Do schemas mediate the relationship between parental bonding and psychopathology in adulthood?

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DECLARATION

"I certify that this is a true and accurate account of the work carried out. This thesis has been composed by myself and the work contained herein is my own."

Signed

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ABSTRACT

The significant influence of early experiences on vulnerability to psychopathology has been proposed in several prominent theories of emotional disorders. One aspect of early experience that has been investigated rigorously with respect to development of psychological difficulties is the nature of parental bonding. Associations between two dimensions of parental bonding – care and overprotection – and psychopathology have been repeatedly documented, with low levels of care and high levels of overprotection more apparent in individuals experiencing psychological distress. However, research investigating the possible mechanisms by which aspects of an individual’s parental bonding experience might result in development of psychopathology is lacking. Given the emphasis on cognitive factors in a number of influential theories of psychopathology, it seems feasible that dysfunctional beliefs might mediate this relationship. Although a few studies have primarily supported this hypothesis, such studies have been conducted using specific populations and without reference to anxiety symptomatology, which is commonly manifested in a large number of individuals seeking psychological input.

Therefore, the current study primarily aimed to examine whether dysfunctional schemas mediated the relationship between parental bonding and both anxiety and depressive symptomatology, using a self-report questionnaire methodology across a sample of general psychiatric outpatients and a comparison group. Such a model was supported for depressive symptomatology, but only partially supported with respect to anxiety symptomatology. Although limitations of the study methodology suggest that conclusions must be tentative, greater focus on schemas in therapy seems indicated.
1 INTRODUCTION

1.1 GENERAL INTRODUCTION

Effective treatment of psychological disorders depends in part on a good understanding of the nature of current symptoms and potential maintaining factors. However, even the more short-term, "here and now" focused therapies would acknowledge benefit from formulation of possible underlying causes or predisposing factors – i.e., the interpersonal and intrapersonal processes involved in the development and maintenance of the presenting problem(s). Clearer understanding of significant risk factors in the development of psychopathology and their interrelation should facilitate identification of the most suitable form of intervention in individual cases. However, the ultimate aim would be to enable appropriate targeting of preventative work (e.g., screening and promotion of protective factors) and early intervention, in an attempt to reduce the likelihood of psychological difficulties developing at a later stage. Emphasis on the importance of psychological well-being across the life course is in accordance with the World Health Organisation’s adoption of a developmental life-span perspective (WHO, 2002).

A variety of risk factors have been implicated in the genesis of psychopathology. A number of prominent psychological theories suggest that early experiences can heighten an individual’s vulnerability to later psychological difficulties. One aspect of early experience that has received a great deal of research interest is the nature of parental rearing styles and their influence on subsequent development. However, although evidence appears to indicate a link between dysfunctional patterns of
parental bonding and vulnerability to psychopathology, little investigation has been conducted into the potential mechanisms underlying this association. Given the importance accorded to cognitive factors in theories of emotional disorders, with respect to the development and maintenance of various forms of psychological problems, it would seem feasible that dysfunctional cognitions might mediate the relationship between negative parenting experiences and subsequent development of psychopathology.

Therefore, in the current study, the possible mediating role of maladaptive schemas in the relationship between parental bonding and psychopathology will be explored across both a clinical and non-clinical population. The first section of the introduction focuses on research investigating the link between parental bonding and vulnerability to psychopathology, with evidence to suggest that perceptions of low parental care and parental overprotection are related to psychopathology, although it remains unclear whether such links are specific to the nature of psychological difficulty or represent a general predisposition. The subsequent section explores prominent cognitive theories of psychopathology, which assume the significance of cognitive processes in the development and maintenance of emotional disorders. In particular, recent research highlighting the relevance of deeper levels of cognition in more complex forms of psychopathology is reviewed along with the need for appropriate assessment of such maladaptive schemas. The final section briefly outlines studies that have been conducted to-date examining the links between parental bonding, dysfunctional schemas and vulnerability to psychopathology. Aims and hypotheses of the current study will then be delineated, followed by a description of the methodology utilised to test the hypotheses. Analysis and interpretation of the
results will then be documented, with respect to both predictions made and with reference to the existing literature, including a critique of the study and possible directions for future research.

1.2 PARENTAL BONDING AND PSYCHOPATHOLOGY

1.2.1 Introduction

Although contrasting in focus with respect to potential causal factors in psychopathology, a number of prominent psychological theories share the view that early life experiences play a significant role in shaping subsequent development and influencing vulnerability to later psychological difficulties. Classic psychoanalytic theory posits that psychopathology emanates from unconscious defences against unacceptable urges that arose early in childhood, in accordance with critical experiences occurring at different stages of development (Freud, 1905). Similarly, the importance of the nature of an individual’s attachment with his/her primary caregiver, with respect to development of internal working models, is highlighted in attachment theory (Bowlby, 1969), with the belief that such mental representations act as a template for subsequent relationships. Indeed, although focusing primarily on current cognitions and their interaction with affective, physiological and behavioural sequelae, cognitive-behavioural theory (Beck, 1967; 1976) also emphasises the significance of early experiences when examining factors that influence an individual’s vulnerability to psychological difficulties. The occurrence of later critical life events congruent with such predisposing vulnerabilities and associated
beliefs are proposed to culminate in specific forms of psychopathology, dependent on the nature of experiences and beliefs developed.

Consequently, a large number of studies have examined the effect of an individual’s early experiences, in particular with respect to quality of attachment as an infant and bonding with parental figures\(^1\) throughout childhood, on his/her susceptibility to development of various forms of psychopathology as an adult. Such research will be detailed in the following sections, including the various methodologies, after a more extensive examination of early attachment processes and their proposed link with development of later difficulties. Measures of parental bonding will also be described, including a detailed critique of the measure utilised in the current study. The section will conclude with an examination of evidence indicating the influence of parental bonding on vulnerability to psychopathology.

1.2.2 Attachment

The idea\(^2\) (Freud, 1905) that an infant’s attachment to his/her primary caregiver (usually the mother) developed solely as a means to ensure fulfilment of biological needs, e.g., alleviation of hunger or pain, was dismissed following emergence of evidence demonstrating that infants could become attached to individuals who were not responsible for feeding them (Bowlby, 1969). Similarly, empirical studies on

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\(^1\) All further references to parents equate to parental figures, i.e., dominant caregivers, as these individuals will not necessarily be biological parents in all instances.

\(^2\) Bowlby (1969) described this view as ‘cupboard theory’.
infant rhesus monkeys raised without their mothers revealed a preference for them to seek proximity to a ‘furry’ terry-cloth model of a monkey over a wire monkey figure with a teet for feeding, particularly when distressed (Harlow, 1958). Such research again indicated that attachment behaviour signified more than mere satisfaction of physical needs.

Bowlby (1969) emphasised humans’ innate capacity to form intimate emotional bonds in his theory of attachment, suggesting that the creation and preservation of such attachments was regulated within a control system in the CNS\(^3\). He postulated that the primary aims of attachment for an infant involved a familiar figure with whom he/she could socially interact, who also provided a secure base from which he/she could explore the world and to whom proximity could be sought as a means of reducing anxiety. Attachment to a primary caregiver should therefore provide an infant with protection, support and security from which to gradually explore the world, whilst ensuring comfort in adverse circumstances.

Of course, inherent to such a theory is the assumption that the nature of attachment would depend on the responsiveness and availability of the primary caregiver, with respect to the expressed needs of the infant, resulting in various forms of attachment behaviour. Assessment of these differing patterns of attachment behaviour in infants at approximately one-year-old was first conducted by Ainsworth and colleagues (Ainsworth & Bell, 1970; Ainsworth, Blehar, Waters, & Wall, 1978), using their concept of the ‘Strange Situation’, which involved examination of the infant’s behaviour on reaction to separation in an experimental setting. The infant and his/her

\(^3\) central nervous system
mother would enter an unfamiliar room containing toys; initially, the infant had the opportunity to explore with his/her mother present, then a stranger would enter the room, followed by a brief 10-minute separation from the mother, with her leaving the room, after which the infant was reunited with the mother. Ainsworth and her colleagues observed several different patterns of behaviour that were apparent during these phases of initial exploration, separation and reunion. The most common pattern, occurring in over two-thirds of the population sampled, was described as secure attachment, whereby an infant readily explored the environment whilst his/her mother was present, making cautious approaches to the stranger and becoming distressed on separation, but responding to the reunion with enthusiasm. The remaining patterns of behaviour were all broadly described as forms of insecure attachment, with further subdivision into anxious-avoidant, anxious-ambivalent and disorganised categories. Infants classified as anxious-avoidant appeared somewhat distant and aloof throughout the assessment, exhibiting minimal distress on separation and actively avoiding or ignoring the mother on her return. Those categorised as anxious-ambivalent tended to focus their attention on their mother, with little exploration of the environment, becoming extremely distressed on separation. On reunion, these infants sought proximity to their mother, e.g., by clinging behaviours, but did not appear to be easily consolable. In addition, a further group of infants who appeared less easily classifiable were described as exhibiting disorganised attachment, whereby they showed a variety of unusual and chaotic behaviours, such as, freezing, stereotypy and confusion, in response to separation and reunion. It was hypothesised that the nature of attachment developed was primarily a

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This pattern of attachment is sometimes also described as anxious-resistant.
function of the different ways in which the attachment figure responded to and interacted with the infant (Main, Kaplan, & Cassidy, 1985). Securely attached infants were believed to have an attachment figure who was sensitive to their needs, responding both appropriately and promptly in a helpful manner. In contrast, it was proposed that anxious-ambivalent infants received inconsistent care from their attachment figure, involving a mixture of responsiveness and unavailability with threats of abandonment often used as a means of discipline. Anxious-avoidant attachment was thought to develop in accordance with neglectful caregiving from the attachment figure, with rejection of the infant being a characteristic feature. Finally, chaotic or abusive parenting was believed to underlie the disorganised pattern of attachment. Matas, Arend, and Sroufe (1978) illustrated such parental responsiveness by observing mother’s interactions with their 2½-year-old children as they attempted to solve a challenging task. They found that the mother’s reactions correlated significantly with the assessment of the child’s attachment towards her 18 months previously (using the ‘Strange Situation’ procedure). Mothers whose child had previously been assessed as securely attached were attentive and encouraging towards their child, whereas those whose child had been categorised as insecurely attached appeared to be inattentive and insensitive towards their child.

Further research examining the link between patterns of attachment and subsequent development led to the proposal that these were relatively stable characteristics, with congruence between behaviours exhibited during the ‘Strange Situation’ assessment and those displayed during early childhood years. Sroufe (1983) highlighted the apparently predictive nature of such early attachment patterns with respect to later patterns of behaviour at nursery (at approximately 4½ years). Nursery staff described
children previously assessed as securely attached as popular, co-operative and resourceful. However, anxious-resistant children tended to display either attention-seeking or passive behaviours, with anxious-avoidant children appearing anti-social and hostile. Likewise, Main and Cassidy (1988) demonstrated consistency of early attachment behaviours with conduct at 6 years, whereby children previously assessed as securely attached appeared to interact well with their parents, anxious-ambivalent children could be either hostile or intimate, those who were anxious-avoidant were somewhat dismissive, with humiliation of the parent tending to arise in children classified as having a disorganised pattern of attachment. Main et al. (1985) also revealed a strong correlation between a parent’s own childhood experiences and the nature of attachment exhibited by their child, using the Adult Attachment Interview (AAI). The AAI examines both narrative style and content when questioning adults with respect to their early attachment experiences. Parents of securely attached infants (as assessed by the ‘Strange Situation’ paradigm) also tended to have had a secure attachment to their parents, as demonstrated by their free-flowing narrative about their childhood, with descriptions of both positive and negative experiences—the former outnumbering the latter. Parents with infants demonstrating anxious-ambivalent attachment seemed more incoherent, with enmeshed negative relationships with their attachment figures prominent in their descriptions. Parents of anxious-avoidant children gave somewhat contradictory reports, with examples of negative experiences belying their statements regarding the happy nature of their childhood.

Bowlby (1988) maintained that the nature of the attachment relationship played a significant role with respect to vulnerability to development of psychopathology,
stating that the “capacity to make intimate emotional bonds with other individuals ... is a principle feature of effective personality functioning and mental health”. He postulated that individuals experiencing disturbed attachments were not only less likely to be able to cope with later stressful life events, but were also more likely to generate such adverse experiences as a consequence of their behaviour and ways of interacting with others.

In a similar manner, Bretherton and Waters (1985) suggested that presence of a secure attachment could function as a protective factor against later psychological difficulties, given that it is likely to engender a sense of self-efficacy, promote better peer relationships and facilitate autonomy.

### 1.2.2.1 Critiques of attachment theory

There are a number of assumptions underlying attachment theory - one of which being the inference of the continuity of caregiving over time, which may not always be the case (for example, following death of the primary attachment figure), although continuity appears more likely if the social environment remains stable (Vaughn, Egeland, Sroufe, & Waters, 1979). Similarly, assumptions of causality have also been disputed, given that a child’s behaviour at nursery could equally be a function of their current caregiving environment rather than purely reflecting earlier attachment patterns, as parental behaviour is likely to be consistent over this period (Lamb, Thompson, Gardner, Charnov, & Estes, 1985). However, it would seem evident that if parental style remains fairly stable over time, persistence of early
attachment behaviours is likely to ensue as a self-perpetuating cycle arises between parent-child behaviour (Bowlby, 1988). For example, a securely attached child is likely to be easier to parent than an insecurely attached child, who instead might elicit further negative responses from the parent, leading to more demanding behaviours and so forth.

1.2.2.1.1 Moderating factors

Freud held a predeterministic stance on causality, with the belief that such early influences solely determined the future course of development. The more prevalent viewpoint, however, appears to be that such early experiences act as ‘templates’ for later development, but can be modified to some degree dependent on subsequent experience. Again this idea corresponds more closely with further studies by Harlow, whereby the early negative effects on socialisation displayed by rhesus monkeys raised in isolation appeared reversible following pairing with monkey ‘therapists’ as appropriate models (Suomi & Harlow, 1972; Novak & Harlow, 1975). Modification of the influence of early experiences appears to be equally possible in humans, with the development of children transferred at an early age from an orphanage (whereby individual care was minimal) to another institutional environment where they were individually adopted (and thereby could form an attachment relationship) appearing to progress in line with ‘normal’ levels, in contrast to the difficulties experienced by those who remained in the orphanage (Skeels, 1966). More recent studies have also demonstrated that the contribution of subsequent life events should not be underestimated - for example, subsequent positive interpersonal relationships
appearing to modify the effects of disrupted early attachments (see section 1.2.5.7). Likewise, environmental factors, such as other significant supports outwith the family, could moderate the effect of early experiences on susceptibility to later adversity (Blatt & Homann, 1992). Such potential for change and modification of early attachment relationships, in line with adaptation of the associated internal working models, is of course meaningful with respect to the possible therapeutic value of psychological input in this area.

Evidence would therefore appear to support the conclusion that early experiences lay the foundation for later emotional and social development, but other factors must also be taken into account when investigating an individual’s development. Indeed, further longitudinal research is clearly necessary to fully elucidate the nature of the relationship between early attachment experiences and later development. However, a cross-sectional study by Hazan and Shaver (1987) revealed that personality characteristics and behaviours of young adults in romantic relationships also correlated significantly with recalled early patterns of attachment, again indicating some degree of continuity of such behaviours across the lifespan.

1.2.3 Parental bonding

As already highlighted, the ways in which a parent (or attachment figure) behaves towards and relates to his/her child is likely to exert a significant influence on the child’s development and socialisation process. Studies have therefore attempted to
elucidate what the crucial characteristics of parental behaviour might be with respect to their effects on offspring development.

Factors suggested as dimensions important to child rearing in early literature, in particular with respect to adequate mothering behaviours, included provision of a 'loving' or 'nurturing' relationship, whilst ensuring protection of the child (Bowlby, 1969; Rutter, 1972). Conversely, Freud (1905) warned that deprivation of affection and excessive 'spoiling' or overprotection by the maternal figure might engender later dysfunction in the child. In line with these early findings, aspects of parental behaviour that consistently emerge in most recent factor-analytic studies are dimensions representing care and control (Parker, Tupling, & Brown, 1979; Arrindell, Perris, Perris, Eisemann, Van der Ende, & von Knorring, 1986). Further discussion of these dimensions will follow in section 1.2.4.

Likewise, models of parenting advocated by Carr (1999) implicate four varying styles, along these two orthogonal dimensions of warmth and control (see Figure 1.1). Authoritative parenting appears to be optimal, representing warmth, age-appropriate responsibility and moderate control. An authoritarian parenting style involves a degree of warmth matched with a high level of control and discipline. In contrast, lack of control alongside warmth was believed to represent a permissive style of parenting, with the final neglecting pattern reminiscent of little warmth and severe or inconsistent discipline.
Figure 1.1. Four varying styles of parenting behaviour relating to orthogonal dimensions of warmth and control (see Carr, 1999).
1.2.3.1 Factors influencing the nature of parental bonding

Although dimensions of parental rearing behaviour are of central concern to the current study, it is also important to recognise which other variables might affect the nature of the parent-child bond or an individual’s ability to parent effectively. Indeed, the reciprocal nature and dynamic qualities of the parent-child bond have been emphasised, whereby bonding is not only influenced by parental behaviours but should be more accurately viewed as an interaction of those behaviours in response to the child’s individual temperament (Cassidy, 1994). Chess and Thomas (1995) identified three principal types of temperament in infants: ‘easy-temperament’ – which equated to expressions of positive affect in response to change and the presence of regular feeding, sleeping and toileting patterns; ‘difficult-temperament’ – corresponding to negative emotional responses to change and poor establishment of basic routines; and ‘slow to warm up temperament’ – representing a midpoint between easy and difficult temperaments. Children classified as having a difficult temperament tended to elicit negative responses from their caregivers, in contrast to those with an easy temperament, with the longitudinal design also revealing that these children were at greater risk for later psychological difficulties.

Other potential influences on parental behaviour, in addition to the child’s own temperament (Kagan, 1984), include the parent’s own childhood experiences (Fonagy, Steele, & Steele, 1991), parental psychopathology, extent of (emotional) support available (Teti, Nakagawa, Das, & Wirth, 1991) and the number of other stresses in the family environment, e.g., financial strain, unemployment, illness.
Perhaps unsurprisingly, the approach assumed by parents towards their own offspring appears to be primarily influenced by their own attachment experiences as children (Bowlby, 1988; Fonagy et al., 1991; Main et al., 1985), which can of course result in transgenerational re-enactment of problematic parenting styles.

A parent’s psychological wellbeing also appears to be significant when considering parenting ability, given that the extent of personal resources available to cope with the intrinsic demands of child rearing is likely to be considerably diminished in such a population (Carr, 1999). For example, a parent’s ability to match his/her infant, with respect to appropriate gestures and responses has been emphasised as a key to optimal parenting (Beebe & Lachmann, 1988) – something which has been demonstrated to be deficient in mothers with depression (Tronick & Gianino, 1986).

1.2.4 Measurement of parental bonding

Generally, three distinct methods have been utilised in the assessment of parental rearing practices: direct observation of parent-child interactions, parental self-report and offsprings’ retrospective reports.

1.2.4.1 Observation of parent-child interactions

Given the significant constraints on time and resources inherent in this method, direct observation of parent-child interactions has tended to be the least frequently used of
these three approaches, although is believed to probably be the most accurate, as actual child rearing behaviour is monitored. Generally, parent-child interaction and parental behaviour has been analysed with respect to the extent to which expression of care or emotional warmth is evident and the degree to which control over the child is manifested. Rapee (1997) reviewed several studies using this method to evaluate potential links between child rearing styles and development of anxiety or depression. He concluded that studies utilising this method were rare and were still subject to methodological limitations, as a ‘snapshot’ of child rearing behaviour under somewhat artificial observational conditions may not be a valid reflection of a child’s upbringing. Rapee also emphasised potential advantages of combining evidence across several methodologies, e.g., offspring self-report, parental or sibling self-report and observational data, in the attempt to obtain a representative picture of child rearing styles.

1.2.4.2 Parental self-report

Another means of evaluating parental bonding has been direct assessment of a parent’s self-reported child rearing behaviour⁵, either via nature of responses to questionnaires or analysis of information gained in semi-structured interviews, although again such studies have been fairly uncommon (see Rapee, 1997). As the design involves direct questioning of parents, offspring in these studies tend to be younger (i.e., children or adolescent populations) than those in studies assessing self-

⁵ Again, assessment has been broadly along dimensions of care and control.
report of adult offspring. A significant drawback of this method is the possibility that parental response may not be an accurate reflection of actual behaviour, given the negative connotations inherent in the majority of questions about child rearing behaviour (e.g., degree of control or intrusiveness). As such, one would anticipate a tendency for parents to rate themselves favourably - a bias that appeared to be evident in a study by Parker (1981a) when mother’s self-ratings were compared with reports given by their offspring (see section 1.2.4.4).

1.2.4.3 Retrospective reports of offspring

The most commonly used of the three methods has been assessment of individual’s recollections of their early experiences with their parents via self-report questionnaires. Although a variety of such questionnaires have been developed, psychometric suitability has only been reliably demonstrated in three measures (Gerlsma, Emmelkamp, & Arrindell, 1990; Rapee, 1997): the Children’s Report of Parental Behaviour Inventory (CRPBI: Schaefer, 1959), the Egna Minnen Betraffande Uppfostran⁶ (EMBU: Perris, Jacobsson, Lindström, von Knorring, & Perris, 1980) and the Parental Bonding Instrument (PBI: Parker et al., 1979).

1.2.4.3.1 The CRPBI

Of these three measures, only the CRPBI was designed for use with children in

⁶ Egna Minnen Betraffande Uppfostran translates as ‘my memories of upbringing’.
addition to adult offspring. Factor analysis of responses to questions assessing whether parental characteristics were / had been present\(^7\) in child and adult populations revealed three dimensions, the first two of which were bipolar in nature: acceptance versus rejection, psychological autonomy versus psychological control and also firm control versus lax control. Further studies examining the psychometric properties of the CRPBI have indicated that the first two factors are more reliable, with the third factor contributing little to the shared variance (Raskin, Boothe, Reatig, Schulterbrandt, & Odle, 1971).

1.2.4.3.2 The EMBU

Development of the EMBU arose as a consequence of research on the CRPBI and was formulated in an attempt to standardise information collated from patient populations regarding early experiences of parenting. Originally the EMBU was devised as 15 subscales in accordance with existing models of parental rearing behaviour. However, subsequent analyses (Perris et al., 1980; Arrindell, Emmelkamp, Monsma, & Brilman, 1983) implicated four significant factors: emotional warmth, rejection, overprotection and favouring subject, of which the first three have been consistently demonstrated cross-nationally\(^8\). The EMBU was originally a Swedish inventory but has since been translated into a variety of languages in an attempt to enable comparison of cross-cultural data (Gerlsma et al.,

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\(^7\) A forced-choice yes / no response format was used to express whether items resembled characteristics 'like' or 'unlike' the parent.

\(^8\) Populations sampled were all from Western cultures.
1.2.4.3.3 The PBI

The PBI was originally designed by Parker and colleagues (Parker et al., 1979) in an attempt to quantify assessment of individual’s perceptions of parental bonding. In line with previous factor-analytic research (Raskin et al., 1971; Roe & Siegelman, 1963) and clinical observation regarding significant constituents of parental rearing behaviours, items relating to the two dimensions of care and of psychological control were piloted on several non-clinical samples, with item reduction following factor analyses. The primary factor revealed consistently in the factor analyses was a bipolar dimension relating to care-rejection, with items at the former pole representing positive affection, empathy and warmth and those at the latter conversely depicting neglect, indifference and lack of approval or encouragement. Amalgamation of the next two factors (due to cross loading of items) resulted in a dimension relating to autonomy and independence at one pole versus overprotection and dependence at the other. The resulting questionnaire therefore contained 25 self-report items encompassing the two dimensions of care and control/overprotection, with responses rated along a 4-point Likert scale in accordance with the extent to which such a characteristic had been apparent. Responses were completed on separate questionnaires with respect to the mother and father figure, although scores could be averaged to provide an estimate of parental care and overprotection. Initial analysis indicated that the two dimensions were significantly negatively correlated, with high scores on the overprotection dimension associated with low scores for care.
- a finding that has been reproduced in subsequent studies (Cubis, Lewin, & Dawes, 1989; Parker, 1989) - and as such could not be said to be independent, but rather interdependent. The relative importance of care and control\textsuperscript{9} dimensions has been questioned (Rodgers, 1996a), with low care scores appearing to be more consistently associated with poor mental health than overprotection scores (see section 1.2.5). However, given the degree of correlation between the two scales and associations between both care and overprotection and psychopathology, Parker (1979a) and others (Rapee, 1997) proposed that focus on parenting styles involving the interaction between the dimensions might be more informative than assessment of care and overprotection in isolation. As such, Parker defined four possible parenting styles (Parker et al., 1979), similar in some respects to those referred to by Carr (1999; see section 1.2.3), using somewhat arbitrary cut-off points along care and overprotection dimensions (see Figure 1.2). Optimal bonding was felt to be represented by reports of reasonably high levels of care in combination with relatively low levels of overprotection and was assumed to be associated with greater likelihood of psychological well-being. The remaining three forms of parenting were anticipated to characterise less favourable childhood environments or distorted patterns of bonding, with the quadrant representing relatively high levels of both care and overprotection described as affectionate constraint, that depicting relatively low levels of both care and overprotection referred to as neglectful parenting / weak or absent bonding and finally that relating to low levels of care in conjunction with high levels of overprotection known as affectionless control. Parker posited that this final quadrant appeared to be the most negative of the parenting styles, with studies

\textsuperscript{9} Control and overprotection tend to be used interchangeably to describe the same dimension on the PBI.
Figure 1.2. Four quadrants of parenting behaviour relating to dimensions of care and overprotection (reproduced from Parker et al., 1979), with inclusion also of a statistically derived 'average' parenting type.
suggesting this approach seemed to exhibit the greatest link with later psychopathology (Parker, 1983a; Parker, 1983b; Plantes, Prusoff, Brennan, & Parker, 1988) – see section 1.2.5.

There have been a large number of studies investigating the psychometric properties of the scale (Gotlib, Mount, Cordy, & Whiffen, 1988; Mackinnon, Henderson, Scott, & Duncan-Jones, 1989; Mackinnon, Henderson, & Andrews, 1991; Parker et al., 1979; Parker, 1986; Plantes et al., 1988), with evidence indicating that the measure is psychometrically ‘robust’ with respect to both its reliability (internal consistency and stability) and validity (concurrent, construct and predictive).

Interestingly, despite the fact that the EMBU and the PBI were developed entirely independently of one another, similarity across the dimensions is clear. Indeed, Arrindell et al. (1986) suggested that their findings for the EMBU in Swedish and Dutch samples also supported a two-factor model of parenting dimensions, namely care (including both emotional warmth and rejection) and protection. However, Livianos-Aldana and Rojo-Moreno (1999) warn against such straightforward comparison between the two measures, having found only moderate, rather than high, correlations between corresponding scales (i.e., PBI care and EMBU affectional warmth; PBI overprotection/control and EMBU overprotection). They urged for cautious interpretation therefore, given that their results suggested that the two measures do not necessarily assess identical factors.

10 See Parker (1989) for a comprehensive review of relevant studies.
However, further support for such a two-factor model has been implicated by several studies utilising different populations. Kazarian, Baker, and Helmes (1987) examined psychometric properties of the scale in a group of schizophrenic patients, concluding that the two factors identified by Parker et al. (1979) accounted for a significant percentage of the variance in their sample. The consistency of their results in a clinical population against Parker’s normative data led the authors to support the internal structure of the measure. Likewise, Mackinnon et al.’s (1989) study of a general community population supported the presence of a two-factor structure and also indicated that the PBI demonstrated a high degree of internal consistency and good test-retest reliability.

