Are overvalued ideas about weight and shape overvalued ideas in the
diagnosis of early onset anorexia nervosa?

Ian Frampton
School of Psychology
University of Exeter
April 1996

Submitted in part fulfilment of the degree of Doctor in Clinical and
Community Psychology
<table>
<thead>
<tr>
<th>Title</th>
<th>Page No.</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Acknowledgements ........................................................................</td>
<td>i</td>
</tr>
<tr>
<td>2. Abstract .......................................................................................</td>
<td>1</td>
</tr>
<tr>
<td>3. Introduction ..................................................................................</td>
<td>2</td>
</tr>
<tr>
<td>3.1 Overview .......................................................................................</td>
<td>2</td>
</tr>
<tr>
<td>3.2 Early Onset Anorexia Nervosa ....................................................</td>
<td>4</td>
</tr>
<tr>
<td>3.2.1 Prepubertal and Intra-pubertal Onset ......................................</td>
<td>4</td>
</tr>
<tr>
<td>3.2.2 Incidence and Prevalence .......................................................</td>
<td>4</td>
</tr>
<tr>
<td>3.2.3 Sex Ratio ...................................................................................</td>
<td>5</td>
</tr>
<tr>
<td>3.2.4 Social Class Bias .....................................................................</td>
<td>6</td>
</tr>
<tr>
<td>3.2.5 Prognosis and Outcome ................................................................</td>
<td>6</td>
</tr>
<tr>
<td>3.2.6 Mortality ....................................................................................</td>
<td>7</td>
</tr>
<tr>
<td>3.2.7 Early vs. Late Onset ..................................................................</td>
<td>7</td>
</tr>
<tr>
<td>3.3 Psychological Theories of Eating Disorder ....................................</td>
<td>9</td>
</tr>
<tr>
<td>3.3.1 Psychodynamic Theories ...........................................................</td>
<td>9</td>
</tr>
<tr>
<td>3.3.2 Feminist Sociocultural Theories ...............................................</td>
<td>12</td>
</tr>
<tr>
<td>3.3.3 Cognitive Behavioural Theories ...............................................</td>
<td>13</td>
</tr>
<tr>
<td>3.3.4 Systemic Theories ......................................................................</td>
<td>14</td>
</tr>
<tr>
<td>3.3.5 Multifactorial Theories ...........................................................</td>
<td>15</td>
</tr>
<tr>
<td>3.4 Weight Concern ..............................................................................</td>
<td>20</td>
</tr>
<tr>
<td>3.4.1 Diagnostic Checklists for Anorexia Nervosa ................................</td>
<td>21</td>
</tr>
<tr>
<td>3.4.2 Diagnostic Checklists for Early Onset Anorexia Nervosa ................</td>
<td>22</td>
</tr>
<tr>
<td>3.4.3 Diagnosti Systems ......................................................................</td>
<td>23</td>
</tr>
<tr>
<td>3.5 Shape Concern ...............................................................................</td>
<td>25</td>
</tr>
<tr>
<td>Section</td>
<td>Title</td>
</tr>
<tr>
<td>---------</td>
<td>-------</td>
</tr>
<tr>
<td>3.6</td>
<td>Introduction Summary</td>
</tr>
<tr>
<td>3.6.1</td>
<td>Research Aims and Design</td>
</tr>
<tr>
<td>4.</td>
<td>Method</td>
</tr>
<tr>
<td>4.1</td>
<td>Participants</td>
</tr>
<tr>
<td>4.2</td>
<td>Materials</td>
</tr>
<tr>
<td>4.3</td>
<td>Procedure</td>
</tr>
<tr>
<td>5.</td>
<td>Results</td>
</tr>
<tr>
<td>5.1</td>
<td>Descriptive Data Analysis</td>
</tr>
<tr>
<td>5.2</td>
<td>Power Analysis</td>
</tr>
<tr>
<td>5.3</td>
<td>Item Analysis</td>
</tr>
<tr>
<td>5.4</td>
<td>Discriminant Validity</td>
</tr>
<tr>
<td>5.5</td>
<td>Subscale Analysis</td>
</tr>
<tr>
<td>5.6</td>
<td>Hypothesis Testing</td>
</tr>
<tr>
<td>6.</td>
<td>Discussion</td>
</tr>
<tr>
<td>6.1</td>
<td>Research Hypotheses</td>
</tr>
<tr>
<td>6.2</td>
<td>Use of the Eating Disorder Examination</td>
</tr>
<tr>
<td>6.2.1</td>
<td>Inter-rater Reliability Study</td>
</tr>
<tr>
<td>6.2.2</td>
<td>Discriminant Validity Study</td>
</tr>
<tr>
<td>6.2.3</td>
<td>Construct Validity Study</td>
</tr>
<tr>
<td>6.2.4</td>
<td>Summary - the Use of the CH-EDE</td>
</tr>
<tr>
<td>6.3</td>
<td>Methodology</td>
</tr>
<tr>
<td>6.3.1</td>
<td>Sample Size</td>
</tr>
<tr>
<td>6.3.2</td>
<td>Measures Used</td>
</tr>
<tr>
<td>6.3.3</td>
<td>Statistical Analysis</td>
</tr>
</tbody>
</table>
6.4 Implications for Theories of Eating Disorders................................. 57
6.5 Implications for the Treatment of Eating Disorders...................... 61

7. References Cited.................................................................................. 64

8. Appendices........................................................................................ 68

8.1 Appendix Index ............................................................................. 68
2. **Abstract**

Objective: The Eating Disorder Examination (EDE) is a reliable and valid semi-structured interview, which measures the specific psychopathology of anorexia nervosa and bulimia nervosa. This study aims to investigate the psychometric properties of the child adaptation of version 12.0D of the EDE (ChEDE 12.0).

Method: The ChEDE 12.0 was administered to 15 children with anorexia nervosa (AN), 15 children with other clinical eating disturbances and two groups of 15 age-matched controls. The groups were compared using a two sample matched groups design.

Results: Alpha coefficients for each of the ChEDE 12.0 subscales indicated a high degree of internal consistency, and inter-rater reliability was found to be high (r=0.91 to r=1.00). The subscale scores of the AN group were significantly higher than those of the other groups, whilst the other eating disturbance group did not differ from its control group.

Discussion: The ChEDE 12.0 differentiates children with AN from children with other forms of clinical eating disturbance and control children. The hypothesis that children with early onset anorexia nervosa would not evidence significant weight and shape concern is refuted.
3. **Introduction**

3.1 **Overview**

The aim of this study is to explore the contribution of weight and shape concern to the core psychopathology of early onset anorexia nervosa. This condition has been well described by several groups of authors in recent years, and the first section below reviews their findings about the nature and prevalence of eating disorders of early onset to help put the present study into context.

The second section of the introduction relates the epidemiological and phenomenological findings to the broad base of theoretical approaches to eating disorders. In particular, an analysis of specifically psychological theories reveals how little supportive research data has been generated, especially for early onset anorexia nervosa. A case is made for integrated theories that predict specific cognitive and behavioural features, with particular reference to Slade's (1982) functional model of anorexia nervosa.

Two theoretically-derived core psychopathological symptoms are then explored in more detail to evaluate their historical origins and the existence of any supportive evidence for them from research studies. For the first of these, *weight concern*, an evaluation is made of the construct "fear of weight gain/weight phobia" over time in different diagnostic systems. The second, *shape concern*, is explored in terms of the research evidence in support of body image disturbance.
The use of standardised assessments of eating, weight and shape pathology is considered in the following section, with particular reference to the Eating Disorder Examination (Fairburn and Cooper, 1987). This investigator-led semi-structured questionnaire assessment has been developed in parallel adult and child versions, allowing for direct comparison between early onset and adult groups.

Since this measure generates subscales for both weight and shape concern, it will be possible to directly test the prediction from Slade's (1982) model and from Cooper et al (1994) that at least a subset of children with major eating pathology might not exhibit the characteristic weight and shape concerns described by older onset groups, and may have attitudes more similar to their healthy peers.

Finally, specific research hypotheses are generated to compare the experimental group with the adult standardisation sample and age matched control participants and the theoretical and clinical implications of the study are considered.
3.2 Early Onset Anorexia Nervosa

This section reviews the epidemiological findings about the nature and prevalence of early onset anorexia nervosa. The relationship between early and late adolescent or adult onset is considered, as well as the rationale for learning about the latter by studying the former.

3.2.1 Prepubertal and Intra-pubertal Onset.

Puberty is a complex process spanning two to three years with distinct developmental stages with a variation in timing from child to child (Tanner, 1962). So, when thinking about early onset anorexia nervosa, Russell (1992) recommends considering the onset in relation to pubertal process rather than simply age. He distinguishes true pre-pubertal anorexia nervosa where the eating disorder starts before the very first signs of puberty. In girls this means that the illness precedes the appearance of pubic hair and breast growth; in boys it precedes the first signs of genital growth.

For intra-pubertal anorexia nervosa, which Russell suggests is more common, the first signs of puberty will have appeared (breast growth in girls and genital enlargement in boys), but puberty will still be incomplete. In girls the eating disorder precedes the first menstrual period so this early form of anorexia nervosa has been called pre-menarcheal (Russell 1985). For the present study, Lask and Bryant-Waugh's definition of onset between ages 8 and 14 is adopted, irrespective of pubertal status.
3.2.2 Incidence and Prevalence

There have been several population-based studies including diagnostic data gathered by psychiatric case registers for anorexia nervosa - e.g. Kendell et al (1979) who found an incidence of 3.4 cases per 100 000.

A large study of the incidence of early onset anorexia nervosa was conducted by Rastam et al (1989) who screened the entire population of schoolchildren in Goteberg in Sweden using questionnaires, growth charts and individual school nurse reports. The population consisted of 4291 children, of whom 23 were found to have an eating disorder after full clinical assessment and interviews with the mothers. Of these, 17 were found to have full anorexia nervosa, 3 had a partial anorexia nervosa syndrome and 3 had bulimia nervosa.

The accumulated prevalence for anorexia nervosa (those who had or who had had the disorder) was 0.84%. This study is particularly strong methodologically as additional searches of local psychiatric and paediatric clinics were made to confirm that no new cases existed that had evaded the community detection process.

Comparable investigations of suspected high risk populations such as Crisp et al's (1976) survey of London private girls' schools found a prevalence of only 0.2% in girls aged 15 years or under. However another study by Szmukler (1983) of London private schools estimated a prevalence rate of 0.8% in girls aged 14 to 18.
There is perhaps a popular view that the incidence of eating disorders in the population as a whole has risen in recent years. This view has received some epidemiological support - for example, Szmukler et al's (1986) study of the Aberdeen case register found an incidence of anorexia nervosa of 1.6 per 100,000 population in 1966-1969 compared to an incidence of 4.06 per 100,000 population in 1978-1982.

However, as Lucas et al (1988) suggest in their similar findings from a study in Minnesota, much of this apparent increase could be accounted for by increases in the young adult female population over this time. They found that controlling for population increase and other intervening variables yielded no significant increase in the incidence rate of anorexia nervosa over a 45 year period.

### 3.2.3 Sex Ratio

Studies of anorexia nervosa in adolescents and adults have consistently shown that 90-95 percent of patients are female (Garfinkel and Garner, 1982). However, clinical series of children with eating disorders reveal more boys relative to the number of girls than would be expected. For example, Jacobs and Isaacs (1986) had six boys in their series of 20 cases with early pre-pubertal anorexia nervosa. Also, Fosson et al's (1987) series of 48 children with early onset anorexia nervosa included 13 males.

By contrast, in his review of seven series of early onset anorexia nervosa, Swift (1982) found an overall ratio of girls to boys of 9.5 to 1, a proportion similar to that commonly reported for older samples.
3.2.4 Social Class Bias

Fosson et al (1987) report that 22 of the 48 children included in their series came from social class I or II (as defined by the Registrar General's Classification of Occupations (1970)). In addition, Gowers et al (1991) report that 80% of the 30 children in their study with premenarcheal onset had Social Class I or II backgrounds.

