PSYCHOLOGY, BEHAVIOUR, AND THE FAMILY ENVIRONMENT IN CHILDREN WIITH DIAGNOSES OF PRECOCIOUS PUBERTAL DEVELOPMENT

by

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ABSTRACT

The aim of the thesis was to identify whether children with a diagnosis of Premature Adrenarche (PA) or Central Precocious Puberty (CPP) presented with an atypical psychological profile in comparison to typically-developing children. A battery of psychometrics was constructed to study several domains, including eating behaviour, self-perception and intellectual ability. Measures of family environment and parental stress were also included. In addition, an interpretative phenomenological analysis was conducted on five interviews with parents to gain a greater insight into the experience of parenting a child with a diagnosis of early puberty.

It was found that several differences between groups, such as weight gain, internalising behaviours and sleep problems, could be attributed to hormonal or behavioural changes typically associated with pubertal development across all groups. Other observations were specific to the pubertal disorders, such as risk of obesity, problem eating behaviours, anxiety and depression, and aggression. Furthermore, being from a family with a single-parent or non-parent care-giver, and increased family stress were related to earlier pubertal development. In summary, children with a diagnosis of PA or CPP may be more likely to display altered behaviour and psychopathology, but some of these difficulties may also occur in typical pubertal development.

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1. LITERATURE REVIEW – PUBERTAL DEVELOPMENT

1.1 Typical Pubertal Development.

Puberty is the stage of a human's life during which they become capable of sexual reproduction. This is an important, yet difficult stage for many individuals, as changes occur in most areas of life (Blondell, Foster & Dave, 1999; Pinyerd & Zipf, 2005); such as morphology, neurological functioning, behaviour, and social interaction. It is generally recognised that normal pubertal development can occur at any point between the ages of 8 and 14 years in females, and 9 and 15 years in males, lasting for a mean duration of 3 years (Heffner & Schust, 2010; Jones & López, 2006; Muir, 2006). There is great individual variation, however, with females reaching puberty earlier, and with atypical puberty tending to be premature in females but delayed in males (Palmert & Boepple, 2001; Pinyerd & Zipf, 2005).

1.1.1 Adrenarche. Pubertal development consists of two stages; adrenarche and gonadarche. Adrenarche is the first stage of puberty, typically occurring between the ages of 6 and 8 years (Belgorosky, Sonia, Baquedano, Guercio & Rivarola, 2007). It is activated by the gradual maturation of the hypothalamo-pituitary-adrenal (HPA) axis, causing the zona reticularis in the adrenal cortex to enlarge and develop from the age of 3 years, and begin to synthesise and secrete adrenal androgens into the bloodstream (Blondell et al., 1999; Plant & Witchel, 2006; Remer, Boye, Hartmann & Wudy, 2005; Zhang, Rodriguez, Ohno & Miller, 1995). These androgens are primarily androstenedione, dehydroepiandrosterone (DHEA), and its sulphate (DHEAS), which are then converted peripherally into sex hormones, such as testosterone and dihydrotestosterone (Auchus & Rainey, 2004). This causes the onset of axillary and pubic hair growth (pubarche), and changes in body odour and skin sebum (Heffner & Schust, 2010; Pinyerd & Zipf, 2005).

Adrenarche is considered independent of other processes of pubertal development, but occurs in parallel to skeletal growth (Forest, 1978; Korth-Schutz, Levine & New, 1976; Srinivasan & Premkumar, 2012).

1.1.2 Gonadarche. The second stage of puberty occurs approximately 2 years after the onset of adrenarche, and is termed gonadarche. This occurs due to disinhibition of the hypothalamo-pituitary-gonadal (HPG) system. Activation of the HPG axis results in subsequent pulsatile release of Gonadotropin-Releasing Hormone (GnRH) from the hypothalamus (Martinez de la Escalera, Choi & Weiner, 1992; Rasmussen, Gambacciano, Swartz, Tueros & Yen, 1989), which in turn increases secretion of gonadotropins from the anterior pituitary gland, predominantly Follicle Stimulating Hormone (FSH) and Luteinizing Hormone (LH). These hormones stimulate the reproductive organs to release gonadal steroids; androgens, oestrogens and progestagens. This process is regulated by negative feedback mechanisms (Heffner & Schust, 2010). Gonadotropin action marks the onset of the physical manifestations of gonadarche. In females, FSH and LH work together during maturation of the ovarian follicles and release of the ova at ovulation, as well as initiating the release of gonadal steroids; oestrogen and progesterone. These hormones are responsible for the development of secondary sexual characteristics, such as altered fat distribution, breast development and menstruation (Bijlani, 2004; Sukkar, Ardawi & El-Munshid, 2000). In males, FSH promotes spermatogenesis and LH stimulates the production of testosterone in the Leydig cells of the testes. This initiates the development of secondary sexual characteristics, such as growth of facial hair, development of genitalia and breaking of the voice, either directly or via conversion by the enzyme 5-alpha reductase to its more potent metabolite, di-hydrotestosterone (DHT) (Bijlani, 2004; Chattopadhyay & Sengupta, 2007; Heffner & Schust, 2010).

1.2 Factors Influencing Pubertal Timing.

1.2.1 Biological Factors. Pubertal development is a process which affects, and is affected by, many physical systems in the body. It has been suggested that 50-88% of variation in pubertal timing is genetically determined (Guo et al., 2006; Mustanski, Viken, Kaprio, Pulkkinen & Rose, 2004; Palmert & Hirschhorn, 2003; Parent et al., 2003; Towne et al., 2005). This genetic contribution is supported, firstly, by the observed discrepancies in pubertal timing between children of different racial backgrounds. Studies on the secular trends of pubertal development have consistently demonstrated that girls of Black heritage begin to develop sexual characteristics roughly a year before their White and Hispanic counterparts (Herman-Giddens et al., 1997; Wu, Mendola & Buck, 2002). Secondly, within-family studies have shown that strong relationships exist between the age at which parents and their offspring develop secondary sexual characteristics (Papadimitriou, Gousia, Pitaouli, Tapaki & Philippidis, 1999; Towne et al., 2005). Additionally, studies recording concordance rates of age of menarche in mono- and dizygotic twins have shown stronger correlations in monozygotic twins compared to dizygotic twins (Eaves et al., 2004; Kaprio et al., 1995; Meyer, Eaves, Heath & Martin, 1991). Other developmental milestones, such as thelarche, pubarche and growth in height have also shown a similar trend (Fischbein, 1977; Loesch, Huggins, Rogucka, Hoang & Hopper, 1995; Sharma, 1983).

Neurological development is another key factor in the onset of puberty, particularly the activation of the HPA and HPG axes described previously. Evidence for these processes can be observed in the occurrence of altered pubertal timing in children with neurological abnormalities, such as hypothalamic hamartomas (Chalumeau et al., 2003; Chemaitilly et al., 2001; Ng, Kumar, Cody, Smith & Didi, 2003; Pescovitz et al., 1986).

Furthermore, neurological structures begin to differentiate between sexes in early puberty. This is predominantly in the limbic system, although it is still unclear whether these structural changes are influenced genetically or through the endocrine system. In a Magnetic Resonance Imaging (MRI) study of participants aged of 4 to 18 years, Giedd and colleagues (1996) reported that hippocampal volume increased over time in females, whilst amygdala volume increased in males. They argued that this was due to the predominance of male sex hormone receptors in the amygdala, and female sex hormone receptors in the hippocampus. This would therefore suggest a bidirectional relationship between endocrine changes and neurological structure.

1.2.2 Environmental Factors. Although an individual's biology plays a large part in the timing of pubertal onset, there is also a great deal of research postulating the involvement of environmental factors, including diet and nutrition, and family stress.

1.2.2.1 Diet and Nutritional Status. With rates of obesity and early puberty increasing at a similar rate, many researchers have suggested a relationship between the two variables. The effects of weight and nutrition on puberty can commonly be seen in the delayed menarcheal onset or amenorrhea in those with a very low body weight, such as athletes, dancers, or those with a diagnosis of Anorexia Nervosa (Copeland, Sacks & Herzog, 1995; Warren, 1980; 1999). Furthermore, earlier pubertal development is frequently observed in those who are at risk of being overweight (Currie et al., 2012; De Simone et al., 1995; Lee et al., 2007), and those with a higher body mass index (BMI) are more likely to be in puberty (Adair & Gordon-Larsen, 2001; Anderson, Dallal & Must, 2003; Harlan, Harland & Grillo, 1980; Himes et al., 2004).

Early research suggested an 'invariant mean weight' had to be attained before individuals would begin to develop (Frisch & Revelle, 1970), whereas later studies

suggested that it was a certain proportion of adiposity which determined pubertal onset (Frisch, 1980). Other authors have investigated the effects of leptin on regulation of body weight and metabolism, and demonstrated that although levels of leptin increase prior to pubertal onset, and a threshold level is necessary for development, there is no causal relationship between increases in leptin and pubertal onset. The influence of leptin is, therefore, more likely to be permissive than causal (Apter, 2003; Garcia-Mayor et al., 1997; Hileman, Pierroz & Flier, 2000). Research in the field of foreign-adoption also suggests a role of nutritional status in pubertal development. Foreign-adopted children are considerably more likely to demonstrate clinically early pubertal development (Baron, Battin, David & Limal, 2000; Krstevska-Konstantinova et al., 2001; Mason, Narad, Jester & Parks, 2000; Proos, Hofvander & Tuvemo, 1991; Teilmann, Pedersen, Skakkebæk & Jensen, 2006), which has been hypothesised as being linked to changes in their nutritional status, and resulting rapid increase in their BMI (Teilmann et al., 2006; Virdis et al., 1998). Proos (2009) reported that a cohort of children adopted from under-privileged backgrounds in India into Swedish families, were considerably below the mean height and weight when compared to national reference data. The authors noted catch-up growth over 2 years after adoption, and found that those with the greatest catch-up growth demonstrated the earliest age at menarche. They concluded that the rate of catch-up growth, and not a weight or adiposity threshold, is responsible for the onset of pubertal development in these children. It could also be argued, however, that entering early puberty initiated a growth spurt in these children, rather than the other way around.

Proos' observations are supported by life history theories, which describe the effect of nutrition on pubertal development through two adaptive processes (Gluckman & Hanson, 2006). The first suggests that if the *prenatal* environment provides less than ideal

conditions and a child is born small for gestational age (SGA), the body will assume that this level of nutrition will continue and the child will become independent at an earlier age, thus able to move to a more thriving environment. The second process suggests that if a child experiences poor *postnatal* nutrition at a time immediately preceding their entry into puberty, their development is likely to be delayed. This is because reproduction is high cost, and will likely be more successful when the environment is prosperous. However, if a child is healthy and living in good conditions, they will develop whilst the environment is favourable. Gluckman and Hanson suggest that children who are adopted from lower socioeconomic status (SES) countries are more likely to experience both prenatal influences and postnatal influences; poor intrauterine nutritional conditions combined with moving from a poor postnatal environment into a thriving one resulting in catch-up growth, which would likely cause earlier pubertal development.

SES is commonly considered to be related to nutritional health, as families of a lower SES are more likely to have a diet which consists of cheaper high-energy foods, resulting in poorer health and greater body weight (Darmon & Drewnowski, 2008; Parsons, Power, Logan & Summerbell, 1999). If nutritional status affects pubertal timing, one might expect children of lower SES families to develop earlier. This was demonstrated by James-Todd, Tehranifar, Rich-Edwards, Titievsky and Terry (2010), who found that girls of a lower SES, or who decreased in SES during childhood, were indeed more likely to reach menarche at an earlier age. Similarly, Braithwaite and colleagues (2009) found greater SES to be associated with later menarche in Caucasian girls, but also demonstrated the opposite pattern for Black girls, who were more likely to experience menarche earlier if living in a high income household. This has also been reported in a review by Parent et al., (2003), implying that the relationship between SES and pubertal onset is complex.

1.2.2.2 Early Environmental Stress. Stress can impact on an individual both physically and mentally, and it has been suggested that these physiological effects can trigger the onset of early pubertal development in children (Ellis & Essex, 2007; Flinn, Nepomnaschy, Muehlenbein & Ponzi, 2011). For example, Ellis (2004) discusses evidence for the stress-suppression theory, and suggests that poor diet or stressful environments are likely to delay sexual maturation to a time when the cost of reproduction is lower. Other authors, however, have found that early stress is related to earlier pubertal onset. For example, studies have found adopted children to be 10-25 times more likely to begin pubertal development earlier than the normal population (Soriano-Guillén et al., 2010; Teilmann, et al., 2006), and, as mentioned previously, this trend is even greater for children adopted from developing countries into European or American families (Baron et al., 2000; Krstevska-Konstantinova et al., 2001; Mason et al., 2000; Proos et al., 1991). However, it must also be acknowledged that a child from an impoverished background is more likely to have experienced poor nutrition as well as stressful living environments, and this may therefore confound studies investigating the effects of stress in the adopted population.

Tremblay and Frigon (2005) have suggested that chronic stressors are likely to influence early puberty to a greater extent than a single stressful event. This is supported by findings that events, such as parental conflict or divorce, adverse family relationships, or absence of the child's father, have been linked to earlier pubertal development (Ellis & Garber, 2000; Ellis, McFadyen-Ketchum, Dodge, Pettit & Bates, 1999; Graber, Brooks-Gunn & Warren, 1995; Moffitt, Caspi, Belsky & Silva, 1992; Romans, Martin, Gendall & Herbison, 2003; Tremblay & Frigon, 2005; Wierson, Long & Forehand, 1993). There have been several articles in the literature suggesting that disrupted family relationships

specifically are a risk factor for earlier development (Ellis & Essex, 2007; Romans et al., 2003; Tither & Ellis, 2008). Belsky, Steinberg and Draper (1991) suggested an evolutionary basis for this, in that it would be advantageous for a young female to become capable of reproduction and to attract a mate of her own, in order to escape less stable family conditions. Other authors, however, have suggested that there may be a genetic factor which is associated with both early puberty in the child and parental relationships. For example, Berenbaum (2011) suggested that if a mother reaches puberty early as an adolescent, she is also at risk for earlier sexualisation and more risk-taking behaviours (Dick, Rose, Viken & Kaprio, 2000; Flannery, Rowe & Gulley 1993; Graber, Seeley, Brooks-Gunn & Lewinsohn, 2004; Westling, Andrews, Hampson & Peterson, 2008; Wichstrøm, 2001). This may cause her to choose a similar partner, likely resulting in an unstable relationship. Berenbaum therefore suggests that marital dysfunction is not a cause of early pubertal development in the child, so much as early pubertal development in the parent encourages marital dysfunction, and this early development is also inherited by the child. Similarly, authors such as Rowe (2002) and Harden and Mendle (2012) have suggested a genetic heritability between pubertal onset and behaviours, such as age at first intercourse, sexual activity, and delinquent behaviour; which implies that these behaviours are likely to run in families. This has also been suggested as an explanation for the prevalence of early puberty in the adoption literature, as early puberty and pregnancy in the mother may lead to the child being put up for adoption. Thus, early development may be inherited by the child, rather than caused by the stress of adoption (Brooker, Berenbaum, Bricker, Corley & Wadsworth, 2012). These genetic accounts therefore provide explanations for the relationship between family dysfunction, delinquent behaviour, and early pubertal development.

1.3 Secular Trends in Pubertal Timing.

There is a growing body of literature suggesting that pubertal onset is occurring progressively earlier. Tanner's work in the 1970's was one of the earliest publications to demonstrate a marked decrease in the age of menarche over a period of 140 years (Tanner, 1973). Subsequent studies have tried to replicate or expand on these findings, recruiting participants from different ethnic and socioeconomic groups and using different methodologies (Anderson & Must, 2005; Freedman et al., 2002; Herman-Giddens et al., 1997; Karpati, Rubin, Kieszak, Marcus & Troiano, 2002). A review of the American literature by Herman-Giddens (2007) suggested that girls are maturing 5-12 months earlier than their counterparts of 3 to 5 decades ago, but that existing findings are inconclusive for boys. Similarly, Euling et al., (2008) reported that there is sufficient evidence to assume an earlier trend of both thelarche and menarche, but insufficient evidence to assume an earlier trend of pubarche in females, and all aspects of male pubertal development. The literature into the secular trends of pubertal maturation not only demonstrates how changes in our environment and public health affect our growth and development as a population, but also has implications on the diagnosis of children with disorders of clinically early puberty, and the threshold at which a child is considered in need of medical and psychological attention.

1.4 Premature Adrenarche.

Premature adrenarche (PA) is a pubertal disorder, where children demonstrate adrenarchal development before the age of 7 or 8 years (Kaplowitz & Oberfield, 1999; Siegel, Finegold, Urban, McVie, & Lee, 1992; Silverman, Migeon, Rosemberg & Wilkins, 1952). The clinical characteristics and adrenal androgen levels of children with PA are the same as those seen in typical adrenarchal development, but are significantly increased for chronological age. PA is predominantly seen in females, at a ratio of ten females to every

male (Kaplowitz, Cockerell & Young, 1985). The physical manifestations of PA occur in isolation and are disconsonant of normal pubertal progression, such as thelarche or menstruation.

1.4.1 Causes. PA is caused by excessive androgen levels at an earlier stage than typically expected. As well as idiopathic PA, medical conditions which lead to premature or excessive levels of adrenal androgens and present very similarly include 21-hydroxylase deficiency or congenital adrenal hyperplasia, virilising adrenocortical or gonadal tumours, Cushing's syndrome or other congenital steroid production abnormalities (Belgorosky, Baquedano, Guercio & Rivarola, 2007; Dacou-Voutetakis & Dracopoulou, 1999). PA itself, is more commonly observed in children who showed intrauterine growth restriction (IUGR) or were born SGA (Ibáñez, Potau, Francois & de Zegher, 1998a; Ibáñez, Potau, Marcos & de Zegher 1999a*;* Koziel & Jankowska, 2002; Persson et al., 1999), however, this may be more applicable to females than males (Charkaluk, Trivin & Brauner, 2004; Persson et al., 1999; Potau, Ibáñez, Riqué, Sánchez-Ufarte & de Zegher, 1999). For example Neville and Walker (2005) found that 35% of girls with a diagnosis of PA had been born SGA, and Ibáñez and colleagues have demonstrated a link between ovarian hyperandrogenism and low birth-weight (Ibáñez, Díaz, López-Bermejo & Marcos, 2009; Ibáñez et al., 1998a). Additionally, several researchers have reported a correlation between low birth weight and higher DHEAS levels in prepubertal children (Dahlgren, Boguszewski, Rosberg & Albertsson-Wikland, 1998; Francois & de Zegher, 1997). This has led some researchers to argue that disorders related to excessive androgen exposure may be predetermined before the individual is born.

Some researchers have suggested that rather than low birth weight itself, it is the rapid growth velocity during the first 2 years of life in SGA children which is responsible

for metabolic problems and increased androgen levels, leading to PA (Ong, Ahmed, Emmett, Preece & Dunger, 2000). The inverse relationship reported between birth weight and androgen levels in childhood would support this, as children born SGA or those who demonstrate IUGR show much faster postnatal catch-up growth than the normal population (Ibáñez, Ong, Dunger & de Zegher, 2006; Ong et al., 2004*)*. Furthermore, typically-developing children begin adrenarchal development at the time when the increase in BMI is at its peak (Remer & Manz, 1999). Other researchers, however, have suggested a relationship between weight or adiposity itself and adrenarchal onset. Charkaluk and colleagues (2004) identified a correlation between BMI z-scores and DHEAS levels in children demonstrating signs of PA, and concluded that BMI was influential in adrenarchal development. Furthermore, the Pediatric Research in Office Settings project demonstrated a relationship between BMI z-scores and features of pubertal development such as breast growth and pubarche (Herman-Giddens et al., 1997).

