm animals bred for leanness are susceptible to stress-induced irreversible self-starvation (Treasure et al., 1997). In humans, nail-biting, gum-chewing and bruxism are all related to stress. Different people respond to
stress by either "going off" their food or by overeating ("comfort eating") (for review see Morley et al., 1986). In one study, 44% of subjects reported an increase in their food consumption under stress, whereas 48% decreased their eating when under stress and only a minority (8%) did not change eating when stressed (Willenbring et al., 1986). Thus, to some extent individual pre-disposition may determine a person's eating response to stress. However, which particular form of eating disturbance occurs may also depend to an extent on type and duration of stressor (for review see Heatherton and Polivy, 1992; Morley, 1990; Morley et al., 1986). For example, in rats mild tail pinching produces overeating, while immobilisation stress or exposure to a novel environment leads to anorexia (Donohoe, 1984; Morley et al., 1983). Similarly, in humans, experimentally induced physical fear did not lead to increased eating in restrained eaters, although it did reduce the eating of control subjects. In contrast, ego threat (i.e. situations threatening an individual's sense of self-esteem or identity) or negative mood inductions did significantly increase the eating by restrained subjects (Heatherton et al., 1991) and eating disordered subjects compared to controls (Cattanach et al., 1988).

It has been suggested that endogenous opioids play an important role in the initiation of stress induced feeding whereas the dopaminergic system is thought to be predominantly responsible for initial chewing (and other oral behaviours) and also facilitatory to the opioid system. Dopamine antagonists block both chewing and feeding behaviours whereas opioid antagonists only decrease the feeding component (Morley et al., 1986).
Much current interest focuses on the serotonergic system. Central nervous system 5-HT is of critical importance in appetite control, with its hypophagic effects being more pronounced in female than in male animals (Treasure and Tiller, 1993). Arousal and stress in rats increases central 5-HT release (Kalan et al., 1989; Dunn and Welch, 1991).

Activation of corticotropin-releasing factor (CRF) is viewed as the final common neurobiological pathway responsible for producing stress-induced anorexia (Morley and Blundell, 1988). Centrally administered CRF is a potent reducer of nocturnal and starvation induced feeding. The hyperphagic effects produced by neuropeptide Y are antagonized by CRF. The effects of CRF on feeding have been localized to the paraventricular nucleus (PVN) where norepinephrine acts to increase feeding and serotonin to decrease feeding. There is evidence that serotonin releases CRF at hypothalamic level.

4.3. Measurement of Life Stress: Some Methodological Considerations

The measurement of life events is complex and difficult (for review see Brown, 1989). Most studies to date have used questionnaire-based "checklist" approaches to obtain information about events. Respondents simply check off whether, say "someone in the family has died". Each class of event is weighted. The weights are derived by averaging the scores of a number of judges asked to assess the disruption caused by each event in relation to a standard maximum score provided by an event such as "death of a spouse". The main problem with this kind of approach is that there is great variability within a particular event category (e.g. "moving house") as to the
meaning and significance for the individual, ranging from very positive to extremely unpleasant. The resulting measures have shown poor reliability at re-interview and also when different judges have rated the "stress" involved in particular events. Moreover, most checklist approaches have not been concerned with the impact of particular events, but with an overall score based on the weights of all events occurring in a given period of time. (This assumes that events have an additive impact, however, there is as yet little empirical data to support this assumption.) The assumption of additivity of stress also diverts interest from the issue of meaning and from the need to date events as accurately as possible vis-a-vis any onset of disorder. Thus, not surprisingly research findings have been extremely inconsistent.

Some questionnaire-based instruments include a rating of impact of the event on the individual to tackle the issue of event variability. However, subjective ratings may be biased by an 'effort after meaning', i.e. the effect of a certain event is exaggerated retrospectively, as a means of coming to terms with recent symptoms.

The Life Events and Difficulties Schedule (LEDS, Brown and Harris, 1978, 1989) is a semistructured investigator-based interview with excellent reliability and validity, which gets around many of the problems described above.

In this approach the threat or unpleasantness of life events (immediate and longer term) and difficulties is rated contextually by taking into account a person's particular life circumstances. Ratings are made using a manual of a large number of different classes of events and degrees of severity. These can then be checked by consensus by a panel of trained raters who are blind to the subject's diagnosis and emotional
reactions. This approach to rating minimizes respondent and investigator bias (Brown and Harris, 1978; Parry et al., 1981; Tennant et al., 1979). The qualities of events rated contextually are also rated in terms of self-report - that is the individual's subjective reaction. Additionally, events and difficulties are checked for 'independence'. An independent event is one that on logical grounds could not be the result of an onset of disorder before symptoms became obvious, because its source is judged to be independent of the subject's agency (e.g., a person who is made redundant when their employer goes bankrupt (independent event) vs. someone who loses their job because of poor time-keeping (not independent)).

The LEDS has pointed towards a specificity of meaning of events or difficulties preceding different disorders. For example, depression is commonly triggered by 'loss' events, anxiety by 'danger' events (Finlay-Jones and Brown, 1981), organic abdominal disorder by 'goal frustration' (Craig and Brown, 1984), functional dysphonia (House and Andrews, 1988) by 'failure over speaking out' and certain menstrual disorders by 'challenge' events (Harris, 1989). Thus, in summary the LEDS is currently the gold standard in the measurement of life events and difficulties.

4.4. Stressful Life Events and Difficulties and the Onset of Eating Disorders

4.4.1. Anecdotal reports and case series

Onset of anorexia nervosa following road traffic accidents (Damlouji and Ferguson, 1985), termination of pregnancy (Thomas, 1983) and torture (Fahy et al., 1988) has been described anecdotally. Ryle (1936) reported that emotional crises, love affairs, and broken engagements preceded onset in many of the young women. He acknowledged, however, that in only 2 of his series of 33 cases could such
attributions be made with certainty. Morgan and Russell (1975) found that 65% of 41 anorexics had an adverse life event in the year preceding the onset of the illness. However, they included events such as being teased about weight, which might have reflected the start of excessive concern about body image and not its precipitant. A retrospective case notes study found 60% of adolescent anorexics to have experienced a major life event in the year preceding the onset of illness e.g. parental separation, change of school or home, or increased academic pressure (Margo, 1985). Beumont et al. (1978) examining a series of 31 anorexic patients noted that the range of stressors before onset "was rather broader than that which had been expected, and includes many factors which have not previously been stressed in the literature. For instance, not only were issues of dependence and independence, sexuality and eating prominent, but so were other less "specific" factors such as losses within the family, religious and quasi-religious constraints on eating, changes in environment, and physical illness of the patient preceding the onset of anorexic behaviour."

As in anorexia nervosa, a wide variety of events and difficulties have been implicated in the onset of bulimia nervosa: In Pyle et al.'s (1981) series 30 of 34 patients recalled some traumatic event associated with the onset of bulimic behaviour, the most common being loss or separation from a significant person in their life. In another series of 50 DSM-III bulimia patients binge eating was preceded by sexual conflict in 72% of patients, a major change in life circumstances in 70% of patients and loss or separation from a significant person in 20% of patients (Lacey et al., 1986).
4.4.2. Controlled studies

Horesh et al. (1995) studied life-events in 21 adolescent anorexia nervosa patients, 79 non-anorexic patients and 40 non-patient controls. Assessment was conducted by semi-structured interview. Events included were bad experiences with parents (physical abuse and cruelty), separations, deaths and parental fighting and verbal hostility not specifically directed at the child. It is uncertain what the cut-off was for inclusion. Patients were asked to discuss their life experiences throughout their life and prior to the year preceding their illness. (Thus precipitants of the illness were specifically excluded as the authors were interested in the "effects of cumulative life trauma on the development of psychopathology"). Anorexics differed from normal controls in having a higher total life events score and more bad experiences with parents and the wider family. The only difference between AN patients and other patients was that anorexics had more bad experiences with parents.

In a comparison of 25 bulimic anorexic adolescents with 25 restricting anorexics life stress was assessed using a 'checklist approach', with each life event being given a weighted severity rating, expressed in Life Change Units (Strober, 1984). Bulimic anorexics had higher Life Change Unit scores than restricters in the period before onset. Events experienced more frequently by bulimics included alienation from peers, parental marital discord, bickering with parents, personal illness requiring hospitalization, serious illness in a parent, involvement with drugs, increased absence of father from the home and personal disappointment in the area of school activities. Greater amounts of life stress correlated with greater severity of bulimia and depression in the bulimic anorexic group.
Russell and Gilbert (1992) examined case notes and found that 83% (15/18) of late onset cases of anorexia nervosa (onset after age 25) experienced a precipitating event, compared with only 14% (5/36) of those who developed anorexia under the age of 20. Likewise, a further case note comparison (Mynors-Wallis et al., 1992) examined life events and difficulties prior to onset in 12 late onset anorexics, 12 anorexics with an early onset and 12 chronic anorexics with an onset below age 25. These authors used standardised criteria for rating life events and difficulties and found that late onset anorexics had significantly more life events prior to the onset of their illness (9/12) than either the early onset anorexics (2/12) or the chronic anorexics, either at initial onset (3/12) or relapse (3/12). The same pattern held if difficulties were included: 11/12 of late onset vs 5/12 of early onset vs 4/12 of chronic patients at onset or 5/12 of chronic patients at relapse.

4.4.3. Other studies

Two non-clinical studies examined the links between stress and the onset of eating problems. In a prospective study of college freshmen Striegel-Moore et al. (1989) found that high levels of perceived stress were associated with eating problems among women but not men. The direction of the relationship is unknown, for example stress may result from attempts to diet. A further prospective study addressed this issue (Rosen et al., 1990). Dieting predicted the degree of stress 4 months later, whereas stress did not predict later dieting behaviour. However, the study relied on self-report measures of life stresses and counting symptoms (rather than caseness) which limits the conclusions one can draw from it.
4.4.4. Shortcomings in existing studies on life events and difficulties in eating disorders

Studies examining the role of life stresses in the aetiology of eating disorders have either been descriptive or used checklist approaches, with the exception of Horesh et al. (1995) who however, have not been interested in precipitating stress.

In reviewing life events and depression (Coyne and Downey, 1991) point out that any event (severe and non-severe) is associated with depressive symptoms whereas only severe events are associated with onset of case depression. Studies using checklists of events and eating disorders symptoms may therefore not extrapolate to women with an eating disorder at case level (e.g. Rosen et al., 1990).

The conclusions that can be drawn from the above studies therefore are extremely limited. There are many unanswered questions about the nature, severity and specificity of stressful life events and difficulties preceding onset of different eating disorders.

4.5. Summary and Conclusions

(1) Stress and eating are intimately linked.

(2) The type of eating disturbance that arises as a result of stress may partly depend on the individual’s disposition and partly on the type and nature of the stressor.

(3) Case series of patients with anorexia and bulimia suggest that a broad range of stressors provoke onset of both disorders.
(4) Different eating disorder subtypes may differ in the rates of pre-onset stress reported.

(5) Existing research on the role of stress in the onset of different eating disorders has methodological shortcomings and there are many unanswered questions about the nature, severity and specificity of stress preceding different eating disorders.
PART II: Experimental Section
Chapter 5: An Investigation into Childhood Adversity and Course of Illness in Different Types of Eating Disorders

5.1. Aims and Hypotheses

Aim 1: To examine the family antecedents of eating disorders.

Hypothesis 1: Bulimia nervosa patients will have had worse childhood care than patients with restricting anorexia nervosa, with bulimic anorexic patients and bulimic patients with a history of anorexia nervosa being intermediate.

Aim 2: To assess whether different types of eating disorders report different rates of childhood abuse (sexual/physical).

Hypothesis 2a: Bulimia nervosa patients will have experienced more childhood physical abuse than those with restricting anorexia nervosa, with other groups in between.

Hypothesis 2b: Different eating disorder subtypes do not differ in the rates of childhood sexual abuse experienced.

Aim 3: To assess whether childhood experiences have an impact on the course of eating disorders.

Hypothesis 3: Patients with more chronic eating disorders will have experienced more childhood adversity than those with a more recent onset.

5.2. Introduction

Bearing in mind the methodological problems in the majority of previous studies assessing family environment and childhood trauma in eating disorder subjects, it was
attempted to design a study that would overcome at least some of the problems of previous research: (a) by using the strategies outlined by Brewin et al. (1993) to help increase reliability of retrospective reports about childhood functioning; (b) by only including pre-onset family problems and (c) by using well-defined and strict criteria of abuse and poor quality of childhood care. Also, unlike the majority of previous studies which concentrated either on the general quality of early family functioning or on childhood abuse, the research presented here combines assessment of the two.

Moreover, there is evidence from the literature on depression that childhood adversity predicts chronicity in women in the community and in female psychiatric patients (Brown and Moran, 1993; Brown et al., 1994). It was therefore of interest, whether in eating disorders too, childhood family disturbance is a predictor of chronicity, a question which has so far been neglected in the research literature.

5.3. Subjects and Method

5.3.1. Sample

A consecutive series of newly referred eating disorder patients with DSM-III-R diagnoses of anorexia nervosa or bulimia nervosa seen at the Maudsley Hospital were enrolled in the study. Patients were subdivided further according to the nosologies of Russell (1985) and Fairburn (1986) into those with restricting anorexia nervosa (RAN), bulimic anorexia nervosa (BAN), bulimic patients with a history of anorexia nervosa (BN/HistAN) and bulimic patients without a history of anorexia nervosa (BN). Patients with eating disorder not otherwise specified or other main diagnoses were excluded. At their first assessment all patients were asked about their childhood
care (from birth to age 17) using the Childhood Experience of Care and Abuse (CECA) Interview described below.

5.3.2. Procedure

The Childhood Experience of Care and Abuse (CECA) Interview: This instrument was designed for the retrospective assessment of childhood care (Andrews et al., 1990; Bifulco et al., 1987; Bifulco et al., 1991; Bifulco et al., 1994; Harris et al., 1986). It is reliable, and agreements between adult pairs of sisters about their childhood suggest that the measures are likely to be a reasonably valid account of early experience (Bifulco et al., 1994). Respondents are encouraged to relate their experiences as 'stories' with questioning continued until a clear picture emerges.

From such accounts the investigator is able to decide whether experiences meet criteria for neglect or abuse (with predefined thresholds; see definitions below and clinical examples in appendix 5.8.) irrespective of how the respondent defines them. The aim is to increase accuracy and to help reduce bias from reporting style or emotional response. The CECA focuses on material which is objective (concerning behaviour) rather than subjective (concerning feelings). The CECA was developed in several stages in two distinct study populations of London women in Walthamstow (Harris et al., 1986) and Islington (Bifulco et al., 1987).

The CECA employs a number of techniques likely to increase recall, as listed by Fisher and Quigley (1992): the respondent is encouraged to recreate the context in which events occurred (Tulving and Thompson, 1972), memory retrieval is better if
there is continued attention to the topic with few distractions or interruptions (Kahneman, 1973) and the more retrieval attempts are allowed, the more successful eventual retrieval will be (Roediger and Payne, 1982).

The length of the interview varies according to the complexity of childhood arrangements and number of surrogate parents. Emphasis is placed on the importance of flexibility in interviewing. Any topic can be reopened at any point in the light of fresh information and although a schedule of questions is provided, questions can be covered in a different order in the light of what happens in the interview itself.

Information is gathered on patients' family structure i.e. number and type of care arrangements, composition of the family and on the quality of childhood care.

The CECA (or elements from it) have now been used successfully to examine a variety of adult conditions: somatising disorder (Craig et al., 1993) conduct disorder (Hepworth, 1992), suicidal behaviour (Tousignant et al., 1988), chronic depression (Brown et al., 1994; Brown and Moran, 1994) and anxiety disorders (Brown and Harris, 1993). Below the elements of the CECA used in this study are outlined.

**Parental mental disorder:** This was rated as present if the parent in question had been treated by a general practitioner or a mental health professional.

**Changes in family structure:** Changes in family structure (e.g. due to death, or separation from a parent, or institutional care) were rated as present if the change
persisted for more than a year. Shorter separations from the parents were not included. For each separate family arrangement the quality of childhood care was investigated and rated on the indicators given below. In cases with several family arrangements the peak negative childhood experience was taken, for example the highest indifference rating across family arrangements in childhood.

**Quality of childhood care:** Ratings were based on concrete examples of parental behaviour, taking into account frequency and duration. The core scales are described below. Clinical examples are shown in appendix 5.8.

**Parental indifference:** This dealt with the amount of neglect or disinterest in the child’s welfare (for example in material care, school work and friendships) exhibited by parents or parent substitute. Both material and emotional neglect were considered with particular attention to examples of neglectful behaviour (Bifulco et al., 1987; Harris et al., 1986). This was rated on a 4-point scale (1: marked, 2: moderate, 3: some, 4: little/none).

**Parental control:** This refers to the level of supervision of the child and parental enforcement of rules and discipline. Questioning covers the extent to which the child plays out of the house alone for lengthy periods of time, was left alone at night, and the presence and enforcement of rules about homework, table manners, going out with boyfriends or returning home late at night. This is a 3-point scale (1: high control, 2: moderate control, 3: lax control).
Discord/tension in the family: This reflects the amount of conflict in the form of arguments and rows. Parents were usually the main protagonists, but children were not uncommonly involved. This was rated on a 5-point scale (0: tension only, 1: marked discord, 2: moderate discord, 3: some discord, 4: little/no discord).

Intrafamilial violence: Two separate ratings were made for this on violence towards the patient or another family member. This was defined as violence by a household member (usually adult) towards the patient or another family member. It covered the patient or other family member being beaten, kicked, burnt, hit with belts or other objects, or being threatened with knives or other weapons. The ratings were made on 4-point scales (1: marked, 2: moderate, 3: some, 4: little/none).

Childhood sexual abuse (CSA): This was defined as a history of sexual activity involving contact, by a perpetrator five years or more older than the victim who was herself 16 years or younger (after Browne and Finkelhor, 1986). (Whilst the CECA does have a sexual abuse module, in this study the Browne and Finkelhor criteria were chosen for greater comparability with other research studies.). Sexual abuse by perpetrators outside the family of origin was included. Non-contact events were excluded. Patients were asked about sexual abuse and the sexual life events questionnaire was also administered (Oppenheimer et al., 1985). Abuse was rated as 'present' or 'absent'.

Reported ratings: Additionally the interviewer made ratings of patients' perceived approval or disapproval by both parents separately on 4-point scales. These ratings
were based on patients' attitudes towards their parents rather than behavioural evidence.

**Institutional care**: Where children had been in institutions (boarding schools, hospitals or children's homes) questions about indifference, control and the general atmosphere were asked to elicit evidence of neglect, harshness or isolation.

**Early onset of eating disorder**: In cases where the eating disorder had started before the age of 17, problems which had arisen after the onset of the eating disorder were not included.

**Socioeconomic status**: This was determined by the Hollingshead 2 factor method and was based on father's occupation (Guy, 1976).

Interviews were conducted by myself, Janet Treasure and Jane Tiller and were tape-recorded. Ratings were made by each interviewer in consensus with a team of raters (i.e. the other two researchers), who had been trained in the use of the method by Tirril Harris and who were blind to the diagnosis of the patient discussed. For all variables rated on a 4-point scale the top two categories of each variable (marked, moderate) were included in the analysis. In the discord rating high tension was also included.

**Statistical analysis**: Data were analyzed using SPSS/PC+ 4.0 (Norusis, 1990). Analysis of variance was used to test for differences between means in the three
different groups for continuous, normally distributed variables. Chi-square statistics
(with Yates' correction where appropriate) were used for categorical data. Odds ratios
were calculated using Gardner et al.'s (1991) microcomputer program (CIA). Forward
stepwise logistic regression was used to model the prediction of diagnostic group
membership by indicators of childhood care. The likelihood-ratio (LR) test was used,
which tests the null hypothesis that the coefficient of the term is 0 and estimates
whether a model which includes a particular variable is an improvement on a model
that does not include that variable. It is reported as -2log LR. The likelihood ratio
rather than the Wald statistic was chosen, as the Wald statistic can lead to Type II
errors when the absolute value of the regression coefficient is large (Norusis, 1990).

5.4. Results

5.4.1. Sample characteristics

64 patients with restricting anorexia nervosa (RAN), 23 patients with bulimic anorexia
nervosa (BAN), 37 patients with bulimia nervosa and a history of anorexia nervosa
(BN/HistAN) and 79 patients with bulimia nervosa without a history of anorexia
nervosa (BN) were interviewed. Apart from 2 RAN patients, all patients were female.
Table 5.7.1. gives information on the patients' clinical and demographic background.