However, some authors have suggested that for anorexia nervosa in general, this social class bias is becoming less pronounced over the past decade (Garfinkel and Garner, 1982). Equally, there are suggestions that middle class families are more able to access health services, though as Lask and Bryant-Waugh (1993) point out, this assertion has yet to be tested experimentally in the context of eating disorders.

3.2.5 Prognosis and Outcome

Hawley (1985) found that for 21 children aged 13 years or less at the onset of anorexia nervosa there was a good nutritional outcome for 67% of them, in that they had remained within 15% of average body weight for their size and age within the preceding six months. Similarly Bryant-Waugh et al (1988) obtained a good outcome in 50% of 34 children with a mean onset of 11.7 years at a mean follow-up time of 7.2 years post treatment. These authors operationalised a "good outcome" in terms of the patient regaining normal body weight. They also found that 17% continued to be underweight and 3% were overweight.

As Bryant-Waugh (1993) points out, the broad range of early onset
outcome findings is remarkably similar to those reported for older patients - for example Hsu's (1990) review of intermediate outcome in five studies yielded a range of normal weight regained in 50 - 62% of patients, 11 - 20% significantly underweight and 2 - 10% overweight.

Of those patients who fail to recover and remain chronically ill, a small number will not complete their puberty and so have a persistence of primary amenorrhoea, some reduction in stature and a failure of breast development (Russell, 1992). If these girls continue to gain weight, they may commence menstruation and become fertile, however it remains unclear whether every anorectic with long term primary amenorrhoea remains capable of reproductive recovery after weight gain. (Russell 1985).

3.2.6 Mortality

Mortality rates reported in the early onset literature have varied from 0% to 18%, although the majority indicate mortality between 0.5%. Swift (1982) estimated a mortality rate of 3.2% among the 186 patients accumulated in the seven studies he reviewed. However, as Russell (1992) points out, this figure needs to be interpreted cautiously as it is not expressed in terms of the average duration of the follow up or in terms of a standardised mortality ratio as proposed by Hsu (1990).

For individual series, Tolstrup et al 's (1985) follow up of 151 patients over the course of 12.5 years revealed a mortality rate of 6%; Bryant-Waugh et al found a mortality rate of 7% over their 7.2 years mean outcome.
As in the outcome literature for older age at onset patients, the main causes of death appear to be suicide, causes secondary to starvation and oesophageal rupture due to vomiting in descending order of frequency (Russell, 1992).

### 3.2.7 Early vs. Late Onset

The descriptive epidemiological data summarised above suggests that in many ways the early onset group are similar to their older cousins with eating disorders - at least in terms of prognosis and morbidity. As discussed elsewhere (Frampton, 1993), there are at least three robust differences between the early and late onset group revealed by research studies, with the latter more likely to have had adverse sexual experiences before the onset of their eating disorder, to have more severe eating pathology and for there to be a lower relative frequency of male sufferers.

Whether or not such differences amount to a distinct pathogenesis for the early onset disorder is in part a question to be investigated by the present study. The use of the Eating Disorder Examination (Fairburn and Cooper, 1987) will make it possible to directly compare the distribution of subscale scores for the two groups, to test the hypothesis that there will be a significant interaction between the groups and specific subscales (that the early onset group will have the same level of Restraint and Eating Concern as the adult standardisation sample, but significantly lower Weight and Shape Concern).

Bryant-Waugh and Kaminski (1993) conclude that the clinical features of children with anorexia nervosa are similar to those occurring in
older adults, with some obvious age-related differences such as the absence of amenorrhoea in pre-pubertal girls. They argue that the core disturbances in behavioural, psychological and physical terms are identical. With the recent development of the Eating Disorder Examination, it is now possible to test the first two components of this assertion experimentally and to speculate about the consequences for theories of eating disorder.

**Key Points:**

- Epidemiological and descriptive studies suggest that there are similarities between the early and late onset group;
- Authors have concluded that the core behavioural and psychological features are largely the same;
- Experimental investigation of this assertion is now possible as a single measure has been developed for use with children and adults.
3.3 Psychological Theories of Eating Disorders:

Having reviewed the epidemiological research into early onset anorexia nervosa, it is important to consider the theories that have been developed to account for the disorder. The present study posits a multifactorial model of predisposing, precipitating and perpetuating factors interacting in the pathogenesis and maintenance of eating disorders, based on Slade’s (1982) functional analysis model. Psychodynamic, Feminist Sociocultural, Cognitive - Behavioural and Systemic theories are all briefly reviewed before being linked by the multifactorial model.

Looking at each theory in turn reveals how little experimental testing has been done of the implicit assumptions or predicted outcomes generated by them. By contrast it is suggested that integrated psychological models that can account for different factors operating at different times and also generate testable hypotheses about differences between adults and children with and without eating disorders in terms of weight and shape concern.

3.3.1 Psychodynamic Theories

Drive-conflict

Early psychodynamic theories of eating disorders were based on Freud’s (1923/61) model of the mind as being made up of three distinct agencies: the id, the ego and the superego. When there is conflict between the impulses or drives of these agencies,
psychological symptoms were thought to result. For eating disorders, Goodsitt (1985) records a variety of early theories that self starvation is a defence against the sexual fantasies of oral impregnation or against oral sadistic and cannibalistic fantasies.

Alternatively, in their brief summary of early psychodynamic theories of eating disorder, Minuchin, Roseman and Baker (1978) suggest that "the psychological factors [of psychodynamic eating disorder theories] have a specific constellation centring around the symbolisation of pregnancy fantasies involving the gastro-intestinal tract." (p.14). They go on to conclude that by the late 1950s, psychodynamic theories had become so widespread that eating had been equated with gratification, impregnation, intercourse, performance, growing, castrating, destroying, engulfing, killing, and cannibalism, with food symbolising the breast, the genitals, faeces, poison, a parent, or a sibling.

Such a broad and inclusive theoretical conceptualisation of eating disorders meant that psychodynamic theories were limited in predicting outcome or generating experimental hypotheses. In addition, as Goodsitt (1985) points out, it is not clear how these broad psychodynamic theories predict the specific symptoms that are characteristic of sufferers of anorexia nervosa, in that they fail to account for "..disavowal of emaciation, delusions of fatness, body image misperception and alienation from inner feelings and sensations."

Object Relations Theory
In contrast to Freudian theory, Selvini-Palazzoli (1978) propounds a developmental theory of how the infant progresses through stages in relating to her mother. She suggests that the future anorectic has unresolved problems in the oral incorporation stage of normal development, which impedes the crucial stage of separation-individuation. The anorectic has a fantasy about an “oral incorporation of a maternal, bad, and overcontrolling object”. This “maternal introject” is then equated by the anorectic as being the same as her body.

Self-starvation thus becomes an attempt by the anorectic to end the feminisation of her body and to minimise her confused ambivalent identification with her mother. The theory suggests that the symptoms of anorexia nervosa result from distorted mental representations of body, self and object that the patient has generated from problems with her early object relations. In this way, object relations theories of eating disorders do lead to specific testable hypotheses (for example that patients with anorexia nervosa should have distorted mental representations). Unfortunately, there is no research evidence that patients confuse their body with their mother's body, or that they have fantasies of oral incorporation.
Self Psychology Theory

Self psychological theories of anorexia nervosa build on the concept of object relations in taking account of how the growing child tolerates separation through the development of specific mental functions and structures including the capacity to provide one's own cohesiveness, soothing, sense of well-being and security, tension and self esteem regulation.

In early life these functions are provided by the mother, and are then transferred to a transitional object, which provides a sense of well-being and security. Over time, these functions are internalised and become part of the child's developing mental structure. However, if this process goes wrong (because of inadequate or overbearing parenting) the child does not learn to provide their own internalised sense of security and self esteem.

Goodsitt (1985) describes such individuals as liable "to feel helpless, ineffective, overwhelmed, unworthy, unreal, incomplete, or empty." He goes on to suggest that these are characteristic features of young people with anorexia nervosa, who lack a reliable sense of self-regulation and thus feel out of control. The psychodynamic concept of loss of control and inadequate sense of self is echoed by Bruch (1978), who understands anorexia nervosa as an attempt to "undo the bodily aspects of adolescent changes... interrupting a development in which [the patient] feels troubled but incapable of making real changes." (p.58)
Her theory centres on the child’s early experience, where their “too good” mother anticipates their needs without the child needing to express them first, generating a sense of a lack of self control. The child no longer feels an active agent in her own development, but rather a helpless product of her parents and other social influences. Food is used as a reward or a response to other needs not related to hunger and as a result the child becomes confused about her bodily sensations and wary of trusting them.

Bruch argues that patients become afraid that they are just ordinary, and not good enough in others’ eyes. Because their lives are enmeshed, their many achievements are not for them but for others; they have a feeling of guilt for not living up to their own impossible standards. She suggests that anorexia nervosa develops in this situation because for such patients, "their own bodies become the arena for their only exercise of control." (p.58), a position echoed by feminist theorists.

3.3.2 Feminist Sociocultural Theory

Sociocultural theorists have pointed to the shift to thinness as a western cultural norm during the twentieth century (Garner et al 1980). Subsequently, feminist theorists have focused on the differential application of sociocultural norms to men and women. Cherzin (1983) suggests that ".the image of women that appears in the advertisement of a daily newspaper has the power to damage a woman's health, destroy her sense of well-being, break her pride in herself, and subvert her ability to accept herself as a woman."
Such theorists have argued from a feminist perspective that these differential norms apply to much more than desirable body image. Orbach (1986) consider gender roles, suggesting that many of the attributes associated with anorexia nervosa are essentially qualities of a woman's expected role. That is to say, women are socialised into deferring to others and restraining their own desires. She writes that: "In the most tortuous denial of need and dependency and the most persistent and insistent expression of independence, women with anorexia [nervosa] live out the contrariness of contemporary cultural dictates" (Orbach 1993. p9)

However, it is possible to argue that these theories, while clarifying the social context in which anorexia nervosa occurs, have insufficient explanatory power. Firstly, sociocultural theorists have suggested that anorexia nervosa is a late twentieth century phenomenon created by the pressures of modern life (e.g. Cherzin, 1983). However, as Brumberg (1988) points out, an historical analysis of eating disorder reveals cases throughout medical history and no clear evidence of a recent increase. Secondly, theories based on broad social forces fail to describe mechanisms by which some individuals develop eating disorders and other do not.
3.3.3 Cognitive Behavioural Theory

Garner and Bemis (1985) suggest that a cognitive-behavioural understanding of anorexia nervosa is not necessarily incompatible with the kinds of models discussed above that view the origins of the disorder in early development. They describe the cognitive model as a "proximal paradigm" that focuses on causal and maintaining factors. They argue that regardless of their theoretical orientation, most investigators would accept that at some stage in the pathogenesis of the illness causal factors converge at the patient's belief that it is absolutely essential to be thin.

Thus they suggest that an "overvalued idea" of the importance of weight and eating can account for the anorectic's apparently bizarre and irrational behaviour. Once maintenance of low weight and fear of loss of control over eating have been established as core beliefs, the typical behaviour of exercise, vomiting and the use of purgatives is maintained by the negative reinforcement of the removal of the aversive stimulus of fear of fatness.

In this way, the anorectic's behaviour serves to maintain a phobic avoidance of the feared outcome of weight gain. However, as Garner and Bemis (1982) point out, in contrast to classic phobic avoidance of a feared stimulus, the patient cannot escape from the phobic object since it is herself at higher weight levels. Since total escape is therefore impossible, control can only be maintained by constant vigilance. In addition, unlike other disorders of phobic avoidance, the anorectic may not want to be relieved of anxiety about food and
weight gain, since this fear is functional in assisting in the difficult task of oral self-restraint, despite voracious hunger.

Finally, they point out that anorexia nervosa can be distinguished from simple phobic disorders in that symptoms are positively as well as negatively reinforced. The ability to maintain a low weight and to be so "special" becomes a source of gratification in its own right. Social reinforcement cannot adequately account for this behaviour since the weight loss attained by most patients goes well beyond what is socially valued. Rather, the sense of mastery, self-control and competence derived from dieting becomes a powerful cognitive reinforcer in its own right. As Garner and Bemis (1985) put it: "The anorexic's extraordinary attempts to control her appetite provide a long-coveted sense of mastery within the context of lifelong feelings of incompetence."