1.4.2 Biological Manifestations. For many years PA was considered a 'benign self-limiting disorder' and simply a variant of typical development (Golub at al., 2008; Silverman et al., 1952; Stanhope & Traggiai, 2004). However, it is now recognised that PA can not only be a problem in itself, but is also commonly associated with several medical disorders. Several research teams have reported a higher incidence of obesity and centrally-distributed adiposity in children both with a diagnosis of PA, and with other forms of early puberty (Biro et al., 2001; Charkaluk et al., 2004; Golub et al., 2008; Ibáñez et al., 2003; Kaplowitz, Slora, Wasserman, Pedlow & Herman-Giddens, 2001). For example, de Ferran and colleagues found that a quarter of their PA group were obese, compared to 4% in the background population (de Ferran, Paiva, Garcia, Gama & Guimaraes, 2011). Similarly, Midyett, Moore and Jacobson (2003) reported that of their

participants presenting with pubarche, 34% had a BMI above the 95th percentile. Cizza and colleagues (2001) found leptin levels in their PA patients to be twice that of controls, as well as the PA patients having a higher BMI. As previously discussed, some researchers have postulated that obesity is a causal factor in the onset of PA, however, others suggest that they are separate mechanisms, and that those with a diagnosis of PA are at an increased risk of obesity through a mediating factor (Sopher et al., 2011; Utriainen, Voutilainen & Jääskeläinen, 2009).

Another medical disorder which is commonly seen in those with early adrenarche is dyslipidaemia; a high level of cholesterol or fat in the bloodstream. Dyslipidaemia is a risk factor for disorders such as metabolic syndrome and type II diabetes mellitus, and can be caused by obesity or an elevated exposure to insulin, both of which are common disorders seen in those with PA (de Ferran et al., 2011; Ibáñez, Potau, Chacon, Pascual & Carrascosa, 1998b; Ibáñez et al., 1998a). Studies have demonstrated increased levels of triglyceride, cholesterol and lipoprotein in children with a diagnosis of early pubarche (Andiran & Yordam, 2008; Denburg et al., 2002; Güven, Cinaz & Bideci, 2005). As a result, Güven and colleagues (2005) have suggested that children with PA may be at risk for high blood pressure and atherosclerosis. Furthermore, children with a diagnosis of PA show a greater reduction in insulin sensitivity than do typically-developing females (Evliyaoğlu, Berberoğlu, Adiyaman, Aycan & Öcal, 2007*;* Ibáñez et al., 1998b; Oppenheimer, Linder & DiMartino-Nardi, 1995; Potau et al., 2003). Ibáñez and colleagues found that girls with PA showed hyperinsulinaemia, earlier action of insulin and also increased uptake of glucose from a young age, and that this was also reflected in their closest relatives (Ibáñez, Castell, Tresserras & Potau, 1999b*;* Ibáñez et al., 1997b). Furthermore, Denburg et al. (2002) found that both boys and girls diagnosed with PA are

susceptible to altered insulin sensitivity, and are therefore at risk of type II diabetes. Furthermore, some researchers have suggested that hyperinsulinism is a common basis for both PA and polycystic ovarian syndrome (PCOS), and that there is an underlying factor influencing both insulin receptor sensitivity and enzyme processes involved in biosynthesis of steroid hormones, leading to hyperandrogenism (Auchus, Geller, Lee & Miller, 1998; Zhang et al., 1995). Ibáñez and colleagues demonstrated both increased androgen synthesis and adrenal hyper-responsiveness, as well as ovarian hyper-responsiveness in girls with PA (Ibáñez et al., 1993; Ibáñez, Potau, Zampolli, Street & Carrascosa, 1997a), and reported that nearly half of PA patients go on to develop ovarian hyperandrogenism, resulting in a steroid profile similar to that seen PCOS. This suggests an underlying factor accounting for the comorbidity of hyperandrogenism and altered sensitivity to insulin, which are key features of PA and PCOS.

PCOS is a disorder affecting 4-8% of females in the general population, compared to 45% of women who have a previous diagnosis of PA in childhood (Asuncion et al., 2000*;* Ibáñez et al., 1993). Women diagnosed with PCOS are at a greater risk of obesity, hypertension, cardiovascular disease and type II diabetes mellitus, as abnormalities in carbohydrate metabolism and impaired glucose tolerance are often observed (Azziz & Saenger, 2000*;* Ehrmann et al., 2006*;* Orio, Palomba & Colao, 2006; Palmert, et al., 2002; Talbott et al., 1998). Furthermore, many features of PCOS are common to metabolic syndrome, such as diabetes mellitus, insulin resistance, high blood pressure and obesity. This is further supported by research showing that children with a diagnosis of PA are at an increased risk of developing metabolic syndrome (Utriainen, Jääskeläinen, Romppanen &Voutilainen, 2007).

A further problem encountered in PA is advanced bone age for chronological age, and resulting decrease in final adult height (Diaz, Bhandari, Sison & Vogiatzi, 2008; Sopher et al., 2011; Utriainen, et al., 2009; Yousefi et al., 2013). Sopher and colleagues (2011) suggested an underlying hormonal factor which enhances the effects of obesity and PA on skeletal growth, as regression analyses found that oestradiol and DHEAS were the strongest predictors of advanced bone age when weight was controlled for. This suggests that there is a complex interaction between weight, height and hormonal influences on adrenarchal development. It can therefore be seen that PA in females is not as benign as was initially thought.

1.5 Central Precocious Puberty.

Another common disorder of early pubertal development is Central Precocious Puberty (CPP), with an incidence of between 1 and 80 per 100,000 live births, and a prevalence of between 1 and 20 per 10,000 (Fuqua, 2013; Gonzalez, 1982; Krstevska-Konstantinova et al., 2001; Soriano-Guillén et al., 2010; Teilmann, Pedersen, Jensen, Skakkebæk & Juul, 2005). CPP is diagnosed when a child enters both adrenarche and gonadarche before the age of 9 years in males and 8 years in females, or reaches menarche before the age of nine (Colaco, 1997; Imel & Bethin, 2007). The disorder is more common in females than males, with a ratio of approximately 10:1 (Partsch & Sippell, 2001).

1.5.1 Causes. The origin of CPP is commonly idiopathic in females, but is likely to have an identifiable cause in males (Blondell et al., 1999; Cisternino et al., 2000; Imel & Bethin, 2007; Pescovitz et al., 1986). Common neurological causes of CPP include hypothalamic hamartoma, astrocytoma, optic glioma, hydrocephalus and neurofibroma (Chalumeau et al., 2003; Chemaitilly et al., 2001; Laue et al., 1985; Ng et al., 2003; Pescovitz et al., 1986; Rieth et al., 1987; Virdis et al., 2003). Other risk factors associated

with the onset of CPP include high BMI, obesity or high leptin levels in females, hyperinsulinaemia or insulin resistance, and being born SGA (Biro et al., 2001; Denburg et al., 2002; DiMartino-Nardi, 1999; Ibáñez, et al., 1998a; Kaplowitz et al., 2001; Koziel & Jankowska, 2002; Midyett et al., 2003; Persson et al., 1999).

It has also been suggested that CPP may be initiated by environmental factors, such as stress, or dietary changes which increase a child's BMI. For example, adopted children are 10-25 times more likely to develop CPP than the normal population, with 13-30% of foreign-adopted children shown to develop idiopathic CPP (Baron et al., 2000; Krstevska-Konstantinova et al., 2001; Mason et al., 2000; Proos et al., 1991; Soriano-Guillén et al., 2010; Teilmann, et al., 2006). This may be due to the stressful process of adoption, or a change in diet when moving to an environment where food is more readily available, and a resulting increase in BMI (Teilmann et al., 2006; Virdis et al., 1998). Furthermore, research has postulated the involvement of leptin in the onset of CPP, as leptin is found in higher concentrations in those with CPP (Palmert, Radovick & Boepple, 1998), and has been shown to stimulate adrenal 17, 20-lyase activity, which plays a key role in sex hormone and steroid production (Biason-Lauber, Zachmann & Schoenle, 2000). There is, therefore, evidence that environmental factors may be connected to the onset of CPP, as well as to earlier pubertal development in the typically-developing population.

1.5.2 Biological Manifestations. There are several physical manifestations of CPP, the most obvious being the development of secondary sexual characteristics atypical for the child's age. Another common consequence of CPP is a reduction in final height, in comparison to both that predicted by parental heights and to that of control groups (Biro et al., 2001; Ghirri et al., 2001; Ibáñez, Ferrer, Marcos, Hierro & de Zegher, 2000a; Midyett et al., 2003; Papadimitriou, Nicolaidou, Fretzayas & Chrousis, 2010). This reduction in

height is due to acceleration in growth velocity, and advanced bone age for chronological age (Paul, Conte, Grumbach & Kaplan, 1995).

Another problem commonly seen in CPP is high BMI or altered distribution of adiposity (Biro et al., 2001; Charkaluk et al., 2004; Golub et al., 2008; Ibáñez et al., 2003; Kaplowitz et al., 2001). Midyett and colleagues (2003) found that half of their patients with a diagnosis of CPP had a BMI above the 95th percentile. Those with early puberty have also been shown to be at an increased risk of obesity in adulthood (Prentice & Viner, 2013), as well as multiple metabolic risk factors, such as high blood pressure, cholesterol and fasting insulin levels (Widen et al., 2012), suggesting that the problems seen during puberty are enduring into adulthood. It is therefore unclear whether being overweight is a consequence of developing CPP, or alternatively, if an underlying cause mediates both weight gain and early development.

Another medical disorder commonly associated with a diagnosis of CPP is PCOS. Franceschi and colleagues (2010) studied the prevalence of PCOS in a group of girls with a diagnosis of CPP, and reported that 37% of their participants showed polycystic ovarian morphology by age eighteen, and 30% showed PCOS, despite there being no signs of hyperandrogenism at the time of diagnosis. Similarly, Lazar and colleagues (1996) reported an exaggerated adrenal response in almost half of their patients with a diagnosis of CPP, as well as physiological characteristics similar to those seen in PCOS. This would suggest that the adrenal system is altered in these patients, as well as the gonadal system.

1.6 Summary.

It is evident that children with a diagnosis of early pubertal development, be it CPP or PA, are at risk for a variety of biological and physiological problems as they progress through adolescence into adulthood. Whilst it is now becoming increasingly common in

medical practice to monitor children for these problems as they develop, it is not as typical to assess the child's mental health. There is existing literature, however, suggesting that these children may need to be monitored for mental health problems, in addition to medical problems.

2. LITERATURE REVIEW – PSYCHOLOGY AND PUBERTAL DEVELOPMENT

2.1 Psychology, Behaviour and Pubertal Development.

Additional factors must be taken into account when considering the psychological health of children with a pubertal disorder. Children with clinically early pubertal development will not only experience the difficulties encountered in 'normal' pubertal development, but also the added complication of being of a considerably younger age. This may exacerbate any difficulties experienced during puberty, as children may not have the cognitive capacity to understand or cope with these bodily and emotional changes at such an early age. In addition, children may feel that they are treated differently by parents, teachers and peers because they appear older. Parents may not know how to explain the diagnosis to their child, and find early sexual development hard to adjust to, putting a strain on family relationships. Furthermore, educational environments are likely to be problematic, as children with early puberty may feel uncomfortable changing their clothing for sports or swimming, and may be teased because they have breast development or skin problems. Primary schools may not provide sanitary facilities in the toilets or separate changing areas, which may be another source of embarrassment. There are clearly many difficulties faced by children with early pubertal development, which may affect both their behaviour and psychological wellbeing, in addition to neurological, hormonal and emotional changes.

2.1.1 Body Image and Eating Behaviour. During puberty, a child's morphology alters considerably, with changes in the amount and distribution of fat, their height and overall shape. These changes, combined with increased interest in conforming to peer-group ideals and in attracting romantic partners, may impact on how the individual perceives their body and the behaviours they may try to carry out in order to change it.

Researchers have found that children as young as 7 years show bodily dissatisfaction (Maloney, McGuire, Daniels & Specker, 1989; Ricciardelli & McCabe, 2001; Thelan, Powell, Lawrence & Kuhnert, 1992), and that children of a larger body mass index (BMI), or who are classified as overweight or obese, are even more likely to be unhappy with their body image (Gualdi-Russo et al., 2008; Vander Wal & Thelan, 2000). Children have been shown to understand the connection between body image and eating behaviour, with evidence that a surprising number of young children will display disordered eating behaviour to influence their weight and appearance (Carper, Orlet Fisher & Birch, 2000; Flannery-Schroeder & Chrisler, 1996; Maloney et al., 1989; Rolland, Farnill & Griffiths, 1998). This disordered eating behaviour is, again, more prevalent in those who are overweight or obese (Decaluwé, Braet & Fairburn, 2002; Lamerz et al., 2005; Vander Wal & Thelan, 2000). As children enter puberty, they typically undergo increases in both height and weight, and it has been reported that those of a higher weight enter puberty at an earlier age (De Simone et al., 1995; Lee et al., 2007), and those who have entered puberty are of a higher weight than those who are prepubertal (Adair & Gordon-Larsen, 2001; Anderson, Dallal & Must, 2003; Harlan, Harland & Grillo, 1980; Himes et al., 2004). For children with a diagnosis of premature adrenarche (PA) or central precocious puberty (CPP), this increase in weight can lead to overweight or obesity (Biro et al., 2001; Charkaluk, Trivin & Brauner, 2004; Cizza et al., 2001; de Ferran, Paiva, Garcia, Gama & Guimaraes, 2011; Golub et al., 2008; Ibáñez et al., 2003; Kaplowitz, Slora, Wasserman, Pedlow & Herman-Giddens, 2001; Midyett, Moore & Jacobson, 2003; Remsberg et al., 2005). We might therefore expect that children who enter puberty earlier are more likely to display body image dissatisfaction.

Berger and colleagues (2009) reported that early-developing girls perceived themselves as bigger, even when BMI was treated as a covariate, and scored higher for body dissatisfaction (Berger, Weitkamp & Strauss, 2009). Similarly, Alsaker (1992) found that early-maturing girls had a more negative body image than those who developed later, or on-time; and that girls of a greater weight presented with body dissatisfaction and negative self-image. Furthermore, Benjet and Hernández-Guzmán (2002) reported that girls with earlier development demonstrated an increase in their bodily self-esteem for the initial 6 months after the onset of menarche, after which their body image satisfaction dropped considerably. More recently, McNicholas, Dooley, McNamara and Lennon (2012) reported that girls who matured earlier showed the greatest desire to be thin and were the most dissatisfied with their body image. It would therefore seem that girls who develop earlier than their peers are more likely to be dissatisfied with their body.

Ge, Elder, Regnerus and Cox (2001) postulated that weight gain commonly associated with the onset of puberty is misperceived by the adolescent as becoming overweight. They reported that this was especially evident for early-maturing girls, even when controlling for actual body weight. Adolescents who perceived themselves as overweight also presented with higher depression scores and lower self-esteem. Similarly, Abraham and O'Dea (2001) found that their female sample only developed a true concept of dieting and weight loss after experiencing menarche and the increase in height, weight and BMI associated with pubertal development. We may therefore hypothesise that the perception of gaining weight at the onset of puberty, may lead these girls to engage in behaviours similar to those seen in eating disorders, such as dieting, weight concerns and a need to exercise. This has indeed been reported multiple times in the literature (Kaltiala-Heino, Marttunen, Rantanen & Rimpela, 2003; Kaltiala-Heino, Rimpel, Rissanen &

Rantanen, 2001; Koff & Rierdan, 1993; McNicholas et al., 2012). Ackard & Peterson (2001) found early-developing girls to prefer a thinner body image and to be more likely to report having an eating disorder then their average- or late-developing peers. Berger and colleagues (2009) also reported that girls who began puberty more than one standard deviation below the mean, scored significantly higher on the Eating Attitudes Test, which assesses problematic eating behaviours and perceptions (EAT-26D; Garner & Garfinkel, 1979). Similarly, research has shown that early-developing girls were more likely to have had a psychiatric diagnosis of an eating disorder, such as Bulimia Nervosa (Day et al., 2011; Graber, Lewinsohn, Seeley & Brooks-Gunn, 1997). It may therefore be that children with PA or CPP are at an increased risk of disordered eating behaviours and negative body image perceptions, due to the prevalence of obesity and overweight in these populations, and the tendency towards maladaptive eating and body image dissatisfaction during puberty.

2.1.2 Self-Perception and Self-Esteem. Puberty is a period of change across several domains, and individuals become increasingly aware of their position in the social world and how they compare to others. This greater awareness of social standing has been described in the literature, and suggests that there is an increase in critical thinking as to how one is perceived by peers (Harter, 1990; Parker, Rubin, Erath, Wojslawowicz & Buskirk, 2006; Rosenberg & Simmons, 1975; Vartanian, 2000). These self-other comparisons may cause dissatisfaction in the individual, resulting in lower self-esteem. We may therefore hypothesise that individuals progressing through puberty would show lower levels of self-esteem than those who are pre- or post-pubertal. This is supported by O'Dea and Abraham (1999), who reported that pubertal status and levels of self-esteem were related, but that this relationship was different for males and females.

One area of self-perception which may become unstable during puberty is physical self-perception. Puberty is a time of bodily change, and it would seem logical that children undergoing this transition "out of sync" with their peers are likely to feel more self-conscious than they would progressing through puberty at the typical age. In the typically-developing population, Graber et al. (1997) reported that early-maturing girls scored significantly lower on measures of self-esteem. Furthermore, Ge and colleagues (2001) reported that girls who developed early and who perceived themselves as overweight, presented with higher depression scores and lower self-esteem. This suggests that girls may develop adjustment problems as their bodies begin to change, particularly if this process begins before the rest of their peer group. This may be an additional problem for those with CPP and PA, due to the combination of early timing and the tendency for obesity and high BMI values in these populations.

In fact, Solyom, Austad, Sherick and Bacon (1980) reported that children with PA were more likely to have a negative self-image than those with CPP, whereas those diagnosed with CPP appeared more self-conscious. They also suggested that children with CPP appear embarrassed and shy when discussing their appearance, whereas those with PA demonstrate more anger and aggression in relation to self-image. Xhrouet-Heinrichs et al. (1997) also reported that their entire sample of CPP patients showed embarrassment regarding their early pubertal development, and over half reported being worried about appearing physically different to their peers, particularly those who had breast development.

Another aspect of self-perception which is likely to fluctuate over pubertal development is one's perceived place amongst peers. During adolescence individuals gradually spend more time with peers and less time with family (Csikszentmihalyi &

Larson, 1986; Larson & Richards, 1991). This may lead to changes in behaviour and attitude in order to "fit in". Those entering puberty early may feel as though they are different to their typically-developing peers, and their satisfaction within the social group may decrease as a result. Furthermore, because they appear older than their years, their place in the social circle is likely to be somewhat different. This may impact on the way in which they are treated by other children.