5.4.2. Childhood care

Parental mental health: Mental illness in the mother occurred in 18/64 (28 %) RAN
patients, 5/23 (22 %) of BAN patients, 13/37 (35 %) of BN/HistAN patients and
22/79 (27 %) of BN patients (Chi-square: 1.3, df = 3; p = 0.72). In all cases this
was before the patient was 17 years of age. Mental illness in the father occurred in
12/64 (19 %) of RAN patients, 6/23 (26 %) BAN patients, 5/35 (14 %) of BN/HistAN patients and 16/75 (21 %) of BN patients (Chi-square 1.4, df = 3, p = 0.70). With the exception of two cases where the patient was 18 years of age when her father developed mental health problems, these problems occurred before the patient reached age 17. (In some of the cases no information was available on the mental health of the father).

**Family structure:** The four groups differed in terms of the number of family arrangements during childhood. The RAN group were less likely to have three or more family arrangements than the other groups (see table 5.7.2.). Parental separation was the most common reason for multiple family arrangements in all groups (see table 5.7.3.).

**Quality of care:** Normal weight bulimia nervosa patients had experienced higher levels of indifference, excessive control, and intrafamilial discord and violence than the other three groups (Table 5.7.4). Typical examples of these are given in Appendix 5.1.

The perpetrator of physical violence against the patient was most commonly the patient's father/stepfather (n=14), more rarely the patient's mother/stepmother (n=8) or other family members (n=5).

The four groups did not differ with regard to low parental control or childhood sexual abuse. The perpetrator of sexual abuse against the patient most commonly was a
family acquaintance (n = 23). Eight patients had been sexually abused by their fathers, two by their stepfathers, ten by other relatives, four by a stranger, and in one case the patient would not divulge the identity of the perpetrator.

A composite measure of childhood adversity was calculated (indifference + discord + high control + low control + violence against patient + violence against other + childhood sexual abuse). Low adversity was defined as 0 - 1 indicators of adversity. High adversity was defined as 2 or more indicators of adversity. 19/64 (30 %) of RAN patients, 9/23 (39 %) of BAN patients, 20/36 (56 %) of BN/HistAN patients and 50/77 (65 %) of BN patients fell into the high adversity group (chi-square 18.9, df = 3, p = 0.0003).

Given a social class difference between the groups which was verging on significance with the greatest difference between RAN and BN patients (RAN: working class background 20 %; BN: working class 38 %) a stepwise fitting procedure using logistic regression analysis was applied, entering the effect of social class first, adding in the effect of diagnosis for each of the indicators of childhood care. A significant effect of diagnosis persisted after controlling for social class.

**Reported ratings (Table 5.7.5.):** The four groups reported similar levels of approval from both parents. However, they differed significantly on felt disapproval from mother and father with normal weight bulimic patients reporting the highest levels of disapproval, RAN patients the lowest and the other two groups being intermediate.
A regression model of childhood care: A logistic regression analysis was carried out to see which (if any) of the indicators of poor childhood care (behavioural or reported) were important in predicting diagnostic group membership (dependent variable: RAN or BN). The following independent variables were included in the analysis: parental indifference, high control, low control, discord, physical abuse, sexual abuse, approval from mother, disapproval from mother, approval from father, disapproval from father. Both physical abuse and parental indifference were necessary to predict diagnostic group. The presence of these variables was more likely to indicate bulimia nervosa rather than anorexia nervosa.

Model if term removed:

<table>
<thead>
<tr>
<th>Term</th>
<th>-2 Log R</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indifference</td>
<td>6.695</td>
<td>0.0097</td>
</tr>
<tr>
<td>Physical Abuse</td>
<td>7.919</td>
<td>0.0049</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th></th>
<th>RAN (n=64)</th>
<th>BN (n=79)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indifference and/or</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Physical Abuse</td>
<td>12/64 (18.8%)</td>
<td>42/79 (53.2 %)</td>
</tr>
</tbody>
</table>

Chi-Square = 16.38; D.F. = 1; p = 0.0001

Childhood care and duration of eating disorder: Patients were dichotomised into two groups based on the median duration of illness which was 4 years (table 5.7.6.). Mental illness in the mother was more common in chronic RAN compared to recent
onset cases, with no other differences between these two groups. In chronic BN, there was also a trend for mental illness of mother to be more common than in recent onset cases of BN. In addition, high indifference, and high discord were significantly more common in chronic bulimics than in those with a recent onset. High adversity during childhood (on the composite measure) was twice as common in the chronic BN group compared to those with a recent onset.

5.5. Discussion

The study has a number of weaknesses which are discussed below.

(1) Research subjects were a clinical sample of eating disorder patients rather than community subjects. It is possible that the decision to seek treatment is affected by childhood adversity. In a study by Fairburn et al., (1996), which compared community and clinic cases of bulimia nervosa, whilst there were no differences between these two groups in terms of childhood sexual abuse (see also Welch and Fairburn, 1994), community cases had significantly higher rates of childhood physical abuse (clinic cases: 19%; community cases: 37%). This would suggest that some forms of childhood adversity may make it more difficult to seek help.

(2) The study was carried out in a tertiary referral centre for eating disorders. This might have biased the sample towards greater symptomatic severity and higher levels of comorbidity (see Fairburn et al., 1996). These factors are associated with greater levels of childhood family disturbance (see chapter 2) and this may have made the findings of this study less readily generalizable.

(3) Different types of eating disorders were compared. It would have been desirable to include other comparison groups i.e. normal controls and psychiatric controls. e.g.
those with depression or anxiety disorders. There are, however, a number of studies using the CECA in community settings (albeit in girls and women of different social background). The findings of the present study are put in context against these studies (table 5.7.7.).

(4) Data collection relied entirely on patients' history and no attempt was made to seek corroboration of patients' stories from other family members. However, as mentioned in the introduction to this chapter, the CECA has previously found good agreement between adult pairs of sisters about their early experiences (Bifulco et al., 1994).

(5) Axis I (e.g. depression) and axis II (personality disorder) comorbidity may lead to biased reporting of childhood care. For example, anorexic patients are often said to be shy and avoidant (Piran et al. 1988), which may be associated with under-reporting of intrafamilial difficulties, whereas bulimic patients' personalities often fit the borderline/histrionic spectrum (Levine and Hyler, 1986; Wonderlich et al., 1990), which may lead to over-reporting of family problems. The possibility that different types and rates of psychiatric comorbidity between the different diagnostic groups may have exaggerated the differences in reported levels of childhood care cannot be discounted in the present study (nor in most studies of different eating disorder subtypes). However, given that the CECA uses investigator-based ratings with behavioural criteria of pre-defined thresholds, this is likely to be less of a problem than with other subject-rated instruments assessing childhood care.

Bearing in mind the above limitations, the results of the present study are going to be discussed in turn.
Parental mental illness: Parental mental illness is likely to affect the quality of childhood care. In the present sample mental illness in the mother occurred in about a third to a fourth of anorexic and bulimic patients, with rates rising in chronic patients to 48% and 37% (for RAN and BN patients respectively). Other studies have also noted that psychiatric morbidity, including depression, alcoholism and eating disorders, is common in the families of patients with anorexia nervosa (Halmi et al., 1991) and bulimia nervosa (Kassett et al., 1989). Strober et al. (1990) found a 43% life-time prevalence of affective disorder in mothers of depressed AN patients and 10% in mothers of non-depressed AN patients. The comparable figures for fathers were 18% and 6%.

Changes in family structure: A considerable proportion of patients - between 22% and 42% - had experienced more than one family arrangement. Previously Piran et al. (1988) had found multiple care arrangements in 23 and 24% for RAN and BAN patients respectively. Interestingly, in the present study whereas in restricting anorexics the majority of those with multiple arrangements had two different arrangements only, in the bulimic subgroups a higher proportion had 3 or more family arrangements. Most of these changes in family arrangements were precipitated by parental separation. Loss of a parent does not necessarily carry a high risk of later psychiatric morbidity, provided it is followed by adequate substitute care (Harris et al., 1986). Clearly, the more changes in family arrangements there were (often through separation of parents and subsequent introduction and loss of step-parents) for a given individual, the less likely there was a chance that the substitute care was stable and adequate.
One question is whether rates of parental separation and loss in eating disorder subjects are higher than in control groups. Dolan et al. (1990) did not find divorce rates of probands with bulimia to be raised compared with the general population, however, Logue et al. (1989) and Bouman and Yates (1994) found rates of early parental loss or divorce to be higher in bulimia nervosa subjects than in normal controls. In this latter study parental divorce and parental mental ill health were associated. However, their study was unable to establish causal relationships.

**The quality of childhood care in context:** Table 5.7.7. puts the findings of the present study into context by comparing them against findings of community studies using the CECA.

Levels of parental indifference in the BN group were high and were comparable to those found in women with depression or panic disorder, and elevated in comparison to non-cases. In restricting anorexia nervosa, in contrast, levels of parental indifference were similar to those in non-cases. Parental indifference and physical abuse were the only variables necessary to model diagnostic group membership. Surprisingly, neither of these variables have been examined in detail as possible antecedents to an eating disorder.

Excessive parental control occurred much more commonly in BN than in RAN cases. Unfortunately, other studies using the CECA have not reported levels of high parental control in their samples. However, studies of the childhood care of eating disorders, using the parental bonding instrument (Parker, 1983) found patients to report high
control from their fathers more commonly than controls (Calam et al., 1990). Harsh parental control has also been found to be an antecedent of depression and alcohol abuse (Holmes and Robins, 1987; Parker, 1983).

Other studies have emphasised the deleterious long-term effects of low parental control (Crook et al., 1981). In the present study there was no difference between eating disorder groups on levels of low parental control, although low parental control occurred more frequently than in depressed or non-depressed working class women in the community (see table 5.7.7.).

High parental indifference and low control have been put together into a 'lack of care' index, which predicts adult depression (Andrews et al., 1990; Bifulco et al., 1987; Harris et al., 1986) (see table 5.7.7.). Levels of 'lack of care' found in the present study in the bulimia nervosa patients (BN: 46% versus RAN 23%) were higher than those found in depressed women in the community and markedly elevated compared to non-cases.

Intrafamilial discord occurred in two thirds of the BN group which is in keeping with previous reports of high levels of conflict in bulimic families in studies using other methodology (see chapter 2).

Childhood physical abuse was present in 25% of bulimia nervosa patients, which is in striking contrast to the very low rate of physical abuse in only 3% of the restricting anorexics. Thus hypothesis 2a was fully supported. Other authors using different
criteria (Fullerton et al., 1995; Piran et al., 1988), have also emphasized that restricters had lower rates of physical abuse than those with bulimic anorexia and bulimia nervosa. Thus the findings of the present study provide further support for a binge-purge and restricter subtypes introduced in DSM-IV. Violence against other family members followed a very similar pattern.

Rates of physical abuse in BN are only slightly raised compared to that found in working class women (table 5.7.7., 15% of total sample; Andrews et al., 1990). Compared with different anxiety disorder subtypes bulimia nervosa patients were intermediate between those with panic disorder and those with agoraphobia or GAD in terms of their rates of physical abuse.

In support of hypothesis 2b there was no difference in the rates of childhood sexual abuse in the four groups of the present study. The rates of 16-30 % of childhood sexual abuse found in the present study, are comparable to those found in some earlier studies of eating disorder patients (McClelland et al. 1991; Vize and Cooper, 1995; Welch and Fairburn, 1994), though lower than the rates of abuse quoted by studies which included non-contact events, abuse during adulthood and abuse by people of comparable age to the patient (see chapter 3). Recent studies using the CECA have found rates between 9 and 15 % in a general population sample (Andrews et al., 1995b; Bifulco et al., 1991; see table 5.7.7.) and rates of 18% for panic disorder patients. 14 % for those with agoraphobia, GAD or social phobia and 3% for those with simple phobia.
Childhood experiences and the course of eating disorders: Childhood adversity was a predictor of chronicity in bulimia nervosa, but not in anorexia nervosa, giving partial support to hypothesis 3. One can only speculate about possible links between childhood adversity and chronic bulimia nervosa. In studies of the course of depression, chronicity was predicted by childhood adversity and interpersonal difficulties during the course of the illness (Brown and Moran, 1994; Brown et al., 1994). Helplessness may be an important mediating variable. Troop and Treasure (1997) found high levels of childhood helplessness in bulimic disorders. This was associated with adult helplessness and a delay in seeking treatment (although this was only a trend, p < 0.08). Tentative support for this might be the finding that cases with physical abuse are frequently not in treatment (Fairburn et al., 1996).

Reported ratings: Bulimia nervosa patients felt more disapproval from their mothers and fathers than anorexic patients. However, they reported similar levels of parental approval. This links in with the notion of inconsistent expression of affect in families of bulimics (Humphrey, 1989; Scalf-McIver and Thompson, 1989). Studies of expressed emotion in families with an anorexic or a bulimic member noted the parents of bulimics to be more critical (Szmukler et al., 1985).

All in all these findings provide evidence that restricting anorexia nervosa and bulimia nervosa have different childhood antecedents, with bulimia nervosa patients experiencing more childhood adversity than those with anorexia nervosa. As predicted in hypothesis 1 the BAN group and BN/HistAN group were intermediate in terms of childhood adversity.
5.6. Summary and Conclusions

(1) Bulimia nervosa patients are exposed to more childhood adversity than those with anorexia nervosa, in particular more parental indifference, excessive parental control, physical abuse and violence against other family members. As predicted the BAN group and BN/HistAN group were intermediate in terms of childhood adversity.

(2) There were no differences between groups in terms of childhood sexual abuse.

(3) Different aspects of adversity tended to cluster in the same patients and 65% of the bulimic group had experienced two or more types of childhood adversity.

(5) Out of all the different indicators of poor childhood care studied only physical abuse and parental indifference were necessary to model diagnostic group membership.

(6) Whilst in both RAN and BN chronicity was associated with more mental illness in the mother, only in bulimia nervosa was chronicity also associated with more childhood adversity.

(7) There were no differences between groups on felt approval from mother and father, however, the groups did differ on felt disapproval with bulimia nervosa patients reporting the highest levels of disapproval from either parent.

These findings support the notion that restricting anorexia nervosa and bulimia nervosa have different childhood antecedents.
### Table 5.7.1: Sample characteristics

*Social Class: Chi-square = 6.9; DF = 3; p = 0.08*
<table>
<thead>
<tr>
<th>No. of Care Arrangements</th>
<th>Restricting Anorexia Nervosa (n = 64)</th>
<th>Bulimic Anorexia Nervosa (n = 23)</th>
<th>Bulimia Nervosa With a History of Anorexia Nervosa (n = 37)</th>
<th>Bulimia Nervosa (n = 79)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>43 (67%)</td>
<td>18 (78%)</td>
<td>22 (59%)</td>
<td>46 (58%)</td>
</tr>
<tr>
<td>2</td>
<td>19 (30%)</td>
<td>1 (4.0%)</td>
<td>7 (19%)</td>
<td>16 (20%)</td>
</tr>
<tr>
<td>3 or more</td>
<td>2 (3%)</td>
<td>4 (18%)</td>
<td>8 (22%)</td>
<td>17 (22%)</td>
</tr>
</tbody>
</table>

Table 5.7.2: Number of family arrangements. Chi-square = 16.0, DF = 6, p = 0.01
<table>
<thead>
<tr>
<th></th>
<th>Restricting Anorexia Nervosa</th>
<th>Bulimic Anorexia Nervosa</th>
<th>Bulimia Nervosa With a History of Anorexia Nervosa</th>
<th>Bulimia Nervosa</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>n (%)</td>
<td>n (%)</td>
<td>n (%)</td>
<td>n (%)</td>
</tr>
<tr>
<td>Boarding School</td>
<td>6/64 (9 %)</td>
<td>0/23 (0 %)</td>
<td>3/37 (8 %)</td>
<td>2/79 (3 %)</td>
</tr>
<tr>
<td>Adopted</td>
<td>0/64 (0 %)</td>
<td>0/23 (0 %)</td>
<td>2/37 (5 %)</td>
<td>2/79 (3 %)</td>
</tr>
<tr>
<td>Parental Separation</td>
<td>11/64 (17 %)</td>
<td>3/23 (13 %)</td>
<td>8/37 (22 %)</td>
<td>21/79 (27 %)</td>
</tr>
<tr>
<td>Parental Death</td>
<td>2/64 (3 %)</td>
<td>1/23 (4 %)</td>
<td>1/37 (3 %)</td>
<td>5/79 (6 %)</td>
</tr>
<tr>
<td>Other Reason</td>
<td>2/64 (3 %)</td>
<td>1/23 (4 %)</td>
<td>1/37 (3 %)</td>
<td>3/79 (4 %)</td>
</tr>
</tbody>
</table>

Table 5.7.3: Reasons for change in family arrangements.
<table>
<thead>
<tr>
<th></th>
<th>Restricting Anorexia Nervosa</th>
<th>Bulimic Anorexia Nervosa</th>
<th>Bulimia Nervosa With a History of Anorexia Nervosa</th>
<th>Bulimia Nervosa</th>
<th>Chi²</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indifference</td>
<td>8/64 (13%)</td>
<td>4/23 (17%)</td>
<td>11/37 (30%)</td>
<td>30/79 (38%)</td>
<td>13.1</td>
<td>0.004</td>
</tr>
<tr>
<td>High Control</td>
<td>12/64 (19%)</td>
<td>5/23 (22%)</td>
<td>9/37 (24%)</td>
<td>34/79 (43%)</td>
<td>11.6</td>
<td>0.009</td>
</tr>
<tr>
<td>Low Control¹</td>
<td>11/64 (17%)</td>
<td>4/23 (17%)</td>
<td>6/37 (16%)</td>
<td>15/78 (19%)</td>
<td>0.2</td>
<td>0.979</td>
</tr>
<tr>
<td>Discord²</td>
<td>30/64 (47%)</td>
<td>9/23 (39%)</td>
<td>22/37 (59%)</td>
<td>51/79 (64%)</td>
<td>7.3</td>
<td>0.064</td>
</tr>
<tr>
<td>Violence against Family Member</td>
<td>6/64 (9%)</td>
<td>4/23 (17%)</td>
<td>8/37 (22%)</td>
<td>23/79 (29%)</td>
<td>8.7</td>
<td>0.033</td>
</tr>
<tr>
<td>Violence against Patient</td>
<td>2/64 (3%)</td>
<td>2/23 (9%)</td>
<td>3/37 (9%)</td>
<td>20/79 (25%)</td>
<td>16.9</td>
<td>0.0007</td>
</tr>
<tr>
<td>Childhood Sexual Abuse¹</td>
<td>10/63 (16%)</td>
<td>7/23 (30%)</td>
<td>11/35 (31%)</td>
<td>20/76 (26%)</td>
<td>4.0</td>
<td>0.258</td>
</tr>
</tbody>
</table>

Table 5.7.4: Indicators of childhood care based on behavioural evidence. ¹: In one case of BN there was not sufficient information to make a rating on presence or absence of low control. ²: The discord ratings also include cases of high tension. ³: No information was available on child sexual abuse in 1 RAN case, 2 BN/HistAN and 3 BN cases.
<table>
<thead>
<tr>
<th></th>
<th>Bulimia Nervosa</th>
<th>Bulimia Nervosa With A History Of Anorexia Nervosa</th>
<th>Chi²</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Restricting Anorexia Nervosa</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>n (%)</td>
<td>n (%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Approval from Mother</td>
<td>43/64 (67%)</td>
<td>12/23 (52%)</td>
<td>50/78 (64%)</td>
<td>0.63</td>
</tr>
<tr>
<td>Disapproval from Mother</td>
<td>12/64 (19%)</td>
<td>9/36 (25%)</td>
<td>31/77 (40%)</td>
<td>0.04</td>
</tr>
<tr>
<td>Approval from Father</td>
<td>42/62 (68%)</td>
<td>12/23 (52%)</td>
<td>38/70 (54%)</td>
<td>0.16</td>
</tr>
<tr>
<td>Disapproval from Father</td>
<td>6/62 (10%)</td>
<td>5/23 (22%)</td>
<td>23/70 (33%)</td>
<td>0.02</td>
</tr>
</tbody>
</table>