This theory predicts that weight and shape concern will necessarily be the presenting psychopathology of eating disorders. However, Garner and Bemis (1985) themselves point out that they are not making any aetiological claims for the underlying psychopathology that led to the establishment of these overvalued ideas. While their theory is practical in the sense that it generates a clear rationale for a treatment approach, it has several limitations.

Firstly, the failure to account for underlying predisposing factors or triggers means that the theory has limited applicability in prevention or early intervention, since treatment cannot begin until weight loss has occurred.
Secondly, there is a danger of a logical error of assigning the thinking errors to core psychopathology rather than seeing them as a “final common pathway” of a range of other underlying factors. This tendency to overvalue the overvalued ideas leads to weight and shape concern being a necessary condition for a diagnosis of anorexia nervosa, when diagnostic systems (as discussed below) have by no means all agreed that this is the case. Such an approach may also undervalue the contribution of other predisposing and precipitating factors, particularly broader contextual and systemic factors.

3.3.4 Systemic Theories

In their introduction to a review of Minuchin's structural approach to therapeutic interventions with families with an anorectic child, Aponte and Hoffman (1973) focus on recurrent, systemic patterns of family interaction and consider how symptoms relate to that structure. Minuchin himself suggests that in such families, the child's psychosomatic symptoms play an important role in maintaining family homeostasis and blocking change. He identifies four key styles of family interacting that are characteristic of such 'psychosomatic' families: enmeshment, overprotectiveness, rigidity and conflict avoidance (Minuchin et al 1978)

Strober and Humphrey (1987) have attempted to test these family styles of interacting experimentally. Their work showed that families with a child with anorexia nervosa tend to be misattuned to the child's emerging affective needs, are less than optimally expressive in
the emotional domain, and may reinforce control and restraint at the expense of autonomous self-expression. However, as Bryant-Waugh and Lask (1995) point out, more work needs to be done to clarify how such systemic factors contribute to eating disorders, since not all families with such communication problems include an anorectic child.

3.3.5 Multifactorial Theories

Garner and Bemis (1985) refer to a number of earlier theorists in support of their assertion that anorexia nervosa cannot be seen as having a unitary cause, but rather that it is multidetermined and develops from the complex interaction of different factors.

This view is shared by Wren and Lask (1993), who distinguish predisposing, precipitating and perpetuating factors in the aetiology of anorexia nervosa. They review the research evidence for different factors operating at different stages in the illness: for example, the possibility that there may be underlying genetic or organic factors that predispose some young people to be more vulnerable to eating disorders; the types of family interaction that have been shown to be more common in families with children with eating disorders than in the general population.

One such integrated model of factors operating at different stages in the course of eating disorders has been proposed by Slade (1982), who begins from the position of attempting to explore eating problems as a secondary behavioural and cognitive response to a
more general set of psychological problems, rather than as a primary disorder.

His core formulation is that "given necessary setting conditions, initial dieting behaviour and weight loss is triggered by specific psychosocial stimuli: that if the dieting behaviour, once commenced, leads to feelings of success and 'being in control', it is reinforced both positively (by the resultant feeling of success/satisfaction) and negatively (through fear of weight gain and avoidance of other problems)."

His model comprises the following elements:

- general setting conditions:

  Slade proposes that general dissatisfaction with life and the self (low self esteem) and perfectionist tendencies are core psychological components. He points especially to child and adolescent developmental issues and the family life cycle as potential settings for these conditions (which would also accommodate psychodynamic and other systemic predisposing factors discussed above).

- need for complete control

  These combined setting conditions result in the individual needing to attain some total success in an area of their life. He points out that bodily functions are about the only area where children especially can attain total control, and goes as far as to argue that if the syndrome
were to be renamed a far more appropriate label would be “pathological self- and bodily control”.

- **specific psychosocial stimuli**

In the face of these setting conditions individuals are influenced by more or less innocuous sociocultural stimuli including media images and comments from peer group. In this way he is able to account for the fact that not all people exposed to these factors go on to develop an eating disorder; for this to occur, the general setting conditions must also be in place.

- **positive reinforcers**

Once the dieting behaviour is begun in response to specific psychosocial stimuli, it is reinforced for emerging anorectics by the context of the setting conditions, in a way that it is not for other dieters. Especially, “feelings of being control in the context of perceived failure in all other areas of functioning" is a potent reinforcer. Or, as Bruch (1978) put it: "..they take an extraordinary pride and pleasure in being able to do something so hard."

- **negative reinforcers**

Firstly, the anorectic's focus on losing weight as a successful behaviour increases the perceived aversive consequence of gaining weight. That is to say, success at losing weight generates a fear that any subsequent gain would signal a collapse of the sense of self
control. Like other two-stage avoidance learning strategies, it is powerfully reinforcing.

Secondly, the characteristic preoccupation with food, eating and weight size at the exclusion of thinking about anything else can be viewed as a form of avoidance behaviour; it enables the anorectic to avoid direct confrontation with all the aversive stimuli that set off the disturbed eating behaviour in the first place. Slade's model is presented diagrammatically in Figure 1. As Wren and Lask (1993) point out, his approach "shifts the wish to be thin from the centre of the phenomenological picture", especially for early onset anorexia nervosa. Equally, his focus on self-control is in concert with theorists from a range of perspectives who, according to Wren and Lask (1993), "..share an understanding of anorexia nervosa as essentially a syndrome of pathological self control."
Figure 1: Integrated formulation of anorexia nervosa (after Slade, 1982)
The aim of this research is to investigate a prediction arising from Slade's model that weight and shape concern may not be part of the core underlying psychopathology of anorexia nervosa, but may act as specific psychosocial stimuli or precipitating factors in launching an eating disorder. If this is the case, then it may also be true that the precipitating stimuli may be different for children compared to adults, and that weight and weight concerns will be different for different age groups.

Having explored the contribution of concepts of weight and shape concern to theories of eating disorders, research hypotheses will be proposed to test how they present in young people with anorexia nervosa compared to how they present in adults with anorexia nervosa and age matched control participants, and what this might tell us about the core psychopathology of eating disorders.

**Key Points:**

- Many theories have been developed to account for eating disorders, but few have generated testable predictions about presentation and progress of the illness;
- Integrated theories can account for different factors operating at different stages in the generation and maintenance of the disorder;
- Slade's theory proposes an underlying psychopathology that is not about weight or shape;
- The nature of proximal triggers may be different for children and
adults.
3.4 Weight Concern

Hsu and Sobkiewicz (1991) consider that weight phobia is "the sine qua non of anorexia nervosa", and conclude that this is the most intriguing aspect of the disorder since "..there is simply no easy explanation of this phenomenon; it is neither an obsessive thought nor a psychotic delusion."

By taking an historical perspective on the inclusion of the overvalued idea of the importance of weight in diagnostic systems, it is possible to show that it is a fairly recent addition to the symptomatology. Indeed, Hsu and Lee (1993) conclude that weight phobia did not emerge as a predominant motive for food refusal until around 1930.

In their review of the history of diagnoses of eating disorders, Beaumont, Garner and Touyz (1994) describe the various stages theories have been through in relation to weight phobia. As the originator of the concept of anorexia nervosa, the physician William Gull focused primarily on physical symptoms. He described anorexia nervosa exclusively in terms of slow pulse rate, skin changes and loss of menstrual periods, and made no mention of underlying psychopathology. By contrast, early work by Wissler in Germany stressed the importance of "Magersucht" or desire for thinness in making a diagnosis of anorexia nervosa (Beaumont et al, 1987). This early interest in psychopathology was revived in the early 1960s, with Bruch's (1961) formulation of anorexia nervosa as a relentless pursuit of thinness and fear of weight gain.
It is interesting to speculate why this particular feature has continued to appear in subsequent reformulations and diagnostic systems, rather than the additional features Bruch identified: body image disturbance, loss of awareness of interoceptive cues and an overwhelming feeling of ineffectiveness. It may be that the focus on the symptom of weight phobia at the exclusion of all others can be understood as a function of recent cognitive and social psychological theories, an hypothesis that will be developed and tested in this study.

It can be argued that Bruch's stance has been widely recruited by subsequent theorists: in 1970, Theander summarised his epidemiological studies of anorexia nervosa solely in terms of the patient's total preoccupation with thinness and body weight. There was no further elaboration of degree of emaciation, age of patient or physical symptom; if the patient had changed her attitude to eating and body weight, and this had caused significant weight loss, then a diagnosis of anorexia nervosa could be made.

3.4.1 Diagnostic Checklists for Anorexia Nervosa

**Dally (1969)**
- refusal to eat enough to maintain normal weight and/or sustained efforts to prevent ingested food from being absorbed.
- loss of at least 10% of previous body weight.
- amenorrhea of at least 3 months - or if menstruation had been irregular, a period of amenorrhea of at least 6 months.
- onset between age 12 and 39 years.
- no organic disease, serious affective disorder, or schizophrenia.

**Russell (1970)**
- behaviours aimed at achieving weight loss - starvation, vomiting,
laxative abuse.
b) an endocrine disorder - amenorrhea in the female and loss of sexual interest in the male.
c) a characteristic psychopathology, manifested by a morbid fear of becoming fat, is often accompanied by a distorted judgement by the patient of her own body size.
d) a specific degree of weight loss is required - 20% of standard body weight.

Feigner et al (1972)
a) onset prior to age 25 years
b) anorexia with weight loss of at least 25% of original body weight.
c) a distorted, implacable attitude towards eating, food, or weight that overrides hunger, admonitions, reassurance, or threats. This could include: denial of the illness with a failure to recognise nutritional needs; apparent enjoyment of weight loss and food refusal; a desired body image of extreme thinness; unusual handling or hoarding of food.
d) no known medical illness.
e) no other known psychiatric disorder.
f) at least two of the following: amenorrhoea, lanugo hair, bradycardia (persistent resting pulse of 60 or less).

Table 1: Diagnostic checklists for anorexia nervosa

As the three diagnostic checklists summarised in Table 1 clearly demonstrate, there was a considerable degree of disagreement between workers at this stage about what features constituted the core psychopathology of anorexia nervosa. In terms of the fear of weight gain/pursuit of thinness dimension, they differ in the necessity of this specific psychopathology: no mention of it by Dally; a necessary "morbid fear of becoming fat" by Russell; an optional "desired body image of extreme thinness" by Feighner and colleagues. This level of confusion is further compounded by the lack of specific operationalised factors necessary and sufficient to make a diagnosis, and also by further conflation with other concepts,
especially body image, which have of themselves proved problematical and are discussed elsewhere.

For the present description, it is reasonable to conclude that the early diagnostic checklists added to rather than clarified the range of assumptions and symptoms needed for a diagnosis of anorexia nervosa.

### 3.4.2 Diagnostic Checklists for Early Onset Anorexia Nervosa.

This level of disagreement has been closely mirrored in the literature on childhood onset anorexia nervosa. As Table 2 details, checklists developed for child populations have been equally heterogeneous in terms of necessary and sufficient conditions.

**Bryant-Waugh et al (1988):** Great Ormond St criteria
1. determined food avoidance;
2. weight loss or failure to gain weight during the period of preadolescent accelerated growth (10 - 14 years) and the absence of any physical or mental illness;
3. any two or more of the following: preoccupation with body weight, preoccupation with energy intake, distorted body image, fear of fatness, self-induced vomiting, extensive exercising, laxative abuse

**Gowers et al (1990):** St George’s criteria
a) phobic avoidance of normal body weight
b) amenorrhoea
c) weight loss to below 85% of mean matched population weight
d) avoidance of weight gain through abstinence, vomiting and/or laxative abuse
e) excessive exercising, rituals and social avoidance

**Jacobs and Isaacs (1986):** The Maudsley criteria:
a) A substantial loss of weight resulting from food avoidance, often accompanied
by excessive exercise
b) an exaggerated and morbid fear of becoming fat
c) no evidence of secondary sexual characteristic development

Table 2: Diagnostic systems for early onset anorexia nervosa
3.4.3 Diagnostic Systems

Beumont et al (1994) begin with the assertion that: "with the best will in the world, it is difficult not to become disillusioned with the diagnostic system for eating disorders." They use the example of a 16 year old female presenting with a variety of eating disorder symptoms, and explore how she would have received different diagnoses in different countries at different times.