Nonetheless, it would appear that the care dimension has been more easily defined than that of overprotection, resulting in the assertion that a two-factor model might not be the most appropriate. Indeed, Cubis et al. (1989) proposed that a three-factor model provided the best fit for their data from a large-scale Australian community adolescent sample, with the care dimension remaining but the original overprotection scale subdivided into two separate factors representing protection in personal and social domains respectively. The authors also concluded that sex differences, which had been obscured by the original two-factor structure, became apparent using such a model - with higher ratings of paternal personal intrusion discernible in female adolescents. Gómez-Beneyto, Pedrós, Tomás, Aguilar, and Leal (1993) also endorsed the view that a three-factor structure should be adopted when using the PBI, given that the predictive power of the measure increased (with respect to identifying those at risk for post-natal depression in their sample of Spanish mothers) when the control scale was divided into dimensions of restraint and overprotection.
However, they did also note that the cross-cultural constancy of normative scores (using the original two-factor structure) indicated the scale’s reliability. More recently, factor-analysis of PBI scores in a student sample also suggested the benefits of a three-factor structure — representing *care, denial of psychological autonomy* and *encouragement of behavioural freedom* — again with respect to the measure’s ability to detect between-group differences and predict psychopathology (Murphy, Brewin, & Silka, 1997). Moreover, Murphy and colleagues emphasised the fact that the two factors relating to control should *not* be viewed as subcategories of the same dimension but instead as distinct factors. However, results were not shown to differ substantially in a study examining associations between lifetime history of depression and parental bonding in Japanese adults (Narita, Sato, Hirano, Gota, Sakado, & Uehara, 2000), whether scores were analysed with respect to either Parker’s original two-factor model or the three-factor model.

Initial assertions by Parker et al. (1979) that demographic factors were not related to scoring on the PBI primarily seem to have been supported. Several studies suggest that responses on the PBI are not significantly influenced by socio-economic status, as assessed by ratings of paternal occupation (Parker, 1983a) and relation to respondents level of education (Mackinnon et al., 1989). Likewise, there do not appear to be any consistent or major differences in PBI scores according to sex of respondent (see Parker, 1989; Parker, 1990; Rodgers, 1996a). Age of respondent also does not seem to exert a significant influence in studies of adult populations (Arrindell, Hanewald, & Kolk, 1989; Parker et al., 1979; Parker, 1990), although it
did appear to have an effect on perceptions of parenting in adolescents\(^{11}\) (Rey, Bird, Kopec-Schrader, & Richards, 1993). (A slight effect of age was also noted by Parker, Kiloh, and Hayward (1987) in their study of neurotic and endogenous depressives, with more favourable ratings of parental bonding apparent with increasing age.) However, the PBI does appear to be somewhat sensitive to cultural influences across parenting behaviours, with differences apparent between female Australian and Greek adolescents (Parker & Lipscombe, 1979).

The PBI has probably been the most widely used measure with respect to examination of the possible influence of parental bonding on later vulnerability to psychopathology. The results of such studies will be detailed in Section 1.2.5.

### 1.2.4.4 Critique of retrospective methods

Criticisms of the PBI and other similar self-report questionnaires primarily centre on the validity of such measures given their retrospective nature, as they only involve assessment of perceived parenting behaviours and therefore accurate assessment of actual parental rearing cannot be assumed. Without evidence of any concordance between perceived reports of bonding and actual experience, potential causal relationships remain undetermined.

Some authors have countered these comments by suggesting that subjective perceptions, with respect to how an individual has interpreted and evaluated their

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\(^{11}\) Such findings were believed to be consistent with other evidence suggesting changes in adolescents' perceptions of their parents over time.
parents’ behaviour, are probably more relevant than how their parents’ actually behaved when investigating the influence of parental bonding on later development (Parker, 1983a; Parker, 1989; Robins, Schoenberg, Holmes, Ratcliff, Benham, & Works, 1985). Indeed, to some degree, individual interpretation will be inevitable in assessment of such characteristics involving parental values, perspectives and feelings. However, there is general agreement as to the need to examine degree of correlation between perceived and actual experiences (Rapee, 1997) and there have been some attempts to achieve such objectives.

Comparison of parents’ (mothers only) ratings of their own behaviour and those of their offspring resulted in a reasonable degree of correlation in a study by Parker (1981a), although, perhaps unsurprisingly, mothers tended to rate themselves more favourably than did their offspring (i.e., more caring and less controlling). Similarly, in the same study, a moderate correlation was found between offspring’s own ratings and those of their sibling, suggesting that PBI scores might reflect actual parenting experiences. However, it was also noted that there was poor discrimination between participants’ self-ratings and their ratings of their sibling, suggesting that further evidence would be necessary to establish the validity of the PBI. Studies examining same-sex twins raised together enable minimisation of variation inherent in comparison of sibling reports, as age and sex are constant, with development progressing at similar rates and the parenting environment comparable. If reports of parental behaviours reflect actual experience, one would expect similar levels of agreement between twin-pair ratings, with ratings between monozygotic (MZ) and dizygotic (DZ) twins roughly equivalent. However, genetic factors might be implicated if MZ ratings exhibit significantly greater concordance than do those of
DZ twins. Significant correlations between twin-pair ratings for both MZ and DZ twins were found in a small-scale study by Parker (1986), with similarity in the degree of correlation apparent between MZ and DZ twins suggesting the PBI to be a valid measure of actual parenting experiences. However, findings of a further study involving a larger twin population (Mackinnon et al., 1991) were less clear cut. Agreement between female twin-pairs was greater than that demonstrated in male twins, with the authors concluding that 3 of the 4 PBI scales	extsuperscript{12} seemed to reflect actual parenting in the female twin sample. Nevertheless, it was felt that further investigation would be necessary to clearly determine validity of the PBI, given the lack of similarity between male DZ twin pairs. Of course, prospective longitudinal studies are the best means of evaluating validity and establishing evidence for degree of concordance between objective parental behaviour and subjective reports.

Another major drawback of retrospective methods is the question of accuracy of an individual’s recollections and the extent to which such recall might be influenced by mood-specific factors. There is some evidence to suggest that social desirability does not tend to affect the nature of an individual’s responses on the PBI (Mackinnon et al., 1989; Parker, 1983a; Parker, 1983b), with Robins et al. (1985) reporting that while recall is not perfect, it is generally significantly better than chance. If the PBI is to facilitate meaningful research into the relationship between parental bonding and vulnerability to psychopathology (see section 1.2.5), it is crucial that the measure can be demonstrated to be independent of mood state (Brewin, Andrews, & Gotlib, 1993; Gerlsma, Kramer, Scholing, & Emmelkamp, 1994). As the PBI relies on memory

\textsuperscript{12} Concordance was significant for all scales except the maternal care scale.
and recall of earlier experiences, it might be anticipated that responding would be congruent with current mood state, given the body of experimental research demonstrating recall biases for mood-congruent information (see Dalgleish & Watts, 1990; Mathews & MacLeod, 1994), with the suggestion that emotional arousal promotes accessibility to mood-congruent information. Likewise, assertions central to cognitive theory as to the presence of negatively distorted cognitions in individuals with depression (Beck, 1967) suggest that mood state tends to bias interpretation of events. Some experimental studies investigating the effect of mood on memory have shown preferential recall of negatively valenced information in both clinically depressed individuals (Williams & Scott, 1988) and in healthy controls, following mood-induction techniques (Perrig & Perrig, 1988; Teasdale & Russell, 1983). However, further research has indicated that selective recall of negatively valenced information only tends to be apparent in depressives when such information is self-referent in nature (Denny & Hunt, 1992). Brewin et al. (1993) therefore suggested that the lack of any obvious bias in depressives’ reports on the PBI might relate to the fact that such information primarily involves memory for others.

In addition, other studies have found that induction of a depressed mood state only seems to result in either increased latency or inhibited recall of positively valenced memories, in comparison with recall following induction of an elated mood, with no apparent effect on recollection of negative memories (Natale & Hantas, 1982; Teasdale & Fogarty, 1979). Such findings might relate to the suggestion that recall could even be more accurate in depressed individuals (depressive realism), reflecting actual negative experiences, in contrast to unrealistically positive memory distortions (illusory glow) in controls (Alloy & Abramson, 1979).
Concerns that self-report on retrospective measures, such as the PBI, is not independent of mood state were highlighted by Lewinsohn and Rosenbaum (1987), following indications from their study of depressed individuals. They found a significant difference between recall of parental care in individuals who were depressed when completing the CRPBI and in those whose depression had remitted at the time of the assessment. Lewinsohn and Rosenbaum therefore concluded that depressed mood state was likely to bias recall in a negative manner, with lower estimates of parental care evident, in turn questioning the validity of such responses. However, it was notable that similarity in recall was apparent between depressives and females who had not been depressed at initial assessment, but who had subsequently developed depression at one-year follow-up.

Evidence from further studies seems to refute Lewinsohn and Rosenbaum’s claims, indicating that individual’s reports of parental bonding do not in fact appear to be influenced by current mood state. Using a longitudinal design, Gotlib et al. (1988) demonstrated stability of perceptions of parental bonding on the PBI in a group of women suffering from post-natal depression\(^{13}\). Participants were followed-up over a period of two to four years and were assigned into one of three groups, according to BDI\(^{14}\) scores over the two assessment points: depressed (at times 1 and 2), remitted (depressed at time 1, non-depressed at time 2) and non-depressed (at times 1 and 2). It was demonstrated that PBI scores remained fairly consistent (irrespective of group) between initial assessment (3 days after giving birth) and follow-up despite

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\(^{13}\) Postpartum depression was assessed according to scores on the BDI, with scores over 9 classified as depressed.

\(^{14}\) Beck Depression Inventory (Beck, Ward, Mendelson, Mock, & Erbaugh, 1961)
fluctuations in depressed mood. Higher overprotection scores were apparent for depressed and remitted individuals than for controls at both times 1 and 2, with lower care scores only apparent in depressed individuals. Gotlib and colleagues therefore suggested that initial reports of low care appeared to predict level of depression approximately 30 months later. Similar findings of stability of PBI scores were apparent in a U.S. study of clinically depressed individuals (Plantes et al., 1988), with responses remaining consistent across a four to six week period despite improvements in depressed mood over this time. Likewise, test-retest stability was demonstrated in responses on the EMBU over a six-month period in individuals with unipolar depression, dysthymic individuals, non-depressed psychiatric controls and healthy controls, with responses seemingly independent of changes in mood state within individuals (Gerlsma, Das & Emmelkamp, 1993). Another study by Gerlsma et al. (1994) attempted to further establish whether mood state influenced recall of parental behaviour, given the implication of such findings with respect to interpretation of associations between parental bonding and psychopathology. A large representative community sample completed self-report measures of depression, anxiety and hostility, in addition to both the PBI and EMBU, on two separate occasions, six months apart. Although results indicated that the care scales were slightly susceptible to changes in anxiety, the authors concluded that such biases had a minimal impact overall and indeed did not appear to be replicated in a selected sample in a second study. Gerlsma et al. therefore commented that results seemed to indicate “...some confidence in the stability of recalled parental rearing styles as measured with the PBI and EMBU”. They also suggested that differences in methods used might account for presence and absence of mood bias in depression,
with studies examining biases in memory often using free-recall procedures in contrast with the more standardised forced-choice format of measures of parental bonding.

Therefore the majority of evidence appears to support the conclusion that parental representations are relatively stable over time, with links between patterns of scoring (i.e., low parental care / high parental overprotection) and depressive state appearing not to merely reflect biases in reporting due to mood state.

Although automatic attentional biases for threat-related information appear to be evident in anxiety (Mathews & MacLeod, 1985; Mogg, Bradley, Williams, & Mathews, 1993), biases in memory recall have not been demonstrated (Mathews & MacLeod, 1994), suggesting that recollection of parental bonding experiences should not be influenced by anxiety states.

Likewise, the possibility of a negative response set significantly influencing reports of parental bonding seems to have been largely rejected. Some evidence against a plaintive response set is suggested by the similarity of findings in both community and patient samples (Duggan, Sham, Minne, Lee, & Murray, 1998; Parker, Hadzi-Pavlovic, Greenwald, & Weissman, 1995). In addition, findings that associations remain between dysfunctional parental styles and psychopathology once personality traits such as neuroticism have been controlled for (Duggan et al., 1998; Parker, 1983a) lend further support that such relationships are not merely mediated by response biases or a personality style influencing reporting.
In addition, the PBI is based on the assumption that parental rearing practices do not substantially differ over time, given that offspring are asked to indicate their responses to the questions in view of the first 16 years of their life. As such, reports assessed by the questionnaire are likely to represent an amalgamation of an individual’s childhood experiences\(^{15}\) (rather than reference to any single specific event – no matter how meaningful) and could therefore be liable to constructive biases (Gerlsma et al., 1990; Rapee, 1997). Such unwitting false reports have been described by Ross (1980), with the suggestion that individuals might implicitly rewrite their past in line with their current expectations and awareness. Again, validity is likely to be enhanced if information is obtained from several sources (i.e., sibling report, parental report) – with such studies appearing to support the validity of the PBI (e.g., Parker, 1981a) – although the use of prospective longitudinal designs is clearly optimal. The fact that responses on the PBI do not appear to be influenced by age of respondent (Arrindell et al., 1989; Parker et al., 1979), in addition to findings that PBI reports are relatively stable over time (Gotlib et al., 1988; Mackinnon et al., 1989), does however suggest that perceptions of parental bonding do not change according to the recency of parenting experience.

\(^{15}\) Rapee (1997) highlighted the possibility that there might be critical stages during childhood at which parental style might exert a greater influence on vulnerability – therefore evaluation of specific experiences within childhood might be more useful than assessment across childhood as a whole.
1.2.5 Influence of parental bonding on later psychopathology

Initially, when investigating the potential influence of early experiences on later psychopathology, focus was directed towards the effect of specific traumatic or adverse life events, such as parental loss or separation (Tennant, Bebbington, & Hurry, 1980). More recently however, emphasis has shifted towards examination of more enduring aspects of the early environment, for example, style of parenting, following recognition that childhood deprivation could encompass more than loss of or separation from parental figures and could include deprived rearing practices (Jacobson, Fasman, & DiMascio, 1975).

Brown and Harris (1993) emphasised the role of early childhood adversity in the aetiology of psychopathology, supported by their finding that parental indifference and abuse (physical and sexual) were significantly associated with presence of both depressive and anxiety disorders in a study of working-class women and single mothers.

The majority of research has primarily focused on aspects of parental bonding when examining the influence of early experiences\textsuperscript{16} on vulnerability to psychopathology. In particular, investigation has centred on the two major dimensions of parental bonding derived from factor-analytic studies, i.e., care and control/overprotection, with most studies having used retrospective self-report measures, such as the PBI. Indeed, Parker and his colleagues have conducted a large proportion of such

\textsuperscript{16} Clearly, early experiences encompass more than just the nature of parental bonding – for example, other meaningful aspects of early experience include significant life events and other facets of the environment (e.g., pertaining to the wider social and physical environment).
research, both with clinical and control populations, which will be detailed below alongside other relevant studies.

1.2.5.1 Relation to depression

A large number of studies have attempted to investigate the relevance of parental bonding in the development of depression. Given the methodological limitations of early research, for example, heterogeneity of samples with respect to type of depression, Parker (1979a) endeavoured to overcome such difficulties by examining whether differential effects were apparent across subgroups of depressives, using the then recently developed and validated PBI. Findings from his study of neurotic and bipolar depressives revealed significantly elevated reports of low parental care, and to a lesser extent high maternal overprotection, in the group of individuals with neurotic depression compared to either bipolar depressives or respective groups of matched controls. Similar results were also apparent in a student population, with low parental care and a trend for higher parental overprotection shown to be associated with depressive experience (assessed via self-report). Another study by Parker and colleagues (Parker et al., 1987) likewise indicated differences between types of depression, with associations between reports of low parental care and high parental overprotection again apparent in a sample of neurotic depressives, but absent in both endogenous depressives and control groups. Parker suggested that associations with dysfunctional parenting were perhaps unsurprising in the group of

17 'Neurotic' (also known as 'reactive') depression was / is believed to relate to a form of depression which has its origins in psychosocial (as opposed to genetic) factors.
neurotic depressives, given the importance of supposed psychosocial aetiological factors when according such a diagnosis (in contrast to the emphasis on genetic and biological factors in endogenous depression). These findings led Parker to speculate that distorted patterns of parental bonding might present a differential risk factor for distinct forms of depression, as opposed to characterising a more general vulnerability (see section 1.2.5.4). However, such specificity was not so evident in a study by Perris, Arrindell, Perris, Eisemann, van der Ende, and von Knorring (1986), examining parental bonding on the EMBU in four different diagnostic subgroups of depressives (unipolar, bipolar, neurotic-reactive and unspecified depressive disorder) and in healthy controls. Emotional warmth was rated as lower across all patient groups compared to controls (although the difference was only statistically significant for the unipolar and unspecified depressed groups) leading the authors to surmise that deprivation of love in childhood appeared to be a significant risk factor for depression, regardless of subtype\textsuperscript{18}. Similarly, Gerlsma et al. (1993) found no difference between unipolar depressives and individuals diagnosed as dysthymic, with respect to reports of parental rearing styles (also assessed by the EMBU) - with lower levels of emotional warmth and higher levels of rejection and maternal overprotection evident in the clinical sample compared to matched controls.

Greater levels of perceived emotional rejection were also apparent on the CRPBI in clinically depressed patients in a study by Lewinsohn and Rosenbaum (1987) – a finding that was particularly evident for females. However, no differences were apparent between depressed and non-depressed individuals with respect to

\textsuperscript{18} The authors did note, however, that their individual sample sizes were relatively small for group comparisons.
dimensions of control or nature of discipline used. Likewise, primacy of the care dimension was evident in a study by Duggan et al. (1998) with perceptions of low care associated with a history of depression in a non-clinical sample predicted to be at familial risk for depression. Similar results have been apparent in community samples, with reports of low care again having the greatest association with depressive experience (Mackinnon, Henderson, & Andrews, 1993; Narita et al., 2000). However, Kerver, van Son, and de Groot (1992) noted maternal overprotection as the greatest risk factor for depression at one-year follow-up in their prospective study of a community sample.

Dimensions of both care and control appeared to be relevant in a further study of neurotic depressives by Parker (1983b), with the interaction of the two parenting dimensions - representing a parental style of affectless control - appearing to discriminate well between clinical and control populations. These findings were replicated in a study of depressed out-patients (Plantes et al., 1988), with reports of both low parental care and high parental overprotection again apparent in the depressed group compared to matched controls. However, low scores (of less than 10) on the care dimension seemed to be the best discriminator between depressed and non-depressed samples (Parker, 1983b; Plantes et al., 1988). Gotlib et al. (1988) also noted that reports of low parental care distinguished between currently depressed women and both non-depressed and remitted groups of women. However, higher levels of parental overprotection were apparent in both depressed and remitted groups compared to the non-depressed sample, leading Gotlib and colleagues to

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19 Depression was assessed according to scores on the BDI rather than clinical diagnoses.
conclude that perceptions of parenting appeared to be remarkably stable across changes in mood state (see section 1.2.4.4. for further discussion of mood state influences).

Although the majority of studies have been carried out in Western cultures, Sato, Sakado, Uehara, Nishioka, and Kasahara (1997) assessed reports of parental bonding in a clinically depressed population in Japan and found remarkably similar results. Reports of low parental care and high parental overprotection were significantly more evident in those with a diagnosis of non-melancholic depression compared to controls, implicating that such associations between dysfunctional parental bonding and depression are independent of culture.

Reports of low parental care also do not appear to be merely a function of help-seeking behaviour or clinical status, given that similar results are evident in community (Parker et al., 1995) and non-clinical (Duggan et al., 1998) samples (see section 1.2.4.4).

Therefore, both low parental care and high parental overprotection seem to have been implicated as potentially meaningful risk factors for depression, with results for care appearing both more consistent and striking than for overprotection. McCranie and Bass (1984) summarised some of the findings for depression, speculating means by which such parental styles could lead to difficulties, stating that "...depression proneness in general is influenced by parental childrearing practices that combine elements of rejection, inconsistent expression of affection, and strict control. Such

20 A similar trend was found for melancholic as with non-melancholic depressives, but differences from controls were not statistically significant in this group.
behaviours could be expected to hinder the development of normal self-esteem in the child, resulting in an increased vulnerability to generalised feelings of helplessness and failure".

1.2.5.2 Relation to anxiety

Several studies have also investigated whether any associations are evident between parental bonding styles and anxiety conditions. Parker (1979b) found overall differences between a combined phobic group\(^{21}\) and controls, with lower ratings of parental care and higher ratings of parental control evident in the phobic population. However, he also noted differences within the phobic sample, with responses from social phobics consistent with the overall pattern (i.e., low parental care and high control) but results from agoraphobics indicating only lower maternal care scores in comparison with controls. Further evidence that parental rearing styles might exert a differential influence on subtypes of anxiety was revealed by Arrindell et al. (1983), on a clinical sample of social, simple and agoraphobics and a non-patient control sample. Ratings were assessed on three of the four scales of the EMBU – emotional warmth, rejection and overprotection. Responses of social phobics and simple (height) phobics were broadly comparable, with lower ratings of emotional warmth in both parents in combination with higher ratings of both parental overprotection and parental rejection compared to the control group. In contrast, although agoraphobics also rated both parents as expressing less emotional warmth, they only

\(^{21}\) It should be noted that although individuals had been assigned clinical diagnoses of agoraphobia and social phobia, this had occurred some 5-7 years previously and therefore individuals could not be assumed to be currently suffering from an anxiety state.
additionally differed from controls with increased ratings of maternal rejection. Arrindell and colleagues therefore stressed the need for independent examination of different anxiety subdiagnoses when evaluating influence of parental rearing characteristics. Silove (1986) also highlighted the likely importance of lack of parental care as a risk factor for agoraphobia, with both low parental care and high parental overprotection apparent in agoraphobic individuals’ reports of parental bonding experiences compared to matched controls. However, associations with low parental care and high parental overprotection were also apparent in two different samples of clinically anxious out-patients (Parker, 1981b; Silove, Parker, Hadzi-Pavlovic, Manicavasagar, & Blaszczynski, 1991), in comparison with matched control samples. Such results, in contrast to those of Arrindell et al. (1983), seem to suggest that parental rearing characterised by an affectionless control style might act as a non-specific risk factor for anxiety in general.

1.2.5.3 Relation to other psychological conditions

Although the majority of studies have been conducted with reference to either depression or anxiety states, the relevance of parental rearing styles has also been examined with respect to eating disorders, personality disorders, offending behaviour and obsessionality traits.

[22] However, Silove et al. (1991) did note some slight, albeit non-significant, differences between generalised anxiety disorder and panic disorder patients, with an affectionate constraint parenting style appearing to better represent those with panic disorder.
Given the importance accorded to degree of perceived control in some theories of eating disorders (Slade, 1982), one might assume evidence of parental overprotection in such individuals. However, results to-date appear to have been somewhat inconsistent. Although reports of low care and overprotection were associated with abnormal eating attitudes in a non-clinical sample (Calam & Slade, 1987), only low levels of care distinguished between perceptions of parental bonding in anorexic and bulimic patients versus those of controls in a study by Palmer, Oppenheimer, and Marshall (1988). A further study by Calam, Waller, Slade, and Newton (1990) also demonstrated that individuals with eating disorders (anorexic, bulimic and mixed) rated their parents as less caring than controls. However, in the latter study, reports of greater paternal overprotection were also apparent in eating-disordered individuals.

Investigation of relationships between parental bonding and personality disorders revealed a significant association between a paternal style of affectionless control (i.e., low care and overprotection) and levels of personality disorder pathology in male psychiatric in-patients, with traumatic childhood experiences and maternal parenting style appearing more crucial in the corresponding female sample (Modestin, Oberson, & Erni, 1998). Similarly, reports of low parental care and parental overprotection were evident in a group of borderline personality disorder patients (Zweig-Frank & Paris, 1991). However, in a later study by the same authors, also examining associations with borderline personality disorder (Zweig-Frank & Paris, 2002), PBI scores did not show any relation to outcome. Low parental care appeared to distinguish between patients with avoidant personality disorder and matched controls, with an absence of differences on the overprotection scale, in a study by Stravynski, Elie, and Franche (1989). Recollections of low levels of
maternal care were also demonstrated in both schizotypal and borderline personality disorder patients (Torgersen & Alnæs, 1992), with reports of maternal overprotection again evident in borderline patients but conversely low levels of overprotection common in schizotypal individuals.

Associations between offending behaviour (as assessed in samples of incarcerated young men) and parental rearing styles have also been demonstrated in a series of studies by Power and colleagues (Biggam & Power, 1998; Chambers, Power, Loucks, & Swanson, 2000, 2001). In contrast with scores from a normative sample of young males (Cubis et al., 1989), a group of young offenders were found to rate their parents as both less caring and less overprotective (Biggam & Power, 1998), although ratings of maternal care were higher in the offender group. The authors consequently suggested that a degree of parental overprotection does not necessarily equate to dysfunctional parenting, but might actually be more beneficial than a permissive style where a lack of clear rules and boundaries are indicated to the child and little control is implemented over behaviour. Further studies examining the relationship between perceptions of parenting and levels of psychological distress (according to scores on the HADS\(^{23}\) and the BHS\(^{24}\)) in young offenders indicated associations between low parental care and heightened psychological distress (Chambers et al., 2000, 2001).

Hypotheses that obsessional personality traits might reflect overprotective patterns of parenting were tested in a study by Cavedo and Parker (1994) on a non-clinical sample. Females scoring higher on measures of obsessionality were indeed shown to

\(^{23}\) Hospital and Anxiety Depression Scale (Zigmond & Snaith, 1983)
\(^{24}\) Beck Hopelessness Scale (Beck, Weismann, Lester, & Trexler, 1974)
report higher levels of parental overprotection on the PBI – a finding that remained after controlling for levels of depression, anxiety and neuroticism. An association between *paternal* overprotection and obsessionality was also apparent for males, although these results were non-significant once levels of neuroticism had been controlled. Cavedo and Parker urged caution in equating such results with obsessive-compulsive disorder, encouraging the need for more studies with such a patient group. However, associations were not apparent between obsessive-compulsive characteristics and parental rearing style in another non-clinical sample (Mancini, D'Olimpio, Prunetti, Didonna, & Del Genio, 2000). Such discrepant findings between the two studies could perhaps be accounted for by the different measures of obsessionality chosen.

1.2.5.4 Specificity or generality of parental bonding influences on psychopathology?

Although results from the majority of these studies appear to propound the negative influence of parental styles characterised by low care and overprotection (although the role of the latter is less consistently advocated), the question remains as to whether such risk factors exert a *differential* effect on *specific* forms of psychopathology or whether they act as a *general* risk factor for psychopathology *per se*. Proponents of the idea that dysfunctional parenting relates in a non-specific manner to development of psychopathology include Enns, Cox, and Clara (2002), who found minimal differences across a variety of clinical diagnoses in perceived parental rearing styles, with lack of care being the parenting characteristic most
consistently correlated with psychopathology. Likewise, Mancini et al. (2000) concluded that “...poor parental bonding, especially regarding care, could constitute a general factor for emotional distress in adulthood, rather than acting as a precursor to a specific disorder”, following their findings that low care and overprotection correlated with high scores for both depressive and anxious symptomatology in a non-clinical population. Gerlsma et al. (1993) also noted that reports of parental rearing were remarkably consistent across unipolar depressives, dysthymic individuals and non-depressed psychiatric controls in comparison with reports from healthy controls. Indeed, given the preponderance of findings implicating a parental style of low care and overprotection (affectionless control) in various psychological conditions, it is perhaps unsurprising that Calam et al. (1990) suggest that “…the PBI may not be particularly useful for predicting different psychopathologies”.