Table 5.7.5: Reported ratings of childhood care. (Ratings were not available for all cases as some patients only grew up with one parent.)
<table>
<thead>
<tr>
<th></th>
<th>Restricting Anorexia Nervosa ≤4 years duration (n = 43)</th>
<th>Restricting Anorexia Nervosa &gt;4 years duration (n = 21)</th>
<th>Odds Ratio (95 % Confidence interval)</th>
<th>Bulimia Nervosa ≤4 years duration (n = 33)</th>
<th>Bulimia Nervosa &gt;4 years duration (n = 46)</th>
<th>Odds Ratio (95 % Confidence interval)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mental Illness in Mother</td>
<td>8/43 (19 %)</td>
<td>10/21 (48 %)</td>
<td>4.0*</td>
<td>5/33 (15 %)</td>
<td>17/46 (37 %)</td>
<td>3.3**</td>
</tr>
<tr>
<td>Mental Illness in Father</td>
<td>9/43 (21 %)</td>
<td>3/21 (14 %)</td>
<td>0.8</td>
<td>4/32 (13 %)</td>
<td>12/43 (28 %)</td>
<td>2.7</td>
</tr>
<tr>
<td>≥ 2 Care Arrangements</td>
<td>16/43 (37 %)</td>
<td>5/21 (24 %)</td>
<td>1.9</td>
<td>10/33 (30 %)</td>
<td>23/46 (50 %)</td>
<td>2.3</td>
</tr>
<tr>
<td>Indifference</td>
<td>3/43 (7 %)</td>
<td>5/21 (24 %)</td>
<td>4.2</td>
<td>5/33 (15 %)</td>
<td>25/46 (54 %)</td>
<td>6.7***</td>
</tr>
<tr>
<td>High Control</td>
<td>8/43 (19 %)</td>
<td>4/21 (19 %)</td>
<td>1.0</td>
<td>11/33 (33 %)</td>
<td>23/46 (50 %)</td>
<td>2.0</td>
</tr>
<tr>
<td>Low Control</td>
<td>9/43 (21 %)</td>
<td>2/21 (10 %)</td>
<td>0.4</td>
<td>3/33 (9 %)</td>
<td>12/45 (27 %)</td>
<td>3.6</td>
</tr>
<tr>
<td>Discord</td>
<td>18/43 (42 %)</td>
<td>12/21 (57 %)</td>
<td>1.85</td>
<td>16/33 (48 %)</td>
<td>35/46 (76 %)</td>
<td>3.4**</td>
</tr>
<tr>
<td>Violence against Close</td>
<td>5/43 (12 %)</td>
<td>1/21 (5 %)</td>
<td>0.4</td>
<td>8/33 (24 %)</td>
<td>15/46 (33 %)</td>
<td>1.5</td>
</tr>
<tr>
<td>Other</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Violence against Patient</td>
<td>2/43 (5 %)</td>
<td>0/21 (0 %)</td>
<td>1.0</td>
<td>6/33 (18 %)</td>
<td>14/46 (30 %)</td>
<td>2.0</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td>0.7 - 5.8</td>
</tr>
<tr>
<td></td>
<td>Restricting Anorexia Nervosa ≤4 years duration (n=43)</td>
<td>Restricting Anorexia Nervosa &gt;4 years duration (n=21)</td>
<td>Odds Ratio (95 % Confidence interval)</td>
<td>Bulimia Nervosa ≤4 years duration (n=33)</td>
<td>Bulimia Nervosa &gt;4 years duration (n=46)</td>
<td>Odds Ratio (95 % Confidence interval)</td>
</tr>
<tr>
<td>--------------------------</td>
<td>-------------------------------------------------------</td>
<td>--------------------------------------------------------</td>
<td>--------------------------------------</td>
<td>---------------------------------------</td>
<td>---------------------------------------</td>
<td>--------------------------------------</td>
</tr>
<tr>
<td>Childhood Sexual Abuse</td>
<td>7/42 (17 %)</td>
<td>3/21 (14 %)</td>
<td>0.8 (0.2 - 3.6)</td>
<td>6/31 (19 %)</td>
<td>14/45 (31 %)</td>
<td>1.9 (0.6 - 5.6)</td>
</tr>
<tr>
<td>High Adversity</td>
<td>14/43 (33 %)</td>
<td>5/21 (24 %)</td>
<td>0.6 (0.2 - 2.1)</td>
<td>13/31 (42 %)</td>
<td>37/46 (80 %)</td>
<td>5.7*** (2.1 - 15.8)</td>
</tr>
</tbody>
</table>

Table 5.7.6.: Differences between recent onset (< 4 years) and chronic (> 4 years) cases. (Restricting anorexics and bulimic patients only). *: p < 0.05; **: p < 0.01; ***: p < 0.001
<table>
<thead>
<tr>
<th>Childhood Factor</th>
<th>% with childhood factor</th>
<th>Sample</th>
<th>Authors</th>
</tr>
</thead>
<tbody>
<tr>
<td>Indifference</td>
<td>(1) RAN: 13%, BN: 38%</td>
<td>present study</td>
<td>Bifulco et al., 1987</td>
</tr>
<tr>
<td></td>
<td>(2) 31% of depressed subjects; 11% of non-cases</td>
<td>community sample of working class women</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(3) 41% of panic disorder subjects; 29% of agoraphobia, GAD or social phobia; 16% of simple phobia or mild agoraphobia</td>
<td>community sample of working class women</td>
<td>Brown and Harris, 1993</td>
</tr>
<tr>
<td>Low Control</td>
<td>(1) RAN: 17%, BN: 19%</td>
<td>present study</td>
<td>Bifulco et al., 1987</td>
</tr>
<tr>
<td></td>
<td>(2) 7% of depressed subjects; 4% of non-depressed</td>
<td>community sample of working class women</td>
<td></td>
</tr>
<tr>
<td>Lack of Care (= high indifference or low control)</td>
<td>(1) RAN: 23%, BN: 46%</td>
<td>present study</td>
<td>Bifulco et al., 1987</td>
</tr>
<tr>
<td></td>
<td>(2) 32% of case depression; 14% of non-cases</td>
<td>community sample of working class women</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(3) 11% of total sample</td>
<td>daughters (age 15 to 25) of Bifulco et al.'s (1987) sample</td>
<td>Andrews et al., 1990</td>
</tr>
<tr>
<td>Physical Abuse</td>
<td>(1) RAN: 3%; BN: 25%</td>
<td>present study</td>
<td>Andrews et al., 1990</td>
</tr>
<tr>
<td></td>
<td>(2) 15% of total sample</td>
<td>daughters (age 15 to 25) of Bifulco et al.'s (1987) sample</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(3) 45% of panic disorder subjects; 26% of agoraphobia, GAD or social phobia, 6% of simple phobia or mild agoraphobia</td>
<td>community sample of working class women</td>
<td>Brown and Harris, 1993</td>
</tr>
<tr>
<td>Sexual Abuse</td>
<td>(1) RAN: 16%; BN: 26%</td>
<td>present study</td>
<td>Bifulco et al., 1991</td>
</tr>
<tr>
<td></td>
<td>(2) 9% of total sample</td>
<td>community sample of working class women</td>
<td></td>
</tr>
<tr>
<td></td>
<td>(3) 15% of total sample</td>
<td>daughters (age 15 to 25) of Bifulco et al.'s (1987) sample</td>
<td>Andrews et al., 1995b</td>
</tr>
<tr>
<td></td>
<td>(3) 18% of panic disorder subjects; 14% of agoraphobia, GAD or social phobia; 3% of simple phobia or mild agoraphobia</td>
<td>community sample of working class women</td>
<td>Brown and Harris, 1993</td>
</tr>
</tbody>
</table>
Table 5.7.7: Indicators of childhood care in community studies using the same methodology as the present study. ¹: RAN = Restricting Anorexia Nervosa; ²: BN = Bulimia Nervosa. * In the present study childhood sexual abuse only included events occurring in a victim who was 16 years or below, whereas in Bifulco et al. (1991)’s study childhood sexual abuse included events occurring up to age 17.
Marked indifference: A patient's mother was totally disinterested in her schoolwork, never went out or played with her and spent the whole day in bed. The patient had to care for her baby brother and herself from age 8 onwards. Her duties included changing his nappies, bathing him, cooking for the family and doing her own washing.

High control: (a) One patient said: "My stepfather was very strict. You had to be perfect, or else he would hit you or send you to bed". Other punishments the stepfather used included locking the patient in the cellar for up to an hour when she was 7 or 8 years old. On one occasion he made her and her siblings eat dog food and orange peel after a minor misdemeanour. On another occasion the children had to pick up all the fluff off the carpet as a punishment. He would never allow the children out or allow them to bring their friends home.

(b) Another patient who described herself as a quiet child who never caused any trouble said her mother was very strict about schoolwork, clothes, make-up and going out with boys. If the mother got angry she would "scream and roar or would threaten to ring up my school or friends to tell them about my behaviour. She'd also threaten me with going to hell or being sent to boarding school".

Low control: (a) A patient who grew up with mother and stepfather said: "My sisters and I used to run riot, we totally looked after ourselves. My mother never had time for us".
Another patient and her siblings grew up with their grandmother abroad whilst their parents were working in England. She described having hardly any adult supervision and mentioned that she and her sibs were left to do everything for themselves. "We were left to do as we pleased all day. If we were hungry we would pick some fruit from the trees. We went to school on our own and played on the beach".

**Marked Discord:** "My parents fought like cat and dog, they were permanently arguing, my mum was the boss, she'd go on and on at my dad, and then he'd lose his control".

**Marked Violence Against the Patient:** One patient was repeatedly hit with a belt by her father. Another patient's mother repeatedly stabbed her with forks and knitting needles. Another patient's father in addition to regularly hitting her, on one occasion threw her to the ground, jumped onto her and smashed her head into the floor.

**Childhood Sexual Abuse:** One patient was repeatedly called into her grandfather's bed to have stories read to her and he would then fondle her breasts and genitals. Another patient was sexually abused by a male servant of the family, who stripped her naked and had intercourse with her. She was threatened in case she told her parents.
Chapter 6: Childhood Adversity and Other Familial Risk Factors in Obese Bulimic Patients: A Retrospective Case-Control Study

6.1. Aims and Hypotheses

Aim 1: To determine whether obese bulimia nervosa patients and those with normal weight bulimia nervosa differ in terms of clinical presentation and associated psychopathology.

Hypothesis 1a: Obese and normal-weight bulimics will differ in their eating symptomatology.

Hypothesis 1b: Obese bulimics will have more associated psychopathology than normal weight bulimics.

Aim 2: To determine whether obese and normal weight cases of bulimia nervosa differ in terms of a family history of psychiatric disorders or obesity, the quality of childhood care and rates of childhood trauma, in particular childhood sexual abuse.

Hypothesis 2a: Obese bulimic patients will be more likely to have a family history of obesity and depression than those with normal weight bulimia nervosa.

Hypothesis 2b: Obese bulimic patients will have had poorer childhood care than those with normal weight bulimia nervosa.

Hypothesis 2c: Obese bulimic patients will have had more childhood sexual abuse than those with normal weight bulimia nervosa.

6.2. Introduction

Binge eating within obesity was first described in the 1950s (Stunkard, 1959). However, interest in overweight binge eaters has only recently developed and there is
confusion over what to call and how to classify these patients ("obese binge eaters", Marcus et al., 1988; "non-purging bulimia", McCann et al., 1990; "binge eating disorder", Spitzer et al., 1992). The debate culminated in the suggestion that binge eating disorder should be included in DSM-IV as a disorder separate from bulimia nervosa (Spitzer et al., 1992; 1993). However, this idea has been criticized (Fairburn et al., 1993) on the basis of a community-based cluster analysis which failed to support it (Hay et al., 1996) and the inclusion of binge eating disorder in DSM has been put on hold until further clarification of its diagnostic usefulness.

In the community obese binge eaters have a prevalence of 2%, but one fourth to one third of obese patients presenting for treatment suffer from severe problems with binge eating (de Zwaan et al., 1992; Gormally et al., 1982; Keefe et al., 1984; Loro and Orleans, 1981; Spitzer et al., 1992; Wilson et al., 1993). Obese binge eaters have more psychopathology and dietary restraint than other obese patients (Loro and Orleans, 1981; Marcus et al., 1985; 1988; 1990; Prather and Williamson, 1988; Specker et al., 1994; Telch and Agras, 1994; Wadden et al., 1993; Yanowski et al., 1993). Compared with normal weight DSM-III bulimics, obese bulimic volunteers used vomiting less often as a method of weight control. Normal weight and obese bulimics had a higher life time prevalence of major affective disorder than non-bingeing obese subjects (Hudson et al., 1988).

Mitchell et al. (1990) compared 25 obese and 25 normal-weight patients with DSM-III-R BN. The obese BN group were older, of lower social class, they binged and vomited less, but took more laxatives and were more depressed with deliberate self
harm. A family history of depression and obesity was more common than in the normal weight bulimics. Marcus et al., (quoted from Wing and Marcus, 1991) found obese binge eaters and normal weight BN patients to be similar apart from more dietary restraint in the normal weight group.

Little is known about the childhood antecedents of obese binge eaters, however, the psychosomatic model of obesity suggests that weight gain is the result of emotional deprivation in childhood, leading the child to eat in response to emotional rather than hunger cues. Eating may thus represent a way of self-soothing in the face of loneliness, anxiety, depression or boredom (Bruch, 1973). It has been suggested that childhood sexual abuse may be an aetiological factor in obesity (Black et al., 1992; Felitti, 1991). In support of this one study, which compared obese patients, with bulimics, anorexics and patients with other psychosomatic conditions found that those with obesity had the highest rates of childhood sexual abuse (Köpp, 1994). It therefore seemed reasonable to hypothesize that obese bulimic patients would have poorer childhood care and higher levels of childhood sexual abuse than those with normal weight bulimia nervosa. On the other hand there is some evidence that childhood sexual abuse is related to the frequency of compensatory behaviours like vomiting in eating disorder patients (Pitts and Waller, 1993; Tobin and Griffing, 1996) and since obese binge eaters have been noted to have a lower frequency of vomiting than normal weight bulimics (Mitchell et al., 1990) an alternative hypothesis might be that they might have lower rates of childhood sexual abuse.
6.3. Subjects and Method

6.3.1. Sample

36 female cases of ICD-10 bulimia nervosa (WHO, 1990) with obesity (OBN; body mass index (BMI: $\geq 25$ kg/m$^2$) were identified from consecutive referrals to the Eating Disorder Clinic at the Maudsley Hospital. (The main difference between ICD-10 and DSM-III-R criteria is the lack of a frequency criterion for bingeing in ICD-10. The more lenient ICD-10 criteria rather than DSM-III-R criteria were used in this study in order to be able to include a broader spectrum of patients).

Female normal-weight bulimia nervosa patients (NBN; BMI 19 - 25 kg/m$^2$) were identified from the same period of time as the index group. Each OBN case was matched with the next case of NBN from the same social class, determined by fathers' occupation as outlined in chapter 5 (Guy, 1976).

6.3.2. Procedure

All cases had a Maudsley Hospital clinical history taken (Department of Psychiatry and Child Psychiatry, Institute of Psychiatry and Maudsley Hospital, 1987). Eating pathology was assessed by clinical interview and clinician-rated scales of bingeing, vomiting and laxative abuse were completed (bingeing/vomiting: 0 - 6 scales; 0 = absent; 1 = less than twice per week; 2 = twice a week; 3 = three to six times per week; 4 = daily; 5 = two to three times per day; 6 = $\geq$ 4 times per day; laxative abuse: 0 - 4 scale; 0 = absent; 1 = mild; 2 = moderately severe; 3 = severe; 4 = extremely severe) (Duphar Laboratories, 1989; unpublished manuscript). Patients' concurrent psychopathology was assessed using the Structured Clinical Interview (SCID; Spitzer et al., 1989). A family history of depression or eating disorder was
rated as present if a first degree relative had been treated by a general practitioner or a mental health professional. A family history of obesity was rated as present if the patient described their parents or siblings as markedly overweight. (Wherever possible, it was attempted to get estimates of weight and height for first degree relatives). Additionally, all patients were assessed with the CECA (Bifulco et al., 1994), details of which, including the definition of the composite measure of childhood adversity, are described in chapter 5.

**Statistical analysis:** As in the previous study data were analyzed using SPSS-PC+4.0 (Norusis, 1990), using the Mann-Whitney U-test for non-parametric data. Odds ratios with 95% confidence intervals were used for categorical data (Gardner et al., 1991).

### 6.4. Results

#### 6.4.1. Sample characteristics

The two groups were similar in age at referral (OBN: 26.2 (±6.9) vs NBN: 25.2 (±5.9) years) and age at onset of their eating disorder (OBN: 18.2 (±5.5) vs NBN: 17.8 (±3.7) years). Sixteen patients (44%) in each group were from a working class background. The mean current BMI was 31.0 (±6.8) kg/m² for the OBN and 21.0 (±1.6) kg/m² for the NBN group. Similarly, the two groups differed in lowest BMI since onset of an eating disorder (OBN: 22.1 (±4.8) kg/m²) vs NBN: 17.5 (±1.9) kg/m²) and highest BMI since onset of an eating disorder (OBN 34.1 (±6.3) kg/m² vs NBN: 24.5 (±3.5) kg/m²).

#### 6.4.2. Clinical features

There were no differences in the frequency of bingeing and laxative abuse between the
two groups, but NBN patients induced vomiting more commonly (Table 6.7.1.).

6.4.3. Associated psychopathology

The two groups were similar in terms of the proportion of patients with concurrent depression, deliberate self harm, substance abuse and stealing (table 6.7.1.).

6.4.4. Family factors

OBN patients had more first degree relatives with obesity than NBN patients, who in turn had a family history of depression and of eating disorders more often than OBN patients (table 6.7.2.).

6.4.5. Childhood care and abuse

There was no difference on any of the individual scales assessing quality of childhood care, however, there was a trend for increased childhood adversity in NBN patients on the summary measure of adversity (table 6.7.2.).

6.5. Discussion

Some of the short-comings of the present study are similar to the ones described in the study in the previous chapter (patients rather than community subjects; tertiary referral centre; lack of normal controls; data collection relied entirely on patients’ history). These will not be repeated here. In addition, the sample size was small and the inclusion of an obese non-bingeing comparison group would have been desirable.

In support of hypothesis 1a there were some differences in the clinical features of the
two groups. Obese bulimics used self-induced vomiting as a method of weight control less than NBN patients. It is thus possible that the differing body mass index at presentation merely reflects the difference in the type of weight control strategies used in the two groups.

The finding of (a) similar levels of concurrent psychopathology and (b) a higher rate of depression and eating disorders in the families of NBN patients rather than OBN patients were contrary to the hypotheses put forward, which had been derived based on the findings by Mitchell et al. (1990). Methodological details may account for this. The patients in the present study were matched for social class, whereas Mitchell’s study did not control for this variable. The increased family history of obesity in the OBN group is not surprising, given the strong genetic contribution to obesity (Stunkard et al., 1986).

Contrary to hypotheses 2b and 2c there was a trend for NBN patients to have more childhood adversity than OBN patients with no differences between the two groups on childhood sexual abuse. This is in keeping with the increase in depression and eating disorders in the NBN families, as poor parental mental health is known to be associated with deficits in childhood care (Andrews et al., 1990).

The findings of the present study suggest, albeit in a small study, that there may be aetiological differences between OBN and NBN. Large community studies are needed to clarify fully the aetiological factors of this group of patients and to determine whether they fit the proposed binge eating disorder spectrum and as such show
6.6. Summary and Conclusions

(1) There were quantitative differences in the clinical features of the two groups, with obese bulimics using self-induced vomiting as a method of weight control less than NBN patients.

(2) Both groups showed similar levels of concurrent psychopathology.

(3) A higher rate of depression and eating disorders was found in the families of NBN patients rather than in those of OBN patients.

(4) OBN patients had an increased family history of obesity.

(5) There were no differences between the two groups in terms of the quality of their childhood care, if anything there was a trend for those with obesity to have less problematic childhoods.