A review of recent diagnostic systems (see Appendix 1) reveals how the emphasis has changed even in recent times. Focusing on the broad dimension of weight concern, ICD-9 (WHO, 1978) describes "unusual eating habits and attitudes towards food" but does not require specific overvalued ideas about weight.

However by 1980 DSM-III (APA, 1980) required (as its first condition), an intense fear of becoming obese (technically shape concern, though linked to weight as the fear "does not diminish as weight loss progresses"). DSM-III-R (APA, 1987) linked the concepts of weight and shape concern more specifically in requiring an "intense fear of gaining weight or becoming fat, even though underweight."

DSM-IV (APA, 1993) by contrast makes a clearer distinction between weight and shape concern by requiring either "intense fear of gaining weight or becoming fat even though underweight" as well as "disturbance in the way in which one's body weight or shape is experienced; undue influence of weight or shape on self-evaluation."
ICD-10 (WHO, 1992) appears to conflate both weight and shape concern in one statement by requiring that "there is a body image disturbance 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."

Thus even since 1978 there has been much disagreement about the nature and centrality of weight concern in the diagnosis of anorexia nervosa. One of the reasons for this is proposed by Beaumont et al (1994), who point out that the eating disorders are good examples of syndromal diagnoses, "where labels are applied to a set of symptoms and signs that appear to be related to each other in a fairly consistent way and that point to a common means of treatment." (emphasis added).

The important word in their definition is "appear", which emphasises a practical clinical focus on making a diagnosis based on the presenting symptoms and getting on with some treatment. However, this approach fails to consider how the presenting symptoms may relate to underlying psychopathology, or indeed to offer any theoretical account of what causes eating disorders. In Garner and Bemis's (1985) terms as discussed above, this "proximal paradigm" is helpful for the busy clinician in giving an immediate focus for treatment.

It may be difficult to get away from placing weight concern at the centre of eating pathology, since it appears casually to "make sense" of why someone stops eating. However, this heuristic reliance on
everyday explanations may be at the expense of having a more complete psychological theory to work from. The following section explores how the related concept of shape concern also has a questionable pedigree in syndromal terms.

**Key Points:**

- weight concern is a relatively recent symptom historically;
- diagnostic systems have differed in making weight concern a necessary or optional symptom;
- weight concern is often recruited as a proximal symptom without any underlying theory to account for the pathogenesis of the disorder.
3.5 Shape Concern

In the same way that weight concern has changed over time and in different diagnostic systems, so too has the construct of shape concern, as exemplified by body image disturbance. A detailed review of the history of this symptom in diagnostic systems would reveal it to be just as variable as the history of weight concern reviewed in the previous section.

As Hsu and Sobkiewicz (1991) point out, the concept itself seems to encompass two distinct definitions: firstly a neurological concept of body schema (some internal representation of one's body); secondly, a psychological concept of feelings and attitudes towards one's body.

Studies focusing on the neurological concept have investigated perceptual distortions in estimating body width using a variety of methodologies. One example is silhouette-card sorting, where the participant sorts through a series of body outlines that are very thin to very fat, and ranks them according to how closely each card matches her own perception of her body size.

The second type of study, adopting a more psychological interpretation of body image disturbance, have attempted to measure attitudes and affect towards the body using a variety of questionnaires, including the Eating Disorder Inventory and more specific body parts satisfaction questionnaires.

Results of 19 perceptual-type studies of body image disturbance
evaluated by Hsu and Sobkiewicz (1991) yield mixed results depending on the perceptual methodology employed. In summary, they found that some patients with anorexia nervosa overestimate their body widths more so than did normals but that this overestimation is not characteristic. Therefore perceptual body width disturbance cannot be pathognomic for eating disorders (a point already argued by Hsu (1982)).

The studies focusing on the psychological components of attitudes and affect towards the body revealed that on average patients with anorexia nervosa are dissatisfied with their body and want to be thinner more so than control participants. However, many of the control participants were equally dissatisfied with their body as patients with anorexia nervosa and wanted to be thinner.

As Hsu and Sobkiewicz conclude: "it is unclear why the normals who demonstrate bodily dissatisfaction do not develop (or, at least, have not developed) an eating disorder." The authors conclude that there is no evidence to support the interchangeable use of "overestimation of body size" and "disturbance of body image", and that neither concepts are pathognomic for eating disorders. They suggest that the studies into the perceptual overestimation of body size have not helped us to understand better the psychopathology of eating disorders. They recommend that it is time to abandon the term "disturbance of body image".

Their conclusion has been echoed by Bowden et al (1989), who suggest that research into body image distortion has itself become so
distorted that it is no longer possible to make meaningful comparisons between studies. They demonstrated that three different techniques for estimating body size (Image Marking, Visual Size Estimation, and Distortion Video techniques) gave different results, implying that research findings can no longer be cross-compared.

Despite these findings, as the section above on diagnostic systems reveals, "body image disturbance" continues to be recruited as a characteristic symptom of an eating disorder. It also has broad casual appeal to sociocultural theorists who formulate eating disorders in terms of societal pressures to attain a certain body size and shape.

As Hsu and Sobkiewicz (1991) point out, though, since this pressure cannot be pathognomic for the disorder (or everyone would have an eating disorder), further psychological explanation is required to account for why this particular individual has an eating disorder rather than someone else.

**Key Points:**

- shape concern has a variable history in diagnostic systems;
- as it is not necessary for or exclusive to eating disorders it cannot be pathognomic;
- the concept is difficult to define and contains perceptual, cognitive and affective components.
3.6 Introduction Summary

The review outlined above indicates that theories about eating disorders are poorly linked to diagnostic systems, which in turn have begun from a medical bias of making syndromal diagnoses based on presenting symptoms. Also, a range of theoretical approaches agree that the development of an eating disorder may be a late 'final common pathway' launched by a precipitating factor in the context of a range of underlying predisposing factors.

In positing a "proximal paradigm", Garner and Bemis (1985) argue that the excess value the patient places on weight and shape as a means of self evaluation would be a good candidate for this kind of precipitating factor. It also has the benefit of being a useful place to start treatment, and can account for the range of bizarre behaviours and heroic self control effort that the patient exhibits. Such models concur with Slade's (1982) approach that identifies distinct underlying problems as setting factors for eating disorders: a general dissatisfaction with life and the self (low self esteem) and perfectionist tendencies. If such psychological stressors cannot be articulated (perhaps because of temperament or dysfunctional family communication style), Slade argues that they can go on to launch an eating disorder in the context of specific psychosocial stimuli (the exhortation to be thin and media images that surround all of us).

However, at the very least Slade's model allows for the nature of these triggering factors to be different for adults and children; that the kinds of psychosocial and psychosexual pressures on young
women are qualitatively different to those on the younger children who make up the early onset group. If this is the case, then the weight and shape concern described by children in the early onset group should also be different to that described for adolescent and adult onset groups.

In addition, it is also possible that the triggering factor that launches an eating disorder may not be in the form of a comment about the child's weight, or in response to peer and social pressure about the importance of being thin and attractive. There may be other reasons why children may begin not eating - for example because they are feeling depressed. Appetite loss secondary to depression has been identified as a common feature of early onset anorexia nervosa (Fosson et al, 1987) and the broader relationship between mood disorder and anorexia nervosa has been extensively covered (e.g. Cantwell et al, 1977).

If a child in a "high risk" group in terms of predisposing factors stops eating for any of a number of reasons and by doing so discovers a source of self control "in the context of perceived failure in all other areas of functioning" (Slade, 1982), this may serve as a sufficient triggering factor in launching an eating disorder.

Two possibilities, then, are presented about the nature and meaning of weight and shape concern in the pathogenesis of early onset anorexia nervosa: either that it is qualitatively different to the adolescent and adult onset group (because psychosocial and especially psychosexual pressures are different) or that it could be
absent (and replaced by another triggering factor).

Either possibility would account for at least some of the children in the early onset group not exhibiting characteristic weight and shape concern (Cooper et al, 1994). It would also be based on the theoretical, historical and research evidence that neither concepts of body image disturbance nor fear of weight gain can be justified as necessary symptoms for a diagnosis of anorexia nervosa.

This study aims to investigate whether such proposed differences in weight and shape concern can be demonstrated between early and later onset groups. If this is shown to be the case, then the rigid adherence to overvalued ideas about weight and shape in the diagnosis of eating disorders may itself represent an overvalued idea that has the characteristic features of being extreme, rigid and imbued with great significance (Cooper and Fairburn, 1987) for diagnosticians and theorists.

3.6.1 Research Aims and Design

The development of the child version of the Eating Disorder Examination (an investigator-led interview that yields four subscales: Restraint, Eating Concern, Weight Concern and Shape Concern) allows for direct comparisons to be made between children with anorexia nervosa and age-matched controls as well as for comparisons to be made between the adult and early onset groups. The present study uses a two sample independent groups design to compare a sample of children with a diagnosis of anorexia nervosa with a control sample.
of age-matched normal participants using the child version of the Eating Disorder Examination. This design is repeated to compare a further sample of children with diagnoses of other eating pathology against age-matched controls, and against the anorexia nervosa sample. A single sample design is used to compare the anorexia nervosa sample with the adult standardisation sample described by Cooper et al (1989).

The research hypothesis predicts that there will be a significant interaction between the anorexia nervosa sample, the eating pathology sample and the control sample on the four subscales of the CH-EDE. Specifically, the hypothesis predicts that the anorexia nervosa sample will have significantly higher scores than the control sample (but not the eating pathology sample) on subscales of Restraint and Eating Concern, and that all groups will have similar scores on subscales of Shape Concern and Weight concern. In addition, it is hypothesised that the anorexia nervosa sample will similar levels of Restraint and Eating Concern, though significantly lower weight and shape concern, when compared with the adult standardisation sample.

The study also provides an opportunity to assess the discriminant validity of the child version of the Eating Disorder Examination by the comparison of the performance of the clinical sample and age matched controls against clinical diagnoses and to test its interrater reliability.
4. **Method**

4.1 **Participants**

A series of 30 attenders at the outpatient Eating Disorder Clinic at the Hospital for Sick Children, Great Ormond St, London participated in the study, which formed the pilot phase of a three year Medical Research Council investigation into the nosology of eating disorders in children. I attended the Clinic at the hospital weekly for 7 months and collected data from 80% (n=24) of the participants. The remaining participants (n=6) were interviewed by other members of the Eating Disorder Research Team.

Ethical approval for the study was gained from the Ethics Committee of the Hospital for Sick Children NHS Trust as a component of the main MRC study and from the Department of Psychology at the University of Exeter (see Appendix 2). I also held an honorary contract as a Research Associate at the Hospital for Sick Children for the duration of the study.

All attenders were contacted by letter, inviting them to participate in the study while they were attending the clinic (see Appendix 3). Informed consent was sought from parents and the young person themselves; families were encouraged to ask further questions about the study before agreeing to participate by signing the research consent form.

Potential participants who did not consent to the research study were excluded. Other attenders were excluded from the study if the clinical team thought that participation would be detrimental to their care. Finally, children with an onset when aged less than 8 years or older than 14 years were excluded. The mean age, gender and matched population weights of the sample are described in Table 1 and 2 in the results section.
Participants were assigned to one of the two experimental groups on the basis of diagnosis made by the Consultant Child Psychiatrist independently of the young person's performance on the child version of the Eating Disorder Examination (CH-EDE). Clinic attenders meeting the criteria for the Great Ormond Street Diagnostic Checklist for Early Onset Anorexia Nervosa were assigned to the Early Onset Anorexia Nervosa Group. The remaining attenders (who could not be diagnosed as suffering from Early Onset Anorexia Nervosa) were assigned to the second experimental group of Other Eating Pathology. This second group was made up of children with a diagnosis of Food Avoidance Emotional Disorder (Higgs, Goodyer and Birch, 1988) or Selective Eating (Bryant-Waugh and Kaminski, 1993).