However, other researchers appear to support the possibility that different forms of dysfunctional parenting might relate to specific types of psychopathology. Indeed, Parker, Roussos, Hadzi-Pavlovic, Mitchell, Wilhelm, and Austin (1997) advocated such a viewpoint25, given the number of findings indicating a degree of specificity of anomalous parenting. Differential associations between parenting style and anxiety diagnoses appeared to be evident in a study by Arrindell et al. (1983). Likewise, Parker demonstrated differences in reports of parental bonding between subtypes of depression (Parker, 1979a; Parker et al., 1987). Alnæs and Torgersen (1990) were

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25 However, Parker et al. (1995) notably emphasised the likelihood that low parental care was a non-specific or “pathoplastic” risk factor for psychiatric disorder per se, given their results in a community sample.
also able to discriminate to some extent between patient groups (with diagnoses of pure depression, pure anxiety and mixed anxiety-depression) according to responses on the PBI. Therefore, findings to-date appear inconsistent with respect to the potential specificity of parental rearing styles on the nature of psychopathology and will undoubtedly require further examination from studies with prospective longitudinal designs. However, it would seem clear that at least some forms of psychopathology are associated with reports of low parental care and perhaps also parental overprotection, enabling predictions to be made in the current study. In addition, the majority of studies investigating associations between scores on measures of parental bonding and psychopathology have examined specific patient populations (with the exception of Enns et al., 2002), where the current study aims to investigate associations across a general patient (with a variety of psychological diagnoses) and control population with respect to levels of depressive and anxiety symptomatology.

1.2.5.5 Potential mechanisms to explain associations between parental bonding and psychopathology

Several explanations have been proposed as to possible mechanisms by which aspects of parental bonding might relate to later psychopathology (Duggan et al., 1998; Parker, 1981a,b, 1982, 1983b; Rapee, 1997), as causality cannot necessarily be assumed. If the validity of individuals’ self-report on measures such as the PBI is accepted, as evidence appears to suggest (see section 1.2.4.4) with respect to stability of reports across fluctuations in mood state and apparent corroboration with actual
parenting, three possible mechanisms could account for the observed associations. One hypothesis is that presence of anxiety or depression in the child (creating a negative temperament) tends to elicit such negative parenting styles, whereby parents perhaps feel less able to be affectionate towards their offspring and are inclined to be more overprotective of them. Although a degree of interaction between parent-child temperaments is likely (Cassidy, 1994; Kendler, 1996), there has been some evidence to suggest that such a mechanism does not entirely account for associations between parental rearing styles and later psychopathology. Parker (1981a) found that an association between parental bonding and depression remained once the effect of childhood dependency traits (which might be predicted to elicit negative parenting) had been controlled for. In addition, Neale, Walters, Heath, Kessler, Perusse et al. (1994) found that a model representing a causal relationship between parental bonding and depression was the best fit for the data in their study of twins, rather than a model characterising depression in offspring as eliciting such dysfunctional parenting. Another possibility that has been proposed is the chance that a third or shared factor could actually be causing a spurious relationship between parental bonding and psychopathology – for example, the possibility that a genetic predisposition for anxiety is shared between parent and offspring. Such a shared factor could account for the variance in both the nature of parenting and level of offspring psychopathology, with parental psychopathology potentially creating difficulties in childrearing (Carr, 1999) and with the existence of a genetic predisposition to psychological disorder in offspring (potentially occurring in childhood and/or adulthood) explaining observed levels of psychopathology. However, the fact that associations between anomalous parenting and trait depression
and anxiety levels remained strong in a non-clinical sample of adoptees (Parker, 1982) suggests that the presence of a third shared hereditary factor might not be the best explanation for observed relationships. The remaining possibility, then, is that dysfunctional rearing styles in some way enhance vulnerability to psychopathology, in line with Bowlby’s (1988) views on the influence of attachment processes on later development. Evidence for such a causal process has only been indirect, being implicated in a sense by gradual exclusion of other explanations (as above) and by research indicating the validity of measures of parental bonding (see section 1.2.4.4), with more conclusive support only achievable from prospective longitudinal studies. However, the possible mechanisms by which low parental care and overprotection could exert an influence on psychological vulnerability seem somewhat logical. Plantes et al. (1988) provided such a rational description, stating that “...low parental care may dispose by impairing self-esteem development, while parental overprotection may slow or impair the normal socialization process to independence, leaving the recipient relatively unprepared to deal with life event exigencies in adulthood”.

Therefore, although evidence from studies using prospective longitudinal designs is required to substantiate apparent findings and non-causal hypotheses cannot as yet be definitively rejected, it would seem that dysfunctional rearing styles, encompassing low levels of parental care and high levels of parental overprotection, in some way exert a pathogenic influence on psychological well-being. However, even if a causal relationship appears the most likely explanation of existing findings, there is general agreement that associations between parental bonding and psychopathology are relatively small (albeit consistent), implicating the need to consider a variety of other
factors when examining predictive factors in psychopathology (Gittleman, Klein, Smider, & Essex, 1998; Mackinnon et al., 1989; Perris, 1988; Rapee, 1997; Rodgers, 1996a).

1.2.5.6 Mediating factors

A number of researchers have suggested that the relationship between parental bonding and psychopathology might be mediated by other factors, such as self-esteem (Chambers et al., 2000; Parker, 1993), social competency (Rodgers, 1996b) or the nature of an individual’s core beliefs or cognitive style (Parker, 1993; Perris, 1988).

The role of low self-esteem as a possible mediator of the relationship between dysfunctional parenting and psychological distress was indicated in a study of young offenders by Chambers et al. (2000). In addition, if offenders rated relationships with other inmates as problematic, they were also likely to have expressed perceptions of low levels of care from their parents and exhibit high levels of psychological distress. Lloyd and Miller (1997) also found some evidence that self-esteem might act as a mediator between perceived parental style and depression in their study of medical students, although such relationships were only apparent in male subjects.

Rodgers (1996b) argued that interpersonal experience and personal relationships throughout adult life, such as extent of emotional support, quality of one’s social network and availability of others for help in crisis, were of equal or greater
importance in determining the likelihood of development of psychopathology compared to the role of early parenting. Rodgers suggested that such interpersonal variables could possibly mediate the relationship between dysfunctional parental bonding and vulnerability to psychological distress. He also noted some differences in the relative importance of contributing variables dependent on sex, with marital status, for example, appearing significant for women but not for men.

Indeed, Perris (1988) stressed the likelihood that a multi-factorial model would best explain findings to-date, given the undoubtedly large number of variables contributing to development of psychopathology. Perris argued that a coherent theoretical framework underpinning the link between anomalous parenting and psychopathology was needed and proceeded to describe a model whereby dysfunctional cognitive schemas were posited as a mediating factor. This idea corresponds with Bowlby’s (1969) proposition of the development of internal working models as representations to understand and guide relationships, the nature of which he believed would be largely shaped by early experiences – a concept which will be elaborated on further in section 1.4.1. Parker (1993) also highlighted the possibility that a negative cognitive style could act as a mediating factor, with partial support for such assertions provided in his study of depressed individuals. As such, the current study aims to establish whether dysfunctional core cognitive schemas do indeed mediate the relationship between parental bonding and psychopathology.
1.2.5.7 Moderating factors

Following a review of the relevant literature, Parker, Barrett, and Hickie (1992) concluded that the impact of negative early experiences could be modified by a number of factors, in particular subsequent interpersonal relationships, in accordance with a dynamic view of development. The moderating effect of later relationships on susceptibility to mental health difficulties has also been documented by Brown and Harris (1978) and Parker and Hadzi-Pavlovic (1984), with good quality marital relationships appearing to counteract negative effects of dysfunctional parenting at least to some degree. Conversely, in the latter study, positive effects of optimal styles of parenting seemed to be invalidated by marriage to an unaffectionate partner. Similarly, presence of a secure adult attachment style (assessed with respect to close relationships) appeared to exert a buffering influence on negative effects of dysfunctional parenting (Gittleman et al., 1998). Consequently these authors argued that both continuity and discontinuity theories appeared to be upheld, with regard the influence of early parenting on later development. Birtchnell (1993) also found that poor maternal care\(^{26}\) appeared to be associated with poorer quality of marital relationships (alongside a tendency for marriage to occur earlier) in a female community sample, even after the significant association between poor maternal care and depression was held constant.

Rodgers (1996a) attempted to determine whether a variety of factors representing childhood adversity, such as low socio-economic status, moderated the relationship between parental rearing styles and psychological well-being in a non-clinical

\(^{26}\) The PBI was used to assess recollections of parenting.
sample. Although associations were not found to be significant for any of the variables representing childhood adversity, high levels of affective symptomatology were evident in individuals who had experienced some kind of adversity (e.g., family disruption) with parental bonding adding little to the predictive value of the relationship. Rodgers therefore concluded that "Reports of relationships between any specific aspect of the early environment and adult disorder should acknowledge the possible contribution of other aspects of adversity". Biggam and Power (1998) also hypothesised that social and environmental variables, in combination with personality characteristics, might determine the influence of parental bonding factors on psychological well-being, i.e., whether an individual is predisposed to anxiety, depression or other psychosocial difficulties. Similarly, the presence of stressful life events (in particular those congruent with negative schemas) might be anticipated to have an interactive effect between early experiences and later psychopathology, in relation with a diathesis-stress model (Brown & Harris, 1978; Parker, 1993; Perris, 1988).

1.3 BELIEF SYSTEMS AND PSYCHOPATHOLOGY

1.3.1 Introduction

The possibility that cognitive schemata might in some manner mediate the relationship between dysfunctional parenting and development of psychopathology has been implicated in several studies (see section 1.2.5.6). Of course, the relevance of belief systems to psychological difficulties has long been propounded, with a
number of prominent schools of thought highlighting the importance of cognitive factors in theoretical accounts of the origins and maintenance of psychopathology. A brief outline of the main cognitive theories and the relation of cognitive factors to psychopathology will be presented in the following sections, in addition to descriptions of measures assessing beliefs, before the potential interrelation of such factors with parental bonding and psychopathology are further examined in the subsequent section (1.4).

1.3.2 Cognitive theories of psychopathology

1.3.2.1 Beck’s cognitive model

The role of cognitive factors in psychopathology became gradually more acknowledged towards the end of the 1960’s and early 1970’s, following awareness of the limitations of application of pure behavioural methods. Undoubtedly one of the most influential cognitive theories is that of Beck (1967, 1976), which initially emanated from his clinical experience with depressed individuals (see Figure 1.3). Beck hypothesised that negative thinking was central to the maintenance of emotional disorders, with affect experienced as a result of the manner in which meaningful events were interpreted and appraised. Correspondingly, different emotions could be evoked by the nature of such interpretations, with perceptions of loss or failure being central to depression and threat or danger more relevant to anxiety. Beck’s model proposed different levels of cognitive processing, with assumptions or schemata representing core cognitive structures, which tend to
Figure 1.3. Beck's (1967, 1976) cognitive model of depression
operate at an unconscious level, and negative automatic thoughts corresponding to
cognitive biases functioning at a more superficial level consistent with underlying
schemas. Schemas were believed to be stable representations of past experience – the
function of which was to enable organisation of incoming information in line with
previous experience and therefore ‘make sense’ of one’s interaction with the
surrounding environment. As such, schemas facilitate interpretation and prediction of
experiences and also govern and regulate behavioural responses. Dysfunctional
schemas were thought to be characterised by their extreme nature and rigidity, being
highly resistant to change, in contrast with more adaptive, flexible schemas. However, Beck (1976) felt that there was some continuity between ‘normal’ and
pathological cognitions, with dysfunctional schema merely exaggerations of more
adaptive beliefs. Beck speculated that the nature of underlying core beliefs was
associated with early experience, with childhood adversity predisposing some
individuals to develop negative views about themselves and the world around them.
However, he believed that such beliefs could remain latent unless activated by a
critical incident(s) that was in some way congruent with such beliefs, for example, a
loss event arousing schema associated with dependency (“I am unable to cope on my
own”). Such significant life events could therefore act as triggers for depressive
experience, stimulating negative automatic thoughts and cognitive distortions, which
in combination with affective, physiological, behavioural and motivational
symptoms were liable to result in a vicious circle, thereby perpetuating emotional
disturbance. Negative automatic thoughts were conceptualised as habitual thinking

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27 An intermediary level of cognitive processing often included in Beck’s model is conditional beliefs, which are believed to represent basic rules by which an individual conducts his/her daily life, e.g. “If someone does not like me, then I must be unlovable.”
errors, which would tend to occur in an involuntary manner in line with underlying assumptions in response to specific events. Examples of such cognitive errors would be discounting the positive in a situation or catastrophising – with such interpretations tending to be more evident in ambiguous or schema-congruent situations. Beck postulated that the cognitive distortions apparent in depression were configured as a *cognitive triad*, with negative biases occurring in cognitions about the self, the surrounding world and the future. Such negative beliefs about the future, combined with the perception that events were personally uncontrollable, were felt to predispose to helplessness (Seligman, 1974).

Beck’s theory has since been applied to other psychological problems, e.g., anxiety (Beck, Emery, & Greenberg, 1985; Clark, 1986), with a specific cognitive conceptualisation for each disorder, and has formed the basis for corresponding therapeutic intervention (Beck, Rush, Shaw, & Emery, 1979), usually in combination with a behavioural approach. Cognitive therapy aims to identify, challenge and correct cognitive distortions (with more accurate alternatives), usually operating - at least initially - at the level of negative automatic thoughts, adopting a collaborative approach with clients. Therapy follows a problem-oriented perspective, has a structured format and is time-limited, with collaborative empiricism encouraging a process of guided discovery. Behavioural elements further aim to rectify problematic behaviour patterns that are contributing to the maintenance of the condition. Cognitive therapy has been repeatedly demonstrated to be effective in the treatment of a variety of emotional disorders, in particular, anxiety (Butler, Fennell, Robson, & Gelder, 1991; Sokol, Beck, Greenberg, Berchick, & Wright, 1989) and depression (Dobson, 1989; Kovacs, Rush, Beck, & Hollon, 1981; Teasdale, Fennell, Hibbert, &
Amies, 1984). Comparisons with pharmacological interventions have been favourable (Beck, Hollon, Young, Bedrosian, & Budenz, 1985; Rush, Beck, Kovacs, & Hollon, 1977), with minimal apparent benefit from combination of the two (Blackburn, Bishop, Glen, Whalley, & Christie, 1981; Murphy, Simons, Wetzel, & Lustman, 1984). Likelihood of relapse also appears to be reduced in contrast with other treatments (Blackburn, Eunson, & Bishop, 1986; Evans, Hollon, DeRubeis, Piasecki, Grove et al., 1992; Hollon & Najavits, 1988; Simons, Murphy, Levine, & Wetzel, 1986), implying the prophylactic nature of cognitive-behavioural methods. However, efficacy of cognitive-behavioural intervention has not been so reliably demonstrated with more complex cases (Kuyken, Kurzer, DeRubeis, Beck, & Brown, 2001; McGinn, Young, & Sanderson, 1995; NelsonGray, Johnson, Foyle, Daniel, & Harmon, 1996; Ratto & Capitano, 1999), in particular individuals diagnosed with a personality disorder, which resulted in the elaboration of Beck’s original model by Young (1990), in order to provide direction for clinical intervention in complex cases.

1.3.2.2 Young’s schema theory

Following recognition of the importance of schemas in work with personality disorders (Beck, Freeman, & Associates, 1991), Young (1990) attempted to extend Beck’s original model by focusing more specifically on the concept of schemas, formalising a model explaining how such schemas develop and function and how they create long-term affective, interpersonal and behavioural difficulties. In addition, Young’s work also clarified means of assessing and identifying schema in
individual cases, with explication of both general schema characteristics and the nature of specific schemas. Young’s conceptualisation of schemas is largely similar to that of Beck (1967, 1976), with the view that schemas are cognitive structures through which an individual implicitly views the world and interprets incoming information. Young, however, proposed that such schemas were *unconditional* in nature, forming the core of an individual’s self-concept. He believed that schemas developed during childhood, in accordance with early experiences, and gradually evolved into enduring, pervasive and stable cognitive themes influencing perceptions and interpersonal relationships and thus, if maladaptive, rendering an individual vulnerable to psychopathology. Young (1990) described such dysfunctional core beliefs as *early maladaptive schemas* (EMSs) and delineated 16 different EMSs in accordance with clinical experience of complex cases: emotional deprivation, abandonment, mistrust/abuse, social alienation/isolation, defectiveness/shame, social undesirability, failure to achieve, functional dependence/incompetence, vulnerability to harm and illness, enmeshment/underdeveloped self, subjugation, self-sacrifice, emotional inhibition, unrelenting/unbalanced standards, entitlement/self-centeredness, insufficient self-control/self-discipline. A hierarchical model was proposed, with EMSs grouped into higher-order factors – Young (1998) proposed 18 EMSs\(^{28}\) divided into five higher-order factors at the most recent revision: disconnection & rejection (abandonment, emotional deprivation, abuse/mistrust, defectiveness/shame, social isolation/alienation), impaired autonomy & performance (dependence, vulnerability to harm, enmeshment, failure), overvigilance & inhibition (negativity/pessimism, emotional inhibition, unrelenting standards, punitiveness),

\(^{28}\) See Appendix 1.1 for a brief description of each of Young’s 18 early maladaptive schemas.
other-directedness (subjugation, self-sacrifice, approval-seeking) and impaired limits (entitlement, insufficient self-control). Young held the view that initially such EMSs are likely to have been adaptive and enabled an individual to cope with stressful or aversive situations, but have gradually become unhelpful, as he/she continues to behave in a manner consistent with the schema, having developed no other means of responding when confronted with different experiences. An example of this pattern might be if schemas of mistrust and emotional inhibition were to develop as a consequence of abusive early relationships. Initially these schemas would have helped to protect the individual against abuse (to some degree), but, over time, were likely to have become increasingly dysfunctional as the individual was faced with new situations incongruent with the early abuse. However, learned behavioural patterns and ways of interacting would have remained unchanged resulting in further interpersonal problems and difficulties and thus perpetuation of the underlying schema.

Young described three processes by which schemas operate to serve a self-perpetuating function: via schema maintenance, schema avoidance and schema compensation. Schema maintenance involves the continual process of unconditional acceptance and magnification of confirmatory evidence (i.e., information consistent with the schema), in conjunction with minimisation and negation of information inconsistent or incompatible with the underlying schema. In a similar manner, an individual is likely to adopt certain behaviours that will result in perpetuation of the schema, e.g., withdrawal from social interaction preserving a sense of being a ‘misfit’ in line with social alienation schema. To some extent, individuals are even likely to solicit information that is consistent with their core beliefs, as this will
enable security in the knowledge that their own appraisals are realistic. Such a pattern was demonstrated in a study by Giesler, Josephs, and Swann Jr. (1996), with depressed individuals choosing to receive negative feedback about themselves in preference to positive feedback in contrast to non-depressed controls. Attempts to prevent activation of schemas, via cognitive, affective and behavioural means, were described by Young as schema avoidance processes, which could range from the extreme of dissociation from upsetting experiences to more conscious acts of avoiding decisions or circumstances which might lead to the schema being challenged. Apparently contradictory behaviour, whereby an individual acts in a manner that appears to be in opposition to the schema was described as schema compensation – for example, an individual appearing defensive or hostile in social interactions as a means of counteracting schema relating to his/her inherent defectiveness or inferiority as a person. Young hypothesised that schemas operate largely outside of awareness, with their activation, via schema-congruent circumstances, resulting in intense experience of negative emotions and further dysfunctional thought patterns.

The nature of schema held is likely to relate to the type of psychological problem exhibited – for instance, one would anticipate themes of emotional deprivation, defectiveness/shame, abandonment, failure and dependency to be fundamental to depressive conditions, whereas themes of vulnerability to harm and unrelenting standards might be more prominent in anxiety-related difficulties. Such themes for depression correspond to Beck’s (1983) idea of sociotropic and autonomous personality types rendering an individual vulnerable to depression, dependent on associated adverse events. Losses, emotional deprivation or rejection were proposed
to be particularly meaningful stressors for sociotropic individuals, with close relationships and dependency highly valued. In contrast, experiences of failure or restrictions imposed by illness were believed to have the potential to trigger a depressive episode in autonomous individuals, who tended to prize achievement, success and independence. Similar distinctions were made from a psychoanalytic or developmental perspective by Blatt (see Blatt & Homann, 1992) in accordance with the type of experiences that enhanced vulnerability to depression in different individuals. It would seem feasible that schemas relating to abandonment, emotional deprivation and dependency would be more prominent in his classification of anaclitic/dependent depression, whereas schemas associated with failure or defectiveness/shame would be more meaningful for an individual with introjective/self-critical depression.

As identification and awareness of the nature of underlying schemas were believed to be highly relevant to facilitate work with complex cases, Young (1990) devised a questionnaire in accordance with his delineation of types of EMS, to enable standardised assessment of such schema. The initial form of the Schema Questionnaire (YSQ)29 consisted of 205 self-report items measuring the 16 originally proposed EMSs, with psychometric properties validated in both clinical and non-clinical populations (Lee, Taylor, & Dunn, 1999; Schmidt, Joiner, Young, & Telch, 1995). Responses on the YSQ were shown to correlate significantly with measures of psychological distress in a student sample, with a substantial proportion of the variance accounted for by EMSs in predicting psychological distress (Schmidt et al.,

29 See section 2.3.2 for further discussion of the Young Schema Questionnaire.
In addition, a different profile of EMSs was indicated in accordance with nature of symptomatology. Schema relating to dependency and defectiveness/shame appeared to be associated with depressive symptomatology (according to scoring on the BDI), whereas schema reflecting themes of vulnerability and incompetence/inferiority were evident in those experiencing anxiety symptomatology (as assessed by the anxiety subscale of the SCL-90-R\(^{30}\)). Lee et al. (1999) also observed some differences in response between individuals with Axis II (personality disorder) and Axis I (other clinical conditions) diagnoses, with scoring generally higher in the Axis II sample as might be predicted given the relevance of schema in assessment and treatment of personality disorders. In particular, schema relating to higher-order factors of disconnection (e.g., emotional deprivation, abandonment, defectiveness) and impaired autonomy (e.g., dependency, enmeshment, failure) were more evident in those with an Axis II diagnosis, which the authors concluded befitted their diagnoses, as attachment difficulties and problems in limit setting were more likely to occur in such a group.

To-date, Young's schema questionnaire has been the only means of assessing such early maladaptive schema. Other assessment tools exist for the evaluation of more accessible cognitions, the most commonly used of which has been the Dysfunctional Attitude Scale (DAS; Weissman & Beck, 1978). The DAS was designed in order to measure depressogenic cognitions, examining the extent to which an individual agrees with contingencies relating to self-worth and acceptance. Analysis of the factor structure of the DAS has revealed two dimensions (Cane, Olinger, Gotlib, &

\(^{30}\) Symptoms Checklist-90 Revised (SCL-90-R; Derogatis, 1983)
Kuiper, 1986) – approval by others and performance evaluation, which appear to relate to the higher-order factors of disconnection and impaired autonomy on the YSQ. However, given that the items on the DAS were derived with specific reference to depression and relate to a more explicit level of cognitions, use of the YSQ would seem to be optimal when examining underlying schemas in a general psychiatric population.

1.3.2.3 Comparison of schema therapy with other psychological models

In many respects, Young’s model of schema therapy is consistent with other influential theories of psychopathology and indeed Young acknowledges the influence of Beck’s cognitive model in the early development of schema therapy (Young, Klosko, & Weishaar, 2003).

Although Young initially believed that schemas were unconditional in nature - thus differing from Beck - at his most recent revision (Young et al., 2003), he proposes that some schema are in fact conditional and arise in response to more fundamental primary schemas which tend to develop at an earlier stage. For example, the development of a secondary schema of subjugation (and corresponding behaviour) as a mechanism to counter unconditional schema relating to one’s inherent defectiveness. However, in spite of some similarities, Young’s model differs in several fundamental respects from that of Beck – even from the revised form of cognitive therapy proposed to address treatment of personality disorders (Beck et al., 1991). Developmental aspects are accorded greater importance in schema therapy,
with respect to the origin of schemas due to specific childhood experiences, e.g., abuse, neglect. Greater emphasis is also placed on the therapeutic relationship within schema therapy, with it viewed as an active component of treatment. Indeed, one facet of the therapist’s role is to provide “limited reparenting”, by acting as a secure base for the client (Young et al., 2003). In addition, coping styles (relating to schema maintenance, avoidance and compensation) feature more centrally in Young’s model, in the manner in which they perpetuate schemas, having the potential to become lifelong repetitive behaviour patterns unless corrected. Schema therapy also differs in its use of experiential techniques, e.g., imagery, as a means of activating and modifying schemas in session.

Similarly, there are obvious links with attachment theory (Bowlby, 1969), given the import placed as to the origins of early maladaptive schemas within an individual’s early interpersonal environment. Bowlby’s conceptualisation of the formation of ‘internal working models’ to guide interpersonal relationships in accordance with a child’s experience with their primary attachment figure corresponds to the idea of development of dysfunctional schema as a consequence of a lack of fulfilment of fundamental emotional needs as a child.

Given the focus on development and the therapeutic relationship in schema therapy, there is also some overlap with psychodynamic theories. However, in schema therapy, the therapeutic relationship is more ‘corrective’ in focus, with the therapist playing a more active role. In addition, psychopathology is not viewed as the lack of resolution of unconscious conflicts and desires but rather as a consequence of core emotional needs not being addressed. As a result, maladaptive coping styles (as
opposed to defences) develop, which, although initially adaptive, serve to effectively ensure that these needs are never likely to be adequately fulfilled.

Cognitive-analytic theory (CAT: Ryle, 1991) also incorporates ideas similar to those in schema theory, with emphasis on the therapeutic relationship and need to address the prolonged use of rigid unhelpful 'procedures'. However, where gaining understanding and insight into these maladaptive patterns is viewed as one of the most important aspects of change in CAT, more weight is given to experiential processes in schema therapy.

Therefore, although Young's schema model encompasses aspects of several prominent psychological theories, it is distinct in its approach, given differences in emphasis.

1.3.3 Relation between schemas and psychopathology

As the concept and corresponding assessment of early maladaptive schemas has only been introduced relatively recently by Young, research in this area is still fairly emergent. The majority of studies have focused on the relevance of core beliefs to eating disorder pathology, following the realisation that cognitions relating to food, weight or shape do not sufficiently explain underlying psychopathology (Cooper, 1997). Leung, Waller, and Thomas (1999) examined the presence and extent of core beliefs in anorexic, bulimic, mixed anorexic/bulimic and control individuals using the original version of the YSQ. Although levels of dysfunctional schemas were
similar across the three eating disordered groups, with greater evidence of maladaptive schemas in these groups in comparison with controls, there did appear to be some distinction according to eating psychopathology with respect to the nature of schemas held. A negative association was found between frequency of bingeing in bulimic individuals and social undesirability beliefs and in bulimic anorexic individuals, a positive association was noted between frequency of vomiting and scoring on the failure to achieve schema. The authors accordingly emphasised the need to embody core beliefs in both the assessment and treatment of eating disorders in the future. Similar findings of greater levels of maladaptive schemas in bulimic individuals (consisting of bulimic anorexia, bulimia nervosa and binge eating disorder groups) in contrast with healthy controls were reported in a study by Waller, Ohanian, Meyer, and Osman (2000). In addition, membership of bulimic groups could be predicted via presence of schemas relating to defectiveness/shame, failure to achieve and insufficient self-control. Meyer, Leung, Feary, and Mann (2001) attempted to extend these studies by examining whether the associations demonstrated between bulimic pathology and core beliefs were mediated by borderline personality characteristics in a student sample, given the recognition of links between borderline personality disorder and bulimic tendencies. Core beliefs relating to defectiveness/shame were no longer predictive of bulimic symptomatology once borderline characteristics had been entered into the regression analysis, leading the authors to speculate that borderline symptoms might mediate the relationship between maladaptive schema and bulimic symptomatology.