These findings support the notion that there may be some aetiological differences between the OBN and NBN patients.
### Table 6.7.1: Clinical features and associated psychopathology

Eating symptomatology was assessed by clinician rated scales (bingeing/vomiting: 0 - 6 scales; 0 = behaviour absent; 1 = less than twice a week; 2 = twice a week; 3 = three to six times per week; 4 = daily; 5 = two to three times per day; 6 = $\geq$ 4 times per day; laxative abuse: 0 - 4 scale; 0 = absent; 1 = mild; 2 = moderately severe; 3 = severe; 4 = extremely severe). *: Mann Whitney U-test; Bonferroni correction applied (original p-value multiplied by 3).

<table>
<thead>
<tr>
<th>Clinical Features</th>
<th>OBN (n=36) Median (Range)</th>
<th>NBN (n=36) Median (Range)</th>
<th>p*</th>
</tr>
</thead>
<tbody>
<tr>
<td>Frequency of Bingeing</td>
<td>3 (1-5)</td>
<td>4 (1-6)</td>
<td>NS</td>
</tr>
<tr>
<td>Frequency of Vomiting</td>
<td>0.5 (0-6)</td>
<td>4 (0 - 6)</td>
<td>0.0006</td>
</tr>
<tr>
<td>Severity of Laxative Abuse</td>
<td>0 (0-4)</td>
<td>0 (0-4)</td>
<td>NS</td>
</tr>
<tr>
<td>Associated Psychopathology</td>
<td></td>
<td></td>
<td>Odds Ratio (95% CI)</td>
</tr>
<tr>
<td>Depression</td>
<td>13/36 (36%)</td>
<td>18/36 (50%)</td>
<td>0.6 (0.2-1.5)</td>
</tr>
<tr>
<td>Deliberate Self-Harm</td>
<td>7/36 (19%)</td>
<td>12/36 (33%)</td>
<td>0.5 (0.2-1.4)</td>
</tr>
<tr>
<td>Substance Abuse</td>
<td>13/36 (36%)</td>
<td>13/36 (36%)</td>
<td>1.0 (0.4-2.6)</td>
</tr>
<tr>
<td>Stealing</td>
<td>2/36 (6%)</td>
<td>5/36 (14%)</td>
<td>0.4 (0.7-2.0)</td>
</tr>
<tr>
<td>Family History of:</td>
<td>OBN (n=36)</td>
<td>NBN (n=36)</td>
<td>Odds Ratio (95% CI)</td>
</tr>
<tr>
<td>----------------------------------------</td>
<td>------------</td>
<td>------------</td>
<td>--------------------</td>
</tr>
<tr>
<td>Depression</td>
<td>4/35 (11%)</td>
<td>12/34 (35%)</td>
<td>0.2 (0.07-0.8)&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Eating Disorders</td>
<td>2/35 (6%)</td>
<td>9/34 (26%)</td>
<td>0.2 (0.03-0.8)&lt;sup&gt;b&lt;/sup&gt;</td>
</tr>
<tr>
<td>Obesity</td>
<td>20/35 (57%)</td>
<td>9/34 (26%)</td>
<td>3.7 (1.3-10.2)&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Family Structure</th>
<th></th>
<th></th>
<th>0.4&lt;sup&gt;d&lt;/sup&gt; (0.1-1.02)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Multiple Family Arrangements</td>
<td>10/36 (28%)</td>
<td>18/36 (50%)</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Quality of Childhood Care</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Parental Indifference</td>
<td>14/36 (39%)</td>
<td>16/36 (44%)</td>
<td>0.8 (0.3-2.0)</td>
</tr>
<tr>
<td>High Control</td>
<td>9/36 (25%)</td>
<td>16/36 (44%)</td>
<td>0.5 (0.2-1.2)</td>
</tr>
<tr>
<td>Low Control</td>
<td>5/36 (14%)</td>
<td>7/36 (19%)</td>
<td>0.7 (0.2-2.3)</td>
</tr>
<tr>
<td>Discord</td>
<td>20/36 (56%)</td>
<td>24/36 (67%)</td>
<td>0.6 (0.2-1.6)</td>
</tr>
<tr>
<td>Violence Against Other</td>
<td>7/36 (19%)</td>
<td>13/36 (36%)</td>
<td>0.5 (0.2-1.4)</td>
</tr>
<tr>
<td>Violence Against Patient</td>
<td>7/36 (19%)</td>
<td>10/36 (28%)</td>
<td>0.6 (0.2-1.9)</td>
</tr>
<tr>
<td>Childhood Sexual Abuse</td>
<td>7/35 (20%)</td>
<td>12/35 (34%)</td>
<td>0.5 (0.2-1.4)</td>
</tr>
<tr>
<td>Summary Scale of Childhood Adversity</td>
<td>16/36 (44%)</td>
<td>24/36 (67%)</td>
<td>0.4 (0.2-1.04)&lt;sup&gt;c&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

**Table 6.7.2.:** Family factors <sup>a</sup>: p < 0.04; <sup>b</sup>: p < 0.02; <sup>c</sup>: p < 0.02; <sup>d</sup>: p < 0.09; <sup>c</sup>: p < 0.10. All Chi-square tests with Yates correction.
Chapter 7: Childhood Adversity and Other Familial Risk Factors in the Development of Early Onset Bulimia Nervosa: A Retrospective Case-Control Study

7.1. Aims and Hypotheses

Aim 1: To determine whether early and typical onset cases of bulimia nervosa differ in terms of clinical presentation and associated psychopathology.

Hypothesis 1a: Early onset cases will be more severely ill than typical onset cases.
Hypothesis 1b: Early onset cases will have more associated psychopathology than typical onset cases.

Aim 2: To determine whether early and typical onset cases of bulimia nervosa differ in terms of a family history of psychiatric or eating disorders, the quality of childhood care and rates of childhood trauma, in particular childhood sexual abuse.

Hypothesis 2a: Early onset cases will be more likely to have a family history of depression, substance abuse and eating disorders.
Hypothesis 2b: Bulimic patients with an early onset will have had poorer childhood care than those with typical onset.
Hypothesis 2c: Early onset cases will have had more childhood sexual abuse than those with a typical onset.

7.2. Introduction

Dieting in preadolescent girls is a relatively new phenomenon (Hill, 1993). It is thought to be the result of increased socio-cultural pressures to be slim in the context of a secular trend for pubertal development to occur at an increasingly lower age.
A significant proportion of girls between the ages of 7 and 12 years are weight conscious and diet (Childress et al., 1993; Collins, 1991; Davies and Furnham, 1986; Hill et al., 1992; Maloney et al., 1989; Wardle and Marsland, 1990). Prospective data from the food diaries of a group of 9-year olds confirm that those who claim to diet actually do so (Hill and Robinson, 1991). Those with a higher body weight (Hill et al., 1989; Hill et al., 1992; Wardle and Beales, 1986) and those perceiving themselves as overweight (Hill et al., 1992; Wadden et al., 1989) are more likely to diet. Other factors associated with dieting in preadolescence and early adolescence include the onset of menarche and dating (Gralen et al., 1990), as during pubertal development girls are frequently distressed about their increase in body fat (Brooks-Gunn and Warren, 1988). Maternal and peer influences are also important (Hill et al., 1990). In one study, over two-thirds of the girls reported that their mother had dieted at some time and 45% that they had a friend who dieted (Maloney et al., 1989). In another study (Attie and Brooks-Gunn, 1989), a relationship between the girls’ pubertal development and mothers’ menstrual status and dieting was found.

Early maturing girls whose mothers were premenopausal dieted less than early maturers whose mothers were menopausal. This was thought to be linked to the greater body fat of the menopausal mothers who, therefore, dieted more.

The preoccupation with weight and dieting in increasingly younger children has been thought to lead to an increase in eating disorders in this group (Lask and Bryant-Waugh, 1992) and there is some evidence from epidemiological studies (Kendler et al., 1991; Turnbull et al., 1996) that the age of onset of bulimia nervosa is indeed decreasing. Until now the typical age of onset of bulimia nervosa has been thought to
be around age 18 (Mitchell et al., 1987), however, case reports of early onset bulimia nervosa have begun to appear. One series described eleven cases with an onset of bulimia nervosa between the ages of 13 and 16 (Remschmidt and Herpertz-Dahlmann, 1990). In a cohort of patients presenting to a tertiary referral centre 25% had started to binge before age 16 (Woodside and Garfinkel, 1992). Premenarchal bulimia nervosa is, however, still a rarity. Only three of a cohort of 323 patients with bulimia nervosa described a pre-menarchal onset (Kent et al., 1992). None of them had a pre-pubertal onset.

It can be argued that subjects who develop a disorder earlier than average may have a higher loading on risk factors, be they genetic or environmental, as is the case in coronary heart disease and Alzheimer type dementia and the present study attempts to assess this. Bulimia nervosa is generally thought to be a multifactorial disorder and, apart from dieting (Patton, 1990), other risk factors include: (a) a family history of affective disorder, alcoholism and obesity (Kassett et al., 1989); (b) deficits in childhood care and childhood trauma (see chapter 5); (c) distressing life events and difficulties (see chapter 4); and (d) acculturation stress (Mumford and Whitehouse, 1988). No attempt was made to study life events and difficulties before onset, as many of the patients included in the present study had a chronic disorder, which would have made it difficult to reliably rate life events before onset.
7.3. Subjects and Method

7.3.1. Sample

Forty cases of early onset bulimia nervosa (EO) were identified from two sources: (a) from a consecutive series of patients referred to the Eating Disorder Clinic (32 cases) and (b) from consecutive referrals to the Department of Child and Adolescent Psychiatry at the Maudsley Hospital (8 cases) over the same time period. All patients were female. In order to qualify for an early onset, subjects had to fulfil DSM-III-R criteria for bulimia nervosa at the age of 15 or below. This cut-off point was chosen because all the subjects in this group would have been expected to live in the parental home (or equivalent) and be in full-time education.

The comparison group consisted of female patients with a typical age of onset (TO), who were also identified from patients referred to the Maudsley Hospital Eating Disorder Clinic and who had been seen over the same period of time as the index group. A typical onset was defined as an onset of DSM-III-R bulimia nervosa between the ages of 17 and 21. Patients with an onset after the age of 21 were not included in the TO group, as they may constitute yet another atypical group (Mitchell et al., 1987). Each case of early onset bulimia nervosa was matched with the subsequently referred case of typical onset bulimia nervosa from the same social class.

7.3.2. Procedure

At the time of patients’ initial assessment, a standard Maudsley Hospital history was taken (Department of Psychiatry and Child Psychiatry, Institute of Psychiatry and Maudsley Hospital, 1987). The CECA (Bifulco et al., 1994), was administered to
sixty-eight of the 72 patients seen in the adult department. Details of the use of this interview in eating disorder patients have already been described above (chapter 5).

Five of the eight cases seen in the children's department were assessed with a semi-structured family interview (Kinston and Loader, 1984). These and the remaining cases (three presenting to the children's department and four presenting to the adult department) were re-rated using the criteria of the CECA to ensure a uniform assessment procedure. Cultural stress was rated as present if the patient was a first or second generation immigrant.

A family history of mental disorder, eating disorder or obesity was rated using the criteria outlined in chapter 6. A personal history of being overweight was rated as present if the patient had a body mass index of $> 25 \text{ kg/m}^2$ before the onset of bulimia nervosa. Socio-economic status was determined using the same criteria as outlined in the study in chapter 5.

Data were analyzed using SPSS/PC+4.0 (Norusis, 1990). Odds ratios with 95% confidence intervals were used for categorical data (Gardner et al., 1991) and $t$-tests for normally distributed data.
7.4. Results

7.4.1. Sample characteristics

At the time of referral, patients in the EO group had a mean age of 20.1 (±4.5) years and patients in the TO group a mean age of 23.9 (±3.8) years. The duration of bulimia nervosa at presentation was comparable in the two groups with 6.4 (±5.1) years in the EO group and 5.1 (±3.8) years in the TO group. The body mass index at presentation was 22.9 (±4.0) kg/m² in the EO and 21.6 (±2.2) kg/m² in the TO group. This difference is not significant. One third of patients in both groups were of working class background.

7.4.2. Clinical features

As expected the groups differed in their mean age at the onset of dieting -- EO: 13.1 (±2.0) years vs TO: 18.1 (±1.1) years -- and their mean age at onset of bingeing -- EO: 13.7 (±1.6) years vs TO: 18.8 (±1.0) years. Mean age at menarche was identical in both groups -- EO: 12.7 (± 1.3) years vs TO: 12.7 (± 1.6) years. Only six patients had a premenarchal onset of their eating disorder.

A comparison of the clinical features between the two groups is shown in Table 7.7.1. The proportion of patients in both groups who induced vomiting, abused laxatives or appetite suppressants, or had a history of anorexia nervosa was almost identical. In contrast, a history of obesity was nearly twice as common in the early onset group as in the typical onset group, although this finding did not quite reach significance.
7.4.3. Associated psychopathology

The early onset group had a significantly higher rate of deliberate self-harm (DSH) than the typical onset group (Table 7.7.1.). Deliberate self-harm usually occurred after onset. The increased level of DSH in the early onset group was not a result of a longer duration of illness. Other impulsive behaviours such as drug and alcohol abuse and stealing were not different in the two groups.

7.4.4. Family factors

These are shown in Table 7.7.2. The two groups did not differ with regard to a family history of depression, substance abuse, eating disorder or obesity. The family structure did not differ between groups. Forty to 45% of patients came from broken homes. Levels of family disturbance were high and almost identical in both groups, with the exception of inadequate parental care, which occurred significantly more often in the EO group. The two groups did not differ with regard to the frequency of cultural stress.

7.5. Discussion

The shortcomings of the present study are similar to those of the studies described in chapters 5 and 6. Moreover, the study is limited by its varying initial assessment procedure.

Contrary to hypothesis 1a the clinical features of the early onset cases resembled closely those of the typical onset cases and there was no suggestion that those with an earlier onset are more severely ill than those with a typical onset. One important trend
which failed to reach statistical significance was: Nearly twice as many early onset cases compared to typical onset cases were overweight before onset. This trend fits in with other research suggesting that higher actual body weight is associated with early onset of dieting (Hill et al., 1989; Hill et al., 1992; Wardle and Beales, 1986) and predisposes to the development of bulimia nervosa (Garner and Fairburn, 1988). Children’s attitude to overweight is extremely negative (Richardson et al., 1961; Staffieri, 1967) and girls are less accepting of overweight same-sex peers than are males (DeJong and Kleck, 1986). Overweight children may be teased and excluded from peer groups and athletic activities (Hill, 1993). Whilst overweight 9 year olds have low body esteem but, in terms of global self-esteem, are no different to their peers (Mendelson and White, 1985) by the age of 13, those with low body esteem also have global low self-esteem. In the present study patients were not asked whether they had been teased about their weight by their peers or families and, therefore, it is not known whether teasing or critical comments about weight and appearance were part of the behavioural chain leading to the youngsters’ decision to start a diet.

Hypothesis 1b, which suggested that early onset cases would have more associated psychopathology, was partially confirmed. Whilst neither substance misuse nor stealing was more prevalent in the young onset group, deliberate self harm occurred in 54% of the early onset group, i.e., considerably more commonly than in the typical onset patients. Interestingly, a recent study which compared multi-impulse bulimics with other bulimics without these features (Widerman and Pryor, 1996), found the multi-impulsive group to have a significantly earlier onset of bingeing, vomiting and
laxative abuse and thus supports the link between an early onset and increased comorbidity.

In the present study deliberate self-harm usually occurred after the onset of the eating disorder. It is well-known that deliberate self-harm often is an impulsive act that happens at a time of crisis in order to communicate distress to close others. In the majority of early onset cases, self-harm was associated with severe family problems. This is in line with a study of adolescent suicide attempters (De Wilde et al., 1992), which found them to differ from depressed adolescents who had never made a suicide attempt and non-depressed adolescents. The suicide attempters had experienced more family difficulties that began in childhood and had not stabilized in adolescence. The deliberate self-harm in the young onset group of the present study may, thus, have been a desperate attempt to signal to others within or outside the family that all was not well.

Another possibility is that depression associated with an eating disorder was a contributing factor in facilitating deliberate self-harm. Unfortunately, life-time depression was not assessed in the present sample, so this point cannot be addressed.

Those young onset patients who do show some outward disturbance, e.g., deliberate self-harm, may be more readily identified and referred for treatment, whereas the older patients who are more independent can more easily refer themselves. Thus, it is possible that the clinic population of young onset cases presented here is
unrepresentative of young onset cases at large and that the increased incidence of deliberate self-harm simply reflects referral bias.

Contrary to hypothesis 2a no differences were found between groups in terms of family history or family structure.

Parental lack of care was nearly twice as common in the early onset group as in the typical onset group. What is the link between parental lack of care and the early development of bulimia nervosa? From a psychodynamic point of view, it has been argued that overeating may represent an attempt to avoid feelings of emptiness and low mood associated with maternal unavailability (Johnson and Connors, 1987). At a more practical level, parents who are emotionally and physically unavailable to their teenage daughter may not provide her with regular meals. Once rigid dieting or an eating disorder have started, these parents may fail to notice it, or if they do notice it, may fail to act appropriately.

7.6. Summary and Conclusions

(1) The clinical features of the early onset cases resembled closely those of the typical onset cases.

(2) Nearly twice as many early onset cases compared to typical onset cases were overweight before onset.

(3) Deliberate self-harm occurred in 54% of the early onset group, i.e., considerably more commonly than in the typical onset patients. Usually it occurred after the onset of the eating disorder.
(4) There were no differences between groups in terms of a family history of depression, substance abuse or obesity.

(5) Parental lack of care was nearly twice as common in the early onset group as in the typical onset group, with no other differences in quality of childhood care.

(6) There was no difference between groups in terms of acculturation stress.

These findings do partially support the notion of early onset bulimia nervosa cases having a higher loading on some of the putative vulnerability factors.
### 7.7. Tables

<table>
<thead>
<tr>
<th>Clinical Features</th>
<th>Early Onset</th>
<th>Typical Onset</th>
<th>Odds Ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Self-Induced Vomiting</strong></td>
<td>33/40 (83%)</td>
<td>35/40 (88%)</td>
<td>0.7 (0.2-2.3)</td>
</tr>
<tr>
<td><strong>Laxatives</strong></td>
<td>19/40 (48%)</td>
<td>23/40 (58%)</td>
<td>0.7 (0.3-1.6)</td>
</tr>
<tr>
<td><strong>Appetite suppressants</strong></td>
<td>6/40 (15%)</td>
<td>9/40 (23%)</td>
<td>0.6 (0.2-1.9)</td>
</tr>
<tr>
<td><strong>History of Anorexia Nervosa</strong></td>
<td>10/40 (25%)</td>
<td>10/40 (25%)</td>
<td>1.0 (0.4-2.8)</td>
</tr>
<tr>
<td><strong>History of Obesity</strong></td>
<td>14/38* (37%)</td>
<td>8/40 (20%)</td>
<td>2.3 (0.8-6.5)</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Associated Psychopathology</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Deliberate Self-Harm</strong></td>
<td>21/39* (54%)</td>
<td>12/40 (30%)</td>
<td>2.7 (1.1-6.7)</td>
</tr>
<tr>
<td><strong>Substance Abuse</strong></td>
<td>13/40 (33%)</td>
<td>11/40 (28%)</td>
<td>1.3 (0.5-3.3)</td>
</tr>
<tr>
<td><strong>Stealing</strong></td>
<td>12/37* (32%)</td>
<td>11/40 (28%)</td>
<td>1.3 (0.5-3.4)</td>
</tr>
</tbody>
</table>

**Table 7.7.1:** Clinical features and associated psychopathology. *a:* no information available in two patients; *b:* no information available in one patient; *c:* no information available in three patients.
<table>
<thead>
<tr>
<th>Family History of:</th>
<th>Early Onset</th>
<th>Typical Onset</th>
<th>Odds Ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Depression</td>
<td>15/39 (39%)</td>
<td>11/37 (30%)</td>
<td>1.5 (0.6-3.8)</td>
</tr>
<tr>
<td>Substance Abuse</td>
<td>7/39 (18%)</td>
<td>10/37 (27%)</td>
<td>0.6 (0.2-1.8)</td>
</tr>
<tr>
<td>Eating Disorders</td>
<td>5/39 (13%)</td>
<td>2/35 (6%)</td>
<td>2.4 (0.4-13.4)</td>
</tr>
<tr>
<td>Obesity</td>
<td>13/39 (33%)</td>
<td>11/35 (31%)</td>
<td>1.1 (0.4-2.9)</td>
</tr>
<tr>
<td>Family Structure</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Multiple Family Arrangements</td>
<td>16/40 (40%)</td>
<td>18/40 (45%)</td>
<td>0.8 (0.3-2.0)</td>
</tr>
<tr>
<td>Adopted</td>
<td>1/40 (3%)</td>
<td>3/40 (8%)</td>
<td>0.3 (0.03-3.2)</td>
</tr>
<tr>
<td>Institutional Care or</td>
<td>5/40 (13%)</td>
<td>3/40 (8%)</td>
<td>1.8 (0.4-7.9)</td>
</tr>
<tr>
<td>Boarding School</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Family Disturbance</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Discord</td>
<td>26/40 (65%)</td>
<td>26/40 (65%)</td>
<td>1.0 (0.4-2.5)</td>
</tr>
<tr>
<td>Lack of Care</td>
<td>20/40 (50%)</td>
<td>11/40 (28%)</td>
<td>2.6 (1.04-6.7)</td>
</tr>
<tr>
<td>Intrafamilial Violence*</td>
<td>10/40 (25%)</td>
<td>9/40 (23%)</td>
<td>1.2 (0.4-3.2)</td>
</tr>
<tr>
<td>Childhood Sexual Abuse</td>
<td>6/40 (15%)</td>
<td>10/38 (26%)</td>
<td>0.5 (0.2-1.5)</td>
</tr>
<tr>
<td>Cultural Stress</td>
<td>13/40 (33%)</td>
<td>11/40 (28%)</td>
<td>1.3 (0.5-3.3)</td>
</tr>
</tbody>
</table>

**Table 7.7.2: Family factors.** a: no information on one patient available because she was adopted; b: no information on three patients available because they were adopted; c: no information available on five patients (three adopted, in two additional cases patients had not been asked); d: in two patients no information was available; *: this is a composite rating of violence against the patient or close other family member.
Chapter 8: Puberty, Psychosexual Development and Childhood

Sexual Abuse: How Are They Related in Eating Disorder Patients?