In the typically-developing population, Graber, Seeley, Brooks-Gunn and Lewinsohn (2004) conducted a longitudinal study, and reported that women who had matured early in adolescence were more likely to report having poor social and family relationships at 24 years. Alsaker (1995a; in Alsaker, 1995b) reported that over half of young adolescents felt as though they had been treated differently since pubertal onset, and the physical changes to their bodies became noticeable. It may therefore be the perception of these physical changes by those around them that causes them to be treated differently, or conversely, it may be the perception of these changes by the individual that causes them to believe that others see them in a different light. Fend (1994) reported that, in a group of adolescents, those that developed earlier showed the lowest bodily dissatisfaction and self-esteem, but also higher perceived social acceptance. This would suggest that early development may make an individual superior to their peers and to fit in with the group, although this does not necessarily reflect the individual's self-esteem. Therefore, a measure of peer acceptance may not necessarily be a good measure of how well a child or adolescent feels that they "fit in" with their peers. Furthermore, for a child of clinically-early development, physical differences may be more likely to make a child stand out in a negative way, because their peers are unlikely to have learned about puberty, and therefore

these changes are seen as a disorder, rather than achieving something before their classmates.

This observation was found by Xhrouet-Heinrichs et al. (1997), who reported that three quarters of their participants with a diagnosis of CPP often reported feeling lonely. Furthermore, Officioso et al. (2004) reported that CPP patients showed 'difficulties in making contact', as demonstrated by them missing out one part of the body when asked to draw a picture of themselves in the Human Figure Test. This somewhat tenuous conclusion, however, is based only on the drawings of 20 girls.

It would therefore seem that pubertal development is related to fluctuations in self-perception across several domains, and for those developing earlier than their peers, these transitions may be more difficult to manage.

2.1.3 Problem Behaviour and Psychopathology. Adolescents are commonly portrayed in the media as being moody and negative, and this transition into adulthood has long been considered a time of turbulent emotional and behavioural change (Dahl, 2004; Dahl & Gunnar, 2009; Larson, Moneta, Richards & Wilson, 2002), often assumed to be associated with hormonal changes. A review by Buchanen, Eccles and Becker (1992) posits several theories of hormonal influence on affect and behaviour, specifically by adrenal androgens, gonadotropins and sex steroids. The first of these is an activation effect, where hormone levels begin to fluctuate to a greater extent than the individual is accustomed to, directly affecting mood and behaviour. The second, is the effect of adjusting to these new hormone levels, suggesting that those further through the developmental process will be less affected as they will be more accustomed to this than those just entering puberty. The third process is that of irregularity in hormone levels at the start of development, as it commonly takes a period of time for these cyclical fluctuations

to become more settled. Finally, there are likely to be complex interactions between each of these effects, alongside influences in the individual's environment, their temperament and their susceptibility to the influence of these hormones.

Studies have shown that many of the hormones involved in the development of secondary sexual characteristics are also implicated in changes in mood and psychopathology (Albert & Beck, 1975; Brooks-Gunn & Warren, 1989; Reardon, Leen-Feldner, & Hayward, 2009; Stice, Presnell & Bearman, 2001). Therefore, puberty has been identified as a trigger for mental health disorders (Angold & Costello, 2006; Graber et al., 1997; Kaltiala-Heino et al., 2003; Mendle, Harden, Brooks-Gunn & Graber, 2010), with one in four adolescents being diagnosed with a mental health disorder (Belfer, 2008). Research by Susman et al. (1985) demonstrated that high levels of adrenal androgens for chronological age were related to greater negative affect in males, and that females with higher gonadotropins demonstrated higher levels of psychopathology. Furthermore, Angold, Costello, Erkanli and Worthman (1999), showed that levels of androgens and oestrogen were better predictors of depression than was pubertal status. Similarly, Graber et al. (1997) reported that girls with earlier onset of pubertal development had a higher incidence of several mental health disorders and symptoms of psychopathology, including depression, externalising behaviour problems and suicidal attempts. The authors also later found that females who had developed early during childhood and adolescence were significantly more likely to have had depression, anxiety, disruptive behaviour disorder, or any psychiatric disorder on Axis I of the DSM-III-R, at some point between adolescence and age 24 years (Graber et al., 2004). Hayward and colleagues (1997) studied a group of female adolescents longitudinally, and reported that participants demonstrating internalising symptoms during the study time-period had reached puberty 5 months earlier,

with those with earlier maturation developing earlier symptomatology. These girls were also more likely to develop depression, phobias or eating disorders. Kaltiala-Heino et al. (2003) observed that internalising and externalising behaviours were more common in girls who experienced menarche at an early, but not clinically premature age, compared to children with on-time or late menarche. Similarly, Blumenthal and colleagues (2011) assessed young people between the ages of 12 and 17 years on measures of social anxiety, and found that girls falling into the category of early puberty demonstrated significantly increased scores of social anxiety in comparison to on-time girls and early-developing boys. Other supporting research provides evidence for increased prevalence of conduct disorder, oppositional defiant disorder and attention disorders in those with earlier development (Benjet & Hernández-Guzmán, 2002; Ge, Brody, Conger & Simons, 2006; Siegel, Yancey, Aneshensel & Schuler, 1999). However, only four of the above studies included participants as young as 9-10 years, despite development at this age now being considered 'typical'. Many research studies have therefore not included the lowest end of the distribution of typical pubertal development, despite hypothesising a link between early development and psychological problems. Including younger participants may therefore have strengthened any observed relationships between early pubertal timing and psychological difficulties. In summary, however, there is a great deal of evidence to suggest that early development is a risk factor for mental health disorders, whether this development is very early at 9 years of age, or slightly early at 12 years of age. It may be that this trend is exacerbated further in those with precocious development.

In addition to alterations in mood and risk of psychopathology, there is a considerable amount of literature on increased behavioural problems during pubertal development, particularly in those with atypical pubertal timing. For example, those who

are maturing earlier than their peers, but still within the normal distribution, are more likely to show externalising behaviours, such as sexual behaviour, smoking cigarettes, drinking alcohol or behaving aggressively (Arim, Tramonte, Shapka, Dahinten & Willms, 2011; Costello, Sung, Worthman & Angold, 2007; Dick, Rose, Viken & Kaprio, 2000; Graber et al., 1997; 2004; Susman et al., 1985; Westling, Andrews, Hampson & Peterson, 2008; Wichstrøm, 2001). A review of the literature by Celio, Karnik and Steiner (2006) concluded that girls who mature earlier are at risk for aggressive and delinquent behaviour for several reasons. These include a need to overcome discrepancies between physical and social maturity, an inability to cope with the changing expectations and perceptions of others, and exposure to environments and social groups in which aggression and delinquency are more common. Other research studies have linked increased sex steroids with behaviours such as alcohol consumption (de Water, Braams, Crone & Peper, 2013). Furthermore, young adolescents become driven towards achieving autonomy, and it may be that their expectations and their parents' expectations of what is an appropriate level of independence become increasingly incongruent.

Another concerning finding, commonly reported in the literature, is that those who begin pubertal development earlier are more likely to have earlier sexual intercourse and commit risky sexual behaviour. For example, Flannery, Rowe and Gulley (1993) reported that adolescents who showed advanced pubertal development for their age reported being more sexually experienced, and that this increased sharply as individuals reached Tanner stage IV. This is somewhat consistent with Angold, Costello and Worthman (1998) who posit that the emergence of psychological problems is most evident after Tanner Stage III. A study of sexual behaviour among Chinese adolescents reported that those who matured earlier also began to date earlier. Of those who had had sex, boys who had matured earlier

also reported having sex at a younger age, but this trend was not observed for girls (Lam, Shi, Ho, Stewart & Fan, 2002). Similarly, Downing and Bellis (2009) found that males who began puberty before the age of 12 years were nearly four times more likely to have unprotected sex before the age of 16 than those who began puberty after 13 years. Deardorff, Gonzales, Christopher, Roosa and Millsap (2005) assessed 666 women who had had an early pregnancy on measures of several factors, including onset of menstruation, substance use and their sexual relationship history. Earlier pubertal development was found to be significantly predictive of early pregnancy, with use of alcohol and age of sexual debut as mediating variables. Other authors, however, propose that it is not pubertal development which is associated with the onset of sexual behaviour in adolescence, but the change in height and appearance which identifies an individual as being a suitable sexual partner (Bingham, Miller & Adams, 1990). This would suggest that puberty is a covariate, rather than a causal factor.

In the literature on children with clinically early puberty, Dorn and colleagues (Dorn, Hitt & Rotenstein, 1999; Dorn et al., 2008) reported higher scores on the Child Behavior Cheklist (CBCL) in girls with PA for the categories of social problems, signs of anxiety and depression, aggression, both internalising and externalising behaviours, and total behaviour problems. In addition, Sonis et al. (1985) reported higher internalising and externalising behaviour in children diagnosed with CPP, with a quarter of girls scoring more than two standard deviations above the mean on the Total Behavior Problem scale of the CBCL. Xhrouet-Heinrichs et al. (1997) found that parents reported several problematic behaviours in their children with a diagnosis of CPP, namely being tearful, shy, overly dependent, and in some cases, aggressive. Additionally, children with early adrenarchal development have been shown to demonstrate greater social problems, signs of anxiety

and depression, aggression and conduct disorder (Cicchetti & Rogosch, 2007; Dorn et al., 1999, 2008; Goodyer et al., 1996; Van Goozen, Matthys, Cohen-Kettenis, Thijssen & van England, 1998). Baumann and colleagues (2001) investigated the long-term behaviours of young adults, who had previously undergone treatment for CPP, and found that elevated behavioural problems persisted several years later. Furthermore, they reported that reduced adult height and later onset of precocious pubertal development were predictors of behavioural problems. The literature on sexual behaviour in the precocious development population includes an early paper by Meyer-Bahlburg and colleagues (1985). They reported that in their sample of girls with CPP, patients were more likely to reach sexual behaviour milestones earlier than controls. Similarly, Cassio and colleagues (2006) observed a higher rate of teenage pregnancy in their CPP patients than they had expected.

Arim and colleagues (2011), however, point out that in a great deal of the research published on increased problem behaviours in early-developing adolescents, the onset of these behaviours is typically only a few months earlier than in control participants. This does not necessarily mean that these behaviours continue for any longer or occur any more frequently than they would in those of on-time development. It would therefore seem that, for at least some of the literature, early maturation is simply associated with earlier exposure to behaviours such as smoking or drinking alcohol, rather than an increased prevalence in this group. Furthermore, other research has found that those developing at the later end of the normal range are more likely to demonstrate behaviour problems than those developing early or on-time (Dorn, Susman & Ponirakis, 2003). It may therefore be that pubertal development "out of sync" with peers is the key difficulty in this population, rather than pubertal development itself. For children with disorders of precocious

development, one might expect these problems to be exacerbated due to the fact that their development is further discordant to that of their peers.

2.1.4 Intellectual Ability. Researchers have long questioned whether pubertal changes have an impact on neurological functioning. Research has shown development across several brain areas during puberty, often peaking around 11 or 12 years which is the average age of pubertal onset (Barnea-Goraly et al., 2005; De Bellis et al., 2001; Giedd et al., 1999; Gogtay et al., 2004; Jernigan, Trauner, Hesselink & Tallal, 1991; Lenroot et al., 2007; Paus et al., 1999; Pfefferbaum et al., 1994; Sowell et al., 1999; Thompson et al., 2000). Furthermore, it is during this time period that higher cognitive functions develop, such as inhibitory control and working memory (Luna, Padmanabhan & O'Hearn, 2010). Studies have identified relationships between hormones implicated in pubertal development and neurological changes. For example, increased testosterone in males has been linked to increased amygdala volume, whereas oestrogen in females is related to grey matter volume in the parahippocampal gyrus (Neufang et al., 2009). More recently, Nguyen and colleagues (2013) reported dehydroepiandrosterone (DHEA) to be related to cortical thickness across several brain areas, and to interact with testosterone in relation to others. Furthermore, sex steroids, such as estrogens and androgens, have been found to be implicated in the structure, excitability or maintenance of the hippocampus, hypothalamus, visual cortex and pathways associated with reward (Blakemore, Burnett & Dahl, 2010; Cahill, 2006; MacLusky, Hajszan, Prange-Kiel & Leranth, 2006; Nuñez, Huppenbauer, McAbee, Juraska & DonCarlos, 2002; Parducz, et al., 2006; Sato, Schulz, Sisk & Wood, 2008). These findings suggest that puberty is a time of neurological development, and we may therefore assume that changes may occur in cognitive functioning as a result. For those entering puberty early, we may see these changes occurring before we would

typically expect, or that the premature neurological exposure to sex steroids has a different effect on the younger brain.

In the literature on early- and late-maturing children within the normal developmental thresholds, earlier development has been linked to advanced intellectual abilities. Douglas and Ross (1964) used national data to compare the educational attainment of those with early and late pubertal onset. The sample was divided according to pubertal status at several time-points, and group performance was compared on school examinations. Earlier maturing children scored higher on measures of intelligence, reading, and maths, and were more likely to stay on for further qualifications after the compulsory leaving age. Those with earlier development were also more likely to leave school with 'good 'O' levels', which are what the authors consider to be English language or foreign languages, maths and a science subject. Waber (1977), however, suggested that earlier puberty prevents hemispheric lateralisation and results in poor spatial intelligence.

In the precocious puberty population, current literature surrounding this topic is inconclusive, with some of the earlier papers poorly defining the parameters of their diagnostic groups, and drawing conclusions from arguably tentative methodology. Nass, Baker, Sadler and Sidtis (1990) studied a group of girls diagnosed with PA, and found that they showed a strong right ear advantage to speech stimuli on dichotic listening tasks. When presented with tonal stimuli, however, young participants showed a typical left ear advantage whilst older participants showed a right ear advantage. The authors concluded that during adrenarche, the right hemisphere is enhanced but the effects of this early androgen exposure results in language later being localised to the left hemisphere to a greater extent than normal. Conversely, Bruder, Meyer-Bahlburg, Squire, Ehrhardt and Bell (1987), reported no difference between CPP patients and control participants on

dichotomous listening tasks, and concluded that there is no resulting difference in verbal ability.

The findings are no less clear when using psychometric measures of intellectual ability. Galatzer, Beth-Halachmi, Kauli and Laron (1984) reported that girls with PA or CPP scored significantly higher than control participants on verbal scales. They also found patients scored significantly higher than control participants on subscales of information, arithmetic, and similarities, with 10% of the altered pubertal development group having an IQ score greater than 130 points compared to just 2% of controls. Other research has failed to demonstrate a difference in verbal ability, but has shown decreased spatial abilities in girls with CPP or early but normal development (Ehrhardt & Meyer-Bahlburg, 1986; Meyer-Bahlburg et al., 1985). Xhrouet-Heinrichs and colleagues (1997) found that their CPP patients scored within the typical range, with a mean IQ score of 102. However, they did report that their patients scored significantly lower on performance scales compared to verbal scales. Research by Dorn et al. (1999) reported significantly lower scores on several other subscales of the Wechsler Intelligence Scales for Children (WISC-III) in nine girls with PA compared to children of typical pubertal development. These subscales were; information and general knowledge, arithmetic, vocabulary and verbal IQ. It is clear that the literature is inconclusive about whether there is an altered intellectual ability in this patient group, and this means that any differences observed may be due to an unintentional bias in sampling, particularly in the smaller samples, or simply by chance.

One must also consider that, if early-maturing children appear older, they are likely to be spoken to as if they are older and may be given more responsibilities requiring higher reasoning skills or forward thinking. This may result in them behaving and speaking in a more mature manner, and being exposed to more advanced language or reasoning

demands. Similarly, if a child has to frequently miss school for hospital appointments and hormone injections, or is bullied at school because of their early development, this could have a negative impact on their academic performance. Any differences observed in the precocious puberty patients may therefore not be exclusively due to hormonal or neurological changes.

2.1.5 Family Environment and Parental Stress. Caring for a child with any form of medical condition is likely to be difficult for a family, and for the family of a child with early pubertal development this is no different. Families may find it difficult to explain the diagnosis to their child, and feel uncomfortable when describing the diagnosis to their child's school or when taking their child for invasive examinations. Furthermore, the nature of early puberty itself may bring discomfort, with a change in what is perceived to be appropriate parent-child interaction, and the associations made between pubertal development and sexual maturation.

Solyom and colleagues (1980) found that parents of children with PA or CPP showed embarrassment over their child's disorder and would deny that their child was showing signs of sexual development. They also reported that parents were concerned about their child beginning to behave sexually. However, this paper was simply a discussion of 16 case studies and employed no experimental methods. Similarly, Officioso et al. (2004) stated that parents of children with CPP showed concern over earlier sexual debut in their children, but again these observations were taken from non-structured interview data of a small number of cases. It would appear however, that similar behaviours are observed clinically suggesting their conclusions are valid. This would imply that being the parent of a child with early puberty may be a source of stress, and in

this way, families may have higher stress levels than those of typically-developing children.

As discussed previously, there are also several studies in the literature which suggest that puberty may be triggered early by stressful life events, as well as early puberty being a cause of family stress. Environmental stressors linked to early puberty in the literature include adoption, family conflict, father absence, low socioeconomic status (SES), and dysfunctional family relationships (Baron, Battin, David & Limal, 2000; Ellis & Garber, 2000; Ellis, McFadyen-Ketchum, Dodge, Pettit & Bates, 1999; Graber, Brooks-Gunn & Warren, 1995; James, Ellis, Schlomer & Garber, 2012; James-Todd, Tehranifar, Rich-Edwards, Titievsky & Terry, 2010; Krstevska-Konstantinova et al., 2001; Mason, Narad, Jester & Parks, 2000; Moffitt, Caspi, Belsky & Silva, 1992; Proos, Hofvander & Tuvemo, 1991; Romans, Martin, Gendall & Herbison, 2003; Soriano-Guillén et al., 2010; Teilmann, Pedersen, Skakkeæk & Jensen, 2006; Tremblay & Frigon, 2005; Wierson, Long & Forehand, 1993). This earlier pubertal development is hypothesised to be an adaptive response to environmental stressors, resulting in the child becoming independent at a younger age, and able to move away from the difficult environment (Belsky, Steinberg & Draper, 1991). It may be, therefore, that the family of a child with early puberty is more likely to experience higher stress and anxiety, whether it be as a result of their child's diagnosis, or a contributing factor.

2.1.6 Summary. The literature on psychology and behaviour in puberty would suggest that those maturing earlier than their peers, but within the range of variation considered 'normal', are at a greater risk of a number of different disorders across multiple domains. Although there are fewer studies on the psychology of children developing clinically early, i.e. before the recommended clinical cut-off of 7-8 years in girls and 8-9

years in boys, the existing publications imply that these patients and their families are just as vulnerable to these problems, if not more so. Further research is therefore needed to assess the prevalence and severity of these problems in the clinical populations. This could then be used to better inform healthcare and educational professionals about the psychological health of these children and their families, and to provide information for the families of children with diagnoses of precocious pubertal development.

3. METHODOLOGY

3.1 Research Design.

The study was a cross-sectional quasi-experimental between-groups design, with participants assigned to their group according to their diagnosis, or lack of diagnosis in the typically-developing group. This allowed for between-group comparisons, to determine whether there were significant differences in performance. The use of two diagnostic groups allowed for comparisons between diagnoses, as well as between patients and controls. This enabled identification, firstly; of biological or environmental influences of having started pubertal development, and secondly; of any impact specific to each diagnosis. Furthermore, analysing pubertal development as a continuous variable, across all groups, enabled observation of any changes in performance relative to changes in puberty.