31 Again, extent and nature of maladaptive schemas were assessed by the original version of the Young Schema Questionnaire.
1.4 LINKS BETWEEN PARENTAL BONDING, DYSFUNCTIONAL SCHEMAS AND CURRENT PSYCHOPATHOLOGY

1.4.1 Introduction

The likelihood that associations observed between parental bonding and psychopathology (see section 1.2.5) might be mediated by cognitive variables has been highlighted by a number of researchers from different theoretical backgrounds.

In accordance with his theory of attachment, Bowlby (1969) hypothesised the importance of internal working models with respect to personality formation and subsequent psychosocial functioning. He believed that early attachment experiences were internalised into working models representing an individual’s expectations and beliefs about both him/herself and other individuals, which would then govern further interpersonal relationships. These mental representations were thought to operate at a largely unconscious level and, along with associated behaviour patterns, were likely to become ingrained over time and develop into stable attributes of an individual’s character with respect to interpersonal functioning. The model therefore predicted that distorted mental representations were likely to form if difficulties were apparent in early attachment, which, in turn, were likely to render an individual vulnerable to psychopathology, due to difficulties in emotion regulation and experience of problematic relationships.

Similar assumptions underlie Beck’s (1967, 1976) cognitive model of emotional disorders, with the hypothesis that early experiences predispose an individual to a
certain cognitive style, which, in conjunction with congruent stressful life events can precipitate experience of psychopathology.

It is, of course, notable that both Bowlby and Beck’s theories refer to attachment experiences and early experiences respectively, as opposed to highlighting parental bonding per se as the relevant factor with respect to vulnerability to develop psychological difficulties. However, the relevance of parental bonding, within the context of more general early experiences, to later psychopathology has been demonstrated in a large number of studies (see sections 1.2.3 and 1.2.5) and consequently is the aspect of early experience focused on in the current study.

Perris (1988) argued the need for a coherent theoretical framework to underlie hypotheses regarding the influence of dysfunctional parental rearing styles on the development of psychopathology, suggesting that such a model would necessarily be multi-factorial, given the improbability of any single linear causal explanation. However, the model proposed by Perris also highlighted the development of maladaptive cognitive structures as “an important pathogenic link” between dysfunctional parenting and vulnerability to psychopathology.

1.4.2 Studies examining relationships between early parental experiences, current beliefs and psychopathology

Despite proposed theoretical links between early experiences, cognitive style and psychopathology, there have been very few studies examining potential causal
processes or mechanisms underlying these relationships. However, several studies have attempted to directly investigate the suggestion that dysfunctional beliefs mediate the relationship between parental bonding and psychopathology.

Whisman and Kwon (1992) received partial support for their hypothesis that a depressive cognitive style mediated the relationship between dysfunctional parental rearing styles and current experience of depression (as assessed by scores on the BDI) in a student sample. Associations found between low parental care and depressive symptomatology appeared to be mediated by both depressotypic attitudes and attributional style, measured by the Dysfunctional Attitude Scale (Weissman & Beck, 1978) and the Expanded Attributional Style Questionnaire (EASQ; Peterson & Villanova, 1988) respectively. However, although parental overprotection was positively associated with depressive symptomatology, this dimension of parental behaviour was not associated with cognitive style and thus did not support a cognitive mediation model. The authors emphasised the need to investigate whether the relationships found were specific to depression or might be generalisable across different forms of psychopathology.

An extension of this study was undertaken by Randolph and Dykeman (1998), again using a student population, to further examine possible causal pathways between dysfunctional parenting and vulnerability to depression. The authors proposed a three-stage causal model, in line with Beck’s (1967, 1976) theory of depression, with dysfunctional attitudes hypothesised as mediating the link between negative parenting experiences and depression-proneness. Results appeared consistent with such a model, although additional parenting dimensions of parental criticism and
perfectionism appeared to provide a better fit for the data than those of care or overprotection, prompting the authors to speculate the need to focus more on such parenting variables. Specificity in development of depressotypic cognitions (as assessed by the DAS) as a consequence of dysfunctional parenting – in contrast to cognitions associated with psychosis – was also indicated in the model. In addition, recall biases congruent with mood state did not appear to account for the relationships found, as associations remained significant after BDI scores were controlled.

A further three studies have specifically examined the possibility that early maladaptive schemas (assessed by the YSQ) might mediate the relationship between parental bonding and psychopathology. Leung, Thomas, and Waller (2000) investigated such relationships in individuals with eating disorder pathology, observing that both anorexic and bulimic groups differed from healthy control subjects by recalling lower levels of parental care, higher levels of maternal overprotection and greater extent of maladaptive schemas. However, regression analysis indicated that parental bonding only reliably predicted core beliefs in anorexic individuals, with low parental care highly predictive of the presence of maladaptive schemas, with links considerably weaker in bulimic and control groups.

Shah and Waller (2000) also utilised a clinical sample in their investigation of the potential mediating role of dysfunctional schemas in established links between reports of dysfunctional parenting and experience of depression. As predicted, the clinically depressed group expressed higher levels of depressive symptomatology according to scores on the BDI and reported lower levels of parental care and slightly
greater levels of parental overprotection than controls. In addition, greater presence of early maladaptive schemas was evident in the depressed sample, with the clinical group appearing to be best discriminated from the control group according to reports of schema relating to defectiveness/shame, self-sacrifice and insufficient self-control. Multiple regression analysis on data from the depressed group revealed that five schemas (dependence, emotional inhibition, failure, unrelenting standards and vulnerability to harm) mediated the relationship between maternal care and parental overprotection and depression. However, the mediating role of maladaptive schemas appeared more limited in the non-clinical group, with only vulnerability to harm partially mediating the relationship between paternal care and depressive symptomatology. Some support for these findings came from a recent study by Harris and Curtin (2002) that similarly examined the relationship between parental bonding, early maladaptive schemas and depressive symptomatology, although in a non-clinical student population. However, the nature of schema proposed to partially mediate the observed relationship between low parental care and overprotection and depressive symptomatology appeared to be somewhat different to those found by Shah and Waller, with defectiveness/shame, insufficient self-control, incompetence and vulnerability to harm appearing relevant in this instance.

1.5 SUMMARY OF MAIN RESEARCH FINDINGS

A greater awareness of factors that influence vulnerability to psychopathology and the interplay between them would benefit selection of appropriate treatment
interventions and might ultimately illuminate crucial health promotion and prevention strategies.

A substantial body of evidence seems to indicate an association between negative parental rearing behaviour and psychopathology of varying forms in the offspring (see section 1.2.5). The majority of such research has been conducted utilising retrospective self-report questionnaires to assess the nature of parental bonding experienced, focusing primarily on the two factor-analytically derived dimensions of care and overprotection/control. A large proportion of these studies have indicated that individuals experiencing a variety of forms of psychopathology report having experienced lower levels of parental care in their childhood than do comparison individuals. Some studies have also suggested that parental overprotection is significantly associated with psychopathology, although findings have been less consistent for this dimension of parental bonding. It remains unclear whether specific patterns of parental bonding have a differential effect on psychopathology.

The possibility that maladaptive beliefs might mediate associations observed between parental bonding and psychopathology has been investigated in a small number of studies and has received some support. However, although the relationship between parental bonding and dysfunctional beliefs has been investigated with regards depressive experience, there has been no comparative examination with respect to the experience of anxiety symptomatology.
1.6 AIMS AND HYPOTHESES

1.6.1 Aims

A large number of studies indicate the importance of a child’s early experiences with his/her parents (or parental figures) with respect to later development, in particular vulnerability to psychological difficulties. Similarly, the relation of dysfunctional beliefs to current psychopathology has been repeatedly documented and forms the basis of several influential theories of psychopathology. However, the possible mechanisms by which negative early experiences might impact on development of later psychological difficulties have yet to be clearly delineated. Some studies have suggested that dysfunctional schemas might be one of several possible mediating factors between early parenting experiences and development of psychological difficulties. However, to-date, such studies have only utilised specific clinical populations (e.g., depressives or individuals with eating disorders) or non-clinical populations rather than examining such issues across both a general clinical population of individuals with psychological difficulties and a non-clinical population. It therefore remains unclear whether the possible mediating role of dysfunctional schemas on early experience relates to the development of specific psychological difficulties or, rather, whether vulnerability is heightened to a range of psychological difficulties. In addition, relationships between parental bonding and dysfunctional beliefs have only been examined with regard to current level of depressive symptomatology, with no examination of other significant types of psychopathology. Given the prevalence of anxiety symptomatology in a variety of mental health problems, investigation of the relationship between perceptions of
parental bonding, dysfunctional schemas and anxiety symptomatology would also appear crucial.

Therefore the current study aimed to replicate and extend previous research by examining firstly whether dysfunctional schemas mediate the relationship between early parenting experiences (as identified by retrospective reports of parental bonding) and development of psychopathology (with reference to current experience of depressive and anxiety symptomatology) across both a general clinical and non-clinical sample. A second aim was to investigate whether specific patterns of rearing practices and the nature of schemas held related to experience of specific psychological symptoms, namely, depressive and anxiety symptomatology, or whether a more general influence on psychopathology was apparent.

1.6.2 Hypotheses

1.6.2.1 Hypothesis 1: Differences between clinical and control samples

Overall, it is anticipated that similar associations between variables will be evident across clinical and control participants. However, responses indicating greater levels of dysfunction are predicted in the clinical compared to control sample. As such, greater levels of anxiety and depressive symptomatology (as assessed by the BAI and BDI), higher levels of dysfunctional schemas (as measured by the YSQ) and

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32 Beck Anxiety Inventory (Beck, Epstein, Brown, & Steer, 1988)
more ‘negative’ parenting (as assessed by the PBI), i.e., lower levels of parental care and higher levels of parental overprotection, are anticipated in the clinical sample.

1.6.2.2 **Hypothesis 2: Association between parental bonding and symptomatology**

Across the whole sample, higher levels of anxiety and depressive symptomatology (as assessed by the BAI and BDI) will be associated with higher levels of ‘negative’ parenting (as assessed by the PBI), i.e., lower levels of parental care (a negative correlation) and higher levels of parental overprotection (a positive correlation).

It is also anticipated that parental care will have a greater association with current symptomatology than parental overprotection, particularly in relation to depressive symptomatology, given existing findings.

1.6.2.3 **Hypothesis 3: Association between dysfunctional schemas and symptomatology**

Across the whole sample, it is anticipated that higher levels of anxiety and depressive symptomatology (as assessed by the BAI and BDI) will be associated with greater presence of dysfunctional schemas (as assessed by the YSQ).

In addition, a degree of specificity is anticipated in any association between schemas and symptomatology. Schemas relating to threat and perfectionism (i.e., *vulnerability to harm, unrelenting standards*) are expected to be more apparent in individuals
experiencing greater levels of anxiety symptomatology. In contrast, schemas relating to loss, rejection or failure (i.e., defectiveness/shame, abandonment, emotional deprivation and failure) are expected to be more pronounced in individuals experiencing higher levels of depressive symptomatology. However, some schemas (i.e., dependence) are anticipated to be related to both anxiety and depressive symptomatology.

1.6.2.4 Hypothesis 4: Relationship between parental bonding, dysfunctional schemas and symptomatology

It is anticipated that dysfunctional schemas will mediate any relationship between perceived parental bonding experiences and current psychopathology (as estimated by level of anxiety and depressive symptomatology), i.e., any direct relationship between parental bonding and current psychopathology will no longer be significant once the effect of the relationship between dysfunctional schemas and current psychopathology has been controlled for. Given the lack of consistency of findings indicating specificity of parental bonding influences on nature of psychopathology, it was anticipated that both parental care and overprotection would be associated with both anxiety and depressive symptomatology and that in all instances, dysfunctional schemas would mediate these relationships.
2 METHOD

2.1 DESIGN

A cross-section correlational design was employed, as relationships between a number of variables across the whole sample were of primary interest. Comparison of control and clinical samples for one of the four hypotheses involved a between-participants analysis. All participants completed four questionnaires assessing perceptions of parental bonding, extent of dysfunctional schemas and current anxiety and depressive symptomatology.

2.2 PARTICIPANTS

2.2.1 Clinical sample

Any individual referred to the Tayside Area Clinical Psychology Department who was offered a new appointment between December 2002 and June 2003 was viable for inclusion in the study. In addition, individuals on the general adult waiting list who opted-into Anxiety Management groups being conducted by Tayside Area Clinical Psychology Department between December 2002 and June 2003 were also eligible for inclusion. However, individuals were excluded from the study if they met

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33 These individuals had been selected from general adult waiting lists, if anxiety difficulties appeared to be the primary reason for referral, and had been invited to attend the groups as part of a waiting list initiative.
any of the following diagnostic criteria\textsuperscript{34}: bipolar disorder, active psychotic disorder or a history of psychosis, active substance abuse, learning disability, or head injury.

As a general adult sample was required, any individual less than 16 years old or over 65 years old was excluded in accordance with Departmental criterion for an ‘adult’ population. In addition, if clinicians felt that inclusion in the study was inappropriate for any reason following first presentation (e.g., case relating to court representation, case requiring a large number of other assessment measures to be completed), they did not invite their client to participate.

151\textsuperscript{35} individuals attending psychology appointments (between December 2002 and June 2003) were invited to participate in the study by their individual clinician. Of these, 46 (30.46\%) completed and returned the four questionnaires, 20 of which were male and 26 female. The mean age of the sample was 40.50 years, with a range of 19-65 years. As a possible bias in the sample was anticipated according to which participants agreed to take part in the study and complete the forms, a comparison of demographic information between responders and non-responders was conducted (see section 3.2.1).

A breakdown of DSM\textsuperscript{36}-IV-R (APA, 2000) diagnoses recorded on SMR00\textsuperscript{37} forms by individual clinicians for the clinical participants is provided in Appendix 2.1.

\textsuperscript{34} Diagnoses were determined by the individual clinician following assessment and were in accordance with DSM-IV-R criteria (APA, 2000).

\textsuperscript{35} Although the initial aim had been to invite consecutive new attenders at adult out-patient appointments to participate, a variety of reasons (bar exclusion criteria) prevented all such individuals from being included; namely, forgetfulness of clinicians, inappropriateness for inclusion and other clinical demands. Consequently, this figure is approximate and probably represents a slight overestimate.

\textsuperscript{36} Diagnostic and statistical manual of mental disorders

\textsuperscript{37} Scottish Morbidity Returns forms are completed for each patient offered an appointment and record attendance at the initial appointment for the Scottish Executive.
However, as diagnostic information was not of central concern to the current study (given that focus was on investigation of extent of anxiety and depressive symptomatology, as opposed to diagnosis per se), no further analysis of diagnostic information has been undertaken.

Participation in the study was entirely voluntary and did not affect the patient’s treatment in any way. Pharmacological treatment was not controlled for and has not been documented as it was felt that medication was not of central concern to the main aims of the current study.

Ethical approval for the clinical sample was sought and obtained from the Tayside Committee on Medical Research Ethics. A minor change to the design was requested, with clinical participants to be given the pack of questionnaires by their clinician, rather than being sent out with the first appointment letter. Approval then followed this amendment. In addition, the principal researcher requested to change a couple of the self-report measures that had initially been intended for use in the study\(^\text{38}\) to more appropriate measures, which was similarly approved. All participants gave written informed consent to take part in the study.

\(^{38}\) The Hospital Anxiety and Depression Scale (HADS; Zigmond & Snaith, 1983) and the Clinical Outcomes in Routine Evaluation (CORE; Core System Group, 1998) had originally been proposed for use as dependent measures in the current study. However, after further consideration, it was decided that the HADS was not an adequate measure of anxiety and depressive symptomatology, in addition to the fact that it had been originally designed for assessment of patients in medical and surgical hospital wards and consequently was not appropriate for the populations sampled here. Therefore, it was decided that the Beck Anxiety Inventory (Beck, Epstein et al., 1988) and the Beck Depression Inventory (Beck et al., 1961) would be more suitable. Given that four questionnaires were already being used and that anxiety and depressive symptomatology were the factors of interest, it was also decided that inclusion of the CORE would be superfluous to the study’s requirements.
2.2.2 Control sample

In order to provide a representative comparison sample, control participants were recruited primarily\textsuperscript{39} from personnel of the local psychiatric hospital (i.e., administrative, domestic, nursing and clerical staff), with attempts made to equate clinical and control samples by age, gender and socio-economic status (according to postcode information\textsuperscript{40}). It was felt that such a sample would enable more effective comparisons than if an undergraduate control population (as frequently chosen for ease of data collection) had been selected.

48 individuals agreed to participate in the research, of which 18 were male and 30 female. The mean age of the sample was 39.85 years, with a range of 18-64 years, which was comparable with the clinical sample (see section 3.2). Participants satisfied criterion for inclusion in the control group if they were not currently receiving input from Tayside Area Clinical Psychology Department\textsuperscript{41}, as stated on the control information form. Individuals were not excluded from the sample if they scored over recognised ‘cut-off’ scores on the BAI and BDI, as extent of symptomatology (rather than categorisation into groups) was the variable of interest. However, if an individual wrote somewhere on the questionnaires that they were experiencing significant levels of psychological difficulty (as occurred in one instance), his/her response was excluded. Ethical approval was requested from and

\textsuperscript{39} To enhance the number of control participants, other individuals known to the principal researcher (who were not receiving psychological input) were also invited to participate.

\textsuperscript{40} Socio-economic status was estimated by relative deprivation category score (Carstairs & Morris, 1991) obtained from postcode information. Scoring ranged from 1-7, whereby 1 equated to the most affluent areas and 7 equated to the most deprived areas.

\textsuperscript{41} However, this statement did not guarantee that individuals were not receiving psychological input from elsewhere (see section 4.2.1 for discussion of such characteristics).
granted by Tayside Health Board Human Resources Department for the recruitment and involvement of hospital personnel for the control sample. As a courtesy, managers of the various departments (e.g., administrative, domestic, nursing) were also informed about the study and their approval was requested prior to invitation of their staff to participate. All participants indicated consent by ticking a box affirming that they had read the information form and another box confirming that they consented to take part in the study (see Appendix 2.7b).

2.3 MEASURES

Responses to four measures were collected in the current study. These were: a shortened form of the Parental Bonding Instrument (Pedersen, 1994), the short form of the Young Schema Questionnaire (Young, 1998), the Beck Anxiety Inventory (Beck, Epstein et al. 1988) and the Beck Depression Inventory Revised (BDI-II; Beck, Steer, & Brown, 1995).

2.3.1 The Parental Bonding Instrument – Short Form (PBI-SF)

The PBI was utilised to evaluate perceptions of early experiences with parents or parental figures. The PBI examines two main dimensions of parenting attitudes and

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42 It was decided, on discussion with Tayside Health Board Human Resources Directorate, that identifying information (i.e., participant’s name) would deter recruitment and was unnecessary to the purposes of the study.
behaviour – care and control/overprotection, providing both overall parental measures, in addition to assessment of these dimensions for both the maternal and paternal figure separately. The original version of this questionnaire was developed by Parker and colleagues in the late 1970’s and contained 50-items (Parker et al., 1979). Psychometric properties of the PBI have been rigorously investigated, with validity and reliability repeatedly established\textsuperscript{43} (see Parker, 1989). Pedersen (1994) subsequently developed a shortened version of the PBI, to enable easier administration, which was demonstrated to retain the original factor structure and have good validity. The short form is a 20-item self-report questionnaire\textsuperscript{44}, which is designed to retrospectively assess offspring’s (over 16 years) perceptions as to the degree of parental care and control/overprotection they received in the first 16 years of their life. 10-items relate to the maternal figure and 10 identical\textsuperscript{45} items relate to the paternal figure, with each scale containing 5-items relating to care and 5 concerning control/overprotection. Each item is rated on a 4-point scale as to the extent to which the individual agrees with a statement relating to his/her parent’s behaviour towards him/her, with options ranging from ‘\textit{strongly agree}’ to ‘\textit{strongly disagree}’. Scores on each item range from 0-3, resulting in total scores for each dimension ranging from 0-15, with higher scores indicating greater levels of care and of control/overprotection.

\textsuperscript{43} See sections 1.2.4.3.3 & 1.2.4.4
\textsuperscript{44} See Appendix 2.2
\textsuperscript{45} except the gender of the 3\textsuperscript{rd} person pronoun
Measurement of the presence and extent of maladaptive schemas was estimated using the short form of the Young Schema Questionnaire\(^{46}\) (Young, 1998). The original version (Young, 1990) contains 205 self-report items and was designed to assess 16 primary maladaptive schemas derived from clinical experience. Schmidt et al. (1995) evaluated the psychometric properties of the YSQ, demonstrating in large clinical and nonclinical samples that it had adequate internal consistency, reliability and validity. However, factor analysis of the original measure consistently supported only 15 of the 16 proposed schemas (Lee et al., 1999; Schmidt et al., 1995). The need for a more concise measure for clinical use led to the development of the 75-item YSQ-SF, which assesses the presence of these 15 early maladaptive schemas and has similarly been shown to have good internal consistency, reliability and validity (Waller, Meyer, & Ohanian, 2001; Welburn, Coristine, Dagg, Pontefract, & Jordan, 2002). In addition, Welburn et al. provided further support for the 15 subscales following factor analysis of the YSQ-SF in a clinical population. The 15 schemas examined are: emotional deprivation, abandonment, mistrust/abuse, social alienation, defectiveness/shame, failure, dependency, vulnerability to harm, enmeshment, subjugation, self-sacrifice, emotional inhibition, unrelenting standards, entitlement, insufficient self-control. Each subscale consists of 5 consecutive items, all of which are evaluated on a 6-point scale, whereby 1=completely untrue of me, 2=mostly untrue of me, 3=slightly more true than untrue, 4=moderately true of me, 5=mostly true of me and 6=describes me perfectly. Higher scores therefore indicate a

\(^{46}\) See Appendix 2.3
greater presence of maladaptive schema, with scores for each individual schema ranging from a possible 5-30 and overall scores ranging from 75-450.

2.3.3 Beck Anxiety Inventory (BAI)

An estimate of the extent of anxiety symptomatology was provided by the Beck Anxiety Inventory\(^{47}\) (BAI; Beck, Epstein et al., 1988). It is a 21-item self-report questionnaire that requires participants to rate the degree to which they have experienced certain symptoms over the past week, including today. The BAI was developed as a clinical measure of anxiety for use in psychiatric populations, with items selected to prevent confounding with symptoms of depression. It is brief, straightforward and easy to use and has been demonstrated to have a high degree of internal consistency, validity and reliability (Beck, Epstein et al., 1988; Fydrich, Dowdall, & Chambless, 1992). In addition, the BAI maintains better discriminant validity against measures of depression, in particular the BDI, in comparison with other existing self-report measures of anxiety (Dobson, 1985; Fydrich et al., 1992; Tanaka-Matsumi & Kameoka, 1986), such as the State-Trait Anxiety Inventory (STAI; Spielberger, Gorsuch, & Lushene, 1970), although a moderately high correlation remains between the measures. However, the BAI and BDI have been demonstrated to load independently using factor analysis with a general psychiatric sample (Hewitt & Norton, 1993). As a central aim of the current study required distinction between anxiety and depressive symptomatology as dependent variables,

\(^{47}\) See Appendix 2.4
the use of the BAI as a measure of anxiety appeared most supported. 13 items describe somatic symptoms (e.g., indigestion or discomfort in abdomen), 5 relate to cognitions (e.g., fear of losing control), and the remaining 3 have both a physiological and cognitive connotation (e.g., terrified / scared). Total scores can range from 0-63, with scoring graded across 4 response options from 0 (‘not at all’) to 3 (‘severely, I could barely stand it’) on each item. Scores of 7 or less are believed to be within ‘normal’ limits for anxiety, scores of 8-15 represent mild to moderate levels of anxiety, 16-25 moderate to severe and 26 or over as severe.

2.3.4 Beck Depression Inventory - Revised (BDI-II)

The revised version of the Beck Depression Inventory48 (BDI-II; Beck et al., 1995) was used to obtain an approximate measure of depressive symptomatology. The BDI-II contains 21 self-report items evaluating different aspects of depressive experience (for example, the extent to which an individual has felt sad, guilty, pessimistic) over the past two weeks. Respondents are required to choose between four possible response options for each item, indicating increasing symptom severity. The BDI is commonly used as an estimate of depressive state in both clinical and control populations (both in its original and revised forms). Revision of the BDI has focused on greater concordance between items and DSM-IV (APA, 1994) diagnostic criteria for major depressive disorders (Beck et al., 1995). Given the more recent emergence of this version, there have been fewer studies examining the use of the

48 See Appendix 2.5
BDI-II with respect to other variables, however, the measure has been demonstrated to be psychometrically robust with respect to both validity and reliability (Beck et al., 1995). The purpose for which the BDI was originally designed is that for which it is being used in the current study - to estimate the extent of depressive symptomatology experienced by an individual\textsuperscript{49}, i.e., whether the depression is of a minimal (scoring less than 14), mild (14-19), moderate (20-28) or severe (29-63) nature.

2.4 PROCEDURE

New patients attending adult out-patient appointments or Anxiety Management Groups at the Tayside Area Clinical Psychology Department between December 2002 and June 2003 were eligible for inclusion in the study, assuming their referral and presentation at first session concurred with inclusion and exclusion criteria. For individual out-patient appointments, individuals were sent the information form describing the study along with the details of their first appointment\textsuperscript{50}, which stated that questionnaires for the study and further information would be provided by their clinician at their first session. Individuals who attended their first appointment were then invited by their clinician to participate in the study and were given a pack containing the relevant forms and questionnaires. Attendees at Anxiety Management Groups were invited by the principal researcher and a clinician to participate in the

\textsuperscript{49} Initially the BDI was developed to examine intensity of depression in clinical (as opposed to nonclinical) populations, although research has since indicated its value in detection of depressive symptomatology in nonclinical populations (Bumberry, Oliver & McClure, 1978; Oliver & Simmons, 1984).

\textsuperscript{50} As this was not always possible, patients were given another copy of the information form in the questionnaire pack.
study at the end of the first group session. A brief verbal description of the study was
provided (outlining aims and requirements) and the voluntary nature of participation
emphasised. Out-patient and group attendees were each provided with a pack
containing the information and consent forms\(^{51}\), a sheet detailing demographic
information\(^{52}\) and the four questionnaires to complete and return in a stamped
addressed envelope to the principal researcher, if they chose to participate.
(Alternatively, participants were able to bring back their questionnaires sealed in the
stamped addressed envelope and hand them to their clinician at their next
appointment, who could then forward them to the principal researcher.) Forms were
not completed during the session – all clients were given the packs to take away and
were therefore able to decide whether they wished to participate in their own time.
The voluntary nature of participation in the study was highlighted and the fact that
participation or non-participation would not affect treatment in any way.

Control participants were approached individually, usually by the principal
researcher but some by their line manager, and given a brief verbal description of the
study. The voluntary nature of participation was emphasised and the fact that no
identifying information would be required. Those indicating they would like to
participate were given a pack containing the control information and consent forms\(^{53}\)
(including demographic information) and the four questionnaires in an addressed
envelope to take away with them and return completed, if they consented to take
part, by internal mail.