8.1. Aims and Hypotheses

Aim 1: To assess whether (a) psychosexual development in anorexia nervosa is delayed compared with normal controls and bulimic patients and (b) whether the delay starts before or after onset.

Hypothesis 1a: Anorexia nervosa patients will show a delay in psychosexual development compared with bulimia nervosa patients and normal controls.

Hypothesis 1b: The delay in psychosexual development is secondary to weight loss and thus starts after onset.

Aim 2: To assess whether bulimia nervosa patients, too, show a delay in psychosexual functioning compared with normal controls.

Hypothesis 2: Bulimia nervosa patients will be delayed in their psychosexual development compared with normal controls but less so than anorexia nervosa patients.

Aim 3: To assess whether traumatic early sexual experiences are associated with a delay in psychosexual functioning in eating disorder patients.

Hypothesis 3: Eating disorder patients who have experienced childhood sexual abuse will be delayed in their psychosexual development compared with eating disorder patients who have not experienced childhood sexual abuse.
8.2. Introduction

The relationship between eating disorders and sexuality has been a matter of interest and speculation since the earliest descriptions of anorexia nervosa. In the most famous case of medieval "holy anorexia" i.e. that of St. Catherine of Siena, suppression of her own sexuality was at least initially a powerful motivating factor in her self-starvation (Rampling, 1985). Both, Lasègue (1873) and Janet (1903) felt that difficulties with heterosexual relationships were aetiologically significant in anorexia nervosa and Janet coined the term 'pudicity' to describe the anorexics' shame and disgust with sexual matters. According to Meyer (1961) one of the central psychological factors in anorexia nervosa is the refusal to grow up and accept the role of a sexually and socially mature woman. Echos of this theme reverberate through the work of Crisp (1980) and some feminists (Boskind-Lodahl, 1976). There is some empirical support for this view. Sexual behaviour involving a partner was much rarer in anorexia nervosa patients than in a control group (Vaz-Leal & Salcedo-Salcedo, 1992). In a series of 31 anorexics 13 patients saw sexual problems as a major precipitant of their illness (Beumont et al., 1981).

An alternative view is that the psychosexual problems of anorexia nervosa are largely a secondary effect, a consequence of the biological regression of starvation, as documented in the Minnesota experiment (Keys et al., 1950). The observation that the early stages of sexual development are normal (Raboch and Faltus, 1991; Tuiten et al., 1992) or even accelerated (Raboch, 1986) support this view. Anorexic patients have a wide spectrum of sexual knowledge, attitudes and behaviour, but report a
decrease in sexual interest and enjoyment following weight loss (Beumont et al., 1981; Tuiten et al., 1992).

Sexual disgust (Buvat-Herbaut et al. 1983; King, 1963) or negative attitudes to sexuality (Leon et al., 1985; Tuiten et al., 1992) have been noted to be more common in anorexia nervosa patients than in controls. Buvat-Herbaut (1983) found sexual disgust to be more pronounced after weight gain, whereas Leon et al. (1985) found the opposite, i.e. an improvement in the anorexics’ attitudes to sexuality with increased weight.

There are thus considerable inconsistencies in the literature regarding the significance of psychosexual disturbance in eating disorders. Scott (1987) concluded that "there is little evidence for many widely held beliefs concerning psychosexual factors within the eating-disordered patient". Some of the inconsistencies between studies may simply be the result of patient selection. For example, Tuiten et al. (1992) deliberately excluded anorexic patients who had been sexually abused or who were currently using oral contraceptives from their survey. Some studies use out-patients, others in-patients. Whilst in-patients are more emaciated and therefore could be expected to be less interested in sex, they may tone down negative attitudes in order to please their therapists or to be discharged sooner (Scott, 1987).

Bulimics are generally thought to be more sexually active than anorexics (Russell, 1979; Wiederman et al., 1996). However, in a small study (Rothschild et al., 1991) hospitalised bulimics showed similar degrees of impairment to hospitalised anorexics
in terms of sexual function and satisfaction, and Lacey (1992) found that bulimic patients were less likely to marry than their sisters or other women from the same catchment area.

Childhood sexual abuse has been thought to contribute to the sexual aversion found in eating disordered patients (Zerbe, 1992). However, to date only one study has examined the links between psychosexual development, sexual behaviour and childhood sexual abuse (De Groot et al., 1992).

8.3. Subjects and Method
8.3.1. Sample
A consecutive series of 168 female outpatients who presented to the Eating Disorder Unit at the Maudsley Hospital took part in the study. As in the study in chapter 5 patients fulfilled DSM-III-R criteria for anorexia nervosa or bulimia nervosa and were subdivided into those with restricting anorexia nervosa (RAN; n=44), bulimic anorexia nervosa (BAN; n=26), bulimic patients with a history of anorexia nervosa (BN/HistAN; n=29) and bulimic patients without a history of anorexia nervosa (BN; n=69). Onset was defined as either weight loss of 15 % or more, amenorrhoea, the start of objective binges, the start of self-induced vomiting or laxative abuse, whichever of these occurred first. Patients with eating disorder not otherwise specified were not included. The control population consisted of 44 female polytechnic students in their early twenties who were approached in the college canteen during breaktime. A further seven students did not wish to participate. Students were not asked about
their father’s occupation and were not screened for the presence or absence of an eating disorder.

8.3.2. Procedure

The Pubertal Development Questionnaire: The questionnaire was developed for the purpose of the study. It contains, amongst others, factual questions about when the various stages of puberty occurred (see appendix 8.9.). Due to time constraints, this questionnaire was given to eating disorder patients only.

The Psychosexual Development Questionnaire: This questionnaire was also developed for this project. It contains 16 factual questions about age of first date, kiss, first sexual intercourse and masturbation, number of partners and homosexual relationships. Attitudes towards sex, masturbation, marriage, children and pregnancy were assessed using visual analogue scales (see appendix 8.9.). This questionnaire was given to eating disorder patients and the control population.

Childhood sexual abuse (CSA): This was defined according to the criteria of Browne and Finkelhor (1986) as outlined in chapter 5. Childhood sexual abuse was only enquired about in the patients; in the control population no attempt was made to assess CSA.

The patients were sent the questionnaires before their first appointment. Psychosexual details were also discussed at the time of assessment. Three patients (2%) did not fill in the pubertal development questionnaire, ten (7%) did not fill in the psychosexual
development questionnaire and two (1.4%) failed to fill in the sexual life event questionnaire. For the controls a 'ballot box' was provided to ensure anonymity of the questionnaire responses.

Statistical analysis: Data were analyzed using SPSS/PC+ 4.0 (Norusis, 1990). Analysis of variance was used to compare group means for normally distributed data. Kruskal-Wallis analysis of variance and Mann-Whitney tests were used for non-parametric data. Chi-square statistics were used for categorical data and Lee-Desu statistics (Lee & Desu, 1972) were used to compare survival curves.

8.4. Results

8.4.1. Sample characteristics

The groups did not differ with respect to age (RAN: 24.8 (±6.3) years; BAN: 23 (±4.3) years; BN/HistAN: 23.1 (±3.2) years; BN: 24.6 (±5.6) years) or age at onset (RAN: 18.6 (±5.7) years; BAN: 17.9 (±3.4) years, BN/HistAN 16.9 (±3.1) years; BN: 17.9 (±3.1) years).

8.4.2. Pubertal development

Figures 8.8.1. and 8.8.2. give survival curves and table 8.7.1. the median survival times of breast development and age of menarche in the four eating disorder groups. The survival curves were compared using the Lee-Desu statistic. The four groups were not different on breast development, but verged on being different with respect to age at menarche. Four RAN patients, three BN/HistAN patients and two BN patients had a pre-menarchal onset. A high percentage of patients in all groups (74%
to 89%) had prior knowledge of menstruation (before their periods started), with no differences between the groups.

8.4.3. Psychosexual development

Figures 8.8.3. to 8.8.7. show survival curves and table 8.7.1. shows the median survival times of first date, first kiss, first genital fondling, first sexual intercourse and first masturbation for eating disorder patients and the control population. Again the Lee-Desu statistic was used for a comparison of the survival curves. The groups were different in all of these variables with the control population more advanced and the restricting anorexia nervosa group the most delayed with the exception of age at first date, where there was only a trend in the same direction.

In order to assess whether there was a delay in sexual activity of eating disorder patients over and above that produced by the effects of their illness, a survival analysis was carried out comparing only those patients who had their first sexual intercourse before onset (RAN: n = 13; BAN: n = 14; BN/ HistAN: n = 11; BN: n = 30) with the controls (Figure 8.7.8.). There was a trend for the group who later developed restricting anorexia nervosa to be delayed compared to the other groups who were all very similar to each other (Median survival age: RAN: 19.3; BAN: 17; BN/ HistAN: 17.3; BN: 17.5; Controls: 17.2; p=0.07).

The median number of sexual partners differed significantly between groups (RAN: 1 (range: 0-6); BAN: 2 (range: 0-20); BN/ HistAN: 4 (range: 0-12); BN: 4 (range: 0-98); controls: 5 (range: 1-56); Kruskal-Wallis Analysis of Variance; Chi-Square:
30.7; p < 0.0001), with all eating disorder groups having significantly fewer sexual partners than the controls (RAN vs controls: Mann-Whitney U test; p < 0.00001; BAN vs controls: Mann-Whitney U test; p = 0.02; BN/HisTAN vs controls: Mann-Whitney U test; p = 0.03; BN vs controls: Mann-Whitney U test; p = 0.04) and restricters also differing significantly from all other eating disorder groups (RAN vs BAN: Mann-Whitney U test; p = 0.04; RAN vs BN/HisTAN; Mann-Whitney U test: p = 0.0008; RAN vs BN: Mann-Whitney U test; p < 0.0001), but no other difference between eating disorder groups.

8.4.4. Attitudes to sex, masturbation, marriage, children and pregnancy

The results of the visual analogue scales are shown in table 8.7.2. All four eating disorder groups found sexual relationships, either current or fantasized, less positive than controls. Restricting anorexics were the least positive of all and also differed significantly from BAN and BN patients. There was a trend for restricting anorexics to be more positive about the idea of getting married than the other groups. There were no differences between groups on attitudes to masturbation, children or pregnancy.

8.4.5. Childhood sexual abuse and its relationship with puberty, sexual behaviour and attitudes

Childhood sexual abuse occurred in 22 to 31% of patients (table 8.7.3.). Repeated abuse was common as was the use of force.

In order to see whether patients who had been sexually abused differed from those who had not been sexually abused in terms of their pubertal and sexual development,
survival analyses were carried out for the total patient group comparing age at breast development, menarche, first date, kiss, masturbation, genital fondling and sexual intercourse in non-abused versus abused patients. However, no differences were found between abused and non-abused groups. There were also no differences between abused and non-abused groups in the number of sexual partners (figures not included).

Similarly, abused and non-abused patients were compared with regards to their attitudes to sex, masturbation, marriage, pregnancy and having children (see table 8.7.4.). Abused patients were significantly more positive about masturbation than those who had not been abused. There was a trend for abused patients to be more negative about pregnancy and having children.

8.5. Discussion

There are several problems with this study. The questionnaires had not been validated before use. Ideally, the visual analogue scales in the questionnaires should have been keyed at random to avoid biased responses (with positive or negative statements appearing on the left or on the right side of the scale equally frequently). The polytechnic students whilst similar in age to the eating disorder patients may not have been comparable to the eating disorder patients in terms of their social class distribution (occupation of father). Social class may, however, affect sexual attitudes and behaviour. A more appropriate control sample would have been a group of young women obtained from a general practitioner's list, matched for social class to the eating disorder group. It would have been desirable to give the pubertal development questionnaire and the sexual life events questionnaire to the control group, too. This
was not feasible due to time constraints. However, there are data available on normal pubertal development (Tanner, 1989). It is probable that the control group is skewed towards those with a more positive attitude to sexuality. Thirteen percent of the controls elected not to fill in the sexual development questionnaire as compared to only 7% of patients. The control group was not screened for the presence of an eating disorder. Given that the point prevalence of eating disorders in young female populations is about 1-2% this is unlikely to have led to any serious distortion of our findings. Since embarking on this study the results of large surveys of sexual behaviour in young women in the community have become available (ACSF, 1992; Johnson et al., 1992) and these are of great interest for comparison, as is discussed below. A further methodological point is common to all sexual surveys (Bancroft, 1992): "To what extent do our data reflect the actual behaviour of the subjects rather than a wish to be seen to conform to the acceptable norms for their particular age and socioeconomic peer groups?" In other words how reliable are the data? As mentioned in the previous chapters, the personality characteristics of the different eating disorder groups may also have distorted the findings. Anorexia nervosa patients may have played down any sexual experiences, whereas bulimics often fit into the histrionic personality spectrum and may be prone to exaggerate their experiences. Despite these caveats the results of the study allow some interesting conclusions. There were no differences between eating disorder groups in the early stages of their pubertal development (breast development). This suggests that the differences in sexual behaviour between the groups cannot simply be interpreted in terms of differences in physical development. There was a trend for the restricters to fall
behind in terms of age at menarche. (The mean age of menarche in the UK is 12.9 years (Tanner, 1989)). This can be explained in terms of the few patients in this group with primary amenorrhoea and an early onset of their disorder who never menstruated in their lives or only menstruated in their twenties. The findings of the present study are thus in contrast with those of Crisp (1970), who found that patients with anorexia nervosa reported an earlier age of menarche than a control group of women with other psychoneurotic or affective disorders.

In terms of the stages of sexual development it is interesting to note that the only variable in which the groups did not differ was age at first date, which is the only milestone not involving any physical contact. This is in line with the results of Katzman and Wolchik (1984) who found bulimics and normal controls to have similar social competency in dating. However, all other milestones showed differences between groups, the greater the contact the greater the differences, especially between restricting anorexics and the other groups. These findings concur with those of Vaz-Leal and Salcedo-Salcedo (1992) and Wiederman et al. (1996), who also noted that anorexics avoided sexual activities involving a partner before onset. At first sight our findings seem to conflict with those of Tuiten et al. (1992), who found that anorexics said that they had a partner as often as controls of similar age. However, their question did not distinguish between a sexual partner and more casual dating and it is possible that the question tapped into the latter.

The life-time number of partners was reduced in all eating disorder patients with restricting anorexics showing the smallest number of partners. A recent survey of
sexual behaviour in Britain found a mean number of life time sexual partners of 4.3 for women aged 25-34 years (Johnson et al., 1992), which is between the figures found in the present study for the BN/HistAN and BN groups (median number of life time partners = 4) and the control group (median number of life time partners = 5). A recent French survey of sexual behaviour found an age at first intercourse for women aged 18-24 of 17.1 years (ACSF, 1992), which compares well with a mean age of first intercourse of 16.9 years in the control group of the present study. (To calculate a mean age at first sexual intercourse for the eating disorder groups would not be meaningful, as many patients had never had sexual intercourse). Attitudes to sexual activities were less disturbed than the behavioural aspects of sexual development. Although restricting anorexics were less positive about sexuality, the median attitude to sexuality was 6 in this group which implies a mildly positive attitude.

In line with a previous study by DeGroot et al. (1992), the present study did not find any difference between sexually abused and non-abused eating disorder patients in terms of sexual activity or attitudes. Thus the possibility that the psychosexual inhibition in anorexia nervosa is due to unresolved conflict over such childhood trauma is not supported. However, the abused patients' more positive attitude to masturbation suggests that this is seen as an unthreatening sexual activity by this group. Clinically one sees some sexually abused women who respond to the abuse by being sexually precocious, whereas others have delayed or no sexual activity. It is possible that the heterogeneity of the abused group in terms of their sexual behaviour is hidden in the apparent similarity between abused and non-abuse patients.
What accounts for the abnormal sexual function in eating disorder patients? In anorexia nervosa, biological regression after onset has some part to play, but it is not the sole explanation for two reasons: firstly, in restricting anorexia nervosa even those who had their first sexual intercourse before onset did so later than the other groups. (This was not the result of these patients being older). Secondly, the bulimic group who were at normal weight also showed a delay/decrease in sexual activity largely secondary to the disorder. Different mechanisms may thus be responsible for the delay in sexual activity in RAN and BN patients. In bulimia nervosa, one can only speculate about the importance of factors like low self-esteem and body-image disturbance in causing psychosexual problems. Patients with other psychiatric disorders, too, show problems with psychosexual functioning (Raboch, 1986) and it would have been useful to have a comparison group of women with depression to see whether bulimics differ from other psychiatric patients in this respect.

In anorexia nervosa, an interesting possibility is that the psychosexual immaturity is the result of an underlying biological liability to the condition which may be associated with a disturbance in 5-HT function (Treasure and Campbell, 1994). There is some support for a familial disturbance in psychosexual functioning in anorexia nervosa. For example, mothers of patients with anorexia nervosa had significantly higher lifetime psychosexual dysfunction (Halmi et al., 1991). Additional support comes from a study of twins discordant for anorexia nervosa. Monozygotic twins without anorexia nervosa have a delay in sexual activity equivalent to that of their sister with anorexia nervosa whereas sexual activity is normal in timing in the unaffected dizygotic twins (Treasure and Holland, 1993). Thus, the findings of this
study suggest that far from being 'red herrings' (Scott, 1987) the observations made hundred years ago by Janet and Lasègue may have considerable validity.

8.6. Summary and Conclusions

(1) The four eating disorder groups were not different in terms of their pubertal development.

(2) Patients and controls did not differ from each other in age at first date, but they did differ in age at first kiss, first genital fondling, first sexual intercourse and first masturbation with control subjects being the most advanced, restricters being the most delayed and the other groups being intermediate.

(3) Similarly, the median number of sexual partners differed significantly between groups.

(4) Eating disorder patients, in particular RAN patients, were less positive about sexual relationships than controls, but did not differ from controls in attitudes to masturbation, marriage, children or pregnancy.

(5) 22-31% of eating disorder patients had been sexually abused during childhood. A history of abuse affected attitudes to masturbation, but did not appear to affect sexual development.