Female Control participants were recruited from an independent school in Bristol. Permission to undertake the study was sought from the Head Teacher and governors, as well as from the pastoral Head of the Middle School. Parents of all 41 girls in the Middle School were contacted by post to give them further information about the study and to encourage them to make contact to discuss any concerns (see Appendix 4). They were encouraged to opt their child out of the study if they had any reservations. In the event two parents made contact to discuss the study in more detail, and one of these withdrew her child from the study. One child was away from school during the whole period of interviewing, so 39 girls were included in the control group. Four male participants were recruited individually from the families of colleagues and friends in the Bristol area.

The final participants in the control group (n=30) were selected from the total number of interviews conducted (n=43) on the basis of match for age as closely to the experimental group as possible, and also for gender.
4.2 Materials

All participants were interviewed using the child version of the Eating Disorder Examination (CH-EDE), a semi-structured interview schedule that has been standardised in its original adult version on a sample of 47 adult females with anorexia nervosa, 53 adult females with bulimia nervosa and 42 matched controls (Cooper and Fairburn, 1987). The adult version of the scale has good discriminant validity and internal consistency of subscales of Restraint, Eating Concern, Weight Concern and Shape Concern.

The CH-EDE has been developed from the adult version by modifying some of the language and introducing a sorting task to assess importance of weight and shape in self-concept (see Appendix 5). An initial pilot study found that this slightly modified version of the EDE can be of use in children and is sensitive to the key psychopathological features (Bryant-Waugh et al, 1996). Training on the administration of the measure was provided by Dr. Rachel Bryant-Waugh who was responsible for the development of the child version.

Participants also completed a diary of events over the previous four weeks (in conjunction with parents before attending for clinic attenders or while waiting for interview for control participants) to help orientate them to the period being examined.

4.3 Procedure

Participants attending the Eating Disorders Clinic at the Hospital for Sick Children were seen individually for interview using the CH-EDE as part of their assessment package. Participants were seen in a quiet interview room immediately after their break for lunch during assessment. Parents and other family members remained in the waiting room.
while the child was seen alone. Having introduced myself and told the participant that I would be asking them some questions, I explained the rationale for the study using a standard rubric (see Appendix 6).

At the end of the interview participants were again thanked for taking part in the study, and returned to the waiting room. Weight and height scores were taken from the record of the patient's visit to the hospital's Growth Clinic in the morning and a standard composite weight for height ratio (taking into account the expected height and weight for the child's age) was generated using a specially written spreadsheet computer program (see Appendix 7).

The interview was summarised in a standard format (see Appendix 8 for an example) and reported back at the case conference to the rest of the clinical team to compare clinical diagnosis with results attained from the CH-EDE. Interview summaries were appended to the clinical notes for future reference.

A random sample of 10 clinic attenders were also enrolled into the interrater reliability study for the CH-EDE. These participants were also scored by Beth Watkins, Research Assistant in the Eating Disorders Programme (who is trained on the use of the CH-EDE by the Oxford Group), who either sat in on the interview and co-rated in vivo, or used an audio tape of the interview for participants who agreed to this.

The Control participants were asked a subset of the CH-EDE omitting the items that are only used for diagnostic purposes and do not contribute to subscale scores. This subset also avoided asking the questions about vomiting and laxative abuse that the school agreed would not be appropriate to ask.

Control participants were seen during lesson time in a quiet room in the Sick Bay of the
school. The purpose of the study was introduced using a standard rubric (see Appendix 9) to stress that they were being seen as normal controls to help us care for the patients at the hospital. A system for alerting the school medical officer and pastoral system about any participants exhibiting caseness for eating pathology was agreed in advance and described to parents in their information letter. At the end of the interview control participants were presented with a certificate in recognition of their contribution to the study (see Appendix 10).
5. Results

The results section below details a descriptive analysis of the data set to compare the samples on demographic factors of age and weight. A power analysis of the data set confirms that the sample sizes are sufficiently large to enable further statistical analyses to be made. Further tests are then made to explore the items and subscales, and to compare the samples in the light of the experimental hypotheses.

5.1 Descriptive Data Analysis

The data set analysed below consists of a Clinical Sample (n=30) made up of two groups: The Anorexia Nervosa Sample - the group of young people attending the clinic who were diagnosed independently of their Eating Disorder Examination score as having anorexia nervosa (n=15); and the Eating Pathology Sample - the group attending the clinic who received a diagnosis of Food Avoidance Emotional Disorder or Selective Eating (n=15).

The control participants (n=43) were assigned to Control Sample 1 (n=15) or Control Sample 2 (n=15) on the basis of the closest age match with the clinical samples. The remainder (n=13) were discounted from further analyses. Summaries of individual responses are contained in Appendix 11.

Table 1 below compares the samples in terms of the age of participants. Probabilities associated with the Student’s t test for independent samples with unequal variance show that the Anorexia Nervosa sample are significantly older than the Eating Pathology sample (t(28) = 2.26, p<0.05) and that they are not significantly different in age from their own control sample Control 2 (t(28) = 0.29, ns). A similar comparison between the Eating Pathology sample and their control sample Control 1 show that
these can also be said to come from the same population (t (28) = 0.84, ns).

<table>
<thead>
<tr>
<th>Group</th>
<th>Number</th>
<th>mean</th>
<th>st.dev</th>
<th>Eating Path.</th>
<th>Control 1</th>
<th>Control 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anorexia</td>
<td>15</td>
<td>12.49</td>
<td>1.28</td>
<td>2.26*</td>
<td>0.29</td>
<td></td>
</tr>
<tr>
<td>Eating Path.</td>
<td>15</td>
<td>10.84</td>
<td>2.35</td>
<td></td>
<td>0.84</td>
<td></td>
</tr>
<tr>
<td>Control 1</td>
<td>15</td>
<td>11.39</td>
<td>0.45</td>
<td>0.84</td>
<td>5.96</td>
<td></td>
</tr>
<tr>
<td>Control 2</td>
<td>15</td>
<td>12.39</td>
<td>0.47</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Table 1: Descriptive data analysis - age of samples**

* p <0.01

**p<0.001

Table 2 shows that the Anorexia Nervosa sample are significantly lighter than both the Eating Pathology sample (t(28) = 3.92, p<0.001) and their control sample Control 2 (t(28) = 5.92, p<0.001). There is however no significant difference in weight between the Eating Pathology sample and the expected weight for their control group (t(28) = 0.94, ns).

<table>
<thead>
<tr>
<th>Group</th>
<th>Number</th>
<th>mean</th>
<th>st.dev</th>
<th>T Test</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eat.Path</td>
<td>15</td>
<td>78.69</td>
<td>1.28</td>
<td>3.92**</td>
</tr>
<tr>
<td>Control 1*</td>
<td>15</td>
<td>97.00</td>
<td>2.35</td>
<td>0.94</td>
</tr>
<tr>
<td>Control 2*</td>
<td>15</td>
<td>100.00</td>
<td>0.45</td>
<td></td>
</tr>
</tbody>
</table>

**Table 2: Descriptive data analysis - weight of samples**

**p<0.001

note 1: weight for control samples extrapolated from tables of expected weight and height (Tanner et al, 1966).

5.2 Power Analysis

Using a conservative critical effect size of 2 units for the subscale scores (based on the adult standardisation sample reported by Cooper et al (1989)), Glass’s effect size for
any two samples is calculated as $\Delta = 0.739$ (see Appendix 12). From tables of critical effect size (Kraemer and Thiemann, 1987), this figure predicts that a total sample size of 28 gives 99% power for a 1% confidence level one-tailed test. Therefore all pair-wise sample statistics are computed from two samples of $n=15$, giving a conservative total sample size of $n=30$. 
5.3 Item Analysis

The mean individual item scores for the combined Clinical sample compared to the combined Control sample distinguish between the two groups in the direction predicted from the adult sample (Figure 1.). In contrast to the adult standardisation sample described by Cooper et al (1989), eleven of the items do not distinguish between the two samples at a statistically significant level. Mean differences for restrained eating, avoidance of eating, food avoidance, losing control, eating in secret, dissatisfaction with weight, desire to lose weight, reaction to proscribed weighing, feeling fat and flat stomach do not achieve significance at the 0.05 level (Table 3). Two items (eating in secret and desire to lose weight) have greater mean item scores for the Control sample than the Clinical sample.

<table>
<thead>
<tr>
<th></th>
<th>Clinical</th>
<th>Controls</th>
<th>T Test</th>
<th>significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>restrained eating</td>
<td>1.87</td>
<td>1.13</td>
<td>1.06</td>
<td>ns</td>
</tr>
<tr>
<td>avoidance of eating</td>
<td>0.77</td>
<td>0.23</td>
<td>1.76</td>
<td>ns</td>
</tr>
<tr>
<td>empty stomach</td>
<td>1.60</td>
<td>0.27</td>
<td>2.44</td>
<td>p&lt;0.05</td>
</tr>
<tr>
<td>food avoidance</td>
<td>1.97</td>
<td>1.07</td>
<td>1.26</td>
<td>ns</td>
</tr>
<tr>
<td>dietary rules</td>
<td>1.40</td>
<td>0.43</td>
<td>1.88</td>
<td>ns</td>
</tr>
<tr>
<td>Preoccup with food</td>
<td>1.63</td>
<td>0.10</td>
<td>3.17</td>
<td>p&lt;0.01</td>
</tr>
<tr>
<td>losing control</td>
<td>0.93</td>
<td>0.37</td>
<td>1.41</td>
<td>ns</td>
</tr>
<tr>
<td>social eating</td>
<td>1.57</td>
<td>0.43</td>
<td>2.27</td>
<td>p&lt;0.05</td>
</tr>
<tr>
<td>eating in secret</td>
<td>0.20</td>
<td>0.33</td>
<td>0.60</td>
<td>ns</td>
</tr>
<tr>
<td>guilt re eating</td>
<td>1.53</td>
<td>0.43</td>
<td>2.41</td>
<td>p&lt;0.01</td>
</tr>
<tr>
<td>diss with weight</td>
<td>1.80</td>
<td>0.83</td>
<td>1.77</td>
<td>ns</td>
</tr>
<tr>
<td>desire to lose wt</td>
<td>1.13</td>
<td>1.23</td>
<td>0.25</td>
<td>ns</td>
</tr>
<tr>
<td>react to weighing</td>
<td>1.70</td>
<td>0.87</td>
<td>1.86</td>
<td>ns</td>
</tr>
<tr>
<td>diss with shape</td>
<td>1.83</td>
<td>0.67</td>
<td>2.83</td>
<td>p&lt;0.01</td>
</tr>
<tr>
<td>preoccup wt/shape</td>
<td>1.67</td>
<td>0.33</td>
<td>2.76</td>
<td>p&lt;0.01</td>
</tr>
<tr>
<td>import of shape</td>
<td>2.40</td>
<td>0.50</td>
<td>3.57</td>
<td>p&lt;0.001</td>
</tr>
<tr>
<td>import of weight</td>
<td>1.87</td>
<td>0.33</td>
<td>3.17</td>
<td>p&lt;0.01</td>
</tr>
<tr>
<td>fear of wt gain</td>
<td>2.13</td>
<td>0.23</td>
<td>3.70</td>
<td>p&lt;0.001</td>
</tr>
<tr>
<td>seeing body</td>
<td>1.97</td>
<td>0.73</td>
<td>2.52</td>
<td>p&lt;0.01</td>
</tr>
<tr>
<td>avoid exposure</td>
<td>1.67</td>
<td>0.70</td>
<td>1.98</td>
<td>p&lt;0.05</td>
</tr>
<tr>
<td>feeling fat</td>
<td>1.67</td>
<td>1.47</td>
<td>0.27</td>
<td>ns</td>
</tr>
<tr>
<td>flat stomach</td>
<td>1.83</td>
<td>0.87</td>
<td>1.66</td>
<td>ns</td>
</tr>
</tbody>
</table>

Table 3: Mean item scores and probability of significant difference for Clinical sample vs Control sample.
Figure 1: Mean individual items scores for combined Clinical vs. Control samples

Figure 2. below indicates that the modal response made by all of the samples to all items was zero. This skew in the overall data set is an important factor to be taken into account in justifying the statistical tests adopted (see appendix 12).
5.4 Discriminant Validity

Participants were assigned to either the Positive CH-EDE Diagnosis or Negative CH-EDE Diagnosis group on the basis of responses to individual items contributing to the criteria contributing to a CH-EDE diagnosis of anorexia nervosa developed by Fairburn and Cooper (1993) (see Appendix 14). Clinical participants were independently rated by the Consultant Psychiatrist responsible for the Clinic to assess their caseness for anorexia nervosa, based on the Great Ormond St. checklist, giving the diagnostic matrix shown in Table 4.