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\(^{51}\) See Appendix 2.6a & b
\(^{52}\) See Appendix 2.6c
\(^{53}\) See Appendix 2.7a & b
Two of the three studies (Leung et al., 2000; Shah & Waller, 2000) conducted to-date examining the role of dysfunctional schemas in the relationship between parental bonding and psychopathology (see section 1.4.2) investigated between-group differences. Sample sizes of 30 (anorexic), 27 (bulimic) and 23 (control) were apparent in the study by Leung and colleagues. In Shah and Waller's study, there were 60 depressed out-patients and 67 control participants. The third study (Harris & Curtin, 2002) investigated the mediating role of schemas across an undergraduate population of 194 participants. Unfortunately, neither effect sizes nor power were reported in any of these studies.

Therefore, sample size was estimated for both the between-participants analysis and also for the regression analysis across the whole sample anticipating a medium effect size. In accordance with Clark-Carter (1997), a sample size of between 45 and 50 participants per group would be required to achieve a medium effect size, with $\alpha = 0.05$ and power of 0.8 when examining whether groups differed in scores across the variables of interest (i.e., parental care, parental overprotection, dysfunctional schemas, BAI and BDI). To determine the potential mediating role of dysfunctional schemas in the relationship between parental bonding and current symptomatology using multiple regression at each node for the path analysis, with $\alpha = 0.05$ and for power to be achieved at 0.8, a total sample size of between 60 and 80 participants would be required. However, it is worth noting that Tabachnick and Fidell (1996) recommend accounting for the number of predictor variables, in addition to desired power, alpha level and effect size, when estimating appropriate sample size for
regression. As such, a sample size of around 74 would be necessary\textsuperscript{54}, assuming the three predicted factors of parental care, parental overprotection and dysfunctional schemas.

\textsuperscript{54} Sample size is calculated according to the equation: \( N \geq 50 + 8m \), where \( m \) is the number of predictors.
3 RESULTS

3.1 ANALYSIS OF DATA

Data were analysed using the Statistical Package for the Social Sciences (SPSS). An examination of minimum and maximum values, means and standard deviations for each of the variables was conducted to determine accuracy of data entry. Prior to statistical analysis, data were examined for normality and variables exhibiting significant skewness\textsuperscript{55} or kurtosis were transformed. Following transformation, remaining outliers were identified\textsuperscript{56} and removed from the data set prior to statistical analysis. In the instance of missing values in an individual’s data set, missing values were assigned a numerical value that excluded these data from statistical analyses, while still allowing them to be easily identified.

Testing of the respective hypotheses were undertaken using parametric tests given that assumptions\textsuperscript{57} of normality were met following the necessary transformations. Multivariate analysis of covariance (MANCOVA) was used to evaluate whether differences existed between the two samples, Pearson’s product-moment correlations were conducted to examine relationships between variables and path analysis (via multiple regression at each node in the model) was undertaken to determine whether dysfunctional schemas played a mediating role between variables. Significance was set at the <.05 level.

\textsuperscript{55} Significance of skewness and kurtosis values are determined by division of the value for skewness or kurtosis by the respective standard error value, with a ratio of 1.96 or greater indicating a significant departure from normality.
\textsuperscript{56} Outliers were determined by examination of stem-and-leaf plots and box plots.
\textsuperscript{57} Indeed, the robustness of parametric tests even when some assumptions are violated has been noted by Clark-Carter (1997).
3.2 DEMOGRAPHIC DATA

94 individuals in total participated in the study, with 48 individuals in the control sample (18 male, 30 female) and 46 (20 male, 26 female) in the clinical sample. Descriptive data will be presented initially for the whole sample (see Table 1), prior to further analyses for both control and clinical samples (see Tables 2a and 2b).

<table>
<thead>
<tr>
<th>WHOLE SAMPLE (N=94)</th>
<th></th>
<th>Age (N=94)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Mean</td>
</tr>
<tr>
<td></td>
<td></td>
<td>40.17</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Standard Deviation (StDev)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>11.99</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Median</td>
</tr>
<tr>
<td></td>
<td></td>
<td>39</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Mode</td>
</tr>
<tr>
<td></td>
<td></td>
<td>38</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Range</td>
</tr>
<tr>
<td></td>
<td></td>
<td>18-65</td>
</tr>
</tbody>
</table>

Table 1: Demographic data for the whole sample

<table>
<thead>
<tr>
<th>sex</th>
<th>Socio-economic status</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>Male</td>
<td>18 30</td>
</tr>
<tr>
<td></td>
<td>7 9 6 5 6 2 0</td>
</tr>
<tr>
<td>Female</td>
<td>20 26</td>
</tr>
<tr>
<td></td>
<td>3 18 4 8 6 4 0</td>
</tr>
</tbody>
</table>

Table 2a: Frequencies relating to sex and socio-economic status for control and clinical samples.
<table>
<thead>
<tr>
<th>Age</th>
<th>Control (N=48)</th>
<th>Clinical (N=46)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>39.85</td>
<td>40.50</td>
</tr>
<tr>
<td>StDev</td>
<td>12.69</td>
<td>11.34</td>
</tr>
<tr>
<td>Median</td>
<td>38</td>
<td>41.50</td>
</tr>
<tr>
<td>Mode</td>
<td>38</td>
<td>36</td>
</tr>
<tr>
<td>Range</td>
<td>18-64</td>
<td>19-65</td>
</tr>
</tbody>
</table>

Table 2b: Descriptive data for age for control and clinical samples.

Pearson's Chi-square analysis revealed that control and clinical samples did not differ according to sex (Chi-square $\chi^2_{(1)} = 0.35$, $p = 0.56$) or socio-economic status$^{58}$ (Chi-square $\chi^2_{(5)} = 5.60$, $p = 0.35$) of participant. Age was found to be normally distributed across both control and clinical samples, with an independent samples t-test (2-tailed equal variance$^{59}$) revealing no significant difference between control and clinical samples according to age ($t_{(92)} = 0.260$, $p = 0.80$).

3.2.1 Responders versus non-responders

It was only feasible to assess proportion of responders versus non-responders in the clinical sample, as demographic information was not available for non-responders in

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$^{58}$ Socio-economic status was assessed from relative deprivation category scores (Carstairs & Morris, 1991) according to postcode information. Postcode information was not available for 13 control and 3 clinical participants. Given the large number of missing data for this variable and the fact that socio-economic status had not been predicted as a significant factor, it was decided that this variable should not be included in further statistical analysis.
the control sample. 151 out-patients who attended new appointments at Tayside Area Clinical Psychology Department were invited to participate in the study, of which 46 returned completed questionnaires (i.e., 30.46%). This response-rate percentage is lower than that usually found in studies utilising questionnaire measures (Cookson, 1997; Jefferis, 1999; Stallard, 1995).

A comparison of demographic information was conducted on a subset of responders (N=25) and non-responders (N=25) to evaluate whether the groups significantly differed on these factors. Pearson’s chi-square indicated no difference in sex of responder versus non-responder (Chi-square \( \chi^2 \) = 2.05, \( p = 0.15 \)) An independent samples t-test (2-tailed unequal variance) also revealed that this subset of responders and non-responders did not significantly differ in age (t(47.58) = 1.49, \( p = 0.14 \)). Likewise, Pearson’s chi-square revealed no difference in socio-economic status (Chi-square \( \chi^2 \) = 5.01, \( p = 0.29 \)) between responders and non-responders. Descriptive data regarding sex, socio-economic status and age for this subset of responders and non-responders are presented in Table 3 (a & b respectively).

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59 Equal variance was assumed, given the normality of the distribution across both control and clinical samples.
60 This figure is approximate and probably represents a slight overestimate as clinicians may have forgotten to invite all appropriate new attendees to participate.
61 Response rates are usually quoted at around 50%.
62 Demographic information was available for individuals who had received new appointments with clinicians in the Perth and Kinross area. As 25 of the total clinical sample (N=46) were out-patients in the Perth and Kinross area, a pseudo-random sample of 25 non-responders was also examined from this area.
63 Equal variance was not assumed, as normality had not been examined for this subset of responders and non-responders.
<table>
<thead>
<tr>
<th></th>
<th>Sex</th>
<th>Socio-economic status</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Male</td>
<td>1 2 3 4 5 6 7</td>
</tr>
<tr>
<td>Non-responders</td>
<td></td>
<td>8 17 2 8 7 4 0 0</td>
</tr>
<tr>
<td>Responders</td>
<td></td>
<td>13 12 1 13 2 5 2 0 0</td>
</tr>
</tbody>
</table>

Table 3a: Frequencies relating to sex and socio-economic status for the subset of responders and non-responders.

<table>
<thead>
<tr>
<th>Age</th>
<th>Non-responders (N=25)</th>
<th>Responders (N=25)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mean</td>
<td>37.44</td>
<td>41.64</td>
</tr>
<tr>
<td>StDev</td>
<td>10.43</td>
<td>9.50</td>
</tr>
<tr>
<td>Median</td>
<td>37</td>
<td>41</td>
</tr>
<tr>
<td>Mode</td>
<td>38</td>
<td>36</td>
</tr>
<tr>
<td>Range</td>
<td>20-55</td>
<td>27-65</td>
</tr>
</tbody>
</table>

Table 3b: Descriptive data for age for the subset of responders and non-responders.
3.3 EXPLORATION OF DATA

Exploratory analysis of a correlation matrix containing all variables revealed that inter-correlations between the average scores for each of the 15 dysfunctional schemas assessed by the YSQ generally achieved either medium or large effect sizes (see Appendix 3.1a). In addition, an exploratory factor analysis similarly determined that all items primarily loaded onto one factor, which significantly contributed to the variance within this measure (see Appendix 3.1b). It was therefore decided that information on dysfunctional schemas would be best represented by one variable encompassing an overall average score across all items of the YSQ. As scoring for each item on the YSQ could range from 1 to 6, the value of the overall average score was between 1-6.

Likewise, correlations between maternal and paternal care and also maternal and paternal overprotection appeared significant (see Appendix 3.2). Therefore, it was decided that maternal and paternal scores should be combined for both care and overprotection resulting in overall parental measures of care and overprotection.

Such combination of maternal and paternal scores is commonly undertaken in studies

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64 As directional predictions had not been made for all the original variables (i.e., age), 2-tailed significance was used in this instance.
65 Given an awareness of the small sample size (N=94), the factor analysis was conducted purely as an exploratory measure to further confirm the need to reduce the 15 possible schema variables to one overall schema variable for further analysis. Tabachnick and Fidell (1996) recommend use of factor analysis only when N>=250.
66 5 participants did not score the paternal PBI measure. Mean sample (control or clinical) scores for the relevant factor replaced the missing values in these instances. (Analysis of data excluding these 5 participants resulted in similar overall results, therefore to maintain the largest possible sample size, the final analysis was conducted including these 5 participants utilising the above method to remove missing values.)
67 There also appeared to be minimal theoretical grounds for division of care and overprotection into maternal and paternal scores, given the lack of any consistent findings indicating a difference according to sex of parent. In addition, the statistical power of further analysis would benefit from fewer variables.
and was indeed intended as one of the possible means of data analysis in the original design of the questionnaire (Parker et al., 1979).

In accordance with the hypotheses and data from the exploratory correlation matrix, five main variables were highlighted for further statistical analysis: parental care68, parental overprotection69, dysfunctional schemas70, BAI and BDI. Cronbach’s alpha co-efficients were demonstrated to be high across all five measures71 indicating good internal reliability of each scale. Although a normal distribution was evident for parental care across control and clinical samples, the other four variables were shown to depart significantly from normality, all exhibiting positive skew and kurtosis (particularly within the control sample). Accordingly, these four variables were subject to transformation across both control and clinical samples. A logarithmic transformation was conducted for dysfunctional schemas and BAI and a square-root transformation for BDI, resulting in a normal distribution for these variables. However, two outliers remained for the transformed BAI variable, which were then removed from further analyses. As transformation of parental overprotection did not result in a normal distribution, it was decided that this variable should remain untransformed (given the minimal positive skew). However, one outlier was also apparent for this variable, which was removed from further analyses. (Removal of this outlier resulted in achievement of normality for parental overprotection.)

Therefore, following transformation and removal of the three remaining outliers, all

68 Parental care equated to the sum of scores for maternal and paternal care for each participant, with a possible range of 0-30.
69 Parental overprotection equated to the sum of scores for maternal and paternal overprotection for each participant, with a possible range of 0-30.
70 Dysfunctional schemas equated to each participant’s overall average score (i.e., the total score divided by the total number of items answered – usually 75) on the Young Schema Questionnaire, resulting in a possible range of 1-6.
statistical analyses were conducted on data from a total of 91 (45 control and 46 clinical) participants. Histograms of the distributions exhibited by the five main variables, once necessary transformations had been completed and outliers removed, can be seen in Appendix 3.4.

3.4 HYPOTHESES-RELATED DATA

3.4.1 Hypothesis 1

A between-participants multivariate ANCOVA\textsuperscript{72} was conducted to establish whether scores on all main variables (i.e., parental care, parental overprotection, dysfunctional schemas, BAI and BDI) significantly differed between control and clinical samples, with more dysfunctional scoring hypothesised in the clinical sample across all measures. Multivariate tests revealed that participant status (i.e., control or clinical) was a significant factor in the model ($F_{(5,82)} = 20.431, p < 0.001$). Sex was also entered as a between-participants fixed factor but was not a significant factor in the model ($F_{(5,82)} = 0.168, p = 0.974$) and did not exert a significant effect on any of the five variables. In addition, the interaction between clinical status and sex was not a significant factor in the model ($F_{(5,82)} = 0.984, p = 0.433$), nor for any of the five variables. Age was entered as a covariate in the analysis, although again it was not a

\textsuperscript{71} See Appendix 3.3

\textsuperscript{72} As there had been 5 data sets with missing paternal PBI values, significance for each between-participants test was calculated manually for the adjusted degrees of freedom (i.e., 81). However, this was not possible for the multivariate tests so data have been included for the unadjusted degrees of freedom (i.e., 86) calculated by SPSS. However, to enable comparison, MANCOVA tables calculated by SPSS for N91 and for N86 (excluding the 5 participants with missing data) are provided in Appendix 3.6. Significance does not change for any of the main results between the two analyses.
significant factor in the model \((F_{(5,82)} = 2.036, p = 0.082)\). However, when examining the effect of age on the individual variables, age did achieve significance for the variable parental care \((F_{(1,81)} = 4.516, p < 0.05; \text{ see Appendix 3.5})\). Since age had not been anticipated as a meaningful variable in the analysis and as there had only been a significant effect with parental care\(^73\) but no overall effect in the model, age was not included as a variable in further analyses.

Participant status achieved significance (and power) for all five variables: parental care \((F_{(1,81)} = 15.784, p < 0.001)\), parental overprotection \((F_{(1,81)} = 10.704, p < 0.005)\), dysfunctional schemas \((F_{(1,81)} = 41.099, p < 0.001)\), BAI \((F_{(1,81)} = 82.196, p < 0.001)\) and BDI \((F_{(1,81)} = 52.727, p < 0.001)\). Examination of mean scores\(^74\) and standard deviations for control and clinical samples on each of the five main variables (see Table 4) revealed that all scores differed significantly in the predicted direction (see Figures 3.1a-e). In line with hypothesis 1, all scores were significantly more dysfunctional in the clinical compared to the control sample (as represented by higher scores on all the variables except parental care, where lower scores indicate more problematic parenting).

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\(^73\) Given the lack of significance of age as an overall factor in the model and the number of comparisons, it is possible that the effect of age on parental care could be a result of family-wise error.

\(^74\) Scores are presented for the original sample of 94, given ease of interpretation prior to data transformation.
Figure 3.1a: Comparison of control and clinical samples mean parental care score, with standard error bars representing the variance.

Figure 3.1b: Comparison of control and clinical samples mean parental overprotection score, with standard error bars representing the variance.
Figure 3.1c: Comparison of control and clinical samples mean dysfunctional schemas (YSQ) score, with standard error bars representing the variance.
Figure 3.1d: Comparison of control and clinical samples mean BAI score, with standard error bars representing the variance.

Figure 3.1e: Comparison of control and clinical samples mean BDI score, with standard error bars representing the variance.
Table 4: Mean scores and standard deviations (stdev.) for each variable across both control and clinical samples.

MANCOVA also reported power and effect size for the five main variables of interest (see ‘corrected model’ in Appendix 3.6). All five variables were significant factors in the model: parental care ($F_{(4,81)} = 5.396, p < 0.001$), parental overprotection ($F_{(4,81)} = 2.994, p < 0.025$), dysfunctional schemas ($F_{(4,81)} = 10.903, p < 0.001$), BAI ($F_{(4,81)} = 21.793, p < 0.001$) and BDI ($F_{(4,81)} = 13.222, p < 0.001$). Power was achieved for all five variables, with the effect size ranging from small (with respect to parental care and parental overprotection variables) to medium (for dysfunctional schemas and BDI) and large (for BAI).

### 3.4.2 Hypothesis 2

Pearson’s product-moment correlations\(^{75}\) were conducted to establish whether

\(^{75}\) Analysis of results was in accordance with Cohen’s (1988) estimate of strength of correlation, with $r = \pm 0.10 - 0.29$ equating to small, $r = \pm 0.30 - 0.49$ equating to medium and $r = \pm 0.50 - 1.0$ representing a large correlation.
parental care was significantly negatively associated with BAI and BDI and also whether parental overprotection was significantly positively related to BAI and BDI across the whole sample, in accordance with predictions. All correlations\textsuperscript{76} were 1-tailed, given the directional predictions made according to the hypothesis.

A Pearson correlation revealed that parental care and BAI were significantly negatively correlated \((r = -0.293, p = 0.002)\). Likewise, a significant negative correlation was apparent between parental care and BDI \((r = -0.407, p < 0.001)\). Parental overprotection and BAI were shown to be significantly positively correlated \((r = 0.251, p = 0.008)\). Similarly, a significant positive association was evident between parental overprotection and BDI \((r = 0.399, p < 0.001)\). These associations are illustrated respectively in Figures 3.2a-d below. It was anticipated that the strength of the association between parental care and current symptomatology (in particular, depressive symptomatology) would be greater than for associations between parental overprotection and symptomatology. However, eye-ballng the data indicated little difference in strength of correlations between parental care or overprotection and symptomatology\textsuperscript{77}, although it is perhaps notable that both appear to have a slightly stronger association (medium effect size) with depressive symptomatology as opposed to anxiety symptomatology (small effect size).

\textsuperscript{76} See Appendix 3.7 for correlation matrix of the five main variables.

\textsuperscript{77} It is notable, however, that parental care is shown to predict both anxiety and depressive symptomatology in the path analysis, whereas parental overprotection only predicts depressive symptomatology (see hypothesis 4).
Figure 3.2a: Graph depicting the correlation between parental care and BAI

Figure 3.2b: Graph illustrating the correlation between parental care and BDI
Figure 3.2c: Graph portraying the correlation between parental overprotection and BAI

Figure 3.2d: Graph illustrating the correlation between parental overprotection and BDI
3.4.3 Hypothesis 3

Associations between dysfunctional schemas and both BAI and BDI were ascertained using Pearson’s product-moment correlations to test predictions that dysfunctional schemas would be positively correlated with both BAI and BDI across the whole sample. Again, correlations were 1-tailed, given the directional predictions made in the hypothesis.

Pearson’s correlations revealed that dysfunctional schemas were significantly positively correlated with both BAI \( (r = 0.632, p < 0.001) \) and BDI \( (r = 0.796, p < 0.001) \), in line with the hypothesis. These correlations are depicted in Figure 3.3a and b respectively.
Figure 3.3a: Graph depicting the correlation between dysfunctional schemas and BAI

\[ R^2 = 0.3990 \]
Rsq = 0.6336

dysfunctional schemas (log transform)

Figure 3.3b: Graph representing the correlation between dysfunctional schemas and BDI

Given the relatively small participants-to-variables ratio and the fact that the majority of inter-correlations between individual schemas achieved medium to large effect sizes (see section 3.3 and Appendix 3.1), further investigation of associations between specific schemas and BAI and / or BDI was deemed unfeasible within the limits of the current study and would not have provided meaningful data. Therefore, the hypotheses relating to possible associations between specific schemas and anxious and / or depressive symptomatology were not tested in the current study.
3.4.4 Hypothesis 4

A path analysis was conducted in order to determine the structure of relationships between the main variables in accordance with theoretical predictions, i.e., any relationship between parental bonding (care and overprotection) and symptomatology (BAI and BDI) would be mediated by dysfunctional schemas. Bramwell (1996) advocated the use of such path analysis, whereby multiple regression is conducted at each node of the model (or relevant factor), when attempting to elucidate the pattern of relationships between variables.

A path analysis was carried out on transformed data from the whole sample (i.e., N=91) as it was anticipated that associations between variables would be similar across the two samples, i.e., dysfunctional schemas would mediate the relationship between parental bonding and psychopathology regardless of group membership (see Figure 3.4).

The proposed model predicted that parental care and overprotection would correlate directly with anxiety and depressive symptomatology, although the effect size of these would be relatively small (see hypothesis 2). Likewise, dysfunctional schemas would be significantly associated with both anxiety and depressive symptomatology with a somewhat larger effect size (see hypothesis 3). Parental care and overprotection would also correlate with dysfunctional schemas (r = -0.452, p < 0.001; r = 0.452, p < 0.001, respectively). (Intercorrelations between all five variables are presented in Figure 3.5.) However, it was anticipated that any relationships discovered between parental care and overprotection and anxiety and /
or depressive symptomatology (c) would no longer remain significant once associations between dysfunctional schemas and symptomatology (b) had been held constant.\textsuperscript{78}

\[ a \rightarrow \text{dysfunctional schemas} \rightarrow b \rightarrow \text{symptomatology} \]

\[ \text{parental bonding} \rightarrow c \rightarrow \text{BAI} \]

\textbf{Figure 3.4: Basic pictorial representation of model with dysfunctional schemas as mediating variable between parental bonding and symptomatology}

Initially the path analysis was conducted for the dependent variable, BAI (see Figure 3.6), with multiple regression conducted at each node (in accordance with Bramwell, 1996). Beta weights indicated that relationships between parental care and dysfunctional schemas ($\beta = -0.306, p = 0.004$) and parental overprotection and dysfunctional schemas ($\beta = 0.306, p = 0.004$) were significant. BAI was significantly predicted by dysfunctional schemas ($\beta = 0.643, p = 0.000$). There was also a trend for BAI to be predicted by parental care ($\beta = -0.225, p = 0.054$). However, parental overprotection did not predict BAI ($\beta = 0.144, p = 0.213$). Once associations between dysfunctional schemas and BAI had been held constant, as anticipated, the trend between care and BAI lost significance ($\beta = -0.028, p = 0.780$). These results therefore indicated that dysfunctional schemas appeared to be a mediator in the near-significant relationship between parental care and BAI. However, no such effect was

\textsuperscript{78} For further description of mediator variables see Baron and Kenny (1986).
Figure 3.5: Model illustrating intercorrelations amongst the 5 main variables across the whole sample (N=91) using transformed data. Pearson Correlation $r$ values are shown. All relationships indicated are significant at $p < 0.01$ level (1-tailed)
Figure 3.6: Path analysis of predictors for BAI (anxiety symptomatology) across the whole sample (N=91) using transformed data. Beta values are indicated to give the weight for each node.

significant relationship = \[\text{solid line}\] trend / relationship approaching significance = \[\text{dashed line}\]

non-significant relationship = \[\text{dotted line}\]
apparent for parental overprotection, given that there was not even a direct association between parental overprotection and BAI (see Figure 3.7).

An identical path analysis was conducted for the other dependent variable, BDI (see Figure 3.8). Beta weights indicated that dysfunctional schemas was also a significant predictor for BDI (β = 0.759, p = 0.000) and likewise, both parental care (β = -0.281, p = 0.010) and overprotection (β = 0.265, p = 0.015) significantly predicted BDI. However, once the association between dysfunctional schemas and BDI had been controlled for, neither of the associations between parental care (β = -0.048, p = 0.531) or parental overprotection (β = 0.033, p = 0.672) and BDI remained significant. Therefore the predicted model that dysfunctional schemas would mediate the relationship between parental bonding (both parental care and parental overprotection) and depressive symptomatology (BDI) appeared to have been supported (see Figure 3.9).

3.5 SUMMARY OF RESULTS

Significant differences were found between control and clinical sample scores for all the five main variables in the predicted direction, with more dysfunctional scores apparent within the clinical compared to control sample, thus lending support for hypothesis 1.

As predicted, significant negative correlations were apparent between parental care and current symptomatology (both anxiety and depressive). Likewise, in accordance
Figure 3.7: Final path model of significant predictors for BAI (anxiety symptomatology) across the whole sample (N=91) using transformed data, whereby dysfunctional schemas act as a mediator between parental care and anxiety symptomatology. (NB. The direct relationship between parental care and BAI just approaches significance, p = 0.054)
Figure 3.8: Path analysis of predictors for BDI (depressive symptomatology) across the whole sample (N=91) using transformed data. Beta values are indicated to give the weight for each node. Significant relationship =
Dysfunctional schemas appear to mediate the relationship between parental bonding (care and overprotection) and depressive symptomatology. Figure 3.9: Final path model indicating significant predictors for BDI (depressive symptomatology) across the whole sample (N=91) using transformed data. Dysfunctional schemas appear to mediate the relationship between dysfunctional schemas and depressive symptomatology.
with hypothesis 2, positive relationships between parental overprotection and current symptomatology (anxiety and depressive) were indicated by significant Pearson’s product-moment correlations. Correlations achieved small effect size with respect to anxiety symptomatology for both parental care and parental overprotection. However, a medium effect size was apparent for correlations with depressive symptomatology. Hypothesis 2 therefore has been supported. However, parental care does not appear to exhibit stronger correlations with current symptomatology (particularly depressive symptomatology) than parental overprotection, suggesting that this part of hypothesis 2 is not supported.

Significant positive associations (both indicative of a large effect size) were apparent between dysfunctional schemas and both anxiety and depressive symptomatology, as predicted. Therefore, hypothesis 3 has also been supported. However, it was not possible to test the second part of hypothesis 3, with respect to the differential specificity of particular schemas to be associated with either anxiety or depressive symptomatology, given the high degree of inter-correlation amongst all 15 schemas assessed.

With respect to anxiety symptomatology, the predicted model was partially supported, with dysfunctional schemas only mediating the relationship between parental care and anxiety symptomatology (although this relationship represented a trend as significance was at $p = 0.054$). The relationship between parental overprotection and anxiety symptomatology did not achieve significance, therefore in this instance, dysfunctional schemas could not be said to play a mediating role.
However, with respect to depressive symptomatology, the predicted model was fully supported, with dysfunctional schemas mediating both the relationship between parental care and depressive symptomatology and also that between parental overprotection and depressive symptomatology.

Hypothesis 4 has therefore received partial support – being fully supported for depressive symptomatology, but only partially supported for anxiety symptomatology.

Further discussion of the results follows in the next section.
4 DISCUSSION

4.1 SUMMARY OF EARLIER RESEARCH AND CURRENT FINDINGS

The significance of an individual’s childhood relationships with parental figures on vulnerability to later psychological difficulties has followed in part from investigation of the importance of early attachment on subsequent development. Research appears to indicate the importance of two particular facets of parental behaviour – care and control/overprotection – when examining links with psychopathology. Uncertainty remains as to whether such characteristics of parental bonding differentially enhance vulnerability to specific forms of psychopathology. However, a substantial proportion of empirical research indicates that reports indicating perceptions of low levels of parental care and (to a lesser degree) high levels of parental overprotection across childhood are evident in individuals experiencing a variety of forms of psychological difficulty.