These findings suggest that psychosexual development is delayed in patients with eating disorders, and in restricting anorexia nervosa this may occur even before onset. Attitudes towards sexuality are less disturbed. Childhood sexual abuse does not account for the delay in psychosexual development.
8.7. Tables

<table>
<thead>
<tr>
<th></th>
<th>RAN</th>
<th>BAN</th>
<th>BN/HistAN</th>
<th>BN</th>
<th>Controls</th>
<th>p'</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>PUBERTAL DEVELOPMENT</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Breast Development</td>
<td>12.8</td>
<td>12.8</td>
<td>12.4</td>
<td>12.5</td>
<td>--</td>
<td>0.43</td>
</tr>
<tr>
<td>Age of Menarche</td>
<td>13.9</td>
<td>13.4</td>
<td>13.3</td>
<td>13.3</td>
<td>--</td>
<td>&lt; 0.06</td>
</tr>
<tr>
<td><strong>PSYCHOSEXUAL DEVELOPMENT</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>First Date</td>
<td>15.3</td>
<td>14.3</td>
<td>16.3</td>
<td>15.3</td>
<td>14.4</td>
<td>0.06</td>
</tr>
<tr>
<td>First Kiss</td>
<td>15.6&lt;sup&gt;c&lt;/sup&gt;</td>
<td>16.5&lt;sup&gt;c&lt;/sup&gt;</td>
<td>14.1&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>14.9</td>
<td>14.4&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>0.01</td>
</tr>
<tr>
<td>First Genital Fondling</td>
<td>19.1&lt;sup&gt;cd&lt;/sup&gt;</td>
<td>17.3&lt;sup&gt;c&lt;/sup&gt;</td>
<td>15.7&lt;sup&gt;b&lt;/sup&gt;</td>
<td>16.5&lt;sup&gt;c&lt;/sup&gt;</td>
<td>15.8&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>0.0001</td>
</tr>
<tr>
<td>First Intercourse</td>
<td>23.7&lt;sup&gt;abc&lt;/sup&gt;</td>
<td>17.9&lt;sup&gt;c&lt;/sup&gt;</td>
<td>18.8&lt;sup&gt;de&lt;/sup&gt;</td>
<td>18.5&lt;sup&gt;e&lt;/sup&gt;</td>
<td>17.2&lt;sup&gt;ab&lt;/sup&gt;</td>
<td>&lt; 0.00001</td>
</tr>
<tr>
<td>First Masturbation</td>
<td>25 +&lt;sup&gt;c&lt;/sup&gt;</td>
<td>16.5&lt;sup&gt;c&lt;/sup&gt;</td>
<td>24.3&lt;sup&gt;de&lt;/sup&gt;</td>
<td>14.7&lt;sup&gt;c&lt;/sup&gt;</td>
<td>14.3&lt;sup&gt;de&lt;/sup&gt;</td>
<td>0.001</td>
</tr>
</tbody>
</table>

**Table 8.7.1: Pubertal and psychosexual development.** Median survival times are given. If overall comparison ('Lee-Desu Statistic (overall comparison)) was significant, a pairwise Lee-Desu comparison was conducted.  
<sup>a</sup>: this group differs from RAN group at p < 0.05. <sup>b</sup>: this group differs from BAN group at p < 0.05. <sup>c</sup>: this group differs from BN/HistAN group at p < 0.05. <sup>d</sup>: this group differs from BN at p < 0.05. <sup>e</sup>: this group differs from controls at p < 0.05.
<table>
<thead>
<tr>
<th></th>
<th>RAN</th>
<th>BAN</th>
<th>BN/HistAN</th>
<th>BN</th>
<th>Controls</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Attitude to Sex</strong></td>
<td>6(^{a,b,c})</td>
<td>7.5(^{a,c})</td>
<td>6.6(^{c})</td>
<td>7.5(^{a,c})</td>
<td>10.0(^{b,c,d})</td>
<td>&lt; 0.0001</td>
</tr>
<tr>
<td><strong>Attitudes to Masturbation</strong></td>
<td>4.5</td>
<td>4.5</td>
<td>5</td>
<td>5.7</td>
<td>5.5</td>
<td>0.17</td>
</tr>
<tr>
<td><strong>Attitudes to Marriage</strong></td>
<td>10</td>
<td>9.5</td>
<td>7.6</td>
<td>7.7</td>
<td>7.5</td>
<td>0.08</td>
</tr>
<tr>
<td><strong>Attitudes to Children</strong></td>
<td>9.6</td>
<td>9</td>
<td>7.2</td>
<td>7.4</td>
<td>8.5</td>
<td>0.37</td>
</tr>
<tr>
<td><strong>Attitudes to Pregnancy</strong></td>
<td>5.4</td>
<td>7.5</td>
<td>5.1</td>
<td>4.7</td>
<td>5.0</td>
<td>0.21</td>
</tr>
</tbody>
</table>

Table 8.7.2: Median scores on visual analogue scales on attitudes to sex, masturbation, marriage, children and pregnancy of eating disorder patients and controls. Ranges were all between 0 to 10 or 1 to 10. A high score denotes a positive attitude. \(^a\): this group differs from RAN group at \(p < 0.05\); Mann Whitney U-test. \(^b\): this group differs from BAN group at \(p < 0.05\); Mann Whitney U-test. \(^c\): this group differs from BN/HistAN group at \(p < 0.05\); Mann Whitney U-test. \(^d\): this group differs from BN at \(p < 0.05\); Mann Whitney U-test. \(^e\): this group differs from controls at \(p < 0.05\), Mann Whitney U-test.
<table>
<thead>
<tr>
<th></th>
<th>RAN</th>
<th>BAN</th>
<th>BN/HisTAN</th>
<th>BN</th>
</tr>
</thead>
<tbody>
<tr>
<td>CSA</td>
<td>10/44 (22%)</td>
<td>8/26 (31%)</td>
<td>9/29 (31%)</td>
<td>17/67 (25%)</td>
</tr>
<tr>
<td>Use of Force</td>
<td>6/10 (60%)</td>
<td>3/7 (43%)</td>
<td>3/9 (33%)</td>
<td>7/16 (43%)</td>
</tr>
<tr>
<td>Repeated CSA</td>
<td>8/10 (80%)</td>
<td>6/8 (75%)</td>
<td>6/9 (66%)</td>
<td>14/17 (82%)</td>
</tr>
</tbody>
</table>

Table 8.7.3: Childhood sexual abuse (CSA) in the eating disorder groups. No information was available about "use of force" in one case of BAN and BN respectively.
<table>
<thead>
<tr>
<th>Attitudes to Sex</th>
<th>Not Abused (n=124) Median (range)</th>
<th>Abused (n=44) Median (range)</th>
<th>p'</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>7.9 (1 - 10)</td>
<td>7.9 (0 - 10)</td>
<td>0.18</td>
</tr>
<tr>
<td>Attitudes to Masturbation</td>
<td>5 (0 - 10)</td>
<td>6.5 (1.9 - 10)</td>
<td>0.01</td>
</tr>
<tr>
<td>Attitudes to Marriage</td>
<td>8.8 (0 - 10)</td>
<td>7.0 (1 - 10)</td>
<td>0.36</td>
</tr>
<tr>
<td>Attitudes to Children</td>
<td>8.3 (0 - 10)</td>
<td>7.0 (1 - 10)</td>
<td>0.13</td>
</tr>
<tr>
<td>Attitudes to Pregnancy</td>
<td>5.0 (0 - 10)</td>
<td>4.0 (0 - 10)</td>
<td>0.11</td>
</tr>
</tbody>
</table>

Table 8.7.4: Attitudinal differences between abused and non-abused patients. Scores are between 0 and 10 and a high score denotes a positive attitude. *: Mann-Whitney U-test.
Figure 8.8.1: Survival curve of breast development in the four eating disorder groups

Figure 8.8.2: Survival curve of menarche in the four eating disorder groups
Fig. 8.8.3.: Survival curve of age at first date in eating disorder and control groups

Fig. 8.8.4.: Survival curve of age at first kiss in eating disorder and control groups
Fig. 8.8.5.: Survival curve of age at first genital fondling in eating disorder and control groups

Fig. 8.8.6.: Survival curve of age at first sexual intercourse (coitarche) in eating disorder and control groups
Fig. 8.8.7: Survival curve of age at first masturbation in eating disorder and control groups.

Fig. 8.8.8: Survival curves of age at first sexual intercourse for those patients with coitarche before onset.

RAN: n = 13; BAN: n = 14; BN/HiSTAN: n = 11; BN: n = 30
8.9. Appendix

**Pubertal Development Questionnaire**

Some people find that the changes that occur during development of puberty are disturbing or upsetting, whereas others find the changes exciting.

1. What age were you and what did you feel about it when:

<table>
<thead>
<tr>
<th>Event</th>
<th>Age</th>
<th>Attitude</th>
</tr>
</thead>
<tbody>
<tr>
<td>Your breasts began to grow</td>
<td></td>
<td>positive/negative</td>
</tr>
<tr>
<td>Your shape changed</td>
<td></td>
<td>positive/negative</td>
</tr>
<tr>
<td>Hair developed under your arms</td>
<td></td>
<td>positive/negative</td>
</tr>
<tr>
<td>Hair developed around your private parts</td>
<td></td>
<td>positive/negative</td>
</tr>
<tr>
<td>Your periods began</td>
<td></td>
<td>positive/negative</td>
</tr>
</tbody>
</table>

2. Did you know enough about these changes? YES/NO

3. Compared to your friends did you develop:
   - ahead
   - average
   - behind

(Please circle appropriate answer)

4. If your periods have ever stopped, how long in months did you have periods before they stopped? ____

5. Were your periods regular? e.g. every 26-34 days YES/NO

   If not, how many days ____

6. How old were you when your periods stopped? ____

7. How old were you when they started again? ____

8. Have you had any treatment for your periods e.g. hormones/
contraceptive pill? Yes/No

When? ____________

What? ____________

How long? ____________

What effect did it have? ____________

Did you have any unpleasant symptoms associated with your periods? (Please write any in spaces below)

1. __________________ mild/severe

2. __________________ mild/severe

3. __________________ mild/severe

4. __________________ mild/severe

5. __________________ mild/severe

10. If you have periods now, please mark the strength of your feelings on this line

__________________________________________________________________________

wish they would go pleased to have them

OR if you have NO periods, please mark the strength of your feelings on this line

__________________________________________________________________________

wish never to have them want them to return

If your periods were not related to your weight, how would you score?

__________________________________________________________________________

wish never to have them want them to return

11. Do you worry that your illness may have affected your fertility? Please mark how you feel.

__________________________________________________________________________

No worries very concerned

12. Do you worry that your illness has damaged you in any other way? (Please indicate your concern in the spaces below)
1. __________________________

slight ______________________ very
worry ______________________ worried

2. __________________________

slight ______________________ very
worry ______________________ worried

3. __________________________

slight ______________________ very
worry ______________________ worried

4. __________________________

slight ______________________ very
worry ______________________ worried

5. __________________________

slight ______________________ very
worry ______________________ worried

(In the original questionnaire the visual analogue scales were 10 cm long.)
Psychosexual Development Questionnaire

I am afraid that some of these questions will be irrelevant for you and there will be some that are difficult to answer. Please skip those that do not apply to you (put N/A) and make a best guess at the others. Please also feel free to write down any comments you want to make.

1. What age were you when you first had a date with a boyfriend? ___

2. How old were you when you first kissed a boyfriend? ___

3. How old were you when you were first fondled (on breasts or private parts/genitals by a boyfriend)? ___

4. How old were you when you first had sexual intercourse with a boyfriend? ___

5. How old were you when you had sexual intercourse with a different boyfriend? ___

6. How many boyfriends have you been physically intimate with? ___

7. Have you ever been physically intimate with a girlfriend? YES/NO

If YES, please answer the questions below.

If NO, go to question 8

   How old were you when you kissed a girlfriend intimately? ___

   How old were you when you were fondled on the breasts and genitals by a girlfriend? ___

   Please record the number of girlfriends with whom you have been physically intimate ___

8. In the past 6 months have you had a sexual relationship? YES/NO

Please indicate on this line your feelings about sexual relationships (either current or in fantasy)
disgusting very pleasurable

9. How old were you when you began to masturbate?

Please indicate on this line your feelings about masturbation

disgusting very pleasurable

10. If you are married, what age were you when you married?

What are your feelings about marriage?

something something you to be want to aim to be avoided for

11. If you have children:
How old were you when you had your first?

How much did you weigh at conception?

How much did the baby weigh?

How old were you when you had your second?

How much did you weigh at conception?

How much did the baby weigh?

12. What are your feelings about having children?

will never look forward to want them having them

13. What are your feelings about pregnancy?

unpleasant pleasant

(In the original questionnaire the visual analogue scales were 10 cm long.)
Chapter 9: The Role of Stressful Life Events and Difficulties before Onset: Is There A Specific Trauma Precipitating Anorexia Nervosa?

9.1. Aims and Hypotheses

Aim 1: To investigate the role of life events and difficulties in the onset of anorexia nervosa and bulimia nervosa.

Hypothesis 1: Bulimia nervosa patients will have had more life stresses before onset than patients with anorexia nervosa and community controls.

Aim 2: To evaluate the role of pudicity events and difficulties in the onset of eating disorders.

Hypothesis 2: Anorexia nervosa patients will have had more pudicity experiences before onset than bulimia nervosa patients and community controls.

9.2. Introduction

As outlined in chapter 4 stressful life events and difficulties have been thought to be implicated in triggering the onset of anorexia nervosa and bulimia nervosa. However, the methodology of most studies has serious drawbacks. The study presented here attempts to overcome some of the problems of previous research in this area by using the LEDS to assess life stresses and by carefully documenting onset.

Whilst the existing literature suggests that a broad range of stressors is implicated in the onset of eating disorders, there is some evidence that sexual stressors may be particularly important (Beumont et al., 1981). Weiner and Stephens (1996) recorded lifetime weight changes in eating disorder patients and found marked weight
fluctuations in response to a variety of sexual life events. The present study tests the
notion that difficulties with sexuality or maturity fears are implicated in the onset of
eating disorders. Based on the ideas of Pierre Janet (1903), who coined the term
'pudicity' to denote the anorexic's shame and disgust with sexuality, a new dimension
to life events and difficulties - 'pudicity' - was developed over the course of the study
(described in detail below).

9.3. Subjects and Method

9.3.1. Sample

A consecutive series of new patients were studied. Patients were diagnosed according
to DSM-III-R (APA, 1987) criteria. Their diagnosis at the onset of the disorder rather
than at presentation was used to group them into those with anorexia nervosa (AN;
n = 72) and those with bulimia nervosa (BN; n = 29). No patient refused to be
interviewed, but there were five further patients in whom due to time constraints the
interview was not completed. Patients with atypical eating disorders were excluded.
Apart from two AN patients all others were female.

Data on life events and difficulties of patients were compared with those of women in
the community from the Camberwell cohort of Brown and Harris (1978). This cohort
included data on 28 women of comparable age and social class background as the
patients in the present study. This subgroup of the Camberwell cohort did not seem an
appropriate comparison group for the pudicity data, as these women were interviewed
in the late 1960s and early 1970s and it seems likely that women's attitudes to
sexuality would have changed markedly by the early 1990s. Pudicity data of the eating
disorder patients were therefore compared with those of a group of 75 young women between ages 15 and 25, daughters of a representative sample of single mothers or women with husbands in manual occupations, who took part in an intergenerational study in Islington in the late 1980s, and had the Life Events and Difficulties Schedule (LEDS) administered to them to cover a period of 12 months before interview (Andrews et al., 1990). As this cohort was very different in social class to the one of the present study, it was not felt to be an appropriate comparison group for life events in general, the rate of which is known to be dependent on social class.

9.3.2. Procedure

Assessment of eating pathology and onset of eating disorder: All patients were given a clinical interview in which the time course of the onset of their eating disorder was carefully charted. Onset was defined as: weight loss of $\geq 15\%$, amenorrhoea, the start of binges or self-induced vomiting or laxative abuse, whichever occurred first. These criteria were chosen rather than the stricter DSM-III-R criteria, to avoid a situation where during the study period patients already had significant symptoms, which might lead to the inclusion of illness-related events and difficulties. Social class was based on father’s occupation (Guy, 1976).

Assessment of life stress: All patients were interviewed with the LEDS at first assessment. The twelve months before onset were covered in the LEDS interview. As there is no significant reduction in the reporting of severe events and difficulties over at least five years (Neilson et al., 1989), patients with an onset of their eating disorder of less than four years prior to interview were included in this study. The threat or
unpleasantness of life events and difficulties was rated contextually (see chapter 4) by a panel of trained raters (U. Schmidt, J. Treasure, M. Blanchard and J. Tiller), who were blind to the subject's diagnosis and emotional reactions. The immediate and longer term threat of life events was rated on a 4-point scale. Life events were rated as severe if they continued to have a high threat rating (top two scale points: = marked or moderate threat) for at least 10 to 14 days after an event had occurred. The qualities of events rated contextually, such as severity or pudicity, were also rated in terms of the individual's subjective reaction. Difficulties were defined as any problem that had gone on for at least four weeks and were rated on a 1 to 7 scale. Difficulties on the top 3 points of the scale were regarded as marked. In the LEDS tradition a marked difficulty with additional characteristics is defined as a major difficulty: (1) a rating of 1 to 3 on overall contextual severity; (2) the difficulty has lasted for 2 or more years continuously; (3) the difficulty is not solely a health difficulty. Events and difficulties were also checked for 'independence' (for definition see chapter 4).

The pudicity dimension: This included events and difficulties with the potential to evoke sexual shame or disgust. Pudicity was rated where events were judged to involve premature, inappropriate or "forbidden" sexuality, or sexual situations which posed a moral dilemma for the patient, or where there was an element of public humiliation. Events where the patient was confronted with her parents' sexuality in an inappropriate fashion were also included (see appendix 9.8 for examples). Patients were rated on contextual and reported pudicity. It has been the tradition in reporting work with the LEDS to use contextual ratings in studies of affective disorder, where the reported emotional reaction (say anxiety or sadness) and the disorder (say anxiety
or depression) are of such an overlapping nature that the data becomes circular. By contrast in studies of physical disorder ratings of reported emotion can hardly be said to overlap at all with dependent variables such as neoplasms or myocardial infarction, and the potential impact of the reported threat or unpleasantness of an event may thus be examined separately from its contextual rating. Eating disorders seem intermediate in terms of this overlap: for example a subject’s subjective feeling of shame about something sexual (high reported pudicity) cannot be seen as 'overlapping' with feelings about food. Rules for rating pudicity were established with raters from the Royal Holloway & Bedford New College (T. Harris, A. Bifulco and B. Andrews), who were involved in developing the LEDS. The agreement study was done in terms of a 3 point rating: marked to moderate vs some vs no pudicity. Inter-rater reliability was good, with a weighted kappa of 0.84.

Statistical analysis: Data were analyzed using SPSS-PC+/4.0 (Norusis, 1990). Chi-square tests were used for categorical data. Independent samples t-tests were used for normally distributed continuous data. Mann Whitney U-tests were used for non-parametric data.

9.4. Results

9.4.1. Sample characteristics

Table 9.7.1. shows eating disorder patients' sociodemographic details. There were no differences between AN and BN patients in terms of age, age at onset or social class. The groups did, however, differ in terms of the duration of their illness with anorexics having a shorter duration of illness than the BN group. As expected the
groups also differed in terms of their body mass index. Comparison subjects from the Camberwell cohort had been chosen to reflect a similar age range (18 to 35 years) and similar social class distribution (25% were working class), whereas subjects from the Islington cohort were aged 15-25 with 95% of working class background.

9.4.2. Number of events and difficulties

The proportion of patients who had severe events and major difficulties before onset is given in table 9.7.2. There was no difference between AN and BN groups or the comparison group in terms of the proportion of patients with at least one severe event. For major difficulties, there were significant differences between the different eating disorder groups and the Camberwell group, with higher proportions of AN and BN patients experiencing these than the Camberwell group.

For marked difficulties data was available only for the eating disorder patients: there was no difference between AN and BN patients in this respect, nor was there any difference between AN and BN patients in the proportion of those with either a severe event or a marked difficulty. Likewise there were no differences between eating disorder groups in terms of the proportion of patients with independent severe stressors in the year before onset.

The median total number of life events and difficulties (ranging from mild to severe problems) over the study period is also given in table 9.7.2. AN and BN patients did not differ in terms of the median total number of events they experienced. However, they did differ in the median total number of difficulties. A further question was
whether the increased median number of difficulties in the BN group was a result of a
greater median number of marked difficulties or of mild difficulties or both. There
was a trend for the AN and BN groups to differ on the median number of marked
difficulties (AN: median 0 (range 0-3); BN: median 1 (range 0-4); Mann-Whitney U
test: p = 0.07). The two groups significantly differed in terms of the median number
of mild difficulties (AN: median 0 (range 0-6); BN: median 1 (range 0-6); Mann-
Whitney U test: p = 0.0024).