The project was sponsored by the University of Birmingham under the Research Governance Framework, and was approved by the West Midlands committee of the National Research Ethics Service; and the Directorate of Research and Development of the Birmingham Children's Hospital (BCH) and University Hospital of Coventry and Warwick (UHCW). The project was funded by the Child Growth Foundation (CGF).

3.2 Participants.

3.2.1 Participant Characteristics and Inclusion Criteria. The participants included in the main study were 101 children and their families (18 male, 83 female; mean age 102.8 months, SD 18.1 months). Participants were divided into three groups; patients with a diagnosis of Central Precocious Puberty (CPP), patients diagnosed with Premature Adrenarche (PA), and those of typical pubertal development (controls). Groups were

matched as closely as possible for age, ethnicity and gender. Of the sixteen participants in the CPP group, eleven participants were receiving hormone treatment at the time of research participation. The treatment usually recommended for those diagnosed with CPP, is a Gonadotropin-releasing Hormone (GnRH) agonist injection, administered once every 8-10 weeks, which reverses the signs of pubertal development by blocking the release of gonadotropins from the pituitary gland. For other participant characteristics, please see Table 1.

The inclusion criteria were that participants were aged 4-10 years, and that both participants and their families had a sufficient standard of English to be able to complete the assessments and questionnaires. The criteria for the patient groups also included a valid diagnosis through the BCH or UHCW, and an absence of a diagnosis in the control group. Participants' pubertal status was validated by scores on the pubertal development questionnaire (see section 3.3.1.1 and Appendix 1). Exclusion criteria included any physical or developmental diagnoses which may have affected participants' pubertal status, behaviour or psychological health, and potentially performance on assessment measures. Participants in the patient groups were excluded if there was an identifiable cause for the diagnosis, as it could not be ascertained whether this biological cause may also affect performance on the assessment measures. Participants in the PA group were excluded from the study if they demonstrated manifestations of pubertal development not typical of adrenarche, that were Tanner stage II or above. Participants in the CPP group were excluded if development had occurred in a disordered sequence, as this would not be considered typical of a diagnosis of CPP.

Table 1: *All participants' demographic information*[1]

	n (m:f)	Mean age in months (SD)	Ethnicity		SES	
Control	50 (14:36)	100.38 (19.82)	White	70%	< £14,000	10%
			Mixed	18%	£14,000-£24,999	10%
			Asian	8%	£25,000-£34,999	10%
			Black	4%	£35,000-£49,999	28%
					£50,000+	42%
PA	35 (3:32)	102.54 (16.00)	White	46%	< £14,000	3%
			Mixed	17%	£14,000-£24,999	18%
			Asian	26%	£25,000-£34,999	23%
			Black	11%	£35,000-£49,999	29%
					£50,000+	27%
CPP	16 (1:15)	111.06 (14.74)	White	50%	< £14,000	13%
			Mixed	13%	£14,000-£24,999	7%
			Asian	13%	£25,000-£34,999	20%
			Black	24%	£35,000-£49,999	27%
					£50,000+	33%

[1] Please note; sample sizes differ for each analysis due to missing data. Total and group sample sizes for each chapter are listed here, with the number of males and females given in brackets.
- Body Composition, Eating Behaviour and Body Image: n= 99 (17: 82), control= 48 (13:35), PA= 35 (3:32), CPP= 16 (1:16).
- Behaviour, Emotion and Sleep: Child Behavior Checklist: n= 95 (16: 79), control= 46 (13:33), PA= 34 (3:31), CPP= 15 (0:15): Teacher Report Form: n= 53 (7:46), control= 27 (6:21), PA= 20 (1:19), CPP= 6 (0:6).
- Self-perception: n= 99 (17: 82), control= 48 (13:35), PA= 35 (3:32), CPP= 16 (1:16).
- Intellectual Ability: n= 101 (18: 83), control= 50 (14:36), PA= 35 (3:32), CPP= 16 (1:15).
- Family Environment and Parental Stress: n= 97 (16: 81), control= 47 (12:35), PA= 35 (3:32), CPP= 15 (1:14).

3.2.2 Recruitment and Sampling. The patient groups were recruited from the BCH and UHCW. Families attending paediatric outpatient and medical day-care clinics were asked if they would be interested in taking part and given an information pack (Appendix 2). The pack included background information, ethical considerations, a full protocol and simplified protocol for participants, parental consent and participant assent forms. The pack also included a demographic questionnaire, contact details form (Appendix 3) and the pubertal development questionnaire. If the pack was not returned within 2 weeks, a telephone call was made to the family to determine whether they were interested in participating. Families in the existing endocrine patient cohort at the BCH who were considered eligible were also sent a letter of invitation from their consultant (Appendix 4) along with an information pack through the post.

Families of participants in the typically-developing group received a letter of invitation (Appendix 5) through a mail-shot of local primary schools. If families wished for more information, they provided their contact details and returned the letter to the school. They were then sent an information pack through the post, as described above. Control participants were offered a book voucher for their school in return for their participation.

The sample size was determined after considering rates of eligible patients attending clinics and those in the existing patient cohort. These figures were compared with those employed in current literature and the respective effect sizes. Power calculations were conducted using a mean value of the significant effect sizes obtained in relevant publications. The effect size calculations demonstrated that a sample size of 102 would give a medium effect size of 0.4 which is comparable to those in similar research

papers. When assuming an uptake rate of 50%, it was decided that a sample of approximately 100 participants would be sufficient.

3.3 Procedure and Measures.

3.3.1 Quantitative Data Collection. Data collection took place either in participants' homes or in the Wellcome Trust Clinical Research Facility at the BCH. Visits took approximately 3 hours, and families could split the session across two appointments if preferred. The study used several measures, each of which is described below. Some of the measures were completed by the participant's parents or carers and others by the participant with the help of the experimenter.

3.3.1.1 Pubertal Development. Parents completed a pubertal development questionnaire on their child's pubertal development (PDQ: Appendix 1) to ensure that they were eligible to take part in the project and that they were assigned to the correct group. There were two versions of the questionnaire; male and female. The questionnaire was made up of five-point rating scales for each secondary sexual characteristic, based on the Tanner stages regularly used in clinical settings (Tanner, 1973). The Tanner stages grade aspects of puberty from pre-pubertal to adult development, including pubic hair, breast and genital growth. These areas of growth are usually assessed and rated by a medical professional during a physical examination; however, this method of measurement is less commonly used in research, as it is often considered invasive and embarrassing for the participant, resulting in high attrition rates. Furthermore, many researchers would not be qualified to conduct these examinations, and would require the co-operation of a physician. Although the reliability of these clinician ratings has been questioned (Hergenroeder, Hill, Wong, Sangi-Haghpeykar & Taylor, 1999), the use of clinician ratings is considered to be optimal when measuring pubertal development (Brooks-Gunn,

Warren, Rosso & Gargiulo, 1987; Desmangles, Lappe, Lipaczewski & Haynatzki, 2006; Dorn, Dahl, Woodward & Biro, 2006; Schmitz et al., 2004).

Another method typically employed to measure pubertal development is that of questionnaires or pubertal development scales. These include either photographs or line-drawings of developmental stages, and are given as either a self- or parent-report format. Brooks-Gunn et al. (1987) report that during their research, only parent-report scales and interview techniques were approved by schools, with self-report questionnaires and physical examinations considered inappropriate. They also demonstrated that agreement between self-report, parent-report and clinician ratings of girls' pubertal development were highly consistent. Other authors, however, have concluded that self-report measures provide little reliable data (Desmangles et al., 2006; Hergenroeder et al., 1999), but are sufficiently robust to provide approximations of pubertal stage when physical examinations are not possible (Schmitz et al., 2004). It was decided that a parent-report scale would be used for the purpose of the current thesis, as the young age of the participants would mean that a self-report format would be inappropriate.

3.3.1.2 Body Composition. Height was measured to the nearest 0.5 cm using a Leicester Height Measure. Weight was measured to the nearest 0.1 kg and body mass index (BMI) to one decimal place, using Tanita WB-100 clinical floor scales. BMI is commonly used to compare individuals to the centiles given on standardised reference curves, to identify overweight and obesity in participants. However, there has been discussion as to whether reference tables should be updated with centiles based on more recent and diverse data. Furthermore, it has been questioned whether the 85^{th} and 95^{th} centiles used as thresholds for classifying overweight and obesity are arbitrary, and not necessarily related to the BMI at which clinical problems develop (Cole, Bellizzi, Flegal &

Dietz, 2000). However, these measurements are still commonly used to measure body composition in patient populations, both clinically and for research (Biro et al., 2001; Charkaluk, Trivin & Brauner, 2004; Golub et al., 2008; Ibáñez et al., 2003; Kaplowitz, Slora, Wasserman, Pedlow & Herman-Giddens, 2001). It was therefore considered appropriate to include them in the current project to identify whether any differences in body composition existed between groups. This would allow for comparison of the current sample to those in the literature, as well as with the standardised percentiles recommended for use in clinical settings.

3.3.1.3 Eating Behaviour. Due to the reported increased height and weight of the early puberty population (Biro et al., 2001; Charkaluk et al., 2004; Golub et al., 2008; Ibáñez et al., 2003; Kaplowitz, et al., 2001), and the increase in disordered eating behaviour during early puberty (Kaltiala-Heino, Marttunen, Rantanen & Rimpela, 2003; Kaltiala-Heino, Rimpel, Rissanen & Rantanen, 2001; Koff & Rierdan, 1993), it was decided that measures of eating behaviour should be included. This was to identify, firstly, whether differences in body composition were related to differences in eating behaviour between groups, and secondly, to assess whether there was an increased risk of disordered eating behaviour in the two patient groups. It was decided to include a modified version of the Dutch Eating Behavior Questionnaire for Children (DEBQ-C: van Strien & Oosterveld, 2008) and the Child Eating Behaviour Questionnaire (CEBQ: Wardle, Guthrie, Sanderson & Rapoport, 2001).

- *Dutch Eating Behavior Questionnaire for Children.* The Dutch Eating Behavior Questionnaire (van Strien, Frijters, Bergers & Defares, 1986; DEBQ) is a well-established questionnaire designed to measure dietary restraint, emotional disinhibition and external disinhibition of eating in adults. This questionnaire has been cited heavily in the literature

and has been translated for use in several countries (Lluch et al., 1996; Sung, Lee, Song, Lee & Lee, 2010; Thøgersen-Ntoumani, Ntoumanis, Barkoukis & Spray, 2009; Viana & Sinde, 2003; Wardle, 1987). Many studies investigating these eating behaviours in children have used a modified version of the DEBQ; the Dutch Eating Behavior Questionnaire for Children (DEBQ-C; van Strien & Oosterveld, 2008), designed for use with children aged 7-12 years. This measure employed a reduced response set, with a three-point rather than five-point Likert scale, and items were simplified to facilitate understanding. The authors confirmed that the model was suitable across age groups, genders and levels of BMI. Other researchers have used the DEBQ-C with even younger participants (Jahnke & Warschurger, 2008), and have translated the measure into several languages (Baños et al., 2011; Czaja, Rief & Hilbert, 2009). The DEBQ-C has been shown to have satisfactory levels of internal reliability both as a whole and as individual subscales, with reported Cronbach's alpha ranging from .69 to .95 (Anschutz, Kanters, van Strien, Vermulst & Engels, 2010; Baños et al., 2011; Jahnke & Warschburger, 2008; Nguyen-Rodriguez, McClain & Spruijt-Metz, 2010; Ouwens, Cebolla & van Strien, 2012; van Strien & Bazelier, 2007; van Strien & Oosterveld, 2008). Test-retest reliability scores ranged from .39 to .96 (Baños et al., 2011).

Despite reports of high reliability and validity when using the DEBQ-C with young children, van Strien and Oosterveld noted that some items were poorly understood by the younger participants, particularly those on the emotional eating scale. In this instance the researchers provided examples to further illustrate the question. It may be, however, that other participants did not voice their lack of understanding and guessed an answer which was not true of their eating behaviour.

The current thesis included a parent-completion format of the DEBQ-C, which has been used previously by other authors (Jahnke & Warschburger, 2008). This new questionnaire was piloted with parents of children aged 4-10 years, recruited from local primary schools, and its factorial structure analysed. Please see Appendix 6 for full details. The findings suggested that the modified DEBQ-C was suitable for assessing external and emotional eating behaviours, although items on the restrictive eating behaviour scale fell into two subscales of altered eating behaviour relating to health awareness, and restrictive eating to alter appearance. Items relating to disordered eating to manipulate appearance were retained, as this most closely reflected the subscale originally proposed by the authors. The final measure consisted of fourteen items; five emotional subscale items, six external subscale items, and three restrictive subscale items (Appendix 7). The scale was shown to have high internal reliability and sufficient factor loadings.

- *Child Eating Behaviour Questionnaire.* The Child Eating Behaviour Questionaire (CEBQ: Appendix 8) has also been regularly cited in the literature, either as a whole or as individual subscales. Furthermore, the scale has been used with children as young as 3 years old. The CEBQ is a 35-item parent-report measure consisting of both approach and avoidance eating behaviour subscales; Food responsiveness, food enjoyment, emotional over-eating, desire to drink, satiety responsiveness, slowness of eating, emotional under-eating and food fussiness. Answers are scored on a five-point rating scale. Cronbach's alpha values range from .57 to .92, and test-retest reliability scores between .52 and .87 (Jahnke & Warschburger, 2008; Santos et al., 2011; Sleddens, Kremers & Thijs, 2008; Spence, Carson, Casey & Boule, 2011; Svensson et al., 2011; Viana, Sinde & Saxton, 2008; Wardle et al., 2001). Several studies have reported similar factorial structures to the original structure (Santos et al., 2011; Sleddens et al., 2008; Svensson et

al., 2011; Viana et al., 2008). Other authors, however, have failed to replicate the original factorial structure and argue that subscales are susceptible to inter-correlation, or are not applicable to other cultures (Cao et al., 2012; Sparks & Radnitz, 2012). However, the CEBQ has proven to be reliable in different languages and across age-groups (Jahnke & Warschburger, 2008; Santos et al., 2011; Sleddens et al., 2008; Soussignan, Schaal, Boulanger, Gaillet & Jiang, 2012; Svensson et al., 2011; Viana et al., 2008). The current project used a sample similar to that originally used by the authors when constructing the measure, and difficulties with cultural biases were considered unlikely. It was therefore decided to incorporate the CEBQ in the project.

The modified DEBQ-C and CEBQ were therefore used to identify whether there were any differences in the eating behaviours between the two patient groups and the control group, and whether any differences were related to the scores on the body composition measures.

3.3.1.4 Body Image. In addition to eating behaviour, it was decided to assess body image and whether this was related to height and weight. There are several psychometrics available in the literature, which have been designed to assess body image. These include the Eating Disorders Inventory - Body Dissatisfaction Scale (EDI-BD: Garner, Olmstead & Olivy, 1983) and the The Body-Esteem Scale (BES: Mendelson & White, 1993; Mendelson, White & Mendelson, 1996; Mendelson, Mendelson & White, 2001). Despite being reported as having good internal reliability with children as young as 8 years (Mendelson & White, 1993; Mendelson et al., 1996, 2001; Wood, Becker & Thompson, 1996), the measures have been criticised as being too inter-correlated and lacking in validity (Lieberman, Gauvin, Bukowski & White, 2001; Smolak, 2004). They were therefore not considered suitable for use in the current study.

- *Pictorial Body Image Scales.* Pictorial figure rating scales are another method of assessing body image, and have been used both in the literature and in the media to study how an individual perceives their body in comparison to others. Pictorial scales consist of a series of figural drawings, ranging from a low body weight to a high body weight. Scales are made up of between five and ten figures, and are gender-specific. The participant is asked to choose which figure they perceive to be most similar to their own figure, and which they would prefer to look like. Each figure is assigned a ranking number, and recorded. Accuracy of perception can be assessed by correlating the self-perception to weight or BMI, and a discrepancy score can be calculated from the difference between the self and preferred figure scores, and used as a proxy measure of body image dissatisfaction. One commonly cited use of pictorial figure scales is that by Collins (1991). Test-retest scores for Collins' figure scales range between .59 and .76 (Collins, 1991; Vander Wal & Thelan, 2000). Additionally, pictorial body image scales are quick and easy to administer, and reduce cognitive load when studying a child population.

Criticisms of the pictorial body image scales, include a lack of realism in the drawings, an inadequate number of figures from which to choose a continuous variable rating, an inflation bias when presenting more favourable figures on the left-hand side of the scale, and inclusion of facial features or hair making the figures appear more similar to some ethnicities than others (Gardner, Friedman & Jackson, 1998; Nicholls, Orr, Okubo & Loftus, 2006; Thompson & Gray, 1995). Despite these limitations, pictorial body image scales are still widely used and are a quick data collection technique (Gardner & Brown, 2010). Furthermore, 95% of children are estimated to be accurate to a level within a 10% range in their body image perceptions, demonstrating that scales are a valid measure of the construct, as well as being practical (Gardner, Friedman & Jackson, 1999a).

To combat the limitations noted above, new body image scales were constructued for use in the current thesis, to assess body image in relation to both weight and physical maturity (Appendix 9). These scales were designed to measure the child's perception of their own body image, their perception of their friend's figure, and their preferred figure. These could then be compared to calculate levels of bodily dissatisfaction. The body image scales were piloted with 300 children aged between 4 and 10 years. For details of the pilot study, please see Appendix 9. It was found that children were able to use the body image scales easily, in order to make judgements about their body image, as well as their friends' and preferred figures.

3.3.1.5 Sleep, Behaviour and Psychological Wellbeing. Many research studies have reported a heightened prevalence of maladaptive behaviours or an increased risk of psychopathology during pubertal development (Angold, Costello & Worthman, 1998; Benjet & Hernández-Guzmán, 2002; Blumenthal et al., 2011; Cyranowski, Frank, Young & Shear, 2000; Ge, Brody, Conger & Simons, 2006; Hayward et al., 1997; Rapoport et al., 1997, 1999; Siegel, Yancey, Aneshensel & Schuler, 1999). It was decided, therefore, to include measures of psychological and behavioural problems in the thesis.

- *The Child Behavior Checklist.* The Child Behavior Checklist (CBCL: Achenbach, 1991; Appendix 10) is a concise screening measure completed by parents, commonly used in clinical settings to assess behavioural difficulties and social competencies in children and adolescents between the ages of 1-5 and 6-18. There are 113 short behavioural statements, which are scored 0, 1 or 2 depending on the relevance of the statement to the child. Items fall under eleven lower-order syndrome subscales, including thought problems, social problems and aggressive behaviour. These syndrome scale scores are used to calculate the total scale score and scores on two higher-order scales;

internalising and externalising behaviour. There are also four competency scales, which are combined to produce a total competence score.