Likewise, links between maladaptive beliefs and emotional disturbance have long been implicated in a number of prominent psychological theories. More recently, the need to identify and address underlying core cognitions or ‘schemas’ about the self and others has been highlighted in therapeutic work with more complex cases, given the prevalence of such dysfunctional schemas in these patient groups. However, empirical research in this area is still in its infancy.

The possibility that associations observed between the nature of an individual’s parental bonding experience and vulnerability to psychopathology could be mediated by an individual’s core beliefs or schemas has been proposed in several recent
studies. (However, similar conceptualisations as to potential predisposing factors in the development of psychological difficulties have been evident in several eminent theoretical models of psychopathology, although emphasis has perhaps been accorded to different factors.) Partial support for such a hypothesis has been evident in the few studies conducted to-date – however, these have focused primarily on specific patient groups or undergraduate populations and have usually examined relationships with respect to diagnosis or depressive symptomatology, with little examination of other manifestations of psychological difficulty.

Therefore, the aim of the current study was to examine whether dysfunctional schemas play a mediating role between nature of parental bonding (as assessed by retrospective self-report) and current level of anxiety or depressive symptomatology. A general adult out-patient population was sampled (therefore including a variety of diagnoses), in addition to a comparative non-clinical sample, as extent of symptomatology was the variable of interest, as opposed to diagnostic category. As such, it was assumed that associations between variables (i.e., parental bonding, schemas and symptomatology) would be similar, irrespective of whether an individual achieved a clinical status or not – therefore, analyses were conducted across the whole sample. However, a between-participants analysis was also undertaken to ascertain that evidence of greater dysfunction would be apparent in the clinical sample, with respect to nature of parental bonding, presence of maladaptive schemas and degree of current anxiety and depressive symptomatology.

Findings indicated that the clinical and non-clinical groups did indeed differ in the predicted direction, as to the extent of dysfunction indicated by scores for parental
bonding, schemas and current symptomatology. Partial support was received for the mediational model with respect to anxiety symptomatology, with dysfunctional schemas only acting as a mediator for the association (which was a trend) between parental care and extent of anxiety symptoms. However, the model appeared to be fully supported for depressive symptomatology, with relationships between both parental care and also parental overprotection and depressive symptomatology being mediated by dysfunctional schemas.

4.2 FURTHER DISCUSSION AND EXPLORATION OF RESEARCH FINDINGS

4.2.1 Hypothesis 1

Research to-date has indicated that dysfunctional parental bonding styles are more apparent in individuals identified as experiencing some form of psychological difficulty (e.g., depression, anxiety, eating disorders) than in comparative control samples (Arrindell et al., 1983; Calam et al., 1990; Gerlsma et al., 1993; Parker, 1979b; Parker et al., 1987; Perris et al., 1986; Plantes et al., 1988; Sato et al., 1997—see section 1.2.5). In particular, patterns of parental bonding characterised by retrospective perceptions of low levels of parental care or emotional warmth consistently appear to be implicated in a range of clinical groups (Arrindell et al., 1983; Palmer et al., 1988; Parker, 1983b; Perris et al., 1986; Stravynski et al., 1989). Similarly, perceptions of greater degree of parental overprotection or control appear more evident in clinical than in control populations (Parker, 1979b, 1983b; Parker et
al., 1987; Plantes et al., 1988; Silove et al., 1991), although it is perhaps notable that increased presence of parental overprotection has not been observed in all studies involving clinical groups (Lewinsohn & Rosenbaum, 1987; Palmer et al., 1988; Stravynski et al., 1989). In the current study, a between-participants MANCOVA revealed that scores of control and clinical samples differed significantly with respect to perceived levels of parental care and parental overprotection experienced throughout childhood. Mean scores indicated that lower levels of parental care and higher levels of parental overprotection were evident in the clinical compared to the control sample, as predicted, in conjunction with previous research.

Likewise, the presence of dysfunctional core beliefs or schemas has been demonstrated to be greater in clinical\textsuperscript{79} compared to control populations (Leung et al., 1999; Waller et al., 2000), although to-date there has been a paucity of empirical research in this area. In parallel with these findings, the clinical sample in the current study exhibited a significantly greater extent of dysfunctional schemas than did those in the control sample.

Given the prevalence of depression and anxiety as presenting difficulties (or comorbid conditions) at secondary mental health services (Boyd, 1986; Lader & Marks, 1971; Sartorius, 2001; Sartorius, Üstün, Lecrubier, & Wittchen, 1996) and the positive association between anxiety and depressive symptomatology (assessed by the BAI and BDI respectively) and clinical diagnoses of anxiety and depression (Beck, Epstein et al., 1988; Beck, Steer, & Garbin, 1988; Oliver & Simmons, 1984),

\textsuperscript{79} All identified studies examining maladaptive schemas using versions of the Young Schema Questionnaire have investigated presence of such dysfunctional core beliefs in individuals with eating disorders.
one would anticipate that greater levels of such symptomatology would be reported within a clinical than a non-clinical population, as has been demonstrated previously (Beck, Epstein et al., 1988; Byerly & Carlson, 1982). Indeed, in the current study, individuals in the clinical sample reported significantly higher levels of both anxiety and depressive symptomatology than did those in the non-clinical sample.

The significant findings in hypothesis 1 of greater levels of dysfunction in the clinical compared to the control sample, as evidenced by higher scores for reports of parental overprotection, dysfunctional schemas, current anxiety and depressive symptomatology and lower scores for parental care, also corroborate with findings by Leung et al. (2000) and Shah and Waller (2000). Higher levels of dysfunctional schemas and parental overprotection, in conjunction with lower levels of parental care were evident in anorexic and bulimic samples in contrast with controls in Leung et al.'s study. Similarly, Shah and Waller demonstrated greater levels of parental overprotection, maladaptive schemas and depressive symptomatology, in addition to reports of lower levels of parental care, in their clinically depressed sample than in their control sample.

There has been little consistent evidence to-date of differential findings according to an individual's gender with respect to response on the PBI (Parker, 1989, 1990; Rodgers, 1996a; although cf. Cubis et al., 1989). No such comparisons appear to have been undertaken with respect to responses on the YSQ or when investigating psychometric properties of the BAI. Some studies have indicated a higher degree of self-reported depressive symptomatology in females on the BDI compared to males (Knight, 1984), although others studies report no differences according to sex (Plumb
& Holland, 1977). Indeed, sex of participant was not a main factor in the current analysis, nor was there a sex by clinical status interaction, again suggesting that participant’s gender does not appear to be a particularly meaningful factor in the investigation of such relationships. However, although an appropriate sample size was achieved (for both the between-groups analysis and the regression analysis across the whole sample), it is notable that a small effect size was evident and power was not achieved for analyses concerning sex of participant (see Appendix 3.6), suggesting that further analysis with a larger sample might be required before the relevance of sex as a factor can be definitively ruled out. Nevertheless, given the small effect size here, it is unlikely that any findings would be particularly meaningful.

Age has also not been indicated in the literature as a significant factor with respect to retrospective perceptions of parental bonding (Arrindell et al., 1989; Parker et al., 1979; Parker, 1990). To-date the possible effect of age on presence of dysfunctional schemas does not appear to have been investigated, neither is there any clear evidence with respect to age effects on the BAI. BDI scores have been shown to be higher in adolescents than adults (Teri, 1982). However, in their review of the BDI, Beck, Steer et al. (1988) comment that although some relationships between demographic variables and the BDI are significant, they are not necessarily meaningful. Likewise, in the current study, age did not appear to be a significant factor in the model, when entered as a covariate in the between-groups analysis. However, a significant negative correlation between age and parental care was evident, with levels of perceived parental care decreasing with increasing age across the whole sample. This finding seems somewhat at odds with that of Parker et al.
(1987), who noted *more* favourable ratings of parental bonding (in general) with increasing age. However, it is consistent with findings by Calam et al. (1990) in their study comparing women with eating disorders and comparison women on the PBI. Perceptions of parents as both less caring and also more overprotective were apparent in older\(^{80}\) females across both groups. It is possible that the current finding might relate to a cohort effect, with changes in values relating to parenting style possibly occurring across the past century, e.g., with respect to variation in the extent to which overt expression of affection or encouragement has been adopted as an integral part of childrearing. Alternatively, perceptions of parenting might become increasingly negative\(^{81}\) as the time period from childhood increases, thus potentially biasing reports. However, such an explanation appears unlikely given evidence that recency of parenting experience does not appear to influence perceptions of parental bonding, due to indications of the stability of reports on the PBI over time (Gotlib et al., 1988; Mackinnon et al., 1989) in addition to lack of findings with respect to age. The significance of the effect for age purely in relation to parental care (as opposed to with other variables of interest) could instead reflect presence of familywise error, given the number of factors tested and therefore may require further analysis in a larger sample to enable more conclusive evidence as to whether or not age is a factor of potential importance with respect to parental bonding. However, the small effect size apparent for age in the current study (see Appendix 3.6) indicates that even if a significant result was to be found with a larger sample, it may well not be a *meaningful* finding (as suggested by Beck, Steer et al., 1988 - see above).

\(^{80}\) It should, however, be noted that a relatively young group was sampled, with the mean age of both samples less than 26 years old.

\(^{81}\) However, it is perhaps equally logical to predict the appearance of more *positive* memories of parenting experiences with increasing time.
Previous research has implied that the nature of parental bonding does not appear to differ according to an individual's socio-economic status (Mackinnon et al., 1989; Parker, 1983a; Rodgers, 1996a). Relationship of socio-economic status to the YSQ, BAI or BDI has not been reported in the respective reviews of their psychometric properties. Given the lack of any predictions regarding socio-economic status (in line with findings for parental bonding) and the fact that a substantial proportion of scores was missing (in particular from the control sample) with respect to estimated socio-economic status, analysis of the potential influence of socio-economic status was not conducted in the current study.

Although significant differences were apparent across the five variables of interest between clinical and control samples, such findings cannot be automatically related to presence (or lack) of clinical status or diagnosis. Such an interpretation would rest on the assumption that none of the control participants met clinical diagnostic criteria – a supposition that was not assessed or evaluated in recruitment of the control sample. Although participants were required to confirm that they had read the information form, which stated that individuals were viable for inclusion in the study if they were not currently receiving input from the Tayside Area Clinical Psychology Department, agreement with such a statement does not necessarily imply that they were not experiencing psychological difficulties at a clinical level. Indeed, it was possible either that individuals could be seeking treatment for psychological problems from another source (e.g., via counselling, GP), or that, even in the absence of seeking any form of intervention, there was evidence of psychological difficulties.

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82 Relative deprivation category scores (Carstairs & Morris, 1991) were assigned in accordance with postcode information.
sufficient to warrant clinical diagnosis. Indeed, control participants were not excluded from the sample according to their scores on the BAI and BDI, even if these exceeded cut-off points commonly utilised to indicate above 'normal' or 'minimal' levels of symptomatology. This policy had been adopted in line with the central focus of the study being the examination of relationships between variables across the whole sample, rather than differences in relationships between variables according to diagnostic category. However, as a consequence of such characteristics of the control sample, it is only really feasible to attribute differences between the two samples to help-seeking behaviour, as opposed to clinical status *per se*. Therefore, apparently greater levels of 'dysfunction', with respect to patterns of parental bonding, presence of maladaptive schemas and current levels of anxiety and depressive symptomatology, in the clinical in comparison with the control sample can only really be ascribed to differences in help-seeking behaviour between the two samples rather than the presence of clinically diagnosable psychopathology. However, it should be noted that findings have been comparable – i.e., indicating an association between dysfunctional parenting style and psychopathology - in several studies utilising general community samples, in an attempt to overcome the possibility of contribution of a higher-order help-seeking variable (Duggan et al., 1998; Parker et al., 1995).
4.2.2 Hypothesis 2

A large proportion of studies conducted to-date investigating associations between parental bonding and psychopathology have focused primarily on evaluation of between-group differences according to clinical disorder, with fewer having concentrated on examination of the relationship between parental bonding experiences and current levels of symptomatology across different populations (i.e., via correlational analyses). However, as part of their meta-analysis of studies examining associations between parental rearing styles and anxiety and depression, Gerlsma et al. (1990) compared several studies that had used correlational designs to relate scoring on measures of anxiety and depression to perceptions of parental bonding. They concluded that a parental style characterised by low levels of parental care and high levels of parental overprotection was apparent for scoring on both state and trait measures of depression in non-clinical populations, which was similar to findings of the meta-analysis of studies examining parental bonding in clinically depressed individuals. Similar findings were evident with respect to correlational studies using trait\(^3\) measures of anxiety, although associations with paternal overprotection on the PBI were not significant overall. A style of 'affectionless control' (i.e., low care, high control) had been identified in the analysis of studies investigating between-group comparisons in individuals with clinical anxiety. However, it should be noted that effect sizes were slightly smaller in the correlational studies (for both anxiety and depression) than those investigating between-group analyses. In addition, none of the studies analysed utilised the measures of anxiety.

\(^3\) No studies were reported that had used measures of state anxiety.
and depressive symptomatology employed in the current study.

Correlations between scores on the PBI and anxiety and depressive symptomatology as assessed by the HADS were also evident in a series of studies by Power and colleagues in a sample of incarcerated young men (Biggam & Power, 1998; Chambers et al., 2000, 2001). Psychological distress⁸⁴ was shown to be associated with reports of lower levels of parental care in offenders (Chambers et al., 2000; 2001), although no correlations with parental overprotection were evident. However, parental overprotection was shown to be positively correlated with psychological distress (again, including anxiety and depression scores) in the study by Biggam and Power (1998), although, with respect to care, only maternal care exhibited a negative association with depression.

Mancini et al. (2000) also examined associations between anxiety and depressive symptomatology (as assessed by the STAI⁸⁵ and BDI respectively) and parental bonding as part of their study, which attempted to investigate links between obsessivity and early parenting experiences in a non-clinical sample. Again, they found significant negative correlations between parental care and both anxiety and depression scores and, in turn, significant positive correlations between parental overprotection and anxiety and depression.

In another study, Rodgers (1996a) demonstrated a graded relationship between dysfunctional early parenting experiences (as assessed by the PBI) and extent of

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⁸⁴ Measures of hopelessness and self-esteem were also included in this composite.

⁸⁵ Both state and trait scales of the STAI (Form Y: Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983) were used to assess anxiety.
psychiatric symptomatology (with items specifically chosen that related to anxiety and depression) across a general population. Correlations exhibited were small but significant, as has been found in other studies of the general population (Mackinnon et al., 1989). However, Rodgers argued that, although small, these associations were still meaningful, given that effect sizes were at least equivalent (if not greater) to those found for other childhood risk factors for adult affective symptoms, e.g., parental divorce (Rodgers, 1994).

In accordance with such findings, it was anticipated that significant correlations would be evident between reports of parental bonding and current anxiety and depressive symptomatology. Such correlations were indeed evident, with effect sizes ranging from medium in associations with depressive symptomatology to small with respect to anxiety symptomatology. Differences in strength of correlation had not been specifically anticipated between anxiety and depressive symptomatology, given comparable findings to-date (Gerlsma et al., 1990). However, considerably fewer studies have examined anxiety and thus less definitive conclusions have been reached with respect to association with early parenting experience than appear to have been for depression. Stronger relationships had been expected between parental care and symptomatology than with parental overprotection, given that parental care appears to have been the more consistently defined of the two parenting dimensions (Cubis et al., 1989; Murphy et al., 1997) and more reliably identified with respect to mental health difficulties (Enns et al., 2002; Gerlsma et al., 1990; Gotlib et al., 1988). However, the effect sizes appeared to be strikingly similar between both parenting dimensions and respective symptomatology in the current study, perhaps lending support to the argument (Parker, 1979a; Rapee, 1997) that investigation of the
interaction between both of these characteristics of parenting is more meaningful than independent analysis (for further discussion see section 4.3.2).

Obviously a critique of such findings would be that these associations, indicating more dysfunctional parental bonding in line with greater extent of current anxiety or depressive symptomatology, could purely represent the operation of a mood bias, resulting in a negative influence across responses. However, although this explanation concurs with findings of a mood-congruent memory bias with respect to depressive experience86 (Perrig & Perrig, 1988; Williams & Scott, 1988), it is unsubstantiated with respect to the body of research strongly indicating that retrospective reports of parental bonding, as assessed by the PBI, remain stable across fluctuations in mood state (Gerlsma et al., 1994; Gotlib et al., 1988; Plantes et al., 1988). Further replication might benefit from reassessment on all the measures at a second time point, in order to determine more definitively whether scores on the PBI (or YSQ) have been influenced by any changes in levels of reported symptomatology over this time period. Another possibility to help determine whether a recall bias is occurring would be to obtain sibling ratings (i.e., asking the participant’s sibling to rate the participant on the PBI) in addition to self-report ratings on the PBI (Rodgers, 1996a). If these reports exhibited a high degree of concordance, one would attribute less likelihood to the presence of a recall bias (due to depressed mood etc.) influencing the participant’s ratings. However, if sibling and self-reports were discrepant, the probability of the existence of a recall bias would be

86 However, such a mood-congruent bias has not been demonstrated in all studies (Natale & Hantas, 1982), with the suggestion that such selective recall might be more likely if information is self-referent and elicited via free-recall procedures (see section 1.2.4.4).
4.2.3 Hypothesis 3

Both theory and clinical experience implicate an association between maladaptive beliefs and psychopathology. However, to-date there have been few empirical studies examining such relationships with respect to more fundamental or unconditional beliefs, such as the early maladaptive schemas proposed by Young (1990). Several studies investigating psychometric properties of the YSQ (both the long and short versions) have however noted associations between dysfunctional schemas and psychopathology. In one such study, Stopa, Thorne, Waters, and Preston (2001) reported modest correlations between individual schemas and psychopathology scores on the SCL-90-R (Derogatis, 1983) in a psychiatric out-patient population. Correlations of a medium effect size were apparent between YSQ scores and bulimic symptomatology in a sample of clinically diagnosed bulimics (Waller et al., 2001). Significant positive correlations were also apparent between most of the 15 schemas identified in the YSQ short-form and estimates of depressive and anxiety symptomatology in a psychiatric population in a study by Welburn et al. (2002). Likewise, in an undergraduate sample, positive correlations achieving a large effect size were evident between the total score on the YSQ and assessments of depressive symptomatology87, with positive correlations of medium strength between schemas

87 Both the BDI and the depression subscale of the SCL-90-R were used to estimate depression.
and anxiety symptomatology (Schmidt et al., 1995).

Findings in the current study of significant positive associations of a large effect size between YSQ total scores and both anxiety and depressive symptomatology are therefore unsurprising, given the comparison with results from the studies outlined above. These findings also fit well with prominent theories of emotional disorders, which emphasise the significance of maladaptive cognitions in the maintenance of psychopathology.

Of course, as with responding on the PBI, there is a possibility that responses on the YSQ could be influenced by mood state, with a greater level of maladaptive schemas endorsed with increasing negative affectivity. However, such an explanation would appear more unlikely given the unconditional, core nature of the phenomenon under investigation. In order to respond appropriately to each item, individuals need to essentially weigh up aspects of their core identity according to the rigid beliefs they hold about themselves. Given that the essence of such maladaptive schemas is their inflexible, enduring and primarily unconscious nature, one would predict that such beliefs would be stable over both time and fluctuations in mood. Nonetheless, until further empirical research has been conducted examining the reliability of scores across changes in mood state, this possibility cannot be ruled out.

Unfortunately, given the relatively small sample size and consequent low ratio of participants to variables, in addition to correlations of a modest to large strength

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88 Anxiety symptomatology was evaluated using the anxiety subscale of the SCL-90-R.
89 One possible means of investigating mood state influences would be examining whether changes in YSQ (and PBI) scores are apparent following successful pharmacological treatment of a clinical episode – e.g., antidepressant therapy providing symptom relief in major depressive disorder.
between the 15 different schemas assessed by the 75-item version of the YSQ, more detailed analysis of relationships between specific schemas and the nature of symptomatology was not possible. Therefore, evaluation of the second part of hypothesis 3 was beyond the scope of the current study, with respect to predictions that schemas congruent with themes identified as symbolic in individuals experiencing depression (i.e., loss, failure) would exhibit more significant associations with depressive symptomatology, whereas those representing beliefs compatible with issues known to be salient in anxiety (i.e., perception of threat) were anticipated to be more strongly correlated with levels of anxiety symptomatology. Such predictions seem to be reasonably well-founded, given both their theoretical basis, in accordance with cognitive models of depression (Beck, 1967) and anxiety (Beck, Emery, & Greenberg, 1985), and some support from corresponding clinical findings (Harris & Curtin, 2002; Shah & Waller, 2000; Welburn et al., 2002).

Support for analysis in the current study to be conducted on a composite schema variable from the exploratory factor analysis (indicating one meaningful factor, see Appendix 3.1b) is perhaps surprising in light of research supporting the 15 subscale factor structure of the YSQ short-form (Welburn et al., 2002). However, the current finding of one main factor is unsurprising given the underlying assumptions of exploratory factor analysis as an empirical data reduction technique. As such, use of confirmatory factor analysis might be more appropriate, in order to investigate specific a priori predictions as to factor structure – although again, a considerably

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90 Limitations of conducting such an analysis with a relatively small sample size have already been highlighted in the results.

91 All items exhibited a high loading on this principal factor, although entitlement also loaded highly on a second factor.
greater sample size (i.e., $N = 250$) would be required for valid analysis. Interestingly, inter-correlations reported by Stopa et al. (2001) between the 15 schemas in the YSQ short-form do not appear to be of the same magnitude as in the current study, with fewer significant correlations reported and generally smaller effect sizes in those which do achieve significance. Such differences might perhaps be explained by the different nature of the populations examined, with a purely psychiatric out-patient sample ($N=67$) in Stopa and colleagues' study as opposed to a mixture of both an out-patient and control sample here. It would therefore appear that further investigation of the psychometric properties and factor structure of the short-form YSQ is required before it can be utilised reliably in research. Moreover, it is perhaps possible that the nature of individual schemas is more meaningful in clinical terms, with respect to enhancing therapists' understanding of the particular idiosyncrasies of each individual and his/her difficulties, than in broader research predictions.

4.2.4 Hypothesis 4

Based on the assumptions of various psychological theories and empirical research indicating the potentially crucial roles of both early parenting experiences and beliefs with respect to vulnerability to psychopathology, the current study aimed to investigate the possible structure between such variables$^{92}$. The central question was therefore whether dysfunctional schemas would act as a possible mediator in

$^{92}$ Path analysis was employed to determine the structure of the variables, given Tabachnick and Fidell's (1996) recommendations against the use of structural equation modelling with a sample size of less than 250.
observed relationships between parental bonding and anxiety and depressive symptomatology.

Although such a model is often hinted at in theory, very few experimental studies have actually investigated such phenomenon. However, some preliminary research appears to support such a mediational model for dysfunctional beliefs (Harris & Curtin, 2002; Randolph & Dykeman, 1998; Shah & Waller, 2000; Whisman & Kwon, 1992). Findings from the current data set also seemed to support such a model with respect to depressive symptomatology, with the effects of both parental care and overprotection on depressive symptomatology shown to be mediated by dysfunctional schemas. However, findings were not so clear-cut for anxiety symptomatology. Although the association between parental care and anxiety symptomatology appeared to be mediated by dysfunctional schemas, the original relationship between care and anxiety was only a trend and thus even though such a mediational pattern seemed to be in operation between these variables, such conclusions can only be at best tentative before further research is conducted. Interestingly, no such model was apparent for parental overprotection, given that this parenting dimension was not even shown to predict anxiety symptomatology outwith the potential effect of dysfunctional schemas.

Of the few studies conducted in this area, the majority have investigated which model appears to best fit the data with regards depression. Some support for the current findings for depression comes from Whisman and Kwon’s (1992) study of a student sample, which demonstrated a mediating role for depressive cognitive style in the relationship between parental care and current experience of depression.
However, it is worth noting that although depressive state was similarly assessed using the BDI, cognitive style was evaluated with respect to more conditional beliefs and attitudes. Given that core schema are believed to underlie such contingency-based assumptions and consequently will relate to congruent themes, it seems logical that such relationships would also be evident, as in the current study, for dysfunctional schemas. However, unlike the current study, Whisman and Kwon did *not* find a corresponding pattern for parental overprotection, given that it did not appear to be associated with cognitive style, even though a direct relationship was apparent with depressive symptomatology. The authors suggested that other mediating variables might be associated with parental overprotection and predispose for depression, for example, deficient social skills - presumably due to lack of opportunity to learn such skills because of over intrusive and controlling parenting. Similar to Whisman and Kwon, analysis of control group data by Shah and Waller (2000) indicated the relevance of parental care, but not parental overprotection in their mediational model. However, like the current study, both parental care and overprotection contributed to depressive symptomatology via the effect of dysfunctional schemas in the clinical sample. Such findings perhaps highlight the need to examine non-clinical and clinical populations independently, given that potentially different factors could be relevant in each case. In turn, such examination according to diagnosed clinical status implies the possibility of *qualitative* rather than merely quantitative differences (as predicted in the current study) across ‘normal’ and psychopathological experience (see section 4.3.1).

Randolph and Dykeman (1998) emphasised the need to examine other facets of parenting behaviour, given findings from a path analysis looking at possible causal
pathways to depression proneness in their study of an undergraduate sample. They found that parenting dimensions relating to criticalness and perfectionistic expectations appeared to have a more significant role than care or overprotection in their mediational model.

One of the caveats of studies focusing on depression has been the need to determine whether such relationships are also evident in other forms of psychopathology. This study aimed to address such issues by also examining this model with respect to anxiety symptomatology, which does not appear to have been examined explicitly to-date. Although dysfunctional schemas appeared to mediate the association between care and anxiety symptomatology, no such relationship was evident for overprotection - indeed there was not even an association between overprotection and anxiety prior to inclusion of schemas in the model. In some respects, this finding for overprotection is perhaps surprising, given associations assumed between excessive parental control and anxiety (Parker, 1983a). However, it perhaps more coherent with findings by Arrindell et al. (1983) and Parker (1979b) where only reports of care seemed to differ in agoraphobic versus comparison samples (whereas individuals with other phobias appeared to exhibit both lower care and higher overprotection). More research is obviously required in order to elucidate more clearly the factors implicated in anxiety proneness, given the relative lack of empirical findings in this area to-date.

In general, therefore, results from the current study indicate that the parenting dimension of care is more predictive of psychopathology than overprotection, in line with previous research indicating the primacy of care relative to overprotection (e.g.,
Duggan et al., 1989; Mackinnon et al., 1993; Narita et al., 2000; Rodgers, 1996a). However, notably, current data seem to indicate that such a relationship is actually indirect, with parental care only influencing vulnerability to psychological difficulties via its influence on the nature of core beliefs that an individual forms about him/herself. However, as in other findings examining parental bonding in depression (Parker, 1983b; Plantes et al., 1988), overprotection also appears to be a relevant factor with respect to depressive symptomatology, but again indirectly, as a consequence of its influence on dysfunctional schemas. It is also worth noting that a negative association between care and overprotection was apparent, as has been reported in other studies (e.g., Cubis et al., 1989; Parker et al., 1979) and indeed was of virtually the same effect size as that found by Rodgers et al. (1996a). As such, this correlation might influence the predicted model, although it is interesting to note that differences between the effects of the parenting dimensions are apparent dependent on symptomatology experienced, lending support to the independent analyses of each factor.