9.4.3. Type of events and difficulties

Serious problems (severe events or marked difficulties) were categorised further
depending on what area of life they affected. There were no differences between the
two eating disorder groups with regards to type of problem. Serious life stresses
occurred most commonly in the area of close relationships with family and friends
(AN: 26/72; 36%; BN: 12/29; 41%). Typical examples include the following:
parental problems (repeated severe arguments/tensions in the presence of the patient,
threat of divorce or actual separation), problems between a patient and a parent (e.g.
one patient disclosed childhood sexual abuse by her paternal grandfather to her parents
and got a very unsympathetic response from her father, who disbelieved her), or less
commonly problems between the patient and a close other outside the family, like a
confiding friend. Health problems were the second most common type of problem
(AN: 20/72; 28%; BN: 10/29; 35%). Typical examples included: one patient was
diagnosed as hyperthyroid, her mother had previously had hyperthyroidism with
dramatic symptoms. Another patient had several operations due to chronic hip
problems. Following the last operation she was on crutches for three months and in a
lot of pain. Another patient's godfather who was her main carer developed a malignancy and died. Other types of severe problems were as follows: partner problems: AN: 13/72 (18%); BN: 5/29 (17%), work or school problems: AN: 9/72 (13%); BN: 4/29 (14%); miscellaneous problems: AN: 1/72 (1%); BN: 1/29 (4%).

Taking a closer look at the most common serious problem, i.e. relationships with family and friends it turned out that there were important differences between the AN and BN groups. As table 9.7.3. shows, in the AN group, patients who had been faced with serious relationship problems were mostly indirectly involved, as the problem concerned two or more significant others e.g. parents’ marital difficulties. In the BN group, however, the majority of patients with relationship problems were directly involved in the relationship that caused problems.

9.4.4. Pudicity problems

Table 9.7.4. shows the distribution of pudicity problems (events or difficulties) rated marked/moderate on reported or contextual pudicity in the eating disorder groups during the year before onset and in the comparison subjects from the Islington cohort during the year before interview. Patients with anorexia nervosa had significantly more pudicity experiences before onset than BN patients or controls. Four of the comparison subjects reached DSM-III-R criteria for bulimia nervosa, one of these had a pudicity problem rated as marked/moderate. In a fifth control subject there was a suspicion that she had anorexia nervosa (her body weight was 83% of matched population mean weight). She also had a pudicity problem rated as marked/moderate. These subjects were not excluded from the analysis as in neither of the two cases with
pudicity experiences the problem occurred before onset. However, had eating disordered subjects been excluded from the control group, the rate of pudicity problems in this group would have fallen from 8% to 5%. One further concern was that the occurrence of pudicity experiences might be class related. This was however, not the case for either the eating disorder group as a whole, or for any of the different eating disorder subgroups.

9.5. Discussion

There are several problems with this study. Firstly, the sample size is relatively small. It was only possible to recruit relatively few cases of BN into the study because women with this disorder usually have a long duration of illness before presenting for treatment. Thus the majority of the BN cases presenting to the Maudsley Hospital Eating Disorder Clinic were excluded from this study. Secondly, sampling bias and referral bias may be at work as discussed in chapter 5. Thirdly, there are some problems with the choice of comparison groups: (a) Control data are not available for all the variables studied. (b) Only a small subgroup of women from the Camberwell cohort was similar to the patients included here in social class and age to be a suitable comparison group. (c) Islington subjects although similar in age to the ones of the present study had a different social class structure.

Despite these caveats some conclusions can be drawn from the findings: Compared to community controls AN and BN patients were no different in the proportion of subjects with at least one severe event before onset. A quarter to a third of the eating disorder patients had experienced at least one major difficulty compared with only 4%
of community controls. This suggests that severe life stress is important in the onset of eating disorders, as it is in other psychological and physical disorders (Brown & Harris, 1978; Craig & Brown, 1984; Finlay-Jones & Brown, 1981; Harris, 1989; House & Andrews, 1988; Ramirez et al., 1989).

BN patients and AN patients were very similar in the proportions of those who had experienced severe stressors, with 67%-76% experiencing at least one severe event or marked difficulty in the year before onset. Moreover, there was no difference regarding the area of life that these serious problems concerned. There was however, an important difference between the AN and BN group in the proportion of patients directly involved in severe relationship problems. This raises the possibility of different mediating variables in these two groups. As mentioned previously anorexics are often shy and avoidant (Piran et al. 1988) whereas many bulimics have borderline or histrionic traits (Levine & Hyler, 1986; Wonderlich et al., 1990). These differences in personality may be associated with an active evasion or seeking of conflictual relationships. In this context it is of note that interpersonal therapy, which addresses interpersonal difficulties, has been used successfully in bulimia nervosa (Fairburn et al., 1991).

Looking at stressful events and difficulties of all levels of severity, there were no differences between eating disorder groups in the median number of life events in the year prior to onset. The picture with regards to difficulties is more complex. Bulimia nervosa patients had more difficulties before onset than AN patients. This was mainly explained by a greater number of mild difficulties in bulimic patients. Starvation is
known to have an effect on memory and may therefore have led to a greater fall-off in the reporting particularly of mild difficulties in anorexia nervosa patients. Although none of the anorexic patients were severely starved when interviewed, memory deficits are known to endure beyond weight restoration (Jones et al., 1991; Szmukler et al., 1992).

The hypothesis that patients with anorexia nervosa would have more pudicity experiences than patients with bulimia nervosa and normal controls was confirmed, suggesting that difficulties with sexuality are relevant in the aetiology of anorexia nervosa. However, pudicity experiences only concerned about a quarter (24%) of anorexic patients in contrast to other studies examining the specific meaning of stressors, where rates of specific events preceding the onset of the disorder under study were in the range of 50-60% (Brown, 1993; House & Andrews, 1988). Putting the findings of the present study into context with the finding of psychosexual delay preceding onset of anorexia nervosa (see chapter 8) a possible conclusion is that a biological vulnerability and a sexual challenge are needed to trigger anorexia nervosa.

9.6. Summary and Conclusions

(1) Anorexic patients, bulimic patients and community controls did not differ in the proportion of patients with at least one severe event. However, significantly more AN and BN patients than community controls had experienced a major difficulty.

(2) 67% of anorexics and 76% of bulimia nervosa patients had either a severe event or a marked difficulty in the year before onset.
(3) In AN and BN the most common serious life stresses before onset concerned close relationships with family and friends with BN patients being significantly more often than AN patients directly involved in the problem (interpersonal events).

(4) Patients with anorexia nervosa had significantly more pudicity experiences before onset than BN patients or community controls.

(5) Whilst serious life stresses commonly precede the onset of anorexia nervosa and bulimia nervosa, problems with sexuality seem to be specific in triggering the onset of anorexia nervosa.
### Table 9.7.1: Sociodemographic details

<table>
<thead>
<tr>
<th></th>
<th>AN (n=72)</th>
<th>BN (n=29)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Age (years)</strong></td>
<td>21.5 (SD 5.4)</td>
<td>20.9 (SD 3.3)</td>
<td>N.S.</td>
</tr>
<tr>
<td>range: 13-42</td>
<td>range: 16-29</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Age at Onset</strong></td>
<td>19.8 (5.3)</td>
<td>18.4 (3.2)</td>
<td>N.S.</td>
</tr>
<tr>
<td><strong>Duration of Illness (years)</strong></td>
<td>1.8 (1.1)</td>
<td>2.4 (1.1)</td>
<td>0.01a</td>
</tr>
<tr>
<td><strong>BMI</strong></td>
<td>16.7 (2.7)</td>
<td>22.0 (2.9)</td>
<td>&gt; 0.000a</td>
</tr>
<tr>
<td><strong>Social class</strong></td>
<td>n (%)</td>
<td>n (%)</td>
<td></td>
</tr>
<tr>
<td>Not working class</td>
<td>57 (79)</td>
<td>21 (72)</td>
<td>N.S. b</td>
</tr>
<tr>
<td>working class</td>
<td>15 (21)</td>
<td>8 (28)</td>
<td></td>
</tr>
</tbody>
</table>

*Table 9.7.1: Sociodemographic details.* a: 2-tailed t-test for independent samples; b: Chi-square test.
<table>
<thead>
<tr>
<th></th>
<th>AN</th>
<th>BN</th>
<th>Camberwell women</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proportion of patients with at least one severe event</td>
<td>31/72 (43%)</td>
<td>14/29 (48%)</td>
<td>9/28 (32%)</td>
<td>0.44*</td>
</tr>
<tr>
<td>Proportion of patients with at least one major difficulty</td>
<td>16/72 (22%)</td>
<td>10/29 (34%)</td>
<td>1/28 (4%)</td>
<td>0.02*</td>
</tr>
<tr>
<td>Proportion of patients with at least one marked difficulty</td>
<td>35/72 (49%)</td>
<td>19/29 (66%)</td>
<td>--</td>
<td>0.19*</td>
</tr>
<tr>
<td>Proportion of patients with at least one severe event or marked difficulty</td>
<td>48/72 (67%)</td>
<td>22/29 (76%)</td>
<td>--</td>
<td>0.5*</td>
</tr>
<tr>
<td>Proportion of patients with an independent severe event or marked difficulty</td>
<td>33/72 (51%)</td>
<td>15/29 (52%)</td>
<td>--</td>
<td>0.75*</td>
</tr>
<tr>
<td>Median total number of life events</td>
<td>4 (1-9)</td>
<td>4 (1-11)</td>
<td>--</td>
<td>0.58b</td>
</tr>
<tr>
<td>Median total number of difficulties</td>
<td>1 (0-6)</td>
<td>2 (0-6)</td>
<td>--</td>
<td>0.0003v</td>
</tr>
</tbody>
</table>

*Table 9.7.2: Life events and difficulties. *: all chi-square tests; b: Mann-Whitney U test.
<table>
<thead>
<tr>
<th>Number of patients with a severe stressor in the domain of relationships</th>
<th>AN (n = 25)</th>
<th>BN (n = 13)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Proportion of patients with direct involvement in relationship problem</td>
<td>8 (32%)</td>
<td>12 (92%)</td>
</tr>
<tr>
<td>Proportion of patients with indirect involvement in relationship problem</td>
<td>17 (68%)</td>
<td>1 (8%)</td>
</tr>
</tbody>
</table>

**Table 9.7.3: Focus of relationship problems.** Direct involvement (= self + other), indirect involvement (other + other). Chi-square test: chi-square: 10.17; DF: 1; p = 0.001
<table>
<thead>
<tr>
<th>Time period before onset or interview</th>
<th>AN</th>
<th>BN</th>
<th>Islington Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-6 months</td>
<td>15/72</td>
<td>0/29</td>
<td>5/75</td>
</tr>
<tr>
<td></td>
<td>21%</td>
<td>(0%)</td>
<td>(7%)</td>
</tr>
<tr>
<td>6-12 months</td>
<td>2/72</td>
<td>1/29</td>
<td>1/75</td>
</tr>
<tr>
<td></td>
<td>(3%)</td>
<td>(3%)</td>
<td>(1%)</td>
</tr>
<tr>
<td>Total (0-12 months)</td>
<td>17/72</td>
<td>1/29</td>
<td>6/75</td>
</tr>
<tr>
<td></td>
<td>(24%)</td>
<td>(3%)</td>
<td>(8%)</td>
</tr>
</tbody>
</table>

Table 9.7.4: Pudicity problems (either self-report or contextual) over the year before onset (patients) or the year before interview (controls). Chi-Square: 12.44, DF: 4, p = 0.01.
9.8. Appendix

High contextual pudicity:

Patient (age 18) develops stomach pain and vaginal discharge and goes to see a private gynaecologist. A day later she is phoned up with an immediate appointment. She is told she has gonorrhoea and is given antibiotics for 1 week. After further tests she is given the medical all clear. She knows she must have contracted gonorrhoea from her boyfriend who is her first and only sexual partner, she also infers that he must have been unfaithful to her. When she confronts him he denies that he might have anything to do with this. A week later she sees him take some tablets which say 'for urinary infections'. He says he is taking them as a precaution. She stops seeing him for a week or two, but then the relationship starts again.

Patient (age 16), strict Catholic upbringing at a Convent school, went on school holiday trip, where she met some boys. She gets involved with one of them and has sex with him (first sexual intercourse). Her friends all know what has happened and are very shocked. After the holiday the boy fails to get in touch again. On self-report she said she felt very let down and used.

Patient (age 16) has a one-night stand with a boy, who as she finds out later, has a girlfriend who she knows. The girl-friend's sister then makes a public scene when patient is in a pub calling her abusive names, implying that she is a "loose woman".
**High reported pudicity:**

Patient (age 18): Her mother confides in her about an affair she has with a work colleague. The mother openly displays soft toys given as a present from this man and spends long times on the phone talking with him in front of the daughter. The mother moves out of the marital bedroom for a while. Although the patient is very concerned about the father's response to this, he keeps a stiff upper lip. She reports feeling repulsed by her mother's behaviour.

Patient (age 23) has a relationship with a married man which is against her principles. Her flatmates were aware of it and advised her to stop. Despite feeling very ashamed and guilty she carried on seeing him and even brought him to stay in the shared house.
Chapter 10: Discussion

In the following some attempt will be made to summarize and link the findings of the five research studies presented in this thesis. The methodological limitations of these studies have already been discussed in each of the five chapters pertaining to these studies and will not be reiterated here. Thereafter, some thought will be given to the direction of future research in this area and the implications for clinical practice will also be discussed.

10.1. Summarizing and Linking the Findings

10.1.1. Childhood care and abuse in eating disorders

Structural, behavioural and attitudinal aspects of childhood family environment were examined in anorexia nervosa and bulimia nervosa (chapter 5) and in different subgroups of bulimic disorders (chapters 6 and 7). These studies provide an advance over previous research in the area in the following respects:

(1) A sophisticated semistructured interview measure was used which facilitates retrieval of childhood memories and allows a distinction between subjective and objective indicators of poor childhood care.

(2) So far, there has been a tendency for eating disorder researchers to examine either family functioning or childhood abuse (especially sexual abuse), despite considerable research evidence to suggest that various forms of child maltreatment tend to cluster in the same families (Briere, 1992). The present studies are amongst the first, to combine the two lines of investigation in eating disorders.

(3) To the best of my knowledge the study in chapter 5 was the first to examine the link between chronicity of eating disorder and problematic childhood care.
In considering the implications of the findings of the study described in chapter 5 it is perhaps useful to concentrate on restricting anorexia nervosa and bulimia nervosa. In restricting anorexia nervosa, the relative absence of physical abuse and neglect and the similarity of anorexic families in this respect to those without psychiatric disorder was striking. Additionally, in RAN, poor childhood care was not associated with later chronicity of the disorder. These findings do not imply that these families are normal in all respects, but what they do suggest is that gross family abnormalities are relatively rare in the histories of those who develop RAN. (Childhood sexual abuse which is not necessarily linked to intrafamilial disturbance is discussed separately below).

In contrast, bulimia nervosa patients had experienced different forms of childhood adversity considerably more often than those with restricting anorexia nervosa and different types of problematic childhood care co-occurred in the same patients, with 65% of bulimic patients having experienced multiple adversity. Interestingly, only physical abuse and parental indifference were necessary to predict bulimia nervosa. This suggests that in bulimia nervosa a family environment resembling that of "affectionless control" may be of aetiological significance as previously found in depression (Parker, 1983; Rodgers, 1996ab). Poor childhood care was associated with chronicity in bulimia nervosa.

These findings provide further evidence for a binger/restricter dichotomy in terms of the childhood antecedents of these disorders. Whilst the studies described here did not include comparison groups of women without psychiatric disorders or with other
psychiatric disorders those comparison data do now exist (see table 5.7.7.; Vize and Cooper, 1995; Welch and Fairburn, 1994) and suggest that the rates of childhood adversity in bulimia nervosa patients are elevated compared to community subjects but not compared to other psychiatric subjects including those with severe anxiety disorder or depression.

Childhood sexual abuse was assessed using strict criteria. Rates of CSA were raised compared to those found in general population samples but did not distinguish between the diagnostic groups. Previously there had been claims in the literature to the contrary, mainly from studies that use more lenient criteria (see chapter 3). The study in chapter 5 thus confirms the results from an earlier case series from the Maudsley Hospital Eating Disorder Unit (McClelland et al., 1991) and has been replicated by Vize and Cooper (1995) who used part of the CECA and also found similar rates of CSA to the patients in the present study with anorexia nervosa and bulimia nervosa.

It could be argued that despite the lack of differences in rates of childhood sexual abuse there might be some more subtle differences between the groups in terms of the severity of the abuse. However, the study described in chapter 8 does not confirm this. Anorexia nervosa and bulimia nervosa patients who had experienced childhood sexual abuse did not differ in terms of the percentage of cases where force had been used or in the percentage who had experienced repeated childhood sexual abuse.

Following on from the study described in chapter 5 which suggested that abuse and neglect and more subtle forms of childhood adversity were common in bulimia
nervosa patients, the studies in chapter 6 and 7 examined different subgroups of bulimic disorders. The study in chapter 7 which compared young onset with typical onset bulimia nervosa further underlined the aetiological importance of inadequate parental care, this time as the only family factor that distinguished those with an earlier onset from those with a typical onset. Childhood sexual abuse failed to distinguish between those with an early and those with a typical onset.

The study on obese binge eaters suggested that if anything there is a trend for this group to have less disturbance in their childhood family environment than normal-weight bulimics. Thus, these obese individuals seem to be those with a genetic predisposition to obesity who are desperately struggling to fit in with cultural norms of slimness. Again, contrary to the hypothesis there was a lack of difference between the two groups in terms of childhood sexual abuse.

In summary, the three studies presented here assessing childhood environment in different eating disorders provide no support for the idea that patients with bulimic disorders or those with increased use of compensatory behaviours, especially vomiting (Pitts and Waller, 1993; Tobin and Griffing, 1996) are more likely to have experienced sexual abuse than individuals with other eating disorders without these features.

As mentioned above very few studies have so far attempted to tease out the relative significance of different types of childhood adversity in the aetiology of different eating disorders and the results do not allow any firm conclusions. Rorty et al. (1994)
in a comparison of bulimic women with normal controls, found that the bulimia nervosa group reported higher levels of physical, psychological and multiple abuse, whereas rates of sexual abuse did not distinguish the groups, except in combination with other forms of abuse. One non-clinical study using a student sample found that childhood sexual abuse and family environment combined in an additive manner to increase the probability of bulimia (Hastings and Kern, 1994). Another non-clinical female cohort (Kinzl et al., 1994) showed a strong association between an adverse family background and high scores on the Eating Disorder Inventory, whereas there was no such link between childhood sexual abuse and eating disturbance. In this context it is perhaps important to consider the findings of a large community study of the adult sequelae of sexual abuse which concluded that although sexual abuse was found to make some independent contribution to adult psychopathology: "The overlap between the possible effects of sexual abuse and the effects of the matrix of disadvantage from which it so often emerges were, however, so considerable as to raise doubts about how often, in practice, it operates as an independent causal element" (Mullen et al., 1993).

10.1.2. Childhood sexual abuse and sexual development

The study described in chapter 8 was an attempt to evaluate the links between childhood sexual trauma and the commonly noted delay in sexual development in eating disorders. The results suggested that there was a delay in psychosexual development in eating disorder patients compared with normal controls and that in anorexia nervosa this may even antedate onset of the disorder. However, the delay in sexual development was not accounted for by a history of earlier sexual abuse. These
findings lend further credence to the assertion that maturity and sexual fears may be implied in the aetiology of anorexia nervosa and may be the result of an underlying 5-HT mediated biological vulnerability.

10.1.3. Life events and difficulties as provoking agents

Family models of eating disorders do not help to explain why in a given individual a particular type of eating disorder develops at a particular point in time. From research in depression we know that whilst different background factors, including disturbance in childhood care, increase the risk for its development, a severe event or major difficulty is required to trigger onset (Brown and Harris, 1978) and it is the event or difficulty that determines the timing of the onset. The life events study presented in chapter 9 is an advance over previous studies into life stresses in eating disorders in terms of the methodology used. It is also the first study to examine the meaning of stressors in addition to the frequency, severity, type and duration of stressors. Whilst compared to a community sample severe life events were not significantly more common in eating disorder patients, major difficulties were. Onset was triggered by a severe event or marked difficulty in 67% of those developing anorexia nervosa and 76% of those developing bulimia nervosa. Troop and Treasure (1997) have since replicated these findings in a separate, smaller study using the same measures and methodology. They found that 58% of their sample developed anorexia nervosa in response to a severe life event or marked difficulty while the rate for bulimia was 74%.