The CBCL has been used repeatedly in research, and translated into 85 languages (Bérubé & Achenbach, 2010). It was cited in over 1000 published articles between 1983 and 1992 (Brown & Achenbach, 1992), with the 1991 manual having been cited over 8000 times since its publication, according to "GoogleScholar" (2013). The scale has been reported to have good internal reliability across scales, with alpha coefficients between .55 and .97 (Achenbach & Rescorla, 2001; Albores-Gallo et al., 2007; Braet et al., 2011; Dutra, Campbell & Westen, 2004; Ferdinand, 2008; Lacalle, Ezpeleta & Doménech, 2012; Nakamura, Ebesutani, Bernstein & Chorpita, 2009; Rescorla et al., 2007; Tyson, Teasley & Ryan, 2010) and test-retest reliability correlation coefficients ranging from .63-1.00 (Achenbach & Rescorla, 2001; Bilenberg, 1999; Leung et al., 2006). Confirmatory factor analyses have supported both the higher and lower order scales suggested by the original authors. For example, Ivanova et al. (2007a) tested the model with 58,051 participants across 30 different societies around the world, and reported a Root Mean Square Error of Approximation (RMSEA) between .03 and .05 for 29 of the 30 countries. This indicated a good fit of the data around the model, with the only exception being Ethiopia with an RMSEA of .06. Twenty-four of the countries had all significant item loadings on to the expected syndrome scale, again suggesting that the model was applicable to other cultures outside the US. However, other authors have failed to replicate the original factorial structure when using the measure across other participant cohorts (Bilenberg, 1999; Tyson et al., 2010). The majority of structures, however, have been largely consistent with that reported by Achenbach and colleagues, and as our sample was very similar to that used in the design of the measure, it was considered appropriate for use.

- *Sleep Problem Scale.* Several research studies have used the items related to sleep problems in the CBCL to devise a sleep subscale, in order to measure the prevalence of sleep problems in their sample (Friedman, Corley, Hewitt & Wright, 2009; Goodnight, Bates, Staples, Pettit & Dodge, 2007; Gregory, Van der Ende, Willis & Verhulst, 2008). It was decided that as the data would already be collected as part of the emotional and behavioural problems chapter, it may be useful to analyse these sleep items separately in order to identify whether problems with sleep were evident in those with early puberty. The various items related to sleep were therefore entered into a factor analysis, in order to assess which were best combined to produce a sleep problem subscale. For more details on this analysis, please see Appendix 12. The final subscale consisted of seven items, and demonstrated good internal reliability. This was therefore deemed suitable for use in this research study.

- *The Teacher Report Form.* The Teacher Report Form (TRF; Appendix 11) of the CBCL was also sent to the teachers of participants over the age of 6 years, to assess behaviour and academic performance at school. The structure of the TRF is similar to the CBCL, but with items falling into a seven subscale model, with an additional attention problem subscale further divided into hyperactivity-impulsivity and inattention. Higher-order scales are internalising, externalising and total behaviour problems, as in the CBCL. Reported internal reliability for the TRF ranges from .62 to .95, with test-retest performance between .52 and .90 (Achenbach & Rescorla, 2001; Liu et al., 2000). Correlational analyses have also demonstrated strong similarities between the factorial structure of the TRF in the original United States (US) sample and other cultures (Liu et al., 2000). In a cross-cultural psychometric analysis, Ivanova and colleagues (2007b) reported a factorial structure consistent with the original model in 16 of the 20 countries

they assessed. This factorial structure also maps onto the CBCL model, allowing for comparisons between behaviour at home and school. Tests of goodness of fit produced RMSEA scores between .03 and .08, which the authors considered as evidence of a good fit when using a cut-off of .08. Again, the TRF was constructed in, and standardised on, a similar population to the sample included in the current project, and it was therefore considered suitable for use. The CBCL and TRF were therefore included in the main battery of psychometrics.

3.3.1.6 Self-Perception and Self-Esteem. Puberty is a time of physical and emotional change, and it was decided to measure self-perception and self-esteem across several areas, including perceived age, physical appearance, physical competence, scholastic competence, and peer acceptance. These were measured using the Perceived Age Questionnaire (Appendix 15), and the Harter scales; the Pictorial Scale of Perceived Competence and Social Acceptance (Appendix 13) for participants aged 4-7 years, and the Self-Perception Profile for Children (Appendix 14) for those over 8 years.

- *The Pictorial Scale of Perceived Competence and Social Acceptance.* The Pictorial Scale of Perceived Competence and Social Acceptance (PSPCSA; Harter & Pike, 1984) is a scale for children aged 4-7 years, consisting of 24 items grouped into four subscales; family and peer acceptance, physical and academic competence, or two broader scales of perceived acceptance and competence. Scales are gender-specific. Each item consists of two opposing pictures, each with a small and a large circle presented beneath. The participant is read a statement such as, 'This girl is good at numbers, but this girl isn't very good at numbers'. The participant then makes two binary decisions; firstly which of the two pictures described in the statement they are most similar to, and secondly how similar they are to the picture they have chosen by selecting either the big or little circle.

Responses are scored from 1-4, with a high score corresponding to a high perceived competence or acceptance.

Reliability values for the lower-order subscales ranged from .43 to .9, and from .63 to .86 for the higher-order subscales. Total scale alpha coefficients were between .83 and .88 (Bart, Hajami & Bar-Haim, 2007; Cadieux, 1998; French & Mantzicopoulos, 2007; Harter & Pike, 1984; Mantzicopoulos, French & Maller, 2004). Factor analyses demonstrated item loadings between .22 and .72 on to the higher-order subscales (Harter & Pike, 1984; Mantzicopoulos et al., 2004), and between .37 and .96 on lower order subscales (Cadieux, 1998). French and Mantzicopoulos (2007) reported that a three-factor model was a better fit of the data, with the family acceptance subscale excluded. Cadieux (1998) also found strong support for a three-factor model, although they excluded the family acceptance subscale a priori. The PSPCSA has been used with a variety of participant groups, including children with learning disabilities, speech and language impairments and from low SES families (Cadieux, 1998; French & Mantzicopoulos, 2007; Rannard & Glenn, 2009). Although the PSPCSA is not recommended for making educational or clinical decisions regarding individual children, the measure is a useful tool for comparing differences in self-perception between groups, and has been cited 494 times in the research literature (Web of Knowledge, 2013a).

- *The Self-Perception Profile for Children.* The Self-Perception Profile for Children (SPPC: Harter, 1985) is a 36 item questionnaire for children over the age of 8 years, used to assess self-perception in six areas: Academic and physical competence, social acceptance, physical appearance, behavioural conduct and global self-worth. Questions are presented in the same format as the PSPCSA, with each item consisting of a positive and negative statement. The participant chooses which statement best describes

them, and then whether this statement is very much like them, or moderately like them. Scoring for each item ranges from 1-4, with higher scores denoting more positive levels of self-perception.

The SPPC has good internal consistency, with Cronbach's alphas between .59 and .92 (Boivin, Vitaro & Gagnon, 1992; Eapen, Naqvi & Al-Dhaheri, 2000; Harter, 1985; Muris, Meesters & Fijen, 2003; Schumann et al., 1999; Van Dongen-Melman, Koot & Verhulst, 1993). Test-retest *r* values ranged from .29 to .86 (Granleese & Joseph, 1994b; Van Dongen-Melman et al., 1993). Confirmatory factor analyses demonstrated stability across ages and cultures (Boivin et al., 1992; Eapen et al., 2000; Granleese & Joseph, 1994a; Muris et al., 2003). Reported item loadings ranged from .27 to .89, with the majority of items loading on to their intended factors (Boivin et al., 1992; Granleese & Joseph, 1994a; Muris et al., 2003; Van den Bergh & Van Ranst, 1998; Van Dongen-Melman et al., 1993). Other authors however, have reported differing structures when comparing children of different ethnic backgrounds (Schumann et al., 1999). These differences could not be attributed to factors such as parental education, and the authors concluded that caution should be taken when using the SPPC to assess self-esteem in non-Caucasian children. However, because the sample in the current project is taken from a predominantly Caucasian population, this was not considered to be problematic.

- *Perceived Age Questionnaire.* In addition to the domains of self-perception mentioned above, the participants' perceived age was also studied, using a perceived age questionnaire (PAQ). This was based on concerns previously raised by parents regarding their child looking older than their class-mates and the various events that had occurred as a result. Similar research in the literature refers to this concept as subjective age, and has been used not only in adolescence, but in older adult samples who perceive themselves as

younger than their chronological age. Subjective age is less concerned with physical perception, but the individual's subjective experience of feeling of a particular age. Research in adolescent samples found that they typically reported feeling older than their chronological age, and that higher subjective age was correlated with behaviours, such as aggression, rule-breaking behaviour, sexual behaviour and alcohol consumption (Arbeau, Galambos & Jansson, 2007; Hubley & Arim, 2012). This implies that the age which an individual perceives themselves to be, as well as their chronological age, is an important factor in determining behaviour.

Previous research investigating the individual's perceived level of physical development in comparison to their peers, rather than their subjective experience of age, has typically used a single item to assess perceived pubertal timing. Examples of this are, 'I look younger than most' compared to 'I look older than most', or 'Do you think your development is any earlier or later than others your age?' (Carter, Silverman & Jaccard, 2013; Dubas, Graber & Petersen, 1991; Siegel et al., 1999). However, this method gives simple nominal data, and therefore no measure of the degree to which a child perceives themselves to be advanced or behind in development. Children may also not be aware of how far their peers are through puberty, and there is no data on how the child feels about this discrepancy in perceived age.

Both parent- and child-report versions of the perceived age questionnaires were constructed, to gather data on whether other adults or children perceived the participating child as older or more developed than their peers, and how the participating parents and children felt about this. This was tested with a pilot population of 95 parents and 349 children, aged 4-10 years, to assess reliability and factorial structure. For full details on construction and analysis of the measures, please see Appendix 15. The findings suggested

that the PAQ was suitable for use in measuring whether a child is perceived as looking bigger or older than their friends, and whether they experience positive or negative consequences of looking older than their peers. The parent version was found to be highly reliable with a stable three-factor structure; whether the child looked bigger or older than their peers, whether they observed negative consequences associated with their child looking older, and whether they observed positive consequences associated with their child looking older. The same three factors were identified in the child participant data; however this model was not as stable as that for the parent data. The model was still considered suitable for use in a patient population, however, and both versions of the PAQ were included in the final analysis.

3.3.1.7 Intellectual Ability. Puberty is a time of great neurological and cognitive change (Giedd, 2004; Gogtay et al., 2004; Lenroot et al., 2007; Luna, Padmanabhan & O'Hearn, 2010), and it was therefore decided to include a measure of intellectual functioning in the current thesis. This would allow investigation as to whether any changes in intellectual functioning could be observed in relation to precocious pubertal development.

- *The British Ability Scales.* The British Ability Scales were introduced in 1978 (BAS: Elliott, Murray & Pearson) in response to the American Wechsler Intelligence Scales (Wechsler, 1949). The BAS produces a general conceptual ability score (GCA), as well as three cluster scores of verbal, non-verbal reasoning and spatial ability, for children aged 3-18 years. These are derived from the child's performance on core scales, such as quantitative reasoning, verbal similarities and pattern construction; diagnostic scales, such as number concepts, digit span tasks and speed of information processing; and

achievement scales of number, spelling and reading. These scores are then compared to the United Kingdom (UK) standardised data for the child's age group and ethnicity.

The third edition of the BAS (BAS3: Elliott & Smith, 2011) was used in the current project, due to its recent publication. Performance is compared to recently standardised data from the same population as the test sample, meaning that factors such as school curriculum, proportion of ethnic groups in the sample and pronunciation of test instructions and stimuli are likely to be more consistent than if the scale had originated earlier or from another country. Furthermore, percentiles are derived from recent UK-based data, and are therefore likely to be more accurate for the current sample. The BAS3 was standardised with 1480 children from ten locations around the UK. The sample was stratified to ensure that there were equal numbers of children in each demographic group, based on characteristics such as age, gender, area of residence, parental education and ethnicity (GL Assessment Limited, 2011).

A further advantage of using the BAS3 is that the child's GCA is based on subscale loadings of different weights, rather than giving each subscale an equal contribution to the global intelligence score. This prevents anomalous performance on one subscale from influencing the overall intelligence score too greatly (Elliott, 1997). Subscales are reliable as stand-alone measures, allowing the administrator to exclude tasks that they do not deem suitable or necessary. Furthermore, the early-years and school-age batteries overlap considerably, allowing for greater consistency when analysing data from different age groups. It was therefore considered that the BAS3 would be the most suitable measure of scholastic intelligence and cognitive functioning.

3.3.1.8 Family Environment and Parental Stress. Three questionnaires on family stress were also included in the data collection; a modified version of the Life Events

Questionnaire (Norbeck, 1984; adapted from the Life Events Scale: Sarason, Johnson & Siegel, 1978), Perceived Stress Scale (Cohen, Kamarck & Mermelstein, 1983) and the Impact of Event Questionnaire (Horowitz, Wilner & Alvarez, 1979). These measures were completed by parents, firstly, to assess whether having a child with a diagnosis of early puberty was related to increased parental stress, and secondly, to identify whether the occurrence of difficult life events was related to earlier pubertal development.

- *The Life Events Questionnaire.* The Life Events Questionnaire (LEQ: adapted from Norbeck, 1984; Appendix 16) is an 82-item scale, which measures the impact of life events which have occurred during the past year. Each applicable item is firstly determined as either being a good or bad event, and then the level of impact it has had on the participant is rated on a four-point Likert scale. Items cover a range of positive and negative events, such as relationship problems, events at work and changes in health. The questionnaire is scored to give a value for the number of positive events, negative events and total events. Test-retest reliability statistics have shown good levels of reliability, with coefficients between .78 and .83 (Norbeck, 1984; Norbeck & Anderson, 1989; Solomon, 2001). Cronbach's alpha ranges from .80 to .85 (Meadows-Oliver, Sadler, Swartz & Ryan-Krause, 2007; Norbeck, 1984).

- *The Perceived Stress Scale.* The Perceived Stress Scale (PSS: Cohen et al., 1983; Appendix 17) is a 14-item questionnaire used to assess how stressful an individual perceives their current life situation to be. Each item is made up of a question asking how often the participant has felt a certain way during the past month, for example, how often they felt nervous or stressed. The participant then rates the frequency of this on a five-point Likert scale. Reliability coefficients between .49 and .86 have been reported (Cohen et al., 1983; Ebrecht et al., 2004; Fogel, Albert, Schnabel, Ditkoff & Neugut, 2003; Golin

et al., 2002; Harris et al., 2006). Test-retest correlations were also adequate, with reported coefficients between .55 and .87 (Cohen et al., 1983; Harris et al., 2006).

- *The Impact of Event Scale.* The Impact of Event Scale (IES: Horowitz et al., 1979; Appendix 18) is a 15-item scale, which is used to measure the extent to which a difficult event has affected the participant. The scale includes items measuring intrusion of the event on the participant's life, such as, 'I thought about it when I didn't mean to', and avoidance items such as, 'I tried not to talk about it'. The frequency of each item is rated on a four-point Likert scale, and a mean score calculated to denote level of impact. Cronbach's alpha values between .70 and .90, and split-half reliability scores of .86 have been reported, suggesting a high level of internal consistency (Dyregrov & Matthiesen, 2008; Horowitz et al., 1979). Test-retest reliability correlations were between .79 and .89 (Horowitz et al., 1979). The measure is used with a variety of patient groups, including cardiac patient, parents of stillborn infants, and breast cancer patients (Bennett, Conway, Clatworthy, Brooke & Owen, 2001; Dyregrov & Matthiesen, 2008; Wenzel et al., 2000). It was therefore considered that a combination of the three scales would provide a suitable overview of the stress level of parents, to determine whether there was any association between a child's onset of pubertal development and parental stress.

3.3.2 Qualitative Data Collection. In addition to the main battery of quantitative psychological and behavioural measures, a subset of five of the participant's families in the patient groups took part in a further qualitative assessment (Demographics are given in Table 60). Families were asked if they would like to participate in an additional home visit after completing the quantative measures. Information on the qualitative stage had previously been provided in the initial information pack, and families were reminded of what to expect during the qualitative interviews. Of the families that

showed an interest in taking part, six were selected, based on their ease with which they discussed the condition, in order to ensure the best chance of obtaining highly detailed data. The aim of this qualitative data collection was to provide a more detailed insight into the experiences that participants and their families have had in relation to the child's diagnosis, including the process of seeking medical care, managing the disorder within and outside of the family, and how families and their children feel about the diagnosis. Through learning about these experiences, we can infer how a family manages with this change, how they reason with and understand the diagnosis, and the impact that it has had on the family. This allows for better understanding of any aspects of the diagnosis which families may find difficult, or areas where families would benefit from extra support, as well as potentially providing insights which may validate conceptual findings suggested in the quantitative data analysis.

Qualitative data collection and analysis were based on the methods used in Interpretative Phenomenological Analysis (IPA; Smith, Flowers & Larkin, 2009), as this approach allows the researcher to explore a participant's experiences and learn how they make sense of these experiences. This concept fit well with the intended outcomes of the interviews. IPA aims to provide a representative, yet interpretative, description of what is important to the participants, through in-depth accounts of the participant's experience and feelings on a topic. This is achieved through open-ended questioning and encouragement for the participant to discuss at length whatever they feel most pertinent. Through these detailed transcripts, the researcher should be able to identify areas of similarity and discrepancy between and across cases, and gradually be able to build a picture of the data, which can be used to understand the themes of significance in the participant's narration. IPA is not designed to produce generalisable findings, but to explore what is most

important to carefully selected individuals and to learn what is interesting about their experience.

Interviews were conducted at the families' homes. The content of the interviews was designed around issues and reoccurring themes that had arisen during informal conversations at CGF family meetings, as well as clinical observations during outpatient consultations at the BCH. Semi-structured parent interviews were conducted by a Masters of Research student in the School of Psychology at the University of Birmingham (see acknowledgements), as part of their course of study. Interviews consisted of open-ended questions focusing on topics such as parents' perception of their child, how they felt the disorder had affected their child and themselves as parents, how their relationship with their child had altered and how others perceive their child (See Appendix 19 for full list of interview questions). Questions were open-ended and of neutral wording to prevent biasing of parents' responses, and had previously been determined through discussion with supervisors and the thesis author. Parents were able to answer freely and in as much detail as they felt necessary. Interviews were recorded on a Dictaphone, transcribed, and then coded and analysed by the current author.

3.3.3 Summary of Measures. A summary of the measures used to assess each domain is provided in Table 2.

Table 2: *Summary of measures used to assess each domain, and the responder for each measure*

	Parent or Carer	Participant	Teacher
Pubertal Development	PDQ		
Body Composition		Height, Weight, BMI	
Eating Behaviour	DEBQ-C, CEBQ		
Body Image		Body Image Scales	
Sleep, Behaviour and Psychological Wellbeing	CBCL		TRF
Self-Perception and Self-Esteem	PAQ	PAQ, PSPCSA/ SPPC	
Intellectual Ability		BAS	
Family Environment and Stress	LEQ, PSS, IES		
Qualitative Data	Optional Interview	Optional Interview	

3.4 Aims and Hypotheses.

3.4.1 Aims and Objectives. The proposed study broadly aimed to expand on current research surrounding disorders of early pubertal development, and to build on the methodology previously employed in the literature. The main objective of the study was to identify a behavioural and psychological profile for children with a diagnosis of PA or CPP, and to determine whether there is a need for psychological intervention within a clinical setting, and educational support in a school setting. This included assessing eating behaviours, body image and body composition, self-perception, behavioural and psychological problems and intellectual ability. A further objective of the study was to look at the levels of stress in the parents and carers of children diagnosed with clinically early pubertal development, compared to the parents of typically-developing children. The conclusions from the study will also be used to provide information for families, to enable parents to prepare for the potential problems they might face as their child progresses through puberty, and help children with diagnoses of precocious puberty to understand why they may experience certain thoughts and emotions and how to manage them.