Scoring on the BAI and BDI exhibited a large positive correlation, in line with previous findings (Beck, Epstein et al., 1988; Fydrich et al., 1992) and the well-documented extent of comorbidity between anxiety and depressive disorders (Sartorius et al., 1996). However, in spite of the interrelation between extent of anxiety and depressive symptomatology, current results also seem to implicate some degree of specificity in the models between these different forms of psychopathology. As such, it would appear that parenting dimensions representing low care and high overprotection (i.e., ‘affectionless control’) are relevant via their influence on dysfunctional schemas in depressive symptomatology, whereas only a
putative relationship is apparent between low care and anxiety symptomatology, again mediated by the effect of dysfunctional schemas. Although such conclusions can only be tentative at best (given limitations in the methodology – see section 4.3), they seem to support the hypothesis that different patterns of anomalous parenting relate to specific types of psychopathology, with respect to the relevance of overprotective parenting. However, given the findings for care, there also appears to be some endorsement of the suggestion that parental care might reflect a non-specific or 'pathoplastic' risk factor for psychopathology (Enns et al., 2002; Parker et al., 1995).

4.3 CRITIQUE OF METHODOLOGY

4.3.1 Design

In addition to limitations in the design that have already been mentioned and addressed, such as the potential influence of retrospective recall biases or mood biases on responses to the self-report questionnaires, a number of other shortcomings will be discussed below.

Perhaps of greatest importance is the caveat regarding assumptions of causality between the factors outlined in the model, given the design employed. As the current study was cross-sectional, involving assessment of all variables of interest at the same time point (including retrospective report of earlier experiences, e.g., nature of parental bonding in childhood), inferences regarding causality cannot be made. Therefore although the path analysis supported a particular causal sequence of
events, in particular with respect to current experience of depressive symptomatology, the actual temporal chain of events still needs to be demonstrated. As such, a prospective longitudinal design, which was beyond the scope of the current study, would be required to determine the nature of underlying causal processes in the development of psychological difficulties. Of course, in a related manner, one cannot assume that the variables assessed are necessarily the only relevant factors in such a causal chain. Indeed, Baron and Kenny (1986) note that the presence of multiple mediating factors is likely if the beta weight of the association between the independent and dependent variable has not been completely reduced to zero, after controlling for the effect of the proposed mediating factor, as was the case here. With respect to mediating factors, other likely variables might be self-esteem (Chambers et al., 2000; Parker, 1993) or social competency – with respect to both interpersonal skills and social support (Rodgers, 1996b; Whisman & Kwon, 1992). However, it is also possible that schemas might function in a sense as a higher-order variable, with respect to the development of appropriate social skills (and in turn the quality of interpersonal relationships) and self-esteem, given the fact that the maladaptive behavioural responses developed are likely to repeatedly elicit negative life experiences. Therefore, it is possible that these other factors might add little to the variance in the outlined model. In addition, parental bonding is only one facet of an individual’s early experiences and thus other potential aspects of childhood adversity, such as abuse, might be relevant to examine with respect to vulnerability to psychopathology within such a model (Rodgers, 1996a).

In addition, the possibility that a third variable might account for the relationships demonstrated here cannot be excluded, i.e., that a genetic determinant underlies
exhibited symptomatology. However, Parker’s (1982) findings that similar associations were evident between parental bonding and psychopathology in a sample of adoptees lends some evidence against such an explanation.

Assessment of the potential influence of moderator variables, such as the quality of current interpersonal relationships or the extent and influence of negative life events, was also lacking in the current study and should be addressed in future replications.

Another potential flaw in the design relates to the fact that differences between the two groups could not be attributed to clinical status, but rather help-seeking behaviour, given the inclusion criteria for the control sample and lack of objective assessment of mental health. Therefore, in order for a more conclusive statement regarding the differences across variables (i.e., the finding of greater extent of ‘dysfunctional’ scores in the clinical sample), more stringent criteria would need to be adhered to with respect to participant recruitment. Unfortunately, greater assessment of the control sample was beyond the scope of the current study. However, given that levels of symptomatology were the dependent variables (rather than clinical diagnosis), with assessment of extent of symptoms having previously been shown to be meaningful using such measures across both clinical and non-clinical samples (Beck, Epstein et al., 1988; Beck et al., 1995; Oliver & Simmons, 1984), and the fact that the main thrust of the current study involved examination of the predicted model across the whole sample, the characteristics of the two samples (with respect to clinical diagnoses) were not so significant.
The fact that many therapeutic approaches intervene at a symptom level, advocates the relevance of use of such a dimensional method, rather than ‘pigeon-holeing’ or classifying individuals according to perhaps somewhat arbitrary diagnostic criteria. (For further discussion of the credibility of diagnostic categories, see Bozarth & Schneider, 2000.) Such a perspective perhaps also enables a greater understanding of an individual’s experience, rather than being constrained by diagnosis, and the corresponding nature of potential contributing factors.

However, an assumption underlying the design employed in the current study, whereby both non-clinical and clinical populations were combined for investigation of the main research question, relates to the continuous nature of psychopathological experience. Amalgamation of both populations was undertaken, given that extent of symptomatology was the variable of interest (rather than clinical status), with the supposition that associations and underlying processes would be similar in both samples even though degree of ‘dysfunction’ (across all variables) would vary on a continuum dependent in part on group membership. There remains considerable controversy as to whether certain clinical conditions, e.g., depression, are continuous in nature, or whether low levels of psychological distress are in fact quite distinct from clinical experience. Coyne (1994) warned against use of findings from non-clinical samples to determine clinical models in depression. However, use of a correlational design, as opposed to division into groups according to relatively arbitrary cut-off values, has been endorsed by Vredenburg, Flett, and Krames (1993),

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93 As Boyle (1999) notes, diagnostic categories were initially developed in medicine with respect to assessment and treatment of distinct physiological difficulties which surely calls into question the attempt to classify human behaviour and experience in a similar manner with psychiatric diagnoses.
therefore allowing examination of factors influencing symptomatology, as opposed to major clinical syndromes. Nonetheless, this methodology still rests on the assumption that identifiable changes will differ in *degree* along a continuum rather than in *kind*. However, following a review of available evidence with respect to depression, Flett, Vredenburg, and Krames (1997) conclude that "...*most relevant literature...is consistent with the continuity perspective*", although they also recommend that a two-factor model accounting for both continuities and discontinuities should be applied to future research.

Nonetheless, as current findings have primarily supported the predicted model that dysfunctional schemas mediate the relationship between parental bonding and symptomatology across a general sample, it would be of interest to further examine whether any differences become apparent in the model when it is analysed independently for control and clinical populations. In addition to ensuring that the control sample did not include any individuals who met diagnostic criteria for mental health disorders, a greater sample size for each group (between 60-80 individuals) than was available in the current study would be necessary to achieve power (see section 2.5) for such an analysis.

In the current study, there was neither categorisation nor analysis of the clinical sample according to diagnostic groupings, with the sample composed of a variety of diagnoses (although primarily consisting of anxiety and depressive disorders - see Appendix 2.1). Analysis according to diagnostic category was not an aim at the outset, given the preferential focus on degree of symptomatology irrespective of diagnosis, although would have been infeasible anyway due to the small size of the
clinical sample and consequently the even smaller number of individuals within each broad diagnostic grouping. However, in order to more definitively address the question of specificity or generality of the model to different psychological conditions, it would be necessary to conduct analysis on different subsamples, categorised according to diagnosis (for example, comparisons between individuals with a primary diagnosis of depressive disorder, anxiety disorder, personality disorder and eating disorder). However, a project of a considerably larger scale would be required to address such issues.

4.3.2 Measures

A general limitation relating to the use of four self-report questionnaires is the possibility of inflation of the relationships between the variables due to common method variance (see Randolph & Dykeman, 1998).

Another difficulty with use of a self-report questionnaire methodology involves the probability that there is likely to be a degree of bias or selectivity in the sample of individuals who have consented to participate, given the percentage who have declined, rather than being a truly representative sample of the population (Barclay, Todd, Finlay, Grande, & Wyatt, 2002). Although demographic comparison of a subset of individuals who had responded versus those who had not within the clinical population sampled did not reveal any significant differences between the two groups (see section 3.2.1), analysis of other potentially more meaningful variables (e.g., diagnostic criteria, marital status, length of time since onset of presenting problem) was not conducted. In addition, such a comparison had only been possible within the
clinical population sampled, as it was not possible to monitor consent in the control sample. Indeed, one might predict that differences between responders and non-responders might be greater within the control sample, given the potentially decreased motivation to subjectively report symptomatology or information relating to aspects of oneself in contrast to the clinical sample. Given that individuals in the clinical sample might be anticipating the discussion of such issues in therapy, and perhaps even possess a greater awareness of such matters due to their current experience (which presumably precipitated their referral), they are likely to have more invested in participation than controls. Therefore, across the whole sample, but perhaps in particular across the control population sampled, individuals who responded to the questionnaires might have differed from non-responders with respect to attributes such as helpfulness, willingness to report on personal experience, or interest in psychology (see Gerlsma et al., 1994). However, given the voluntary nature of involvement in research, it would be extremely difficult to attempt to control for such factors.

A number of studies indicate the validity and reliability of the PBI (see sections 1.2.4.3.3 and 1.2.4.4). However, given that it involves retrospective recall, use of multiple methods of assessment, such as sibling reports or a semi-structured interview, would enhance the extent to which data regarding aspects of parental behaviour can be regarded as accurate94 (Brewin et al., 1993).

94 Of course, it should be noted that use of sibling report rests on the assumption that such accounts are valid or accurate representations of actual experience.
In addition, there is a possibility that *current* relationships with parents could bias an individual’s account of his/her earlier experiences with parents (Rodgers, 1996b). It might therefore be interesting in adult populations to firstly ascertain whether parents are still alive and, if so - assuming that contact is ongoing - also assess an individual’s *concurrent* experiences or perceptions of how his/her parents currently treat him/her.

The original two-factor model of the PBI was used in the current study, which has been called into question by some researchers who believe that three factors (with control divided into two separate factors) better represent the structure of the measure (Cubis et al., 1989; Gómez-Beneyto et al., 1993; Murphy et al., 1997). However, such factor-analytic studies have only been reported with respect to the original version of the PBI, not the shortened version utilised here. In addition, the fact that different predictions were apparent between the two parenting dimensions in the current results suggests the validity of using the original model with this version.

In addition, given the correlational design of the study, it was not possible to examine the interaction of parental bonding dimensions with respect to the four parenting styles defined by Parker et al. (1979), as this would have necessitated division of participants according to cut-off values on the two dimensions. However, it might be of interest to investigate the interaction between care and overprotection in future studies, given the apparent pertinence of the ‘affectionless control’ quadrant

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95 However, clearly further use of the shortened version of the PBI would benefit from factor-analytic studies of this version. Such analysis was beyond the scope of the current study given the small sample size.
in various forms of psychopathology (Parker, 1983b; Plantes et al., 1988). However, despite the significant negative correlation between the two dimensions in the current study, differences were also apparent as to their relative importance in the path analyses, with care more consistently associated with psychopathology.

Data entry also revealed some patterns of responding that seemed indicative of inconsistencies on the PBI. In particular, discrepancies were occasionally observed between individuals’ responses on similar items, such as “he/she liked me to make my own decisions” and “he/she let me decide things for myself”. However, as these potentially discrepant responses were rare, they are unlikely to have significantly influenced the data and indeed may have reflected a genuine response on both occasions, given the subtle nuances between the two statements.

In order to elucidate more information with respect to whether specific schemas might differentially mediate such relationships according to nature of symptomatology or psychological disorder, it might be necessary to utilise the original version of the YSQ (i.e., 205-items). The factor structure of the long version appears to have been more rigorously validated (Lee et al., 1999; Schmidt et al., 1995), with analysis reliably supporting 15 of the 16 schemas and revealing several higher-order factors in line with those proposed by Young (1994). Nonetheless, Stopa et al. (2001) and Waller et al. (2001) indicated that long and short forms

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96 Three were apparent in the Schmidt et al. (1995) study and four in Lee et al.’s (1999) study.
exhibited similar psychometric properties across patient samples and therefore the short-form was selected for the current study given its greater ease of use$^97$.

A few participants did directly comment on the negative wording and connotation across all items on the YSQ, which might have resulted in a response-set bias, given increased predictability as to direction of response. However, equally, adaptation of the YSQ to include positively worded items might also lead to confusion with regard to ratings.

The YSQ was utilised in the current study as the aim had been to assess core schemas. Using the current methodology, its use was also preferable over the DAS (which assesses conditional beliefs) due to the similarity in item content between the DAS and the BDI, which might result in artificially inflated relationships (Whisman & Kwon, 1992).

A further criticism of the YSQ regards the fact that if such schemas operate at a primarily unconscious level, how accurate can a self-report measure based on conscious awareness be (Welburn et al., 2002)? Nonetheless, the YSQ has been demonstrated to be a clinically useful tool with respect to both the assessment of complex cases and also as a guide for appropriate intervention (Young, 1994). Indeed, it would seem highly unlikely that schemas operate entirely outwith awareness, given that they are proposed to direct everyday behaviour and interactions.

$^97$ Indeed, Roszkowski and Bean (1990) found lower response rates for long versus short versions of the same questionnaire.
There has been some criticism of the BAI’s ability to accurately measure general anxiety symptomatology, given that most items relate primarily to panic symptomatology (Cox, Cohen, Direnfield, & Swinson, 1996), with less emphasis given to cognitive aspects such as those more apparent in generalised anxiety disorder or obsessive-compulsive disorder. Indeed, Cox et al. suggested that the BAI is at best confounded with, or even largely measures, panic symptoms, which has significant implications for both assessment of anxiety and evaluation of treatment outcome. However, Steer and Beck (1996) challenged Cox et al.’s findings, stating that many of the items supposedly merely representing symptoms of panic on the BAI were also criteria used in diagnosis of GAD. Supporting the use of the BAI, is the fact that it appears to be relatively good at discriminating anxiety from depression98 (Beck, Epstein et al., 1988) in comparison with other anxiety measures (such as the STAI). Nonetheless, as was evident here, scoring on the BAI and BDI is significantly correlated, which might reflect the comorbidity of the two conditions (Sartorius et al., 1996). Indeed Eaton and Ritter (1988) report little basis for distinction between anxiety and depression from their data from a community survey, with respect to differences regarding socio-demographic factors, life events or previous psychological difficulties. Of course, another implication of intercorrelation between self-reported symptoms of anxiety and depression in a non-clinical sample is the fact that it might represent a more general construct of negative affectivity (Watson & Clark, 1984).

Several of the criticisms associated with the original version of the BDI (e.g., similar

98 One should note that these comparisons were conducted with the original version of the BDI, rather than the BDI-II as used in the current study.
tautology of items, neglect of somatic symptoms) appear to have been addressed in the revised version utilised in the current study. However, given that the validity of the BDI has been systematically examined primarily in clinically depressed populations (and was indeed devised for assessment of the severity of depressive symptomatology in such samples), caution must be used in extrapolation of results when assessing non-clinical populations.

4.4 DIRECTIONS FOR FUTURE RESEARCH

In accordance with suggestions made in the previous section with regards appropriate changes to the methodology, some possibilities are indicated for extension and replication of the current study. In particular, analysis of the potential mediating role of dysfunctional schemas could be conducted across different diagnostic categories, whilst allowing for a more strictly controlled comparison (non-clinical) sample and ensuring more participants in each group. In addition, use of different assessment measures (for example, the original version of the YSQ) might be implicated to enable more detailed analysis with respect to the specific nature of schemas involved.

In addition, the tentative conclusions that can be drawn from the current study, with respect to the mediating role of dysfunctional schemas in the relationship between parental bonding and psychopathology, suggest several possible avenues for further investigation. One such area could be examination of other variables that might also act as mediating factors between early experiences of parenting (or, assuming a wider scope with respect to early experience, e.g., childhood adversity) and
psychopathology. Given Bowlby's (1977) suggestion that the propensity to form and maintain social relationships was crucial in the link between attachment and later psychological wellbeing, exploration of the potentially mediating role of interpersonal relationships or social support seems meaningful. Indeed, with respect to depression, Coyne and Gotlib (1983) stress the need to expand focus beyond cognitive processes to interpersonal factors, such as social skills, which, while relevant in the maintenance of depression, might also function causally.

Another relevant factor worth further investigation as a mediating variable might be self-esteem (Chambers et al., 200; Lloyd & Miller, 1997), given its link with experience of psychopathology. Indeed, it would appear reasonably logical that low levels of parental care might engender a negative self-concept and a corresponding lack of confidence in one's own abilities, which might then result in psychological difficulties if effective mechanisms were not in place to facilitate competence to cope with critical events.

Indeed, examination of the contribution of stressful life events (in line with a diathesis-stress model; Brown & Harris, 1978, 1986; Parker, 1993; Perris, 1988) to the mediational model investigated in the current study would be of interest – for example, elucidation of whether stressful experiences are more likely to be elicited in individuals who hold a number of maladaptive beliefs about themselves via prospective studies. In addition, a related question would be whether these life events need to be schema-congruent to influence vulnerability, as suggested in a study by Hammen, Marks, Mayol, and deMayo (1985)\textsuperscript{99}. It would be of interest to determine

\textsuperscript{99} Schemas were assessed differently in this study.
what weight, if any, life events add to the current model, given that many individuals experience negative events without subsequently developing psychological difficulties. Within such a model, determining which factors (both internal, e.g., self-esteem, and external, e.g., social support) can be protective (Rutter, 1985), against those that appear to engender vulnerability to psychopathology, would be of relevance. As such, focus on individual strengths, resources and other areas of competency that might moderate vulnerability to psychopathology, would be informative. Investigation of all such possible factors, if undertaken with prospective longitudinal methodologies, would help contribute to models of developmental psychopathology.

Another important line of inquiry would be examination of the ability of such a model to predict treatment outcome, given the emphasis on determination of efficacy of psychological interventions in the current health service climate. Indeed, Leung et al. (1999) highlighted the need to examine response to various forms of treatment according to the nature of core beliefs held to enable evaluation of the most appropriate intervention dependent on such characteristics. The predictive value of dysfunctional beliefs (Lam, Green, Power, & Checkley, 1997) and parental bonding styles (Chambers, Power, & Durham, in preparation; Sakado, Sato, Uehara, Sakado, & Someya, 1999) has already been indicated with respect to treatment outcome. Given the current findings, it would be of interest to determine whether the interaction of both the nature of parental bonding and of dysfunctional schemas would enhance predictions regarding treatment response further, and also whether
this would be irrespective of the severity of the presenting problem as suggested by Lam et al.’s results.

If the results of the current study are replicated and causal pathways more conclusively determined via longitudinal studies using prospective designs, there is a strong implication for health promotion work and early intervention. If particular patterns of parental bonding, e.g., an affectionless control style, are indicative of the development of maladaptive schemas and a poor self-identity, thereby enhancing vulnerability to psychological difficulties, screening for such characteristics in childhood could help address such issues before they become ingrained and result in a psychopathological chain. For example, early intervention could be targeted at parental management of the child, such as use of positive parenting techniques (Sanders, 1999). In addition, schema-focused therapy (Young, 1990) could potentially be adapted for use with children and adolescents to facilitate modification of unhelpful beliefs.

4.5 SUMMARY AND CONCLUSIONS

Associations between early parenting experiences and offspring psychopathology have been well documented. However, little empirical research has been conducted to establish the mechanisms by which parental bonding might exert an influence on vulnerability to psychopathology. In line with several theoretical perspectives (Beck, 1967; Bowlby, 1969; Young, 1990), investigation of the potential mediating role of dysfunctional beliefs seemed warranted.
Results from the current study across a general psychiatric out-patient and non-clinical population indicated that maladaptive schemas mediate the relationship between dimensions of parental bonding (care and overprotection) and depressive symptomatology. The pattern of results for anxiety symptomatology appeared less straightforward, with an indication that dysfunctional schemas might play a mediating role on the effects of parental care. However, no such relationship was apparent for parental overprotection.

Although causal interpretations cannot be implied due to the cross-sectional design, the results seem to support this structure amongst the variables. However, prospective longitudinal studies are clearly required to enable more definitive conclusions. Further studies would benefit from comparison of such models between non-clinical and clinical groups to determine whether similar associations are apparent, as believed in the current study. In addition, investigation of the efficacy of such a model for prediction of diagnostic category would inform whether the nature of such factors is specific to particular psychological difficulties or rather whether a general vulnerability to psychopathology per se is apparent. Use of the longer version of the YSQ might also enable elucidation of the relevance of specific schemas in such a model and identification of salient schemas for particular disorders. Incorporation of additional corresponding measures or semi-structured interviews would also enhance the validity of the self-report measures.

Further investigation of the predictive value of such a model with respect to treatment outcome is necessitated to ensure the efficacy of individual therapies. In addition, the possible significance of preventive work is highlighted by such a model, with potential for use of screening measures or early intervention in child or
adolescent populations. Such innovations would be in accordance with the increasing focus on a developmental life-span perspective to health (WHO, 2002).

However, it should be noted that, although the model presented here appears quite elegant, a multifactorial model would seem more probable (Perris, 1988), given the relevance of biological, psychological, social and cultural factors in the genesis of psychopathology. Vulnerability may also not represent a static concept, but evolve throughout an individual’s life course dependent on the reciprocal interplay of transactions between an individual and the surrounding environment.

Nonetheless, some potentially important implications for therapy (Platts, Tyson, & Mason, 2002) can perhaps be inferred from these findings - in particular, the suggestion of the important role of dysfunctional schemas from the current model. Clinicians perhaps need to have a better awareness of such underlying core beliefs and ensure greater focus is accorded to schemas in assessment, formulation and intervention of individual cases.
REFERENCES


APPENDIX 1.1: BRIEF DEScriptions OF EACH OF YOUNG'S (1998)

18 EARLY MALADAPTIVE SCHEMAS

EARLY MALADAPTIVE SCHEMAS
NOVEMBER, 1998

DISCONNECTION & REJECTION
(Expectations that one's needs for security, safety, stability, nurturance, empathy, sharing of feelings, acceptance, and respect will not be met in a predictable manner. Typical family origin is insecure, discriminating, unloving, exploitive, unpredictable, or abusive.)

1. ABANDONMENT / INSTABILITY (MA)
The perceived instability or unreliability of those available for support and connection.

- Involves the sense that significant others will not be able to continue providing emotional support, connection, strength, or practical protection because they are emotionally unstable and unpredictable (e.g., angry, outbursts), unreliable, or erratically present; because they will die imminently; or because they will abandon the patient in favor of someone better.

2. MISTRUST / ABUSE (MA)
The expectation that others will hurt, abuse, humiliate, cheat, lie, manipulate, or take advantage. Usually involves the perception that the harm is intentional or the result of unjustified and extreme negligence. May include the sense that one always ends up being cheated relative to others or "getting the short end of the stick."

3. EMOTIONAL DEPRIVATION (ED)
Expectation that one's desire for a normal degree of emotional support will not be adequately met by others. The three major forms of deprivation are:

- A. Deprivation of Nurturance: Absence of attention, affection, warmth, or companionship.
- B. Deprivation of Empathy: Absence of understanding, listening, self-disclosure, or mutual sharing of feelings from others.
- C. Deprivation of Protection: Absence of strength, direction, or guidance from others.

4. DEFECTIVENESS / SHAME (DS)
The feeling that one is defective, bad, unwanted, inferior, or invalid in important respects; or that one would be unfivable to significant others if exposed. May involve hypersensitivity to criticism, rejection, and blame; self-consciousness, comparisons, and insecurity around others; or a sense of shame regarding one's perceived flaws. These flaws may be private (e.g., selfishness, angry impulses, unacceptable sexual desires) or public (e.g., undesirable physical appearance, social awkwardness).

5. SOCIAL ISOLATION / ALIENATION (SI)
The feeling that one is isolated from the rest of the world, different from other people, and/or not part of any group or community.

IMPAIRED AUTONOMY & PERFORMANCE
(Expectations about oneself and the environment that interfere with one's perceived ability to separate, survive, function independently, or perform successfully. Typical family origin is enmeshed, undermining of child's confidence, overprotective, or failing to reinforce child for performing competently outside the family.)

6. DEPENDENCE / INCOMPETENCE (DI)
Belief that one is unable to handle one's everyday responsibilities in a competent manner, without considerable help from others (e.g., take care of oneself, solve daily problems, exercise good judgment, tackle new tasks, make good decisions). Often presents as helplessness.

7. VULNERABILITY TO HARM OR ILLNESS (VH)
Exaggerated fear that imminent catastrophe will strike any time and that one will be unable to prevent it. Fears focus on one or more of the following: (A) Medical Catastrophes: e.g., heart attacks, AIDS; (B) Emotional Catastrophes: e.g., going crazy; (C) External Catastrophes: e.g., elevators collapsing, victimized by criminals, airplane crashes, earthquakes.
4. ENMESHMENT / UNDEVELOPED SELF (E)

Excessive emotional involvement and closeness with one or more significant others (often parents), at the expense of full individual or normal social development. Often involves the belief that at least one of the enmeshed individuals cannot survive or be happy without the constant support of the other. May also include feelings of being smothered by, or fused with, others or insufficient individual identity. Often experienced as a feeling of emptiness and floundering, having no direction, or in extreme cases questioning one's existence.

9. FAILURE (FA)

The belief that one has failed, will inevitably fail, or is fundamentally inadequate relative to one's peers, in areas of achievement (school, career, sports, etc.). Often involves beliefs that one is stupid, inept, untalented, ignorant, lower in status, less successful than others, etc.

IMPAIRED LIMITS

(Deficiency in internal limits, responsibility to others, or long-term goal-orientation. Leads to difficulty respecting the rights of others, cooperating with others, making commitments, or setting and meeting realistic personal goals. Typical family origin is characterized by overindulgence, permissiveness, lack of direction, or a sense of superiority—rather than appropriate confrontation, discipline, and limits in relation to taking responsibility, cooperating in a reciprocal manner, and setting goals. In some cases, child may not have been pushed to tolerate normal levels of discomfort, or may not have been given adequate supervision, direction, or guidance.)

10. ENTITLEMENT / GRANDIOSITY (ET)

The belief that one is superior to other people; entitled to special rights and privileges; or not bound by the rules of reciprocity that guide normal social interaction. Often involves insistence that one should be able to do or have whatever one wants, regardless of what is realistic, what others consider reasonable, or the cost to others; OR an exaggerated focus on superiority (e.g., being among the most successful, famous, wealthy) in order to achieve power or control (not primarily for attention or approval). Sometimes includes excessive competitiveness toward, or domination of, others: asserting one's power, forcing one's point of view, or controlling the behavior of others in line with one's own desires—without empathy or concern for others' needs or feelings.

11. INSUFFICIENT SELF-CONTROL / SELF-DISCIPLINE (IS)

Pervasive difficulty or refusal to exercise sufficient self-control and frustration tolerance to achieve one's personal goals, or to restrain the excessive expression of one's emotions and impulses. In its milder form, patient presents with an exaggerated emphasis on discomfort-avoidance: avoiding pain, conflict, confrontation, responsibility, or overexertion—at the expense of personal fulfillment, commitment, or integrity.

OTHER-DIRECTEDNESS

(An excessive focus on the desires, feelings, and responses of others, at the expense of one's own needs—in order to gain love and approval, maintain one's sense of connection, or avoid retaliation. Usually involves suppression and lack of awareness regarding one's own anger and natural inclinations. Typical family origin is based on conditional acceptance: children must suppress important aspects of themselves in order to gain love, attention, and approval. In many such families, the parents' emotional needs and desires—or social acceptance and status—are valued more than the unique needs and feelings of each child.)