Pudicity problems, i.e. events and difficulties which implied a shameful, and
conflictual sexuality, were significantly more common in anorexia nervosa subjects than in BN or in community subjects. The greater rate of pudicity problems in anorexia nervosa patients was not simply the result of these patients having been sexually sensitized by a higher rate or greater severity of childhood sexual abuse (see chapter 5).

In bulimia nervosa interpersonal events were more common than in anorexia nervosa. There is some suggestion from research into adult affective symptoms that interpersonal life events in adulthood are preceded by parental affectionless control in childhood and that these interpersonal events (in addition to availability of emotional support and social network) in turn are associated with adult affective symptoms (Rodgers, 1996ab). It would have been desirable in the present study to combine the study on childhood problems with the data on life events to see whether there were any continuities or discontinuities in terms of experiences of adversity. For example, are those patients with bulimia nervosa whose onset is triggered by an interpersonal event those who have had to face severe problems in their childhood relationships? The reason that the data weren’t combined was to do with the decision to limit the life event study to those patients with a relatively recent onset, to minimize problems with recall of events and difficulties. This meant that most of those patients with a seriously disturbed background weren’t included in the life events study as it was mainly those patients with a duration of bulimia nervosa of more than 4 years who had high levels of childhood adversity.
10.1.4. Bulimia nervosa: Childhood adversity, life events before onset and potential mediators

In a review article on the links between childhood abuse and neglect and later development of bulimia nervosa, Wonderlich (1992) made the point that: "Perhaps the greatest limitation of ... family conceptualizations of bulimia is that they do not thoroughly account for how the affected individual chooses dieting, bingeing, and purging as symptoms rather than depression, drug use, or some other breakdown in personal functioning...". And of course there is a wealth of studies showing that childhood abuse and neglect is common in the histories of adults with depression, alcohol problems or personality disorder (e.g. Andrews et al., 1990; Bifulco et al., 1987; Crook et al., 1981; Harris et al., 1986; Holmes and Robins, 1987; Parker, 1983; Zweig-Frank and Paris, 1991). The family disturbance found in bulimia nervosa is thus by no means specific.

A two-stranded model for the links between childhood care and adult psychiatric disorder (depression) through environmental and cognitive pathways has been put forward by Harris et al. (1990). In the case of bulimia nervosa this may mean that childhood adversity may be linked to later eating disorder by either setting the scene for further environmental hazards (such as a premature flight from the family into further abusive relationships) or by cognitive/emotional pathways affecting personality development (leading to borderline personality organization or to more subtle deficits like abnormal defense style, poor problem solving or coping and low self esteem) (Bifulco et al., 1991). Clearly, those two pathways are not mutually exclusive and may often occur in combination.
The finding that bulimia nervosa patients experienced significantly more severe interpersonal events and difficulties than anorexia nervosa patients could either be a result of a continuation of environmental hazards from childhood to adulthood or might have been mediated by cognitive-emotional pathways or might be the result of both.

Two studies using the CECA have explored the cognitive pathways between childhood adversity and later onset of eating disorders. Schmidt et al. (1993b) examined childhood environment (using the CECA) and adult defense style (Andrews et al., 1989). Defense mechanisms are constructs derived from psychodynamic thinking and are thought to be unconscious, homeostatic mechanisms which help individuals to regulate affect and behaviour. Defense style predicts adult adjustment and mental health (Vaillant et al., 1986). Bulimia nervosa patients had a significantly less mature defense style than patients with anorexia nervosa and female controls and more immature defenses than the control group. In restricting anorexia nervosa no childhood predictors of adult defense style were found, whereas in bulimia nervosa, excessive parental control during childhood was a negative predictor of mature defenses and physical abuse a positive predictor of immature defense style. This would suggest that those who develop bulimia nervosa may be vulnerable when encountering stressful situations and this vulnerability seems to be the result of earlier deficits in childhood care.

Examining constructs (helplessness, mastery) derived from a different theoretical model, Troop and Treasure (1997) compared women with and without a history of
eating disorders using similar methodology to the present study (CECA, LEDS).

Despite similar levels of childhood adversity in eating disorder and control subjects (the latter group had deliberately been biased towards high levels of adversity) there was a higher rate of childhood helplessness and a lower rate of childhood mastery in women with eating disorders compared to those without. In a subsample for whom helplessness and mastery were also rated in response to a severe life event or marked difficulty (provoking agent), it was found that helplessness and mastery in childhood were associated with helplessness and mastery in response to a provoking agent. However, a regression analysis showed that it was only this later helplessness and mastery that was required to predict onset of eating disorders. Unfortunately, the number of patients in that study was too small to look at potential differences between bulimics and anorexics.

In addition to individual environmental and cognitive-emotional factors predisposing an individual for the onset of an eating disorder contemporary cultural factors may shape the form of the symptomatic reaction. This is exemplified by an intergenerational community study of mothers and their teenage or young adult daughters. Parental neglect and abuse (physical or sexual) had an effect on daughters' psychiatric health (Andrews et al., 1990). Whilst the most common disorder found in the mothers was depression, in the next generation bulimia and depression were equally common. This suggests that contemporary cultural pressures have changed the clinical manifestation of neurosis (Russell, 1995). The increasing incidence of bulimia nervosa over the last three decades adds further support to this hypothesis (Bushnell et al., 1990, Kendler et al., 1991; Turnbull et al., 1996). Dieting which increases the
risk of bulimia nervosa 8-fold may be the single most important cultural factor which is operative in this context (Patton et al., 1990). In a large community based case-control study of risk factors for bulimia nervosa one of the main differences between individuals with bulimia nervosa and psychiatric controls was with respect to risk factors that increased the likelihood of dieting, e.g. premorbid obesity or family history of obesity (Welch and Fairburn, 1997).

10.1.5. A model of anorexia nervosa

Treasure et al. (1997) draw our attention to a fascinating animal model of anorexia nervosa. Pigs (and other farm animals), especially those bred for leanness, can develop irreversible emaciation and their clinical features strongly resemble those of humans with anorexia nervosa. Affected animals restrict their intake of normal food and some consume large amounts of straw. They also spend more time on non-nutritive 'hyperactive' behaviour, like incessantly moving around the pen. Their hair becomes coarse and long and many do not return to heat. The onset of this wasting syndrome is stress-related. 5-HT2 receptor antagonists successfully treat these wasting conditions. This is perhaps not surprising, given the pivotal role of serotonin, and in particular 5-HT2 receptors in the central control of appetite. 5-HT is also implicated in the central control of locomotor activity and sexual behaviour.

Several of the findings of the studies presented here are consistent with this model. Firstly, there is an absence of gross family disturbance in anorexia nervosa, secondly, even before onset anorexics' sexual development is more hesitant than that of bulimics and normal controls. Thirdly, severe stress before onset is a trigger of the disorder in
the vast majority of cases and fourthly, in a quarter of cases this is stress of a psychosexual nature (pudicity problems).

10.2. Future Research

10.2.1. Childhood adversity

Research in the area of family adversity in eating disorders is moving away from the study of patient populations into the study of community subjects (see Welch and Fairburn, 1994), as this circumvents some forms of bias (Fairburn et al., 1996). However, study of community cases is not without its own difficulties as many community cases - in particular of anorexia nervosa - escape detection (Beglin and Fairburn, 1992). Studies of patient and untreated community populations can complement each other as they will inform about factors that drive people into or prevent them from seeking treatment.

Future studies in the area of childhood adversity in eating disorders need to get away from mere 'head counts' and needs to focus more strongly on the mediators between abuse and neglect and the development of eating disorders. Whilst there is a growing literature on mediators between childhood sexual abuse and later eating disorders, more emphasis needs to be given to the assessment of mediators relating to other forms of childhood adversity that form the "matrix of disadvantage" (Mullen et al., 1993) from which especially bulimia nervosa so often arises.

10.2.2. Attachment and eating disorders

One account of how parental behaviour may influence adult psychopathology is
offered by attachment theory. Bowlby (1988) sees the development and organization of emotional bonds between the infant and its care givers, in particular the mother as a major determinant of an individual’s childhood and adult mental health. The primary function of the attachment relationship is protection, the goal of attachment is subjective 'felt security' and the outcome of attachment is behavioural and/or psychological proximity. Secure attachments are the result of parents being available and responsive when called upon. Inadequate parenting (variable responsivity of parents, separations or threats of abandonment, repeated maltreatment, rejection or institutionalization) can lead to three forms of insecure attachment, anxious resistant, anxious avoidant and disorganized.

"Attachment theory has always been presented as a psychological theory of how certain aspects of interpersonal relationships are represented intrapsychically.....Such theorizing links attachment research to psychodynamic formulations, particularly object relations theory and Mahler’s work on the process of separation-individuation. But attachment concepts, such as Bowlby’s notion of "working models", are also compatible with recent attempts within empirical psychology to understand the internalized (i.e. cognitive) structures and functions that organize attachment relationships and behaviors. In broad terms, working models of attachment at a cognitive level include memories of attachment-related experiences, beliefs, attitudes and expectations about the self and others in relation to attachment, attachment goals and needs, and plans and strategies to achieve these goals and satisfy these needs" (O’Kearney. 1996). Individuals who have experienced disturbances in their early attachment are at risk of later disturbances in their concepts of self and others. their
ability to form and maintain social relationships and are also at risk of developing psychiatric disorder in particular neurotic and personality disorders and delinquency.

Several groups have developed an interest in early attachment in eating disorder patients, but most existing studies have serious methodological shortcomings (O'Kearney, 1996) and are based on questionnaires or projective tests. A small study by Armstrong and Roth (1989) found that compared to normal controls eating disorder patients overreacted to minor separations. In a study of mixed psychiatric patients using the Adult Attachment Interview (a well-validated interview measure), there was a positive association between idealization of parents and diagnosis of an eating disorder, whereas a diagnosis of depression was negatively associated with parental idealisation (Fonagy et al., 1996). Intergenerational studies of attachment in mothers and their eating disordered daughters using the Adult Attachment Interview are under way looking at adolescent (Pugh, personal communication) and adult eating disorder patients (Ward, personal communication).

10.2.3. Nature and nurture

We also do not understand well how environmental (family and individual) factors interact with other aetiological factors. Evidence for a genetic component in the aetiology of anorexia nervosa and to a lesser extent bulimia nervosa has gradually accumulated (Treasure and Holland, 1995). The "other" important message from genetic research is that nurture is important, too. Future research efforts should be directed at untangling the threads of nature and nurture. While traditional views of parenting have emphasized the role of the parent, implicitly assuming that children are
passive "recipients" of parenting there is increasing evidence of the interactive nature of the parent child relationship. "Parenting is a complex, dyadic process likely to be influenced by a range of factors, including cultural beliefs of the parent about child rearing, genetic temperamental characteristics of the parent (i.e., genetic factors influencing the provision of parenting), and genetic-temperamental characteristics of the child (i.e. genetic factors influencing the elicitation of parenting)" (Kendler, 1996). In a large population-based study genetic factors in both parent and child were more important for parental warmth than for protectiveness or authoritarianism, the latter may be more influenced by social and religious attitudes (Kendler, 1996).

In this context an interesting line of research is that into non-shared family environment, as there is now increasing evidence that non-shared family factors are much more influential in determining personality and psychopathology than shared family factors. The Sibling Inventory of Differential Experience (SIDE) was developed to assess non-shared (differential) experiences of siblings, not just in relation to parents but also to each other and to peers (Daniels and Plomin, 1985). The novel aspect of the SIDE is that it asks siblings to rate their experiences relative to their siblings rather than in an absolute terms. One small study (Wonderlich et al., 1994) using the SIDE suggested that lack of paternal affection may be a source of non-shared environmental experience in bulimia nervosa patients. Further investigations along these lines are needed, using for example the CECA and LEDS in twins or siblings of comparable age who are discordant for the development of an eating disorder. Such studies are currently under way (Treasure, personal communication).
10.2.4. Life events and difficulties

Further work needs to be done in assessing the role of life stresses as provoking agents in the onset of eating disorders as outlined below.

**Taking a closer look at symptoms before onset:** In the present study onset was defined as the occurrence of one of a number of possible happenings, i.e. onset of bingeing, vomiting, weight loss, etc. It might be possible to refine this and to look at whether different types of events or events with different meaning precipitate different types of symptoms, as there is some suggestion from the literature that different types of stress do precipitate different types of eating disturbance.

**Patients without pre-onset events or difficulties:** Whilst in the study in chapter 9 most patients with anorexia and bulimia nervosa did have a precipitating severe life event or marked difficulty, there were some who did not and it would be interesting to know what is different about these cases.

**Age factors:** As outlined earlier (see chapter 4) there is some suggestion that precipitation of onset by a severe event or marked difficulty may be more common in cases of late onset anorexia nervosa than in those with typical or early onset (Mynors-Wallis et al., 1992; Russell and Gilbert, 1992). However, Troop et al. (in preparation) point out that there are methodological problems with both of these studies in that they relied on case note descriptions of triggering events and difficulties rather than on a standardized interview like the LEDS. As the assessing clinicians completing the casenotes may have been aware of the notion that precipitating events
might be more common in late onset cases, this may have biased the degree of questioning and the recording of this detail in the notes. In addition, there was no control of length of recall as there was in the present life events study and in that of Troop and Treasure (1997).

It is at present also unknown whether such age effects are involved in the onset of bulimia nervosa. It would have been desirable to include the study of life events before onset in the case-control study of young onset BN, however, this was not done in view of the fact that in most of the cases the disorder was very chronic. The number of patients in the present study (see chapter 9) was not large enough to disentangle these age effects. However, it may be possible to answer these question by pooling the data from the present study and the data of Troop and Treasure (1997).

**Meaning of stress:** Previous writing on stress as a precipitant of eating disorders has always emphasized the broad range of stressors and to a large extent the present study confirms this. However, until now what has been studied is mainly the domain of the event or difficulty (e.g. health or relationships) as opposed to the meaning of the event and the present study is the first to study the potential meaning of events (see pudicity problems). It would be of interest to build up a more detailed picture of the meaning of stresses preceding different eating disorders in future studies.

**Life events and relapse, recovery, and maintenance of symptoms:** Further work needs to be done to assess the role of life events and difficulties in relapse, recovery and maintenance of symptoms of eating disorders. Certainly in depression
interpersonal difficulties at onset and during the course of the disorder predict chronicity of the disorder (Brown and Moran, 1994) and positive life events - particularly relating to a reduction in the level of a marked difficulty or offering a "fresh start" - have been implicated in recovery or improvement of depressive symptomatology (Brown et al., 1988).

10.3. Implications for Clinical Practice

Empirical research cannot - and need not - answer questions of individual meaning (Rorty and Yager, 1996) and thus the lack of a specific association between childhood neglect and abuse and eating disorders does not suggest that in those cases where abuse or neglect occurs this is somehow irrelevant. On the contrary, given the relative frequency of experiences of abuse and neglect in eating disorder patients - particularly those with bulimia nervosa - a thorough assessment of family background is indicated in every case. In every single case clinicians together with their patients have to decide whether abuse is significant in formulating the eating disorder (Waller et al., 1994) and will affect plan of treatment.

Physical abuse perhaps has less of a stigma attached to it than sexual abuse, and may be perceived as something that a therapist can ask about more easily. Interestingly, a large proportion of bulimic patients who had received harsh physical treatment as children did not define themselves as abused when asked directly (Rorty et al., 1995) and physical abuse may be an obstacle to seeking treatment (Fairburn et al., 1996). This might suggest that these women may have internalised a sense that they are bad, worthless and blameworthy and therefore deserving of the 'punishment' they receive.
It is therefore important that physical abuse is asked about sensitively, and in some detail, giving individuals multiple prompts.

Similarly, in cases of childhood neglect, it is unlikely that patients will spontaneously volunteer this experience, they may not have the words, the awareness or the necessary self-esteem to do so.

Particularly in cases of suspected childhood sexual abuse, the clinician may have to face a number of difficult questions and decisions. What is to be done if the clinician has a suspicion that the patient may have been abused, yet she does not volunteer this? Should therapists address their suspicion, if so when is the best time to do so and how should they go about raising their suspicion? Addressing the topic too early, when there is no relationship of trust between therapist and patient, may simply lead to the patient "clamming up" and being unable to reveal what has happened to her. On the other hand a therapist who colludes with the patient's silence may reinforce her view of herself as unacceptable. In reaching a decision as to how to proceed it is important for the therapist to bear in mind that although the majority (60%) of eating disorder patients with childhood sexual abuse presenting for treatment will have tried to confide in a close other, most of them (93%) will have had a negative or absent response to their revelation (Waller and Ruddock, 1992). This experience will undoubtedly for some of these patients raise their threshold for trying to confide again.

One further important question is often whether, in the presence of significant abuse
and/or neglect, this ought to be addressed before, after, or in conjunction with any symptomatic work. This is particularly important in the context of a relatively brief outpatient treatment, whereas in in-patient or day-patient work more symptom-oriented treatment and an exploration of the patient's past can occur in parallel. Symptomatic progress often provides patients with a sense of achievement and self-esteem and symptomatic work may therefore, for some, be the safest starting point from which to develop trust in their therapist. Treatment which exclusively focuses on structured symptom management without giving the patient the option of addressing their childhood abuse or neglect may be experienced by the patient as coercive or irrelevant. On the other hand, if the therapist insists too strongly on the importance of exploring the childhood traumata in depth, the patient may feel that the therapist is only interested in her past and may experience this as intrusive, controlling or at worst as further abuse. Additionally, there may be a danger of inducing false memories of childhood sexual abuse, an issue which has recently received much public attention (Pope and Hudson, 1996).

Decisions as to whether or not family involvement is indicated and if so what format this should take (family therapy or family counselling) have to be carefully weighed up, especially if there is the suggestion of past or present family disturbance. In anorexia nervosa, in those with an onset at a young age family therapy is superior to individual supportive treatment (Russell et al., 1987). However, high expressed emotion in the family is a predictor of drop-out from family treatment and poorer outcome (for review see Eisler, 1995) and in those cases family counselling may be more appropriate (Le Grange et al., 1992). Families will often feel that they are to
blame for their daughter's disorder and great care needs to be taken as to how family involvement is presented to them.

There is little empirical data on the family treatment of patients with bulimia nervosa (See Dare and Eisler, 1995). It is therefore difficult to know whether or not to recommend this form of treatment, especially if the family background is very disturbed. There is some suggestion that in bulimia nervosa (Blouin et al., 1994) a highly controlled or discordant family environment predicts poor outcome following group cognitive behavioural therapy, although Turnbull et al. (1997) who studied predictors of outcome following individual cognitive therapy failed to support this. Interpersonal therapy is an effective alternative to cognitive behavioural treatment in bulimia nervosa (Fairburn et al., 1991) and given that interpersonal events and difficulties are common triggers of bulimia nervosa (see chapter 9) it should be easy to provide patients with a credible rationale for introducing this treatment.

In summary, the treatment of eating disordered women who have had experiences of neglect or abuse is a considerable challenge to the therapist, not at least as these patients often have personality disorders (McClelland et al., 1991) which will result in more prolonged and perhaps more "rocky" treatment.
References


Blair, C., Freeman, C. and Cull, A. (1995). The families of anorexia nervosa and

nervosa: The role of depression. *International Journal of Eating Disorders* **9**, 649 -
658.

Barlow, J. and Perez, E. (1994). Prognostic indicators in bulimia nervosa treated with
cognitive behavioral group therapy. *International Journal of Eating Disorders* **15**, 113
-123.


- 356.


Jaeger, R., Lischer, S., Muenster, B., and Ritz, B. (1976). *Biographisches Inventar zur Diagnose von Verhaltensstörungen (Handanweisung).* Verlag für Psychologie (Hogrefe), Göttingen.


Kilpatrick, D. (1985). *Crime Victimization Inventory*. Department of Psychiatry and Behavioral Sciences, Medical University of South Carolina, Charleston, S.C.


New York.