Due to the variability of findings observed in the current literature, and the number of novel measures being included in the present thesis, exploratory analyses were conducted in addition to hypothesis-driven analyses. Because the participant pubertal development data was suitable for both comparative and correlational analyses, this allowed for more in-depth and data-led analyses of puberty in relation to the other variables being measured. As a result, the empirical findings of each chapter of the thesis are presented in two sections; firstly the findings driven by previous observations reported in the research literature and resulting hypothesis-testing, and secondly, any findings of interest taken from more exploratory analyses of relationships within the data.

3.4.2 Hypotheses. The empirical hypotheses and areas of exploratory analysis for each chapter are presented below.

Chapter 4.1: Body Composition, Body Image, and Eating Behaviour. The experimental hypotheses for this chapter were that, firstly, children with a diagnosis of PA or CPP would have significantly greater height, weight and BMI values than children in the typically-developing group, and would be more likely to be classified as overweight or obese. Secondly, it was hypothesised that children in the PA and CPP groups would perceive themselves as looking fatter, and score greater for body dissatisfaction on the body image scales. Finally, it was hypothesised children in the PA and CPP groups would score significantly higher for disordered eating behaviours, specifically restrictive eating behaviour.

Exploratory analyses for this chapter aimed to identify whether puberty as a continuous variable was related to variables of body composition, and whether the nature of this relationship differed in the two patient groups. Furthermore, relationships between pubertal development, BMI, body dissatisfaction ratings and DEBQ-C and CEBQ disordered eating behaviour subscale scores were explored, both for the dataset as a whole, and for each individual group.

Chapter 4.2: Behaviour, Emotion and Sleep. The experimental hypotheses for this chapter were that children with a diagnosis of PA or CPP would score significantly higher than those in the typically-developing group, on CBCL subscales of anxiety, depression, and internalising and externalising problem behaviours. The two patient groups were also hypothesised to be more likely to score above borderline and clinical thresholds for these variables.

Exploratory analyses for this chapter looked to identify whether the groups scored any differently on the other behavioural and emotional subscales of the CBCL, and whether there were any significant differences between groups on subscale scores of the TRF. Comparisons were made between problem behaviours at school and at home. New thresholds were also constructed based on the distribution of the present sample, and whether the two patient groups were more likely to score above the new thresholds was analysed. Furthermore, pubertal development as a continuous variable was correlated against scores on the subscales of the CBCL and the TRF, to explore whether any of these behavioural or emotional difficulties increased as a child passes through puberty. Finally, scores on the newly-constructed sleep scale were analysed, to identify whether the two patient groups scored significantly higher for sleep problems than the typically-developing group, or whether puberty correlated with sleep problem scores.

Chapter 4.3: Self-Perception: Perceived Age and Self-Esteem. The experimental hypotheses being tested in chapter 4.3 were that the PA and CPP groups would show significantly lower scores of self-esteem, specifically bodily self-esteem and social acceptance. Puberty was also analysed against these self-perception variables in an exploratory correlational analysis. Furthermore, the newly-constructed perceived age questionnaires enabled exploration of whether children in the two patient groups perceived themselves as looking and being treated as older than did the typically-developing participants, and whether they experienced positive or negative consequences to this if so. The parent-completion version of this scale allowed for comparison of the children's perceptions to that of the parents'.

Chapter 4.4: Intellectual Ability. The empirical hypotheses for the intellectual ability chapter were that children in the PA group would score significantly lower on the

BAS III, specifically the verbal subscale, in comparison to the CPP and typically-developing groups. In addition, further exploratory analyses looked to identify whether the patient groups scored differently to the typically-developing group on the remaining subscales of the BAS III, and whether the continuous variable of pubertal development was related to performance on these subscales when controlling for factors, such as SES.

Chapter 4.5: Family Environment and Parental Stress. It was hypothesised in chapter 4.5, that children in the typically-developing group would be more likely to live in a household with both biological parents, compared to the two patient groups. Furthermore, the PA and CPP groups were predicted to be of a lower SES, and to score significantly higher on measures of parental stress and significant life events.

Exploratory analyses were also conducted to identify whether any associations existed between pubertal development, the type of care-givers and number of siblings living in the household and family SES. Furthermore, correlational and regression analyses were conducted to explore the relationships between pubertal development in the child, SES of the family, age of the child and measures of parental stress and family life events.

4. QUANTITATIVE DATA ANALYSIS AND RESULTS

4.1 Body Composition, Body Image, and Eating Behaviour.

Weight and body mass index (BMI) have been the focus of a great deal of recent research, and there are several publications in the literature suggesting a rise in the prevalence of both overweight and obesity in children, with some studies suggesting an increase of as much as 30% over the last 30 years, and in children as young as 3 years of age (Bundred, Kitchiner & Buchan, 2001; Flegal & Troiano, 2000; Kautiainen, Rimpelä, Vikat & Virtanen, 2002; Ogden & Carroll, 2010; Ogden et al., 2006; Reilly, Dorosty & Emmett, 1999; Stamatakis, Primatesta, Chinn, Rona & Falascheti, 2005; Troiano, Flegal, Kuczmarski, Campbell & Johnson, 1995; Wang & Lobstein, 2006; Zhang & Wang, 2013). Furthermore, Jollifee (2004) reported that not only is the prevalence of overweight increasing, but also the extent of this weight. This suggests that not only are more children overweight, but these overweight children are also much heavier than in previous generations.

Children who are overweight may be more likely to experience dissatisfaction with their body image. Research has shown that children as young as 6 years old are able to accurately and reliably judge which figure is most like their own body shape, and have a preference as to which body shape they like best (Collins, 1991; Gardner, Sorter & Friedman, 1997; Gualdi-Russo et al., 2008; Veron-Guidry & Williamson, 1996). At age 10 years, children already demonstrate a preference for thinner figures, and dissatisfaction when this differs from their own body image (Thelan, Powell, Lawrence & Kuhnert, 1992). Vander Wal and Thelan (2000) reported higher levels of bodily dissatisfaction in obese children than in children of a typical weight. Similarly, Gualdi-Russo et al. (2008) found that overweight or obese participants were more likely to report being dissatisfied

with their figure, with 74% of them wishing to be thinner, compared to 3% who wanted to be bigger. In a review, Ricciardelli and McCabe (2001) reported that 28-55% of girls and 17-30% of boys preferred a slimmer body size to their own, compared to 4-18% of girls and 13-48% of males who preferred a larger body size. Similarly, Maloney, McGuire, Daniels and Specker (1989) reported that nearly half of their sample of children aged 7-13 years wanted to be thinner, and 37% reported previously attempting to lose weight. If this is true for children across the population, an increase in the average weight is likely to increase the proportion of children who are dissatisfied with their weight, putting them at risk for developing unhealthy behaviours. Furthermore, children appear to be highly perceptive of body image and how others perceive body image. A review by Feldmann, Feldmann and Goodman (1988) reported that children as young as 6 years of age perceive heavier figures more negatively than average weight figures. This suggests that children with a higher BMI may not only be unhappy with their body image, but also concerned with how they are perceived by others.

Body composition and body image satisfaction are likely to be inter-related, and children may become motivated to manipulate their weight as a result of bodily dissatisfaction in the same way as do adults. Children seem aware of the relationship between eating and body shape, and evidence has shown surprisingly high numbers of children attempting to change their weight and, in turn, their bodily satisfaction. Furthermore, research has demonstrated that children have an awareness of dieting and exercise carried out by their parents (Flannery-Schroeder & Chrisler, 1996; Hill & Pallin, 1998). Smolak, Levine and Schermer (1999) observed that parents' weight loss strategies were positively related to their daughters' attempts at weight loss. This suggests that children are aware of weight ideals, and may learn, firstly, that lower body weights are

desirable, and secondly, that weight loss is a typical everyday experience. In fact, research by Hill and Palin (1998) found that 8 year olds recommended dieting and exercise when given a vignette about someone who 'feels fat'. Dieting therefore becomes an acceptable way of altering body image, and the evidence suggests that surprisingly high numbers of children control food intake to lose weight. For example, Maloney and colleagues (1989) found that one in eight children as young as 7 years old reported limiting their calorie intake in order to reduce their weight, and two in five children would exercise to achieve the same effect. Research by Carper, Orlet Fisher and Birch (2000) found that girls as young as 5 years reported disordered eating behaviours, with one third restricting their food intake. Similarly Flannery-Schroeder and Chrisler (1996) reported that as many as one in five first-graders responded positively to items regarding restrictive eating behaviour. This implies that not only is the weight of children increasing, but awareness of body image and disordered eating behaviour is also more prevalent in children than one might expect.

Disordered eating behaviours appear to be even more prevalent in children of a higher BMI. Research by Lamerz and colleagues (2005) found that children with a BMI over the 90th percentile were four times more likely than those under the 50th percentile to demonstrate binge-eating behaviours. Similarly, Decaluwé, Braet and Fairburn (2002) reported that over a third of their obese child and adolescent participants reported binge-eating, with one in six admitting a bingeing episode more than twice weekly, and several admitting purging behaviours in order to manipulate weight. Herpertz-Dahlmann et al. reported that 36% of overweight and 54% of obese adolescents in their sample scored above the recommended cut-off for eating disturbances, in contrast to 10% of severely underweight and 6% of underweight participants (Herpertz-Dahlmann, Wille, Hölling,

Vloet & Ravens-Sieberer, 2008). Furthermore, Vander Wal and Thelan (2000) reported significant differences between their obese and average-weight participants in dieting behaviour, restrictive eating, and anxiety over gaining weight. It would therefore appear that attention needs to be paid, not only to the eating behaviours of underweight children, but also to the overweight or obese.

Other disordered eating behaviours observed in young children include emotional and external eating behaviour. Emotional eating behaviour occurs when an individual eats in response to emotions, rather than hunger cues. The literature on emotional eating is primarily concerned with psychosomatic theory, and the observation that despite emotional states typically leading to loss of appetite, some individuals will increase their food intake due to a lack of awareness of hunger and satiety (Bruch, 1964), leading to overweight. This may stem from a common parenting technique of using food, typically unhealthy treat foods, to soothe or distract a child when they are upset. The child learns that food functions to improve mood, and they begin to associate foods with positive emotions. In contrast, external eating occurs when an individual responds to environmental cues, such as the smell of cooking or the amount of food on a plate, as a way of determining when and how much they should eat. Parents' use of treat foods as a method of rewarding their child, is a further example of how children may learn to attend to external cues instead of hunger and satiety signals (Birch, McPhee, Shoba, Steinberg & Krehbiel, 1987). These individuals may therefore be at a heightened risk of obesity. For example, Herpertz-Dahlmann and colleagues (2008) suggested that children in a high weight group experience a loss of internal control surrounding food, implying that external cues have a strong influence over their eating behaviour. Furthermore, Orlet Fisher, Rolls and Birch (2003) found that children who were given a larger portion of food would eat 15-25%

more than they would when they served themselves a portion. Carper et al. (2000) also found that three quarters of girls as young as 5 years old were reported to eat in response to external disinhibition. Furthermore, eating in the absence of hunger has been found to be predictive of weight status, when children were given free access to several high calorie snack foods (Orlet Fisher & Birch, 2002).

There is a well-established link between puberty and weight; however, the specific nature of this relationship is less clear. Research has shown that those of a higher weight are more likely to enter puberty at an earlier age (De Simone et al., 1995; Lee et al., 2007), and that those who are pubertal are of a higher BMI than those who are prepubertal (Adair & Gordon-Larsen, 2001; Anderson, Dallal & Must, 2003; Harlan, Harland & Grillo, 1980; Himes et al., 2004). This increase in weight, and resulting difference in size compared to peers, may lead the child to become dissatisfied with their body shape. In contrast, those of a low body weight may have delayed, or even halted pubertal development (Copeland, Sacks & Herzog, 1995; Warren, 1980, 1999). Other research on puberty and weight status has suggested a positive relationship between the hormone, leptin, and the onset of pubertal development, whereby an increase in leptin is permissive of pubertal onset but not directly causal (Apter, 2003; Garcia-Mayor et al., 1997; Hileman, Pierroz & Flier, 2000). Furthermore, rapid weight gain is often observed in children who move from a low socioeconomic environment to affluent conditions where food is readily available, and this weight gain has been associated with earlier onset of puberty (Proos, 2009; Teilmann, Pedersen, Skakkebæk & Jensen, 2006; Virdis et al., 1998). This risk of being of a greater weight may therefore lead to body dissatisfaction and disordered eating behaviour in puberty.

In addition to puberty being associated with greater weight gain, puberty has been identified as a risk factor for problematic eating behaviours and attitudes, such as dieting, weight concerns, a need to exercise and bulimic tendencies (Day et al., 2011; Kaltiala-Heino, Rimpel, Rissanen & Rantanen, 2001; Kaltiala-Heino, Marttunen, Rantanen & Rimpela, 2003; Koff & Rierden, 1993). Berger and colleagues (2009) reported that girls who began puberty earlier scored higher for disordered eating behaviour and maladaptive perceptions. Early-developing girls also perceived themselves as bigger and reported higher body dissatisfaction. When considering the evidence, we might posit that children with a diagnosis of clinically early puberty are at an increased risk for disordered eating behaviour and dissatisfaction with their body image, due to an increased prevalence of overweight and obesity in this group, as well as a tendency for disordered eating to manifest itself during puberty. The aim of this study therefore, was to assess whether children with disorders of premature pubertal development were more likely to have a larger body composition, and show higher levels of disordered eating behaviour or bodily dissatisfaction.

It was hypothesised that the two clinical groups would be significantly taller, heavier and of a higher BMI than in the control group, and would be more likely to be fall above standardised thresholds for overweight and obese. The premature adrenarche (PA) and central precocious puberty (CPP) groups would also be more likely to rate themselves as fatter than the control group, and score higher for body dissatisfaction. Finally, the two patient groups would show a different pattern of eating behaviours, specifically higher levels of restrictive eating behaviour. Exploratory analyses were also conducted to investigate the relationships between pubertal development, measures of body

composition, body dissatisfaction and disordered eating behaviours, and whether these relationships differed between groups.

Method. For a full description of the recruitment process, please see section 3.2. Participant demographics are presented in Table 1. Parents were asked to complete a demographic questionnaire and the pubertal development questionnaire, as well as a modified version of the Dutch Eating Behavior Questionnaire for Children (DEBQ-C), and the Child Eating Behaviour Questionnaire (CEBQ). For information on these measures, please see 3.3.1.3. The participants' heights were measured on a Leicester Height Measure to the nearest 0.5 centimetres, and weight taken on Tanita WB-100 clinical weighing scales to the nearest 0.1 kilograms. BMI was either measured using the weighing scales, or calculated using the formula weight/height^2. The percentiles for these measurements were determined using standardised percentile charts for each gender (Child Growth Foundation, 2009; Appendix 20). Participants also completed the pictorial body image scale consisting of five figures varying in weight, where they had to choose the figure that looked most like themselves, their friends and the figure they would prefer to have.

Analysis and Results. Tests of normality of distribution and homogeneity of variance were calculated prior to each analysis. Where these tests were significant, non-parametric tests were employed. No statistically significant differences were observed between the three groups for age, SES and ethnicity.

- *Body Composition- Hypothesis Testing.* The experimental hypotheses for the body composition data were tested first. Descriptive statistics are given in Table 4. A Kruskal-Wallis comparison of the body composition measures between the three groups demonstrated that there was a significant difference in height ($H(2)= 11.61$, $p= .003$), weight ($H(2)= 11.47$, $p= .003$) and BMI ($H(2)= 7.15$, $p= .028$). Post-hoc analyses with a

Bonferroni correction and adjusted alpha level of .017 demonstrated a significant difference between the control group and CPP group for height (U(48, 15)= 169.50, z= -3.08, p= .002) and weight (U(48, 15)= 172.00, z= -3.03, p= .002), and approaching significance for BMI (U(48, 15)= 213.50, z= -2.39, p= .018). Differences were not significant for other group comparisons.

Table 3: *Means and standard deviations for each group on the body composition measures: height, weight and BMI*

	Height (cm)	Weight (kg)	BMI
Control	131.51 (11.09)	31.68 (9.86)	17.89 (3.02)
PA	136.81 (11.22)	36.69 (11.10)	19.13 (3.42)
CPP	142.24 (10.45)	42.98 (12.45)	20.81 (3.98)

Participants were then coded as overweight or not overweight, with a cut-off for overweight as falling on the 91st percentile or higher. Chi-squared analyses found a significant association between group and whether a participant was classified as overweight (X^2(2, 98)= 6.90, p= .032), where control participants were least likely to be overweight and the CPP group most likely (Table 4).

Participants were then coded as obese or not obese, with a cut-off for obese as falling on the 98th percentile or higher. Chi-squared analyses showed no significant association between group and whether a participant was classified as obese (X^2(2, 98)= 4.57, p= ns), however, there did seem to be a far higher proportion of the clinical groups falling in the obese category in comparison to the control group, with nearly half of CPP patients classified as obese (Table 4).

Table 4: *Proportion of each group falling in the overweight compared to not overweight categories, and obese compared to not obese categories*

	Overweight		Obese	
	No	Yes	No	Yes
Control	75%	25%	85%	15%
PA	57%	43%	74%	26%
CPP	40%	60%	60%	40%

- *Body Composition- Exploratory Analysis.* Exploratory analysis of the body composition data was then conducted. Jonckheere-Terpstra analyses found a significant trend across the three experimental groups for all three body composition measures; height (J= 1968.0, z= 3.41, p= .001), weight (J= 1968.0, z= 3.41, p= .001) and BMI (J= 1863.5, z= 2.70, p= .007), suggesting a positive relationship between pubertal development and body composition. Spearman's correlational analyses across the three groups found significant relationships between pubertal development, as assessed by mean Tanner score, and height (r= .38, p< .001), weight (r= .51, p< .001) and BMI (r= .37, p< .001). However, when groups were analysed separately these significant associations were not evident in the CPP group, and BMI was no longer significantly related to puberty in the control group. Relationships were maintained in the PA group (Table 5). When this correlation was repeated whilst partialling out the effects of age, these associations with pubertal development all became non-significant, except for weight in the PA group (r= .38, p= .025).

Table 5: *Spearman's correlations for each group, between pubertal development and the three measures of body composition*

	Height	Weight	BMI
Control	.45**	.39**	.15
PA	.50**	.55**	.43*
CPP	.12	.07	.27

Note: *p< .05 **p< .01 ***p< .001

Eating Behaviour- Hypothesis Testing. Analysis of the scores on the DEBQ-C and CEBQ was conducted to assess any differences in eating behaviour between groups. Descriptive statistics are given in Tables 6 and 7. Kruskal-Wallis analyses identified no significant differences between the three groups on any of the DEBQ-C subscales, Similarly, no significant differences were found on the CEBQ subscales, except for the satiety subscale (H(2)= 8.18, p= .017), where the PA group showed lower levels of satiety than the control (U(47, 35)= 560.00, z= -2.47, p= .013) and CPP groups (U(35, 15)= 154.50, z= -2.30, p= .021).

Table 6: *Means and standard deviations for each group on the three DEBQ-C subscales*

	External	Emotional	Restrictive
Control	.90 (.54)	.12 (.27)	.11 (.39)
PA	.86 (.60)	.24 (.44)	.19 (.44)
CPP	.81 (.64)	.33 (.58)	.27 (.46)

When the PA and CPP groups were collapsed into one clinical group, Mann-Whitney analyses demonstrated a significant difference between the clinical group and

controls on the restrictive eating subscale of the DEBQ-C ($U(47, 50)= 984.0$, $z= -2.08$, $p= .037$), in that the combined clinical group showed significantly higher levels of restrictive eating. No significant differences were identified on the CEBQ.