<table>
<thead>
<tr>
<th></th>
<th>Positive clinical diagnosis</th>
<th>Negative clinical diagnosis</th>
</tr>
</thead>
<tbody>
<tr>
<td>Positive CH-EDE Diagnosis</td>
<td>7</td>
<td>5</td>
</tr>
<tr>
<td>Negative CH-EDE Diagnosis</td>
<td>8</td>
<td>44</td>
</tr>
</tbody>
</table>
Table 4: diagnostic matrix for all samples

5.5 Subscale Analysis

Items were grouped into the subscales derived by Cooper et al (1989) - Restraint, Eating Concern, Weight Concern and Shape Concern. The Cronbach Alpha coefficient for each subscale, calculated using the full data set of 60 participants, were 0.80, 0.91, 0.90 and 0.88 respectively.

<table>
<thead>
<tr>
<th>Subscales</th>
<th>Restraint</th>
<th>Eating</th>
<th>Weight</th>
<th>Shape</th>
</tr>
</thead>
<tbody>
<tr>
<td>Restraint Items</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>restrained eating</td>
<td>0.88</td>
<td>0.78</td>
<td>0.90</td>
<td>0.92</td>
</tr>
<tr>
<td>avoidance of eating</td>
<td>0.70</td>
<td>0.65</td>
<td>0.70</td>
<td>0.68</td>
</tr>
<tr>
<td>empty stomach</td>
<td>0.87</td>
<td>0.79</td>
<td>0.76</td>
<td>0.79</td>
</tr>
<tr>
<td>food avoidance</td>
<td>0.93</td>
<td>0.85</td>
<td>0.83</td>
<td>0.84</td>
</tr>
<tr>
<td>dietary rules</td>
<td>0.86</td>
<td>0.83</td>
<td>0.81</td>
<td>0.80</td>
</tr>
<tr>
<td>mean</td>
<td>0.85</td>
<td>0.78</td>
<td>0.80</td>
<td>0.81</td>
</tr>
<tr>
<td>Eating Concern</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>items</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>preoccup with food</td>
<td>0.73</td>
<td>0.85</td>
<td>0.68</td>
<td>0.72</td>
</tr>
<tr>
<td>losing control</td>
<td>0.70</td>
<td>0.74</td>
<td>0.64</td>
<td>0.63</td>
</tr>
<tr>
<td>social eating</td>
<td>0.71</td>
<td>0.86</td>
<td>0.61</td>
<td>0.66</td>
</tr>
<tr>
<td>eating in secret</td>
<td>0.15</td>
<td>0.29</td>
<td>0.19</td>
<td>0.14</td>
</tr>
<tr>
<td>guilt re eating</td>
<td>0.83</td>
<td>0.81</td>
<td>0.91</td>
<td>0.84</td>
</tr>
<tr>
<td>mean</td>
<td>0.62</td>
<td>0.71</td>
<td>0.61</td>
<td>0.60</td>
</tr>
<tr>
<td>Weight Concern</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>items</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>diss with weight</td>
<td>0.81</td>
<td>0.74</td>
<td>0.86</td>
<td>0.80</td>
</tr>
<tr>
<td>desire to lose wt</td>
<td>0.64</td>
<td>0.53</td>
<td>0.76</td>
<td>0.63</td>
</tr>
<tr>
<td>react to weighing</td>
<td>0.64</td>
<td>0.68</td>
<td>0.76</td>
<td>0.72</td>
</tr>
<tr>
<td>preoccup wt/shape</td>
<td>0.90</td>
<td>0.86</td>
<td>0.86</td>
<td>0.88</td>
</tr>
<tr>
<td>import of weight</td>
<td>0.77</td>
<td>0.76</td>
<td>0.87</td>
<td>0.84</td>
</tr>
<tr>
<td>mean</td>
<td>0.75</td>
<td>0.71</td>
<td>0.82</td>
<td>0.78</td>
</tr>
<tr>
<td>Shape Concern</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>items</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>seeing body</td>
<td>0.79</td>
<td>0.71</td>
<td>0.81</td>
<td>0.86</td>
</tr>
<tr>
<td>avoid exposure</td>
<td>0.76</td>
<td>0.67</td>
<td>0.75</td>
<td>0.76</td>
</tr>
<tr>
<td>feeling fat</td>
<td>0.67</td>
<td>0.57</td>
<td>0.71</td>
<td>0.72</td>
</tr>
<tr>
<td>flat stomach</td>
<td>0.70</td>
<td>0.67</td>
<td>0.70</td>
<td>0.78</td>
</tr>
<tr>
<td>diss with shape</td>
<td>0.83</td>
<td>0.83</td>
<td>0.87</td>
<td>0.88</td>
</tr>
<tr>
<td>import of shape</td>
<td>0.76</td>
<td>0.72</td>
<td>0.82</td>
<td>0.85</td>
</tr>
<tr>
<td>preoccup wt/shape</td>
<td>0.90</td>
<td>0.86</td>
<td>0.86</td>
<td>0.88</td>
</tr>
<tr>
<td>fear of wt gain</td>
<td>0.78</td>
<td>0.81</td>
<td>0.81</td>
<td>0.89</td>
</tr>
<tr>
<td>mean</td>
<td>0.78</td>
<td>0.73</td>
<td>0.79</td>
<td>0.83</td>
</tr>
</tbody>
</table>

Table 5: Correlation of individual items with mean subscale totals
Table 5 records the individual and mean correlation of subscale items with their own subscale totals compared to other subscales as a measure of internal consistency. The results indicate that all items with the exception of restrained eating (Restraint subscale), guilt re eating (Eating Concern subscale) and preoccupation with weight/shape (Weight Concern and Shape Concern subscales) correlate more highly with their own subscale totals than with others.

### 5.6 Hypothesis Testing

The research design established an hypothesis that there would be an interaction between the samples and individual subscale scores. In order to test this hypothesis, a multivariate analysis of variance was performed on the total data set of mean subscale scores for all samples (recorded at Appendix 13). The analysis did not reveal a significant interaction between samples and subscales ($F(9,168) = 0.73$ N.S.). However there is a significant main effect for group ($F(3,56) = 8.12$ p<0.001), which is also reflected in Figure 3.

![Figure 3: Mean subscale scores by sample](image)
Further univariate ANOVAs were therefore performed to compare the Clinical and Control samples, and are recorded in Tables 6 - 8. Table 6 shows that there are significant differences between the Anorexia Nervosa and Eating Pathology samples on all subscales at a significance level of at least $p<0.05$.

<table>
<thead>
<tr>
<th>Subscale</th>
<th>Anorexia Nervosa Mean</th>
<th>St Dev.</th>
<th>Eating Pathology Mean</th>
<th>St Dev.</th>
<th>F value</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Restraint</td>
<td>2.43</td>
<td>1.97</td>
<td>0.80</td>
<td>1.95</td>
<td>5.1312</td>
<td>0.0314</td>
</tr>
<tr>
<td>Eating Concern</td>
<td>1.74</td>
<td>1.44</td>
<td>0.46</td>
<td>1.12</td>
<td>7.2348</td>
<td>0.0119</td>
</tr>
<tr>
<td>Weight Concern</td>
<td>2.35</td>
<td>1.69</td>
<td>0.93</td>
<td>1.84</td>
<td>5.1303</td>
<td>0.0314</td>
</tr>
<tr>
<td>Shape Concern</td>
<td>2.68</td>
<td>1.76</td>
<td>1.19</td>
<td>1.78</td>
<td>5.1668</td>
<td>0.0309</td>
</tr>
</tbody>
</table>

**Table 6: Univariate ANOVA for Anorexia Nervosa vs Eating Pathology sample**

<table>
<thead>
<tr>
<th>Subscale</th>
<th>Anorexia Nervosa Mean</th>
<th>St Dev.</th>
<th>Control 2 Mean</th>
<th>St Dev.</th>
<th>F value</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Restraint</td>
<td>2.43</td>
<td>1.97</td>
<td>0.68</td>
<td>0.95</td>
<td>9.6597</td>
<td>0.0042</td>
</tr>
<tr>
<td>Eating Concern</td>
<td>1.74</td>
<td>1.44</td>
<td>0.33</td>
<td>0.61</td>
<td>12.1778</td>
<td>0.0016</td>
</tr>
<tr>
<td>Weight Concern</td>
<td>2.35</td>
<td>1.69</td>
<td>0.81</td>
<td>1.08</td>
<td>9.0045</td>
<td>0.0055</td>
</tr>
<tr>
<td>Shape Concern</td>
<td>2.68</td>
<td>1.76</td>
<td>0.80</td>
<td>1.09</td>
<td>12.6256</td>
<td>0.0013</td>
</tr>
</tbody>
</table>

**Table 7: Univariate ANOVA for Anorexia Nervosa vs Control 2 sample**

Table 7 shows that there are significant differences between the Anorexia Nervosa sample and their matched controls on all subscales at $p<0.01$ or better; by contrast Table 8 shows no significant differences between the the Eating Pathology sample and their matched controls on any of the subscales.
Table 8: Univariate ANOVA for Eating Pathology vs Control 1 sample

Single sample t test comparisons of the Anorexia Nervosa sample with the adult standardisation population described by Cooper et al (1989) are recorded in Table 9. These show that there is no evidence that the Anorexia Nervosa sample have a significantly lower score for either Weight or Shape Concern as compared with the Adult sample, and so does not support the research hypothesis.

Table 9: T Test for Anorexia Nervosa vs. Adult Standardisation sample

Finally, comparisons between the Anorexia Nervosa sample and the combined Control samples on the individual items of the Weight and Shape Concern subscales are shown in Figures 4 and 5 below.
Figure 4: Comparison of anorexia nervosa sample and combined controls on individual weight concern items.

Figure 5: Comparison of anorexia nervosa sample and combined controls on individual shape concern items.
6. Discussion

6.1 Research Hypotheses

The main hypothesis predicted that there would be a significant interaction between the anorexia nervosa sample, the eating pathology sample and the control sample on the four subscales of the CH-EDE. Specifically, the hypothesis predicted that the anorexia nervosa sample would have significantly higher scores than the control sample (but not the eating pathology sample) on subscales of Restraint and Eating Concern, and that all groups would have similar scores on subscales of Shape Concern and Weight Concern.

However, this predicted interaction did not emerge from the experimental samples. Tables 5 to 8 confirm that the relationship between the samples is more simple: the anorexia nervosa sample score significantly higher than the control sample and the eating pathology sample on all subscales; the eating pathology sample could not be statistically distinguished from the control sample; and the child anorexia nervosa sample could not be distinguished from the adult anorexia standardisation sample (Table 9).

Two types of conclusions can be drawn from these results, firstly concerning the nature of eating disorders in children and adults and the comparison of children with and without eating problems, and secondly concerning the adequacy of the measure to indicate such distinctions. This second issue is considered in more detail in the section on the Use of the CH-EDE below, by reviewing the reliability and validity evidence gathered from the research data. The similarities and differences between children with and without eating disorders is considered first, though the subsequent caveats made about the measure need to be taken into account in drawing out conclusions.
The evidence from this study suggests that the young people in the early onset anorexia nervosa sample cannot be distinguished from the adult anorexia nervosa sample reported by Cooper et al (1989). This finding has important implications for the debate about how similar the illness is in children and adults. The findings do not support the hypothesis that weight and shape concerns in children and adults are different, in that the overall levels of concern of the two groups are equal. This finding is a challenge to any of the theories reviewed above (including the theoretical stance adopted for the present study) which would have predicted an age-related difference in these concerns.