12. SUBJUGATION (JS)

Excessive surrendering of control to others because one feels coerced—usually to avoid anger, retaliation, or abandonment. The two major forms of subjugation are:

A. Subjugation of Needs: Suppression of one's preferences, decisions, and desires.

B. Subjugation of Emotions: Suppression of emotional expression, especially anger.

Usually involves the perception that one's own desires, opinions, and feelings are not valid or important to others. Frequently presents as excessive compliance, combined with hypersensitivity to feeling trapped. Generally leads to a build up of anger, manifested in maladaptive symptoms (e.g., passive-aggressive behavior, uncontrolled outbursts of temper, psychosomatic symptoms, withdrawal of affection, "acting out", substance abuse).
3. SELF-SACRIFICE (SS)
Excessive focus on voluntarily meeting the needs of others in daily situations, at the expense of one's own gratification. The most common reasons are: to prevent causing pain to others; to avoid guilt from feeling selfish; or to maintain the connection with others perceived as needy. Often results from an acute sensitivity to the pain of others. Sometimes leads to a sense that one's own needs are not being adequately met and to resentment of those who are taken care of. (Overlaps with concept of co-dependency.)

14. APPROVAL-SEEKING / RECOGNITION-SEEKING (AS)
Excessive emphasis on gaining approval, recognition, or attention from other people, or fitting in, at the expense of developing a secure and true sense of self. One's sense of esteem is dependent primarily on the reactions of others rather than on one's own natural inclinations. Sometimes includes an overemphasis on status, appearance, social acceptance, money, or achievement— as means of gaining approval, admiration, or attention (not primarily for power or control). Frequently results in major life decisions that are inauthentic or unsatisfying; or in hypersensitivity to rejection.

OVERVIGILANCE & INHIBITION
(Excessive emphasis on suppressing one's spontaneous feelings, impulses, and choices OR on meeting rigid, internalized rules and expectations about performance and ethical behavior—often at the expense of happiness, self-expression, relaxation, close relationships, or health. Typical family origin is grim, demanding, and sometimes punitive; performance, duty, perfectionism, following rules, hiding emotions, and avoiding mistakes predominate over pleasure, joy, and relaxation. There is usually an underemphasis of passion and worry—that things could fall apart if one fails to be vigilant and careful at all times.)

15. NEGATIVITY / PESSIMISM (NP)
A pervasive, lifelong focus on the negative aspects of life (pain, death, loss, disappointment, conflict, guilt, resentment, unsolved problems, potential mistakes, betrayal, things that could go wrong, etc.) while minimizing or neglecting the positive or optimistic aspects. Usually includes an exaggerated expectation— in a wide range of work, financial, or interpersonal situations—that things will eventually go seriously wrong, or that aspects of one's life that seem to be going well will ultimately fall apart. Usually involves an inordinate fear of making mistakes that might lead to: financial collapse, loss, humiliation, or being trapped in a bad situation. Because potential negative outcomes are exaggerated, these patients are frequently characterized by chronic worry, vigilance, complaining, or indecision.

16. EMOTIONAL INHIBITION (EI)
The excessive inhibition of spontaneous action, feeling, or communication—usually to avoid disapproval by others, feelings of shame, or losing control of one's impulses. The most common areas of inhibition involve: (a) inhibition of anger & aggression; (b) inhibition of positive impulses (e.g., joy, affection, sexual excitement, play); (c) difficulty expressing vulnerability or communicating freely about one's feelings, needs, etc.; or (d) excessive emphasis on rationality while disregarding emotions.

17. UNRELENTING STANDARDS / HYPERCRITICALNESS (US)
The underlying belief that one must strive to meet very high, internalized standards of behavior and performance, usually to avoid criticism. Typically results in feelings of pressure or difficulty slowing down; and in hypercriticalness toward oneself and others. Must involve significant impairment in: pleasure, relaxation, health, self-esteem, sense of accomplishment, or satisfying relationships.

Unrelenting standards typically present as: (a) perfectionism, inordinate attention to detail, or an underestimate of how good one's own performance is relative to the norm; (b) rigid rules and "shoulds" in many areas of life, including unrealistically high moral, ethical, cultural, or religious precepts; or (c) preoccupation with time and efficiency, so that more can be accomplished.

18. PUNITIVENESS (PU)
The belief that people should be harshly punished for making mistakes. Involves the tendency to be angry, intolerant, punitive, and impatient with those people (including oneself) who do not meet one's expectations or standards. Usually includes difficulty forgiving mistakes in oneself or others, because of a reluctance to consider extenuating circumstances, allow for human imperfection, or empathize with feelings.
APPENDIX 2.1: DIAGNOSTIC INFORMATION FOR THE CLINICAL SAMPLE (N46)

Diagnostic information was only available for 33 of the 46 participants in the clinical sample (71.74%) as SMR00 forms had not been returned for some individuals by their clinician. Of these 33, 20 were female and 13 male.

With respect to primary diagnosis, 21 individuals were diagnosed with anxiety disorders (63.64%), 8 with depressive disorders (24.24%), 2 with adjustment disorders (6.06%), 1 with an eating disorder (3.03%) and 1 with significant relationship difficulties (3.03%).

15 of the 33 individuals were also accorded a secondary diagnosis: 4 of anxiety disorders, 3 of depressive disorders, 3 of adjustment disorders, 1 of significant relationship difficulties, 1 of sleep difficulties, 1 of postviral fatigue syndrome and 1 of significant difficulties relating to work.

A tertiary diagnosis was provided for 3 individuals: 1 anxiety, 1 depression and 1 adjustment disorder.
## APPENDIX 2.2: PARENTAL BONDING INSTRUMENT (PBI)

### Parental Bonding Instrument (Parker et al., 1979)

#### RELATIONSHIP WITH MOTHER

For each statement please fill in the circle that best describes how you remember your **MOTHER** in the first 16 years of your life or mother figure (e.g., stepmother, aunt, grandmother)

<table>
<thead>
<tr>
<th>Strongly Agree</th>
<th>Agree</th>
<th>Disagree</th>
<th>Strongly Disagree</th>
</tr>
</thead>
<tbody>
<tr>
<td>She did not help me as much as I needed.</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>She appeared to understand my problems and worries.</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>She was affectionate to me.</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>She liked me to make my own decisions.</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>She tried to control everything I did.</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>She tended to baby me.</td>
<td>□</td>
<td>□</td>
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<tr>
<td>She did not seem to understand what I needed or wanted.</td>
<td>□</td>
<td>□</td>
<td>□</td>
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<tr>
<td>She let me decide things for myself.</td>
<td>□</td>
<td>□</td>
<td>□</td>
</tr>
<tr>
<td>She did not talk to me very much.</td>
<td>□</td>
<td>□</td>
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</tr>
<tr>
<td>She was very overprotective of me.</td>
<td>□</td>
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</tbody>
</table>

Cont’d/
Parental Bonding Instrument (Parker et al., 1979)

RELATIONSHIP WITH FATHER

For each statement please fill in the circle that best describes how you remember your FATHER in the first 16 years of your life or father figure (e.g., stepfather, uncle, grandfather)

<table>
<thead>
<tr>
<th>Statement</th>
<th>Strongly Agree</th>
<th>Agree</th>
<th>Disagree</th>
<th>Strongly Disagree</th>
</tr>
</thead>
<tbody>
<tr>
<td>He did not help me as much as I needed.</td>
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<td></td>
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<tr>
<td>He appeared to understand my problems and worries.</td>
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<tr>
<td>He was affectionate to me.</td>
<td></td>
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<td></td>
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<tr>
<td>He liked me to make my own decisions.</td>
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<tr>
<td>He tried to control everything I did.</td>
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<tr>
<td>He tended to baby me.</td>
<td></td>
<td></td>
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<tr>
<td>He did not seem to understand what I needed or wanted.</td>
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<tr>
<td>He let me decide things for myself.</td>
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<tr>
<td>He did not talk to me very much.</td>
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<tr>
<td>He was very overprotective of me.</td>
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</tbody>
</table>
YSQ-SI
Developed by Jeffrey Young, Ph.D.

INSTRUCTIONS: Listed below are statements that a person might use to describe himself or herself. Please read each statement and decide how well it describes you. When you are not sure, base your answer on what you emotionally feel, not on what you think to be true. Choose the highest rating from 1 to 6 that describes you and write the number in the space before the statement.

RATING SCALE:
1 = Completely untrue of me
2 = Mostly untrue of me
3 = Slightly more true than untrue
4 = Moderately true of me
5 = Mostly true of me
6 = Describes me perfectly

1. _____ Most of the time, I haven't had someone to nurture me, share him/herself with me, or care deeply about everything that happens to me.
2. _____ In general, people have not been there to give me warmth, holding, and affection.
3. _____ For much of my life, I haven't felt that I am special to someone.
4. _____ For the most part, I have not had someone who really listens to me, understands me, or is tuned into my true needs and feelings.
5. _____ I have rarely had a strong person to give me sound advice or direction when I'm not sure what to do.
6. _____ I find myself clinging to people I'm close to because I'm afraid they'll leave me.
7. _____ I need other people so much that I worry about losing them.
8. _____ I worry that people I feel close to will leave me or abandon me.
9. _____ When I feel someone I care for pulling away from me, I get desperate.
10. _____ Sometimes I am so worried about people leaving me that I drive them away.
11. _____ I feel that people will take advantage of me.
12. _____ I feel that I cannot let my guard down in the presence of other people, or else they will intentionally hurt me.
13. _____ It is only a matter of time before someone betrays me.
14. _____ I am quite suspicious of other people's motives.
15. _____ I'm usually on the lookout for people's ulterior motives.
16. _____ I don't fit in.
17. _____ I'm fundamentally different from other people.
18. _____ I don't belong; I'm a loner.
19. _____ I feel alienated from other people.
20. _____ I always feel on the outside of groups.
21. _____ No man/woman I desire could love me one he/she saw my defects.
No one I desire would want to stay close to me if he/she knew the real me.

I'm unworthy of the love, attention, and respect of others.

I feel that I'm not lovable.

I am too unacceptable in very basic ways to reveal myself to other people.

Almost nothing I do at work (or school) is as good as other people can do.

I'm incompetent when it comes to achievement.

Most other people are more capable than I am in areas of work and achievement.

I'm not as talented as most people are at their work.

I'm not as intelligent as most people when it comes to work (or school).

I do not feel capable of getting by on my own in everyday life.

I think of myself as a dependent person, when it comes to everyday functioning.

I lack common sense.

My judgment cannot be relied upon in everyday situations.

I don't feel confident about my ability to solve everyday problems that come up.

I can't seem to escape the feeling that something bad is about to happen.

I feel that a disaster (natural, criminal, financial, or medical) could strike at any moment.

I worry about being attacked.

I worry that I'll lose all my money and become destitute.

I worry that I'm developing a serious illness, even though nothing serious has been diagnosed by a physician.

I have not been able to separate myself from my parent(s), the way other people my age seem to.

My parent(s) and I tend to be overinvolved in each other's lives and problems.

It is very difficult for my parent(s) and me to keep intimate details from each other, without feeling betrayed or guilty.

I often feel as if my parent(s) are living through me—I don't have a life of my own.

I often feel that I do not have a separate identity from my parent or partner.

I think if I do what I want, I'm only asking for trouble.

I feel that I have no choice but to give in to other peoples' wishes, or else they will retaliate or reject me in some way.

In relationships, I let the other person have the upper hand.

I've always let others make choices for me, so I really don't know what I want for myself.
50. I have a lot of trouble demanding that my rights be respected and that my feelings be taken into account.
51. I'm the one who usually ends up taking care of the people I'm close to.
52. I am a good person because I think of others more than of myself.
53. I'm so busy doing for the people that I care about that I have little time for myself.
54. I've always been the one who listens to everyone else's problems.
55. Other people see me as doing too much for others and not enough for myself.
56. I'm too self-conscious to show positive feelings to others (e.g., affection, showing care).
57. I find it embarrassing to express my feelings to others.
58. I find it hard to be warm and spontaneous.
59. I control myself so much that people think I am unemotional.
60. People see me as uptight emotionally.
61. I must be the best at most of what I do; I can't accept second best.
62. I try to do my best; I can't settle for "good enough."
63. I must meet all my responsibilities.
64. I feel there is constant pressure for me to achieve and get things done.
65. I can't let myself off the hook easily or make excuses for my mistakes.
66. I have a lot of trouble accepting "no" for an answer when I want something from other people.
67. I'm special and shouldn't have to accept many of the restrictions placed on other people.
68. I hate to be constrained or kept from doing what I want.
69. I feel that I shouldn't have to follow the normal rules and conventions other people do.
70. I feel that what I have to offer is of greater value than the contributions of others.
71. I can't seem to discipline myself to complete routine or boring tasks.
72. If I can't reach a goal, I become easily frustrated and give up.
73. I have a very difficult time sacrificing immediate gratification to achieve a long-range goal.
74. I can't force myself to do things I don't enjoy, even when I know it's for my own good.
75. I have rarely been able to stick to my resolutions.

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APPENDIX 2.4: BECK ANXIETY INVENTORY (BAI)

Below is a list of common symptoms of anxiety. Please carefully read each item in the list. Indicate how much you have been bothered by each symptom during the PAST WEEK, INCLUDING TODAY, by placing an X in the corresponding space in the column next to each symptom.

<table>
<thead>
<tr>
<th>1. Numbness or tingling.</th>
<th>NOT AT ALL</th>
<th>MILDLY</th>
<th>MODERATELY</th>
<th>SEVERELY</th>
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</thead>
<tbody>
<tr>
<td>2. Feeling hot.</td>
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<tr>
<td>3. Wobbliness in legs.</td>
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<tr>
<td>4. Unable to relax.</td>
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<tr>
<td>5. Fear of the worst happening.</td>
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<tr>
<td>6. Dizzy or lightheaded.</td>
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<tr>
<td>7. Heart pounding or racing.</td>
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<tr>
<td>8. Unsteady.</td>
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<tr>
<td>11. Feelings of choking.</td>
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<td>14. Fear of losing control.</td>
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<tr>
<td>15. Difficulty breathing.</td>
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<td>17. Scared.</td>
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<tr>
<td>18. Indigestion or discomfort in abdomen.</td>
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<tr>
<td>19. Faint.</td>
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<tr>
<td>20. Face flushed.</td>
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<tr>
<td>21. Sweating (not due to heat).</td>
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</tbody>
</table>

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## APPENDIX 2.5: BECK DEPRESSION INVENTORY REVISED (BDI-II)

<table>
<thead>
<tr>
<th>Name:</th>
<th>Date of Birth:</th>
<th>Sex:</th>
<th>Date:</th>
</tr>
</thead>
</table>

Instructions: This questionnaire consists of 21 groups of statements. Please read each group of statements carefully, and then pick out the one statement in each group that best describes the way you have been feeling during the past two weeks, including today. Darken the circle beside the statement you have picked. If several statements in the group seem to apply equally well, darken the circle that has the highest number for that group. Be sure that you do not choose more than one statement for any group, including Item 16 (Changes in Sleeping Pattern) or Item 18 (Changes in Appetite).

1. **Sadness**
   - I do not feel sad. ③
   - I feel sad much of the time. ③
   - I am sad all the time. ③
   - I am so sad or unhappy that I can’t stand it. ③

2. **Pessimism**
   - I am not discouraged about my future. ③
   - I feel more discouraged about my future than I used to be. ③
   - I do not expect things to work out for me. ③
   - I feel my future is hopeless and will only get worse. ③

3. **Past Failure**
   - I do not feel like a failure. ③
   - I have failed more than I should have. ③
   - As I look back, I see a lot of failures. ③
   - I feel I am a total failure as a person. ③

4. **Loss of Pleasure**
   - I get as much pleasure as I ever did from the things I enjoy. ③
   - I don’t enjoy things as much as I used to. ③
   - I get very little pleasure from the things I used to enjoy. ③
   - I can’t get any pleasure from the things I used to enjoy. ③

5. **Guilty Feelings**
   - I don’t feel particularly guilty. ③
   - I feel guilty over many things I have done or should have done. ③
   - I feel quite guilty most of the time. ③
   - I feel guilty all of the time. ③

6. **Punishment Feelings**
   - I don’t feel I am being punished. ③
   - I feel I may be punished. ③
   - I expect to be punished. ③
   - I feel I am being punished. ③

7. **Self-Dislike**
   - I feel the same about myself as ever. ③
   - I have lost confidence in myself. ③
   - I am disappointed in myself. ③
   - I dislike myself. ③

8. **Self-Criticalness**
   - I don’t criticize or blame myself more than usual. ③
   - I am more critical of myself than I used to be. ③
   - I criticize myself for all of my faults. ③
   - I blame myself for everything that happens. ③

9. **Suicidal Thoughts or Wishes**
   - I don’t have any thoughts of killing myself. ③
   - I have thoughts of killing myself, but I would not carry them out. ③
   - I would like to kill myself. ③
   - I would kill myself if I had the chance. ③

10. **Crying**
    - I don’t cry anymore than I used to. ③
    - I cry more than I used to. ③
    - I cry over every little thing. ③
    - I feel like crying, but I can’t. ③

11. **Agitation**
    - I am no more restless or wound up than usual. ③
    - I feel more restless or wound up than usual. ③
    - I am so restless or agitated that it’s hard to stay still. ③
    - I am so restless or agitated that I have to keep moving or doing something. ③

12. **Loss of Interest**
    - I have lost interest in other people or activities. ③
    - I am less interested in other people or things than before. ③
    - I have lost most of my interest in other people or things. ③
    - It’s hard to get interested in anything. ③

13. **Indecisiveness**
    - I make decisions as well as I ever did. ③
    - I find it more difficult to make decisions than usual. ③
    - I have much greater difficulty in making decisions than I used to. ③
    - I have trouble making any decisions. ③

CONTINUED ON BACK
14. Worthlessness
I do not feel I am worthwhile.
I don't consider myself as worthwhile and useful as I used to.
I feel more worthless as compared to other people.
I feel utterly worthless.

15. Loss of Energy
I have as much energy as ever.
I have less energy than I used to have.
I don’t have enough energy to do very much.
I don’t have enough energy to do anything.

16. Changes in Sleeping Pattern
I have not experienced any change in my sleeping patterns.
I sleep somewhat more than usual.
I sleep somewhat less than usual.
I sleep a lot more than usual.
I sleep a lot less than usual.
I sleep most of the day.
I wake up 1-2 hours early and can't get back to sleep.

17. Irritability
I am no more irritable than usual.
I am much more irritable than usual.
I am irritable all the time.

18. Changes in Appetite
I have not experienced any change in my appetite.
My appetite is somewhat less than usual.
My appetite is somewhat greater than usual.
My appetite is much less than before.
My appetite is much greater than usual.
I have no appetite at all.
I crave food all the time.

19. Concentration Difficulty
I can concentrate as well as ever.
I can’t concentrate as well as usual.
It’s hard to keep my mind on anything for very long.
I find I can’t concentrate on anything.

20. Tiredness or Fatigue
I am not more tired or fatigued than usual.
I get more tired or fatigued more easily than usual.
I am too tired or fatigued to do a lot of the things I used to do.
I am too tired or fatigued to do most of the things I used to do.

21. Loss of Interest in Sex
I have not noticed any recent change in my interest in sex.
I am less interested in sex than I used to be.
I am much less interested in sex now.
I have lost interest in sex completely.
TAYSIDE AREA CLINICAL PSYCHOLOGY DEPARTMENT INFORMATION FORM

Examination of the role of perceived parental bonding in the development of core beliefs in individuals experiencing psychological difficulties

We would like to ask you to take part in a research project. To help you understand what the research is about, please read the following information.

Why are you asking me to participate?
We are interested in examining how early bonds formed with parents (or parental figures) affect beliefs people hold about themselves and how this relates to any difficulties they might be experiencing currently.

What will the research involve?
The study involves you completing four fairly short questionnaires, which will probably take between 15 and 30 minutes. Two of the questionnaires relate to how you have been feeling recently (e.g., the extent to which you have felt ‘sad’ or ‘uptight’ over the past 1-2 weeks), one concerns your perceptions of your parents (e.g., “She tried to control everything I did”) and the final questionnaire looks at your beliefs about yourself (e.g., “I don’t fit in”). All of the questionnaires should be straightforward, but if you have any queries or difficulties completing the forms, please contact the principal researcher (details below). The 4 completed questionnaires should then be returned in the stamped addressed envelope provided, or alternatively you could bring them with you to your second appointment and hand them sealed in the stamped addressed envelope to your clinician, who will give them to the principal researcher.

Who will have access to the questionnaires that I complete?
All of your responses will be treated as highly confidential. Only 3 researchers will have access to your data and it will be stored using a code rather than your real name. The results will not be entered into your medical records.

Participation in this study is entirely voluntary and you are free to refuse to take part or to withdraw from the study at any time without having to give a reason and without this affecting your care or your relationship with health care staff looking after you. The Tayside Committee on Medical Research Ethics, which has responsibility for scrutinising all proposals for medical research on humans in Tayside, has examined the proposal and has raised no objections from the point of view of medical ethics.

If you have any further questions or queries, or would like to discuss any aspect of the study, please contact the principal researcher at the address / telephone number below:

Dr Lindsey Murray
Clinical Psychology Department
Murray Royal Hospital
PERTH, PH2 7BH
Tel: 01738 562383
The role of perceived parental bonding in the development of core beliefs in individuals experiencing psychological difficulties

Consent Form

(Please complete this form yourself) PLEASE CROSS OUT AS NECESSARY

Have you read the Information Sheet? YES/NO

Have you been provided with the opportunity to ask questions and discuss this study? YES/NO

(If you have contacted the researcher to ask questions or discuss this study) have you received satisfactory answers to all of your questions? YES/NO

Have you received enough information about the study YES/NO

Who have you spoken to? Dr./Mr./Mrs. ........................................

Do you understand that participation is entirely voluntary? YES/NO

Do you understand that you are free to withdraw from the study:
* at any time?
* without having to give a reason for withdrawing?
* without this affecting your future medical care? YES/NO

Do you agree to take part in this study? YES/NO

Signature ....................................................... Date ..........................................................

Name in block letters .................................................................

Telephone number where you can be contacted:
........................................ (Home) ........................................... (Work)
Demographic Information

NAME: .................................

Please note all responses will be kept strictly confidential and no identifying information will be attached to your data once it has been entered into the database.

AGE: ...................

SEX: male / female (please circle as appropriate)
APPENDIX 2.7A: CONTROL INFORMATION FORM

TAYSIDE AREA CLINICAL PSYCHOLOGY DEPARTMENT

Healthy Volunteer Information Form

Examination of the role of perceived parental bonding in the development of core beliefs in individuals experiencing psychological difficulties

We would like to ask you to take part in a research project. To help you understand what the research is about, please read the following information.

Why are you asking me to participate?
We are interested in examining how early bonds formed with parents (or parental figures) affect beliefs people hold about themselves and how these relate to current psychological well being. We are examining these issues in a clinical population and also need comparison information from individuals, such as yourself, who are not currently receiving input from Tayside Area Clinical Psychology Department.

What will the research involve?
The study involves you completing four fairly short questionnaires, which will probably take between 15 and 30 minutes. These questionnaires will be given to you in a pack for you to take away and complete in your own time. Two of the questionnaires relate to how you have been feeling recently (e.g., the extent to which you have felt ‘sad’ or ‘uptight’ over the past 1-2 weeks), one concerns your perceptions of your parents (e.g., “She tried to control everything I did”) and the final questionnaire looks at your beliefs about yourself (e.g., “I don’t fit in”). All of the questionnaires should be straightforward, but if you have any queries or difficulties completing the forms, please contact the principal researcher (details below). The 4 completed questionnaires should then be returned in the addressed envelope provided via internal mail, or directly to the Psychology Department mailbox in the mailroom at Murray Royal.

All forms should be completed and returned as soon as possible.

Who will have access to the questionnaires that I complete?
All of your responses will be treated as highly confidential. Only 3 researchers will have access to your data and it will be stored using a code.

Participation in this study is entirely voluntary and you are free to refuse to take part or to withdraw from the study at any time without having to give a reason.

If you have any further questions or queries, or would like to discuss any aspect of the study, please contact the principal researcher at the address / telephone number below:

Dr Lindsey Murray
Clinical Psychology Department
Murray Royal Hospital
PERTH, PH2 7BH
TEL: 01738 562383
APPENDIX 2.7B: CONTROL CONSENT AND DEMOGRAPHIC FORM

Demographic Information

Please tick to confirm you have read the information form about this study

Please tick to confirm you have agreed to take part in this study

AGE: .................

SEX: male / female (please circle as appropriate)

POSTCODE: .................. (the last two digits are not necessary)
### APPENDIX 3.1A: CORRELATION MATRIX OF 15 YSQ SCHEMAS

#### Correlations (2-tailed)

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** Correlation is significant at the 0.01 level (2-tailed). * Correlation is significant at the 0.05 level (2-tailed). (See Key for abbreviations)

Given the high proportion of correlations, variables that do not show a significant correlation are highlighted in **bold**.
APPENDIX 3.1B: EXPLORATORY FACTOR ANALYSIS OF YSQ

Scree Plot: Factor analysis of YSQ components

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Extraction Method: Principal Component Analysis.
3 components extracted.
As can be seen via exploratory analysis, the majority of the variance is accounted by one main factor, onto which all 15 dysfunctional schemas load. Therefore an overall dysfunctional schemas variable was utilised which was an average of scores on all 15 dysfunctional schemas.

Key:

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APPENDIX 3.2: CORRELATION MATRIX FOR MATERNAL AND PATERNAL MEASURES OF THE PBI

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** Correlation is significant at the 0.01 level (1-tailed).

Non-significant correlations are highlighted in **bold**.
APPENDIX 3.3: CRONBACH'S ALPHA CO-EFFICIENTS FOR THE FIVE MAIN VARIABLES

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Key:

care = parental care

o/p = parental overprotection

YSQ = dysfunctional schemas
APPENDIX 3.4: HISTOGRAMS ILLUSTRATING DISTRIBUTION OF EACH VARIABLE

Distribution of parental care across whole sample (N91)

Distribution of parental overprotection across whole sample (N91)
Distribution of dysfunctional schemas across whole sample (N91)

YSQ (logarithmic transform)

Distribution of BAI across whole sample (N91)

BAI (logarithmic transform)

Distribution of BDI across whole sample (N91)

BDI (square-root transform)
APPENDIX 3.5: GRAPH OF CORRELATION BETWEEN AGE AND PARENTAL CARE

Correlation of parental care and age across the whole sample (N91)

Rsq = 0.0519
APPENDIX 3.6A: ESTIMATES OF SIGNIFICANCE, POWER AND EFFECT SIZE FROM MANCOVA FOR N91

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a Computed using alpha = .05, b Exact statistic, c Design: Intercept+AGE+STATUS+SEX+STATUS * SEX
Tests of Between-Subjects Effects

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b R Squared = .210 (Adjusted R Squared = .174)
c R Squared = .129 (Adjusted R Squared = .088)
d R Squared = .350 (Adjusted R Squared = .320)
e R Squared = .518 (Adjusted R Squared = .496)
f R Squared = .395 (Adjusted R Squared = .367)
APPENDIX 3.6B: ESTIMATES OF SIGNIFICANCE, POWER AND EFFECT SIZE FROM MANCOVA FOR N86

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a Computed using alpha = .05
b R Squared = .204 (Adjusted R Squared = .165)
c R Squared = .126 (Adjusted R Squared = .083)
d R Squared = .363 (Adjusted R Squared = .331)
e R Squared = .551 (Adjusted R Squared = .528)
f R Squared = .410 (Adjusted R Squared = .381)
APPENDIX 3.7: CORRELATION MATRIX FOR THE FIVE MAIN VARIABLES

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**Correlation is significant at the 0.01 level (1-tailed).**

Correlations have been performed on the transformed variables for the whole sample (N=91).

Key:

care = parental care

0/p = parental overprotection

YSQ = dysfunctional schemas