Table 7: *Means and standard deviations for each group on the CEBQ subscales*

	Food Responsiveness	Emotional Over-eating	Food Enjoyment	Desire to Drink	Satiety	Slowness of Eating	Emotional Under-eating	Food Fussiness
Control	1.56 (.94)	.73 (.61)	2.90 (.74)	1.35 (.87)	1.70 (.71)	1.64 (.89)	1.34 (.67)	1.64 (.84)
PA	1.85 (1.21)	.94 (.88)	2.85 (.87)	1.47 (.83)	1.31 (.75)	1.30 (.98)	1.16 (.71)	1.53 (.86)
CPP	1.75 (1.20)	1.15 (1.08)	2.55 (1.07)	1.38 (1.41)	1.88 (.79)	1.80 (.96)	1.37 (.77)	2.09 (1.12)

Eating Behaviour- Exploratory Analysis. Exploratory analysis of the eating behaviour data was then conducted. Jonckheere-Terpstra analyses identified a significant trend across the three groups for the restrictive eating behaviour subscale of the DEBQ-C, with controls showing the lowest restrictive eating behaviour, and the CPP group the highest ($J= 1656.5$, $z= 2.26$, $p= .024$). Spearman's correlational analyses between the mean Tanner rating and DEBQ-C subscale scores across all three groups, suggested weak positive relationships between pubertal development and both emotional ($r= .31$, $p= .002$) and restrictive ($r= .27$, $p= .007$) eating behaviours. When looking at these relationships within each participant group, the relationship between pubertal development and emotional eating behaviour remained in the control and PA groups, but was not evident in the CPP group. The relationship was no longer significant between Tanner rating and restrictive eating for any group, however, the control group did display a weak association with external eating (Table 8). Correlational analyses for the whole sample between BMI and the DEBQ-C showed moderate significant associations between BMI and all three subscales (External: $r= .32$, $p= .001$; emotional: $r= .37$, $p< .001$; restrictive: $r= .35$, $p< .001$). These relationships were maintained in the control group when analysing each group individually, and strengthened in the PA group. However, there was no significant relationship between BMI and the DEBQ-C subscales for the CPP group (Table 8).

Table 8: *Spearman's correlations between BMI and pubertal development, and the subscales of the DEBQ-C*

		External	Emotional	Restrictive
Control	Puberty	.32*	.38**	.15
	BMI	.30*	.36*	.33*
PA	Puberty	.19	.45**	.21
	BMI	.46**	.46**	.35*
CPP	Puberty	-.43	-.19	.17
	BMI	.20	.13	.10

Note: *p< .05 **p< .01 ***p< .001

Analysis between mean Tanner stage and CEBQ subscale scores presented with a similarly complex outcome. Pubertal ratings for the whole sample were only weakly associated with reduced satiety, with all other relationships non-significant. However, group by group analysis showed slightly different eating behaviour patterns per group. Control participants showed a significant moderately strong relationship between pubertal development and enjoyment of food, but no other significant associations. Similarly, the CPP group showed increased food fussiness as they progressed through puberty, but no other significant eating behaviours. The PA group, however, showed an increased responsiveness to food, increased food enjoyment and reduced satiety which may explain the increased weight gain in this group (See Table 9). In the CEBQ correlational analysis with BMI, several associations were identified between BMI and eating behaviours within the entire sample, including food responsiveness, emotional over-eating, food enjoyment, and negative relationships with satiety and slowness of eating. In the between group analysis, each of these associations was strengthened for the PA group. The control group

maintained significant relationships between food responsiveness, emotional over-eating and satiety, but the CPP group only demonstrated a significant negative relationship with satiety (See Table 9).

Table 9: *Spearman's correlations between BMI and pubertal development, and the subscales of the CEBQ*

		Responsive to Food	Emotional Over-eating	Food Enjoyment	Desire to Drink	Satiety	Slowness of Eating	Emotional Under-eating	Fussiness
All	Puberty	.19	.19	.13	.15	-.24*	-.14	-.29	.04
	BMI	.48***	.42***	.33**	.15	-.46***	-.30**	.00	-.17
Control	Puberty	.19	.20	.40**	.24	-.22	-.10	.16	-.11
	BMI	.35*	.30*	.26	.22	-.44**	-.16	.01	-.31*
PA	Puberty	.42*	.32	.40*	.23	-.37*	-.21	-.06	-.09
	BMI	.66***	.50**	.50**	.05	-.50**	-.44**	-.03	-.12
CPP	Puberty	-.12	-.23	-.42	.11	.19	-.01	-.49	.53*
	BMI	.39	.45	.34	-.16	-.68**	-.53	.07	-.18

Note: *p< .05 **p< .01 ***p< .001

Body Image Satisfaction- Hypothesis Testing. The data from the pictorial body image scales were compared between groups. Descriptive statistics for each group and for each figure rating are given in Table 10. The figures were scored from one to five, where one was the thinnest figure, and five the fattest.

Table 10: *Means and standard deviations for the body image ratings on the weight scale*

	Self Figure Rating	Friends' Figure Rating	Preferred Figure Rating
Control	2.96 (1.25)	3.05 (1.28)	2.91 (1.37)
PA	3.04 (1.18)	2.65 (1.09)	2.27 (1.12)
CPP	3.37 (1.22)	2.53 (1.04)	2.85 (1.20)

Kruskal-Wallis analyses of body image scores, as measured on the weight pictorial body image scales, identified no significant differences between groups in the figure ratings for self-perception, perception of friends or preferred figure. Chi-squared analyses found no significant associations between group and whether participants rated themselves as looking fatter, the same size or thinner than their friends, but there was a significant association between groups on participants' comparison between self and preferred figure ($X^2(4, 98)= 11.14$, $p= .025$). From the proportion of each group falling into each category, it could be seen that the clinical groups were twice as likely to perceive themselves as fatter than their preferred figure (Table 11).

Table 11: *Proportion of each group who reported perceiving themselves as thinner, fatter or the same size as their preferred figure*

	Self-Rating Compared to Preferred Figure		
	Thinner	Same Size	Fatter
Control	27%	42%	31%
PA	14%	23%	63%
CPP	20%	13%	67%

The differences between the participants' self-rating and their friends' and preferred figure ratings were then calculated to produce weight comparison ratings. These were calculated by taking the friends' and preferred figure ratings away from the self-rating. This took into account the direction of the difference between the perceived own figure and that of the friends' or preferred, with a negative value denoting that the participant perceived themselves as thinner than the comparison figure, and a positive value as fatter. A Kruskal-Wallis analysis between groups of the weight comparison ratings identified a significant difference between groups for the comparison between perceived own figure and preferred figure ($H(2)= 6.48$, $p= .039$). Mann-Whitney analyses with a Bonferroni correction demonstrated that the significant difference lay between the control and PA groups, with the PA group perceiving themselves as bigger than their preferred figure to a greater extent than did the control group ($U(48, 35)= 584.5$, $z= -2.42$, $p= .016$) (See Table 12).

When the direction of the difference was discounted, and the magnitude of the difference between the chosen own figure and that of the friends' or preferred figures was analysed irrespective of whether this was thinner or fatter, it was found that there was a

significant difference between groups for the difference between perceived friends' figure and own figure (H(2)= 6.19, p= .045), with the difference lying between the control and PA groups. However, this difference was non-significant after Bonferroni correction of the p-value (U(48, 35)= 596.5, z= -2.30, p= .022).

Table 12: *Means and standard deviations for the comparison ratings and magnitude of differences, for the comparison between self and friends' figures, and self and preferred figures.*

	Self-Friends		Self-Preferred	
	Comparison Rating	Magnitude of Difference	Comparison Rating	Magnitude of Difference
Control	-.09 (1.9)	1.5 (1.1)	.05 (1.6)	1.1 (1.1)
PA	.39 (1.4)	1.0 (.9)	.83 (1.6)	1.4 (1.2)
CPP	.83 (1.9)	1.6 (1.2)	.52 (1.8)	1.5 (1.1)

Body Image Satisfaction- Exploratory Analysis. Exploratory analysis of body image data was then conducted. Jonckheere-Terpstra analyses identified no significant trends across the three groups for the participants' rating of their own size or their preferred size on the body image scales, although there was a near significant trend in the perception of friends' size (J= 1185.5, z= -1.90, p= .058), whereby the CPP group rated their friends as thinner than did the PA and control groups. Trend analyses for the differences between self ratings, and ratings for the friends' or preferred figures found a significant trend across groups for the comparison between the perceived own figure and preferred figure (J= 1804.5, z= 2.35, p= .019), and a near significant trend for perceived own figure compared to perceived friends' figures (J= 1745.0, z= 1.92, p= .055). In both of

these comparisons, the clinical groups perceived themselves as bigger than their friends' and preferred figures to a greater extent than did the control group.

Spearman's correlational analyses for the whole dataset demonstrated significant relationships between BMI and both the perceived comparisons between the self-rating, and friends' and preferred figure ratings, as well as the magnitude of the difference between these ratings. When these relationships were analysed between groups, they were only maintained in the PA group (Table 13).

Table 13: *Spearman's correlations between BMI and the comparison ratings and magnitude of differences, for comparisons between the self-figure rating, and the friends' and preferred figure ratings*

	Self-Friends		Self-Preferred	
	Comparison Rating	Magnitude of Difference	Comparison Rating	Magnitude of Difference
All	.34**	.24*	.45***	.31**
Control	.17	.14	.25	.26
PA	.48**	.39*	.61***	.46**
CPP	.29	.50	.36	-.01

Note: *p< .05 **p< .01 ***p< .001

Discussion. This study aimed to identify whether having a diagnosis of precocious pubertal development has an impact on body composition, eating behaviours or perception of body image. The analysis supported the evidence for an increased risk of being taller, heavier and overweight in children with clinically early pubertal development. Children with a diagnosis of CPP were of a greater body composition than the control group, with the PA group falling inbetween, suggesting that body composition increases alongside

pubertal development. However, there was also a significant association between group and whether a child was classified as overweight, suggesting that although children typically grow in height and weight as they progress through puberty, they may not become overweight, as observed in the two patient groups.

The analysis showed that a quarter of the control group were overweight, which is considerably higher than the 9% presented on standardised percentile charts, suggesting that our control group were slightly heavier than the normal population. However, this proportion of overweight in the control group is far lower in comparison to the 43% in the PA group and the 60% in the CPP group. Furthermore, although not statistically significant, almost half of the CPP group were classified as obese, in comparison to 15% of controls. This implies a higher risk of overweight and obesity in those with diagnoses of clinically early puberty.

Correlational analysis of the whole sample also suggests that further development through puberty is related to increased body composition, although considering that puberty and age are highly related this finding is to be expected. What is interesting, however, is the way in which this relationship changes when analysing the groups individually. The PA group grow more rapidly and to a greater extent as they progress through puberty in comparison to the control group, demonstrated by the significant positive correlation between Tanner rating and BMI. This suggests that the PA group gain weight to a greater extent than is typically observed during puberty, causing them to become overweight or obese. The CPP group, however, do not appear to grow in height and weight in relation to their pubertal development, despite demonstrating higher values for all body composition measures. This implies that these patients grow in height, weight and BMI prior to, or early on, in their pubertal development, and therefore no relationship

between pubertal progression and growth is observed. This weight gain in the very earliest stage of puberty may explain why researchers have put forward the theory that increases in weight are causal in pubertal onset.

The relationship between puberty and eating behaviour appears to be more complex. There seems to be little difference between groups in eating behaviour, although the PA group presented with lower awareness of satiety signals than the other groups, and there was a trend for the clinical groups to demonstrate more restrictive eating. When looking at the correlational data for each group, the typically-developing participants showed a relationship between greater pubertal development and increased food enjoyment, whereas the CPP group increased in food fussiness as they progressed through puberty. This, in combination with increased restrictive eating, may reflect attempts to manipulate weight in this group. The PA group, however, demonstrated increased responsiveness to external food cues, increased enjoyment of food, and reduced satiety as they progressed through puberty. This combination of behaviours may be related to their greater increase in BMI.

When analysing the relationship between BMI and eating behaviour, it was found that all three subscales of the DEBQ-C were positively correlated with BMI, with the PA group demonstrating the strongest associations, and the control group almost as strong. The CPP group, however, demonstrated no associations between eating behaviour and BMI, again suggesting that the increased weight in this group is not necessarily related to disordered eating behaviours. Similarly, in the CEBQ data analysis, relationships were evident in the control and PA groups between BMI and food responsiveness, emotional over-eating, and negatively with satiety, and additionally food enjoyment and speed of eating in the PA group. It may be that those of a higher weight become more preoccupied

with their relationship with food, or that their parents restrict food in order to manage their weight. This would result in many of the behaviours described above, such as eating quickly and without recognising satiety, as soon as food becomes available. The CPP group, however, only showed a negative relationship between BMI and satiety, which may again suggest eating food when it is available, rather than when hungry. Several parents reported trying to limit their child's food intake in the patient groups, and this therefore may account for these disordered eating behaviours. It would appear that having a larger BMI is related to increased amount and speed of eating as a result of environmental and emotional cues, as well as reduced awareness of satiety cues in typically-developing children and to a greater extent those with a diagnosis of PA. It is unclear whether these behaviours precede the increase in BMI, particularly in the PA group, or whether there is an additional factor such as an impending growth spurt, that influences weight gain as well as eating behaviour. In the CPP group these tendencies were not evident, suggesting that the higher BMI in this group may also be related to an additional factor, such as hormonal changes.

Body image analysis suggested that the two clinical groups did not perceive themselves as any fatter than the control group. Furthermore, there was little difference in how participants perceived their friends' figures or the figure they chose as their preferred body shape, although there was a tendency for the CPP group to perceive their friends as thinner than did the control group. There was also a significant association between group and whether participants perceived their body shape as thinner, the same size or fatter than their friends' figures or preferred figures, in that participants in the PA and CPP groups were twice as likely to rate themselves as fatter than their preferred figure in comparison to the control group.

When looking at the BMI and body-image comparison correlation data in each group, the PA group perceived a bigger difference between their own body-image and their friends' or preferred figures than did the control and CPP groups, suggesting that children with a higher BMI in the PA group are more aware of the difference between their own figure and their friends' or ideal figures. Considering that children with CPP tend to be heavier than those with PA, one might expect these participants to be more aware of differences between their body shape and that of their friends, particularly as there was a tendency for this group to perceive their friends as thinner than the other groups. This implies that children with a diagnosis of CPP are not necessarily as concerned with their larger size, in comparison to the PA group.

To summarise the findings, children with clinically early puberty are taller and heavier than their peers, but this increased size affects the two clinical groups differently. Children in the PA group with higher Tanner ratings showed higher levels of disordered eating behaviour, by eating more in response to external and emotional cues and showing reduced satiety. Those with a higher BMI were also more aware of the discrepancy between their body shape and that of their friends' or preferred body shape. Children with a diagnosis of CPP, however, showed increased fussiness but reduced satiety, and did not show the same awareness of their size in comparison to their friends' or preferred figures. When looking at the sample as a whole, children are more likely to develop disordered eating behaviours as they progress through puberty, which is consistent with the literature. Furthermore, there were significant relationships between BMI and several eating behaviours, again supporting the literature which suggests that those of a higher weight are at an increased risk of disordered eating behaviours and altered body image.

This study demonstrates the complex relationship between eating behaviour, body image, BMI and pubertal development. A strength of the study is the variety of measures employed, allowing for a more in-depth understanding of how these variables are related, and how changes in biological factors such as puberty or weight gain can be related to changes in eating behaviour and bodily satisfaction. Furthermore, the inclusion of two puberty groups allowed for the inference that it is not simply being taller, or looking differently to peers that is related to body image and eating behaviour, as the PA and CPP groups perform very differently. From this we can assume that the different biological profiles of the two groups have some influence on participants' relationship with food and the way each participant perceives their body in comparison with others'. However, there are several limitations to this project. One such limitation is the difference in group sizes. Because the CPP group was considerably smaller than the PA and control groups, it may be that other individuals in this population who did not participate in the project may have performed more similarly to the PA or control groups. This may be because families of children with CPP could have more concerns over their child's eating behaviour and therefore felt uncomfortable in taking part, which would explain the smaller group size. A further limitation of the study is the dichotomous nature with which overweight and obesity was coded. As Jolliffe (2004) points out, the extent to which a child is overweight is important and should be considered in the analysis. For example, Decaluwe et al. (2002) included an adjusted BMI in their analysis to code for the extent to which their participant was overweight, which then allowed them to identify not only differences in prevalence of overweight, but also the extent of each participant's weight. This may demonstrate that a child who is on the borderline of being classified as overweight performs very differently to a child who is morbidly obese. Future research should consider this method in the early

puberty population, as it may be that those with clinically early pubertal development are overweight to a greater extent to those who are overweight in the typical population. Furthermore, it may be that those who are more overweight also show a different eating behaviour profile, or an altered level of body image satisfaction.

4.2 Behaviour, Emotion and Sleep.

Adolescents are commonly perceived as 'moody' and temperamental, and the transition from childhood to adulthood has long been considered a time of emotional instability and behavioural impulsivity, as early as the works of Rousseau (1762/ 2009) and Shakespeare (1623). The phrase, "storm and stress" was coined by Hall in 1904, and has been cited ever since. This phrase summarises the typical perception of adolescence, and Hall wrote of adolescents reliving historic troubles through the heritable memories of previous generations, and hormonal awakenings leading to unacceptable sexual feelings. Although we now understand that memories are not passed from one generation to the next, Hall's description of adolescent problem behaviour and resistance to authority is still considered applicable today. The strength of this perception of adolescence is so pervasive that Freud believed an absence of this stressful presentation was indicative of underlying psychological problems (Freud, 1958; 1968).

Another suggested universal basis for adolescent difficulties is hormonal fluctuations at the onset of puberty. Buchanen, Eccles and Becker (1992) suggest that this influences emotion and behaviour through three mechanisms; activation effects, where a change in hormone levels directly activates changes in emotion and behaviour; adjustment effects where becoming accustomed to the initial changes is more problematic than changes occurring further through development; and finally initial irregularity in hormone levels at pubertal onset, which gradually settle into a more stable rhythm as puberty progresses. The authors also suggest that these three mechanisms are likely to interact with each other, as well as with individual factors such as cell receptor sensitivity, the child's environment and velocity of pubertal development. Studies have reported relationships between hormones released during puberty and various measures of emotion and affect,

for example, Susman et al. (1985) observed increased negative affect in males with greater adrenal androgens, and higher scores for psychopathology in females with increased gonadotropins. Furthermore, Angold, Costello, Erkanli and Worthman (1999) found that androgen and oestrogen levels were predictive of depression, to a greater extent than pubertal status. This suggests that the endocrinological aspect of puberty influences emotion and psychological wellbeing during adolescence.

Other research has posited neuro-endocrinological bases for adolescent changes. It is widely accepted that the hormones implicated in puberty affect neurological function, either through activational or organisational effects (Cameron, 2001). The hypothalamus and its connecting structures contain a high number of both oestrogen and androgen receptors (Doncarlos, Monroy & Morrell, 1991; Fernández-Guasti, Kruijver, Fodor & Swaab, 2000; Simerly, Chang, Muramatsu & Swanson, 1990), suggesting a function for the binding of hormones to these receptor sites. Furthermore, several sexually dichotomous changes occur as a result of sex steroid influence, both neurologically and behaviourally (Cameron, 2001; Collaer & Hines, 1995; Cooke, Hegstrom, Villeneuve & Breedlove, 1998; Matsumoto, 1991; McEwen & Alves, 1999). It may therefore be that there is a link between these neurological changes and changes in observable behaviour.