However, a more detailed analysis of the responses to individual Weight and Shape Concern items by the anorexia nervosa sample and the control sample (recorded in Figures 4 and 5 above) suggest that the pattern may not be as clear cut as the former group being exactly the same as the latter and totally different to other children. The individual item analysis shows that on some of the questions, the mean score for the anorexia nervosa sample was not significantly different to the mean score for the control sample. Of these, the items Desire to Lose Weight ("Over the past four weeks, have you wanted to lose weight?") and Feeling Fat ("Have you felt fat over the last four weeks?") were scored on average as highly by the control sample as by the anorexia nervosa sample.

These results concur with the study by Wardle and Marsland (1990), which showed that concerns about weight and fatness are normative in this population. They also mean that the overall significant differences that emerge for Weight and Shape Concern cannot be accounted for by either of these items. An inspection of Figures 4 and 5 and the correlation of individual items with subscale totals in Table 3 suggest that the biggest mean differences between the groups (i.e. those contributing most to the
overall significant differences) are the *Importance of Weight and Shape to Self Concept* and the *Fear of Weight Gain* items.

This dominant contribution of the Importance of Weight and Shape to Self Concept appears to be an unambiguous endorsement of theories that place overvalued ideas of weight and shape at the centre of the core phenomenology of eating disorders, and contra the theoretical stance of the present study.

However, the additional contribution of the Fear of Weight Gain item (which is statistically the item with the smallest probability of a Type 1 error in distinguishing between the samples) could be said to be reflecting the fear of gaining *any* weight (even while on average only 78.69% weight for height) for the anorexia nervosa sample. That is to say, to a fear of becoming fatter/heavier (relative to current shape and weight) rather than a fear of becoming fat/heavy (relative to average shape and weight for age). In this sense, the fear may be more about losing control as predicted by Bruch and other theorists, rather than being about becoming objectively fat and overweight (with all its sociocultural and personal consequences).

In this construction, concerns about weight and shape do not themselves constitute the core psychopathology of eating disorders. Rather, such concerns are recruited as good indices of being in control for the nascent disordered eater precisely because they are such common ideas in the population. Such a conclusion is supported by the findings of Wardle and Marsland (1990) that concerns about weight and shape are normative in this population as well as the results of the present study indicating a uniform distribution of Desire to Lose Weight and Feeling Fat items in the clinical and control samples.

If this is the case, then there may be important implications for the treatment of eating
disorders, as well as for theories. These theoretical and clinical issues are considered in more detail below, after taking into account the strengths and weaknesses of the CH-EDE and the current research design.
6.2 The Use of the Eating Disorder Examination

This section critically reviews the use of the child version of the Eating Disorder Examination (CH-EDE) by identifying some of the positives and problems associated with its use, before discussing the interrater reliability results and validity study.

**Positives**

Administering the CH-EDE proved to be a positive and useful experience. The young people came willingly to the session and it appeared that they valued an opportunity to have their own say in a context where their opinion was listened to carefully. Although there are no data to support this assertion, it seemed to be helpful that the questions focused on details of eating and worries about eating; that rather than being asked to think about other problems the young person had clear permission to think just about their worries and problems related to food and eating.

The CH-EDE was relatively quick and simple to administer, with most interviews lasting about an hour. The construction of the subscales made it easy to subsequently organise responses into groups of statements for each of the subscales for subsequent report to the clinical case conference (see Appendix 8 for an example).

The generation of subscale scores for Restraint, Eating Concern, Weight Concern and Shape Concern means that the CH-EDE is useful in research studies that aim to compare these variables across different populations. The inclusion of diagnostic items and criterion-referenced cut-offs generated by the adult standardisation sample means that the CH-EDE can potentially provide an index of "caseness" for childhood onset anorexia nervosa, valuable in both research and clinical practice (though see the discussion of the validity study below).
Finally, the close relationship between the child and the adult version of the Eating Disorder Examination made it possible to compare these two populations directly for the first time, and to explore the similarities and differences between childhood and adult onset eating disorders.

**Problems**

By contrast, some features of the CH-EDE proved more problematical. Several questions track behavioural components which may be hard for children to remember, even with the use of the diary. It may just be too hard for a child at the bottom of the age range to remember whether he or she ate lunch on a particular day a month ago. We know from developmental psychology that especially younger children are poor historians. In this case self report may not be the method of choice for recording the purely behavioural components of the examination.

A similar issue is presented by the complexity of some of the core concepts questions are trying to tap. A particular difficulty arises for questions that conflate behavioural, cognitive and emotional components. For example, question 12 aims to "ascertain whether the child has felt concerned about other people seeing them eat, and whether they have avoided eating in front of others". The probe questions are recorded in table n below:

- over the past four weeks have you been worried about other people seeing you eat?
- Have you tried putting off (or getting out of) eating with other people?
- Would you avoid eating in front of others if you were allowed to by your parents?
- Why were you concerned about others seeing you eat?

**Table 10: probe items for question 12 of the CH-EDE**
The scoring key for this question requires the interviewer to take account of emotional elements (feelings of concern), cognitive elements (the reasons for concern are about weight or shape) and behavioural elements (avoiding eating) as the scoring key recorded in table 11 below indicates.

In practice, this conflation may make it hard for children to grasp which element of the question (cognitive, emotional or behavioural) they should focus on, since it may be computationally difficult to answer all three simultaneously. Equally, experience of administering the question suggests that researchers may have similar difficulties in separating out the three components of the child's response.

0 - No concern about being seen eating by others and no avoidance of such occasions
1 -
2 - Has felt slight concern about being seen, but no avoidance
3 -
4 - Has felt definite concern, and has avoided some such occasions
5 -
6 - Has felt definite concern, and has avoided all such occasions

**Table 11: scoring key for question 12 of the CH-EDE**

A further problem about how participants may respond to the probe question emerges from the theoretical stance taken by the authors of the original adult version that weight and shape concern are the core specific psychopathology of eating disorders. This stance means that emotional and behavioural components assessed by the examination can only be included if they are based on these core cognitions. The examiner is therefore required to verify "did you feel this way because of your weight or shape?"/"is this to try to change your weight or shape?" before scoring 12 out of the 34 questions.

The danger of concluding a complex enquiry with a closed question is that the child who has not fully grasped the essence of the enquiry is probably more likely to respond
positively to conclude the question rather than to ask for further clarification. The dangers of "yea-saying" and same-sense closed questions have been well documented in studies of questionnaire design.

A related problem comes from the nature of eating disorders themselves. One of the intriguing features of anorexia nervosa is its incomprehensibility; the difficulty in understanding why the sufferer goes to such heroic lengths to avoid eating. Some of the psychological theorists reviewed above posit an underlying core psychopathology such as feelings of loss of control that are not available to consciousness and therefore that cannot be articulated.

Such theorists would argue that this very inability to express underlying psychological distress is part of the core pathogenesis of eating disorders. Therefore, interview questions that provide a ready-made reason for the young person to help them make sense of their behaviour ("I stopped eating because I was afraid of getting fat") may generate erroneously inflated scores relating to these issues.

**6.2.1 Interrater Reliability Study**

Appendix 15 shows that the correlation between two raters was uniformly high for all questions, ranging from n1 for question a to n2 for question b. To some extent, this finding reduces the concern expressed above about the conflation of some of the questions, since two raters were able to independently agree how to separate out behavioural, cognitive and emotional components of answers. The high level of agreement also confers on the CH-EDE a viability for use in research contexts; that although the construct validity of some of the questions can be challenged (i.e. what are they measuring?), by extension of the interrater results the examination could be said to be measuring the same thing in different participants (i.e. if two participants
said the same thing to different raters they would get the same scores).

### 6.2.2 Discriminant Validity Study

The purpose of the discriminant validity study is to assess the sensitivity, specificity and positive predictive value of the CH-EDE. The authors of the revised 12th version of the adult Eating Disorder Examination (which the CH-EDE is based on) have developed operationally defined eating disorder diagnoses (Fairburn and Cooper, 1993) from their measure.

The diagnostic system established by these authors uses the DSM-IV criteria for anorexia nervosa, and is based on responses to specific questions as set out in Appendix 14. Based on these criteria with the exception of the weight for height index (i.e. focusing on the cognitive and behavioural indices rather than physiological), the present study showed that the CH-EDE has a sensitivity of 47%, a specificity of 90% and a positive predictive value of 58%.

The low sensitivity of the CH-EDE is discussed by Bryant-Waugh et al (1996), who point out that one of the difficulties of assessing eating disorder patients is that a proportion deny having any problems or worries, despite sensitive questioning.

The remaining eight subjects clinically diagnosed with anorexia nervosa who received a negative CH-EDE diagnosis all had a mean individual item score of less than 1, suggesting that they were tending to rate all items at a low level. For this group, the denial of any of the behavioural components of their eating disorder despite being within the fasting range of weight for height indicates that they were not able to acknowledge the problems they had.
The theoretical model developed by the present study (that anorexia nervosa is the symptomatic response to underlying unconscious psychological distress) could account for the tendency of these participants not to report eating problems or associated worries: since they could not make sense of their own behaviour, they had adopted a strategy of denial in response to questioning.

Although the development of an interviewer-led rather than self-report questionnaire was motivated by a desire to overcome some of these methodological difficulties (Cooper and Fairburn, 1987), problems clearly remain for patients who are denying their illness. Attempts to circumvent such denial have recently been reported by Chesters et al (1994), who developed a form of the Stroop task for assessing bulimia nervosa.

Other work by Cooper and Fairburn (1992) demonstrated that Stroop task latencies could distinguish between adult patients with anorexia nervosa, bulimia nervosa, current dieters and non dieters. It may be that the use of appropriate computer-based stroop tests with children with eating disorders could be developed as a similar diagnostic tool; a pilot investigation of such a test is planned for the Medical Research Council study for which the present study is piloting the use of the CH-EDE.

The specificity of the CH-EDE in the present study is less than 100% because the condition of weight for height of 15% below that expected was not included in making the diagnostic grouping. In addition one participant from the eating pathology sample scored sufficiently highly on CH-EDE measures to attain caseness for anorexia nervosa; however she was 105% weight for height and so could not be included in an anorexia nervosa diagnosis.
Four participants from the control samples attained sufficiently high scores to indicate caseness for anorexia nervosa. This proportion (i.e. 10% of the total control sample) was higher than had been expected given Crisp et al's (1976) survey of London private girls' schools which had found a prevalence of only 0.2% in girls aged 15 years or under. However, since the control participants were not weighed in the current study, it is not possible to verify whether any of the four attaining caseness on the basis of their CH-EDE scores were also sufficiently below the expected weight for their age and height to merit a diagnosis of anorexia nervosa.

It may be that the CH-EDE has a valuable role in screening young people who have not yet embarked on a major weight-loss programme, but who are at risk of developing an eating disorder in response to other psychological stressors. Certainly, my clinical impression was that none of these four participants were currently exhibiting major eating pathology in their behaviour, but that their attitudes about themselves, food and weight (as indicated by their CH-EDE responses) suggested that they might be at future risk. The issue of screening and the early primary and secondary prevention of eating disorders is returned to in the section on Clinical implications below.

6.2.3 Construct Validity

The internal consistency of the four subscales as assessed by Cronbach alpha coefficients shows that they are satisfactory. However, in contrast to the adult sample described by Cooper et al (1989), further analysis reveals that the internal consistency of all subscales could be improved by removing items, a finding that suggests that the original grouping of items for the adult sample may not necessarily be appropriate for children.

This issue is further examined by Table 4 in the Results, which records the correlation between individual item scores and subscale total scores. As for the adult sample, the
mean level of correlation for subscale items was highest against their own subscale totals than against others. However, in contrast to the adult sample, some individual items correlated more highly with other subscale totals than with their own (for example, both Restrained Eating and Guilt About Eating scores correlated more highly with the Weight Concern Subscale than with their own subscale).

Further work may be needed with a larger sample to perform a full factor analysis of the individual questions. Indeed, the relatively uniform and high positive correlation for many of the items suggests that it may be possible to simplify the questionnaire and ask fewer questions if many appear to load on the same factor.

6.2.4 Summary - the use of the CH-EDE

The present study has demonstrated that the CH-EDE can be used with young people with anorexia nervosa and other eating pathology. The measure is relatively quick to use, and is responded to well by most participants. The questions are relevant to the concerns of young people with eating problems, and the measure is clinically useful in giving them a chance to describe their fears and thoughts in a context where they are being listened to carefully.

It has been argued that some of the questions may require additional 'translation' from the adult version to make them sufficiently comprehensible to younger participants.

Also, the techniques developed to focus attention on the time span in question and to elicit self concepts may need further refinement. The reliance on closed questioning to confirm whether responses meet the "is this because of weight/shape?" condition may need further modification, especially for younger respondents.

However, interrater reliability results suggest that independent raters have a good
level of agreement on how to translate responses into scores for most questions. The analysis of subscales suggests that these retain a good level of internal consistency in this version, though further factor analysis of a larger sample may indicate a degree of overlap and permit some items to be dropped.

The diagnostic use of the measure indicates that it potentially has low sensitivity, because of the nature of eating disorders and the likelihood that individuals may deny their problems. It may be that the CH-EDE needs to be used in conjunction with a more direct measure of the emotional loading of issues around eating, weight and shape such as a stroop task. The use of the measure with a control sample indicates that it may be valuable as a screening tool to detect cognitions and fears about food, weight and shape before major eating pathology sets in.

These limitations and strengths of the measure are taken into account in the following section, which critically reviews the overall research design. Having taken these limitations into account, it is then possible to explore the theoretical and clinical implications of the study.
6.3 Methodology

The aim of this section is to critically review the methodology adopted, to clarify the subsequent clinical and theoretical interpretations. Issues of sample size, measures used and statistical analysis are all briefly discussed.

6.3.1 Sample Size

The critical effect size discussed in Appendix n shows that the sample sizes of n=15 were minimally adequate to permit statistical comparison of the samples. However, sample size was in practice also determined by the prevalence of clinical patients during the data collecting period. Visits were made to the Eating Disorder Clinic at Great Ormond St Hospital on 24 separate occasions over an eight month period. It is fortunate in clinical if not research terms that severe eating problems in children continue to be very rare, and only 30 subjects were recruited during this period.

Further research would need to extend the data gathering period over several years and perhaps adopt multi-centre methodologies to see enough cases to permit factor analytic and other advanced statistical manipulations. It would also then become possible to evaluate the relative sensitivity of the EDE and distribution of weight and shape concerns, without the risk of the findings being an artefact of the sampling.

6.3.2 Measures Used

The research design was weakened by having only one measure of eating pathology, which was itself under investigation to assess its reliability and validity. The availability of a second, independent measure of eating pathology from a well established instrument such as the Eating Attitudes Test (Garner and Garfinkel, 1979) would have
facilitated the analysis of concurrent validity. Equally, the use of other independent measures of general child psychopathology or specific experimentally hypothesised constructs such as self control or self esteem would have strengthened the research design.

However, the research was conducted in a clinical setting, and clinical considerations needed to remain paramount. The original research methodology proposed the inclusion of an interview to assess locus of control and weight phobia. However, in discussion with the clinical team it was felt that the additional measures would not be appropriate, since the young people attending the clinic were already participating in a large range of research led assessment.

Attendees at the clinic have just begun to be screened using a measure of general psychopathology as well as the EDE. However, this research forms a large funded Medical Research Council study and was beyond the resources of the present study.

6.3.3 Statistical Analysis

Appendix 12 gives the rationale for using Student's T test and analyses of variance in the research design. Computation of Glass's Effect Size confirms that sample sizes are sufficiently large to permit the research hypotheses to be tested.

Having evaluated the methodological limitations of the study, it is now possible to go on to draw out its theoretical and clinical implications.
6.4 Implications for theories of eating disorders

The analysis of responses to individual Weight and Shape Concern by the anorexia nervosa and control samples reported in section 6.1 above indicates that mean responses to some items (e.g. *Desire to Lose Weight, Feeling Fat*) did not distinguish between the two samples; by contrast mean scores on other items were clearly different for the each sample (e.g. *Fear of Weight Gain, Importance of Shape*). Theories of eating disorders need to account for why some components of weight and shape concern only apply once an eating disorder is established, while others apply to all young females.

For the present study, it is theorised that ideas about the importance of weight and shape are overvalued by anorectics because they are good indices of being in control (rather than being of themselves any more desirable for anorectics than their contemporaries). Such an approach has important implications for the way eating disorders are formulated. As Wren and Lask (1993) conclude: "A number of authors from very diverse perspectives share an understanding of anorexia nervosa as essentially a syndrome of pathological self-control. Crucially, this shifts the wish to be thin from the centre of the phenomenological picture..."

They also go on to record Bruch's (1974) conclusion that: "The main issue is a struggle for control, or a sense of identity, competence and effectiveness. Many of these youngsters had struggled for years to be perfect in the eyes of others. Concern with thinness and food refusal are late steps in this maldevelopment.."

Such a realignment of the location of issues about thinness and lightness in the phenomenology of eating disorders is supported in this study by the finding that control participants felt equally fat and had the same desire to lose weight as the
clinical participants. If wanting to lose weight and have a thinner shape are desires common to both groups, then these constructs cannot amount to a sufficient condition for causing an eating disorder.

Cognitive theorists would probably argue that their theories do not disagree about the equal distribution of the desire to lose weight and not be fat across the whole population. What characterises disordered eaters in cognitive theories is the centrality of these desires to self concept (illustrated in Figure 4 above for the current sample).

However, to claim that the importance of weight and shape are key overvalued ideas to the anorexia nervosa sample does not explain why they are more important to this group than to the control sample. There is a danger that the recent suggestion that eating disorders should be recharacterised as dieting disorders (Beumont et al, 1994) will discourage further investigation into why young people with anorexia nervosa place such greater importance on having a thin shape and weighing very little as key self concepts than their contemporaries.

Further research may be needed to account for the equal feelings of fatness and desire to lose weight between the two samples, given the differing importance they place on these in evaluating self concept. It could be that the anorexia nervosa sample have 'regressed back' to the mean level of feeling fat and wanting to lose weight because they are successfully keeping their weight down.

This problem of interpreting the statements of successful anorectics who may not report a great desire to lose weight since they are currently successful at losing weight is discussed by Bryant-Waugh et al (1996). Adopting a longitudinal approach to evaluating these concepts at different stages across the career of an anorectic may clarify how they operate at different stages in the progression of the illness.
Further work could also investigate how the intensity of the cognitions trapped by the Weight and Shape Concern subscale of the CH-EDE relate to measures of more general psychopathology. The range of theories focusing on fear of losing control as a precipitating factor for anorexia nervosa have made some suggestions about the potential underlying core psychopathology (for example Slade's model), though this has largely remained untested experimentally.

If the need to exert control is itself a product of a more profound underlying psychopathology, then the anorexia nervosa sample's significantly higher levels of Shape and Weight Concern could be construed as an intermediate set of cognitions mediating between an underlying emotion (fear of loss of control) and an overt set of symptoms (the behavioural components of the measure such as Restraint). In turn the fear of loss of control could itself be the expression of core underlying schemas that are not available to consciousness.

Since the underlying psychopathological schemas are not available to consciousness, the overt restraint and food avoidance behaviours in anorexia nervosa represent the conversion of psychological distress into physical symptomatology, and the disorder itself may be classified best as a subtype of somatoform disorder. If this is the case then the anorectic may have no more conscious access into their motivation for not eating than the child with another somatoform disorder can “know” why he or she has lost the function of a limb for example. The crucial difference, though, for the anorectic patient, is that he or she has a ready-made set of culturally-syntonic explanations to help make sense of his or her symptoms: that the relentless pursuit of thinness is a result of a fear of fatness.

The proposition that the cognitions about weight and shape made by anorectics are
attributions to help them make sense of their behaviour is presented diagrammatically in Figure 6 below.

The model suggests that cognitions about weight and shape impact at two distinct levels in the pathogenesis of anorexia nervosa: generic concerns that are shared by the majority of all young people impact as precipitating factors that help to establish anorexia nervosa; different specific concerns operate as perpetuating factors to articulate the anorectic’s fear of loss of control.

Theories that try to account for the pathogenesis of anorexia nervosa need to be able to predict how different factors impact at these stages. Such a formulation of eating disorders would also have important implications for treatment, which is discussed in the section below on the clinical implications of the study.
Perpetuating Factors

- avoidance of other issues
- physiological effects of starvation
- fear of loss of control

ANOREXIA NERVOSA

- enhanced sense of self control

Precipitating Factors

- psychological distress
- sociocultural factors

- reduced sense of self control

Predisposing Factors

- early experience, e.g., attachment
- systemic factors
- individual differences, including genetic predisposition

Figure 6: Schematic attribution model for anorexia nervosa

6.5 Implications for the treatment of eating disorders
The suggestion that weight and shape concerns can be divided into generic concerns that all young people are likely to carry and specific attributions linked to fear of loss of control in anorexia nervosa also has important implications for treatment.

In the light of these findings, creators of treatment approaches need to clarify how they distinguish between generic and specific concerns. Focusing on the limitations of the way society constructs thinness and lightness (the sociocultural approach) is likely to lead only to frustration at the limited gains possible against such a pervasive and powerful alliance of media, fashion and advertising. As Garner and Bemis (1983) point out, anorexia nervosa has become a "culturally syntonic" disorder, and therefore it may be hard to shift an individual's thinking away from such attitudes that are so widely held.

The distinction also supports the cognitive therapy approach described by Garner and Bemis (1983) that moves away from disputations about the patient's weight and shape concern itself in favour of discussions about self concept and self esteem, and attempts to create ways of evaluating these that are not couched in weight and shape terms.

In essence, treatment approaches informed by the prediction of this study would adopt the cognitive therapy approach of viewing the weight and shape concern itself as a "proximal paradigm" and final common pathway of more profound psychological distress. Although the presenting concerns way offer a useful way to join with the patient, it is important clinically to keep in mind the underlying issues that need to be tackled. As Garner and Bemis (1983) conclude: "Since these topics [weight and shape concern] occupy so much of the starved patient's thinking, they provide "common ground" around which therapeutic trust may evolve; however prudence must be
exercised to ensure that these do not become a means of avoiding other psychological issues such as low self esteem, inadequate personal trust, anxiety, depression, and poor socialisation."

However, the low specificity of the Eating Disorder Examination found in this study suggests that a significant subset of patients clinically diagnosed as having anorexia nervosa might not share these typical weight and shape concerns, or at least do not admit to having them in the context of a clinical interview. For such patients, it is questionable whether the therapist needs to somehow convince them that they do indeed have these concerns, only to then move beyond them to the more fundamental issues.

In practice, it might be possible that all patients would benefit from the techniques developed by cognitive theorists to explore self concept and self esteem, whether or not they initially held the beliefs necessary for EDE diagnosis.

In addition, the model proposed from the current study also generates important predictions for the early identification and prevention of eating disorders in a way that existing theories are not able to do. Because the weight and shape concerns associated with attributions of maintaining control are late-emerging symptoms (or not even necessary symptoms, according to the results found in the present study) in the pathogenesis of anorexia nervosa, focusing diagnosis and treatment on them means that nothing can happen until significant weight loss has occurred and the dysfunctional attitudes are in place.

However, if more profound underlying psychopathology can be identified as distal predisposing and precipitating factors in promoting eating disorders, then it may be possible to screen for children who reach a criterion index of potential caseness on a
range of measures such as family functioning, self esteem and self-attributional style. Those children who also identify weight and shape concerns above the general population norms may be considered to be at risk of developing an eating disorder, and monitoring and early intervention could then be deployed.

In addition, the shift away from weight and shape concerns to areas of psychopathology related to self concept, acceptance of self in the light of failure and ability to identify and label emotions and internal states has broader implications for the theory and practice of child development: we may need to explore more about how children learn to recognise and label emotions and to differentiate between internally and externally focused self-evaluation, and where necessary include such issues in parent training packages; schools may need to review the relative importance they place on external achievement verses the development of self esteem in their pupils; in broader social terms we may need to explore how children can develop appropriate autonomy and control over their lives as well as to clarify the optimum level of parental structure and boundaries.

Above all, the evidence from the present study suggests that focusing on weight and shape concerns at the exclusion of all other aspects of cognitive and emotional functioning in the diagnosis of anorexia nervosa may lead to an erroneous focus on these same issues in the treatment of the disorder, as well as to missed opportunities for early diagnosis and prevention.