It has also been suggested that the dopaminergic pathways in the limbic and ventral striatal areas form a socio-emotional system, implicated in impulsivity and response to reward. This develops over mid-adolescence, and results in increased attraction to potentially hazardous, yet highly rewarding behaviours during this time (Casey & Jones, 2010; Steinberg, 2004, 2007). Alternatively, Ernst and colleagues suggested that the amygdala is less developed in this age group, and that this impairs adolescents' motivation to avoid dangerous situations and behaviours (Ernst, Pine & Hardin, 2008).

In contrast to universal biological bases for adolescent difficulties, research has studied the influence of external factors such as family and school environments, or individual factors such as self-esteem or natural temperament (Booth, Johnson, Granger, Crouter & McHale, 2003; Forehand et al., 1990; Leve, Kim & Pears, 2005; Lohman & Jarvis, 2000; Patton et al., 2008; Simmons, Blyth, van Cleave & Bush, 1979). This approach would suggest that individuals may all present slightly differently as they progress through adolescence, and this does appear to be the case. Dahl suggests that only 20% of individuals experience significant emotional and behavioural difficulties during adolescence, but also reports that rates of mortality or ill health double during this time, commonly in relation to psychological problems, such as substance misuse, self-harm or disordered eating (Dahl, 2004). Puberty therefore does seem to be a high-risk time for individuals, and there is agreement that changes in emotion, behaviour and cognition are evident in adolescents to varying degrees, but these difficulties are not universal and should not be over-generalised.

It is argued that increases in emotional experience during puberty can influence ability to reason and make logical choices about appropriate behaviour (Dahl, 2004; Loewenstein & Lerner, 2003; Steinberg, 2004). In addition to the type of emotions experienced, it has been suggested that the intensity of the emotions is heightened, leading to a stronger impact of these emotions (Dahl & Gunnar, 2009). Peper and Dahl (2012) describe how this heightened stress response may be due to an increased sensitivity to the environment. This “experience-expectant” period reflects changes in the prefrontal cortex, and functions to facilitate learning, in order to reach the social competencies of adulthood (Crone & Dahl, 2012). However this can also lead to maladaptive changes in behaviour, cognition and emotional control, such as displaying argumentative and antisocial

behaviour, or behaving in a risky and sensation-seeking manner (Arnett, 1999; Bauman & Phongsavan, 1999; Dahl, 2004; Moffitt, 1993; Young et al., 2002). Additionally, puberty has been identified as a trigger for the onset of psychopathologies, including depression, phobia, eating disorders, social anxiety, conduct disorder and attention disorders (Angold, Costello & Worthman, 1998; Benjet & Hernández-Guzmán, 2002; Blumenthal et al., 2011; Cyranowski, Frank, Young & Shear, 2000; Ge, Brody, Conger & Simons, 2006; Hayward et al., 1997; Rapoport et al., 1997, 1999; Siegel, Yancey, Aneshensel & Schuler, 1999). However, it seems that there is an interaction between puberty, genetic predispositions, environmental changes, and neurological and endocrine developments, each of which impact on psychological health, rather than puberty being a direct cause of psychopathology (Walker & Bollini, 2002; Walker, Sabuwalla & Huot, 2004). Furthermore, other authors have suggested that it is a perceived asynchrony of pubertal timing, rather than pubertal stage, which links problem behaviour and psychopathology (Carter, Silverman & Jaccard, 2013; Michael & Eccles, 2003).

In addition to emotion and behaviour, families often observe a change in the sleeping pattern of their adolescent offspring, as they frequently stay up later of an evening and get up later in the morning (Carskadon, 2011; Carskadon, Acebo & Jenni, 2004; Laberge et al., 2001). This may result in asynchrony between the adolescent's and family members' sleeping patterns. Furthermore, research has shown a bidirectional relationship between negative emotions, such as irritability and aggression, and the amount of good quality sleep an individual is getting (Ireland & Culpin, 2006). It may be that changes in the adolescent's sleeping pattern also affect their temperament, and that the emotional changes associated with puberty are affecting the adolescent's ability to fall asleep. There

are therefore several potential difficulties facing parents when their child enters puberty, with regards to their sleep pattern, emotions and behaviour.

Research in the early pubertal development population suggests that they are at an even greater risk of these negative changes. For example, research has shown that early-maturers are more likely to suffer mental health disorders or show symptoms of psychopathology than their on-time peers (Albert & Beck, 1975; Brooks-Gunn & Warren, 1989; Reardon, Leen-Feldner, & Hayward, 2009; Stice, Presnell & Bearman, 2001). Research by Graber and colleagues identified an increased prevalence of mental health diagnoses and psychopathology in those with earlier pubertal onset, and reported that this increased risk continued up until the age of 24 years (Graber, Lewinsohn, Seeley & Brooks-Gunn, 1997; Graber, Seeley, Brooks-Gunn & Lewinsohn, 2004). Conversely, Hayward et al. (1997) found that those who demonstrated higher levels of internalising symptomatology were more likely to have started puberty earlier. Additionally, those who develop earlier are more likely to display behaviours, such as delinquency, aggression, tobacco and alcohol use or sexual behaviours (Arim, Tramonte, Shapka, Dahinten & Willms, 2011; Copeland et al., 2010; Costello, Sung, Worthman & Angold, 2007; Dick, Rose, Viken & Kaprio, 2000; Graber et al., 1997; 2004; Kim & Smith, 1998; Patton et al., 2004; Stice et al., 2001; Westling, Andrews, Hampson & Peterson, 2008; Wichstrøm, 2001). For example, those who have reached Tanner stage III earlier than their peers are more likely to have had more sexual experiences and are at a greater risk of psychological dysfunction (Angold et al., 1998; Flannery, Rowe & Gulley, 1993). Downing and Bellis (2009) further reported that males whose pubertal onset was before the age of twelve were three and a half times more likely to have underage unprotected intercourse than those who entered puberty after the age of thirteen. Westling, Andrews and Peterson (2012) suggest

that problem behaviour in early-maturing adolescents is mediated by lower social competence, suggesting that those developing earlier are exposed to challenging situations before acquiring the social knowledge to cope with them. Alternatively, early-maturers may display problem behaviours as a result of the older peers with whom they socialise, (Skoog, Stattin, Ruiselova & Özdemir, 2013), implying that early puberty itself, has little impact on problem behaviours. Hochberg and Belsky (2013), however, suggest that from a life history perspective, problem behaviours, such as aggression and promiscuity, may be adaptive in reproduction. The authors suggest that factors causing pubertal timing to advance, such as stressful home environments, may influence earlier development, reproduction, and independence from the home environment. In this way, they suggest that early puberty should not be considered as a disorder under the medical model.

When considering that those who enter puberty at the earlier end of 'normal' are at an increased risk of multiple difficulties in comparison to their on-time peers, it may be that those below the clinical threshold are at an even greater risk. The little psychological research on clinically early puberty does support this theory. One example is the work by Dorn and colleagues (Dorn, Hitt & Rotenstein, 1999; Dorn et al., 2008), which compared the performance of typically-developing girls and those with a diagnosis of premature adrenarche (PA) on the Child Behavior Checklist (CBCL; Achenbach, 1991). Those with a diagnosis of PA scored higher on subscales of social problems, anxiety and depression, aggression, and higher-order subscales of internalising, externalising and total behaviour problems. Similarly, Sonis et al. (1985) found a quarter of their participants with a diagnosis of Central Precocious Puberty (CPP) scored more than two standard deviations above average for total problem score, and were significantly higher on internalising and externalising behaviour. Research by Xhrouet-Heinrichs et al. (1997) reported that those

with a diagnosis of CPP showed negative emotions, such as tearfulness and aggression, and Baumann et al. (2001) found that behaviour problems persisted even after hormone therapy. Furthermore, those with a diagnosis of CPP are more likely to have earlier sexual experiences than control participants (Cassio et al., 2006; Meyer-Bahlburg et al., 1985). Research by Sontag-Padilla and colleagues (2012), however, posited an interaction between executive functioning and pubertal development on the the behavioural and emotional problems observed. They reported that signs of psychopathology were more commonly found in participants with a diagnosis of PA who also had lower executive functioning, implying that a discrepancy between physical age and cognitive age is problematic for these children. Similarly, those with lower cognitive ability and earlier pubertal onset were at greatest risk for use of substances, sexual activity and difficulties at school (Orr & Ingersoll, 1995). It would appear that those with precocious pubertal development are at increased risk of altered emotion, cognition and behaviour, but that there are protective factors, such as high executive functioning, which increase a child's resilience against these difficulties.

The aim of the current study was to identify whether children with a diagnosis of precocious pubertal development were at a greater risk of emotional, behavioural or sleep problems than typically-developing children. It was hypothesised that those in CPP and PA groups would demonstrate significantly higher T-scores for emotional and behavioural problems, specifically anxiety, depression, internalising and externalising behaviours, as measured on the parent and teacher versions of the CBCL. It was also hypothesised that the two clinical groups would be more likely to score above standardised borderline and clinical thresholds, and thresholds derived from the variance of the present dataset, for these subscales. Exploratory analyses were also conducted to explore whether the two

patient groups would score differently to the typically-developing group on the other subscales of the CBCL and the Teacher Report Form (TRF), and whether any differences could be observed between behaviour at home and at school. Furthermore, correlational analyses were conducted to study whether pubertal development was related to behavioural or emotional scores on the CBCL and TRF. Finally, analyses were conducted to identify whether the two patient groups scored any differently on items measuring sleep problems, compared to the typically-developing group.

Method. For information on the recruitment process, please see 3.2, and for demographic information, see Table 1. Parents or carers were asked to complete the parent version of either the 1-5 years, or 6-18 years CBCL, depending on their child's age. If families consented, the teachers of children over the age of 6 years were sent a copy of the TRF to complete on the child' behaviour at school; 56% of teacher questionnaires were returned. Please note the smaller age range of participants in the TRF dataset. Families also completed a demographic questionnaire. For a review of each measure, please see 3.3.1.1 and 3.3.1.5.

Results. Tests of distribution and variance were calculated prior to each analysis. Where these tests were significant, non-parametric tests were employed. No statistically significant differences were observed between the three groups for age or socioeconomic status (SES).

- *CBCL Competence Scales- Hypothesis Testing.* First, the competence subscales of the CBCL were analysed between groups, which included items on activities, social group, school performance, and a total competence score. As the CBCL scoring materials provided enables researchers to derive T-scores for each participant as standardised with a United States (US) population, the analysis of the competence data included both these

pre-standardised T-scores, which are referred to as comparative T-scores, as well as T-scores calculated from the distribution of our sample, referred to as calculated T-scores. This allowed for comparison of the clinical groups' performance against other participants in the sample, as well as against standardised thresholds derived from the population in Achenbach's original analysis.

Calculated T-scores were analysed for the competence subscales for each group (Descriptive statistics in Table 14). A higher score indicates a higher level of competence, and all subscales had a standardised mean of 50 and a standard deviation of approximately 10.

Table 14: *Means and standard deviations for the calculated competence T-scores of the CBCL*

	Activities Subscale	Social Subscale	School Subscale	Total Competence
Control	50.70 (9.80)	50.56 (9.27)	51.74 (8.30)	51.13 (9.34)
PA	49.99 (9.15)	50.06 (9.99)	49.34 (9.64)	49.42 (9.70)
CPP	50.23 (9.33)	48.29 (12.38)	46.40 (14.11)	48.02 (12.51)

Non-parametric between-group analyses identified no significant differences between the groups on any of the three subscales, nor the total score for competence. This was also evident when the two clinical groups were collapsed into a single group and compared with the control group. There were no significant associations between group membership and whether participants scored more than one standard deviation below the mean for the activities subscale, the social subscale, or the total competence score. There was however, a significant association between membership of the three groups and score

on the CBCL school subscale ($X^2(2, 90)= 6.31$, $p= .043$). Please see Table 15 for the proportions of each group falling below the normal range.

Table 15: *Proportion of each group scoring within the normal range, and less than one standard deviation below the mean on the school subscale of the CBCL, on the Chi-squared analysis of the calculated CBCL competence T-scores*

	Within normal range	>1 SD below the mean
Control	93%	7%
PA	84%	16%
CPP	67%	33%

Analyses were then repeated for the comparative T-score values taken from the original scoring procedure, in order to compare the current sample with the recommended borderline and clinical thresholds. Descriptive statistics are provided in Table 16.

Table 16: *Means and standard deviations for the comparative competence T-scores of the CBCL*

	Activities Subscale	Social Subscale	School Subscale	Total Competence
Control	45.14 (9.55)	49.40 (8.14)	50.72 (5.83)	47.93 (9.67)
PA	43.60 (10.33)	49.13 (9.03)	49.16 (6.95)	46.03 (10.06)
CPP	45.00 (11.70)	47.40 (11.33)	47.13 (9.21)	45.20 (12.50)

As with the calculated T-scores, no significant differences were identified in the comparative T-scores between the three groups on the three subscales or total competence T-score. No significant differences were found when the control group was compared to

the two clinical groups combined. Chi-squared analyses showed no association between group membership and whether a participant scored in the normal, borderline, or clinical ranges as suggested in the original scoring framework. This was also observed when the two clinical groups were collapsed to form a single clinical group and compared with controls.

- *CBCL Competence Scales- Exploratory Analysis.* The data for the groups were then combined and a Spearman's correlational analysis conducted, in order to identify whether a relationship existed between pubertal development and the competence subscale T-scores, both comparative and calculated. It was found that pubertal development was significantly negatively correlated with the school subscale on both the calculated T-score ($r= -.32$, $p= .002$), and also the comparative T-score ($r= -.35$, $p= .001$).

When analysing the predictive value of pubertal development on competence scale performance, it was found that a quadratic model of regression fit both the calculated and comparative T-score data for the school subscale, explaining 23% of the variance on both measures (Calculated T-score: $R^2= .23$, $F(2, 89)= 13.18$, $p< .001$; Comparative T-score: $R^2= .23$, $F(2, 89)= 12.76$, $p< .001$). The curve of the predicted model began to curve downward at mean Tanner stage II, suggesting that it is at this point in pubertal development that school competence begins to decline.

- *CBCL Syndrome Scales- Hypothesis Testing.* The syndrome scales of the CBCL were analysed in the same manner as the competence scales, with the calculated T-scores analysed first, followed by the comparative T-scores. A high score in the syndrome subscales and higher-order scales indicates the presence of problem behaviours, and a clinical score is therefore a higher value than a non-clinical score. Again, the standardised

mean is 50 and standard deviation 10. The descriptive statistics for the calculated T-scores are presented in Table 17.

Non-parametric between-groups analyses identified significant differences between the calculated T-scores for the anxious-depressed subscale ($H(2)= 7.42$, $p= .024$) and the aggression subscale ($H(2)= 6.74$, $p= .034$). Post-hoc analyses were carried out with a Bonferroni correction, and a critical value of .017. It was found that differences on the anxious-depressed subscale lay between the control group and the PA group ($U(46, 34)= 540.50$, $z= -2.37$, $p= .018$) and the control group and the CPP group ($U(46, 15)= 224.50$, $z= -2.04$, $p= .041$), however, these differences were not significant after Bonferroni correction. Post-hoc analyses for the aggression subscale identified significant differences between the control and PA groups ($U(46, 34)= 510.50$, $z= -2.66$, $p= .008$). The two clinical groups were then combined and compared with the control group, and significant differences were again observed between the two groups in the anxious-depressed ($U(43, 47)= 765.00$, $z= -2.72$, $p= .007$) and aggression subscales ($U(43, 47)= 802.00$, $z= -2.43$, $p= .015$), with the clinical group scoring higher than the control participants.

Table 17: *Means and standard deviations for the calculated syndrome scale T-scores on the CBCL*

	Anxious-Depressed	Withdrawn-Depressed	Somatic Problems	Social Problems	Thought Problems	Attention Problems	Rule-breaking Behaviour	Aggression
Control	47.31 (8.63)	48.94 (8.24)	48.38 (7.32)	49.30 (8.93)	48.83 (9.19)	48.94 (10.09)	48.67 (8.15)	47.10 (6.67)
PA	51.90 (9.49)	50.81 (8.73)	51.52 (10.30)	50.41 (9.53)	51.66 (11.13)	50.86 (9.42)	51.13 (9.12)	53.56 (12.19)
CPP	53.95 (13.07)	51.39 (16.32)	51.53 (15.24)	51.14 (13.85)	49.81 (9.88)	51.31 (11.30)	51.41 (15.54)	50.83 (10.90)

Table 18: *Means and standard deviations for the comparative syndrome scale T-scores on the CBCL*

	Anxious-Depressed	Withdrawn-Depressed	Somatic Problems	Social Problems	Thought Problems	Attention Problems	Rule-breaking Behaviour	Aggression
Control	52.63 (3.64)	53.70 (5.68)	54.09 (6.35)	54.89 (6.09)	55.00 (5.85)	55.65 (6.24)	55.07 (7.39)	53.07 (4.11)
PA	56.68 (7.53)	56.94 (6.91)	55.50 (6.73)	57.00 (7.87)	55.63 (6.24)	57.59 (7.47)	56.62 (6.86)	54.53 (4.62)
CPP	55.13 (6.76)	57.47 (9.05)	55.87 (12.06)	57.93 (12.14)	56.47 (9.32)	56.47 (7.17)	57.27 (8.26)	54.67 (7.14)

The analysis was repeated using the comparative T-scores, and the recommended clinical thresholds used for comparison. The descriptive statistics for these comparative T-scores are presented in Table 18. Non-parametric analyses were conducted between the three groups for each syndrome subscale, and significant differences were identified between groups for the anxious-depressed ($H(2)= 6.25$, $p= .044$) and withdrawn-depressed subscales ($H(2)= 6.82$, $p=.033$), with clinical groups scoring higher than the control group. Post-hoc Mann-Whitney analyses with a corrected alpha of .017 were then conducted. The anxious-depressed analysis demonstrated a significant difference between control and PA groups ($U(46, 34)= 525.00$, $z= -2.56$, $p= .010$). Post-hoc analyses for the withdrawn-depressed subscale suggested differences between control and PA groups ($U(46, 34)= 551.50$, $z= -2.29$, $p= .022$) and control and CPP groups ($U(43, 15)= 231.00$, $z= -1.95$, $p= .051$), however, these were non-significant after Bonferroni correction. Analysis of all clinical participants compared with the control group also identified significant differences between groups on the anxious-depressed ($U(43, 47)= 817.50$, $z= -2.36$, $p= .018$) and withdrawn-depressed subscales ($U(43, 47)= 782.50$, $z= -2.61$, $p= .009$).

Between-group analyses of the calculated and comparative T-scores for the higher-order subscales were then carried out, with the calculated T-scores analysed first. Descriptive statistics are given in Table 19 and 20.

Table 19: *Means and standard deviations for the calculated higher-order scale T-scores on the CBCL*

	Internalising	Externalising	Total Syndrome Score
Control	47.65 (7.44)	47.35 (6.66)	47.74 (8.35)
PA	51.66 (8.89)	53.15 (11.56)	52.06 (10.07)
CPP	53.46 (16.41)	50.99 (12.85)	52.28 (13.25)

Table 20: *Means and standard deviations for the comparative higher-order scale T-Scores on the CBCL*

	Internalising	Externalising	Total Syndrome Score
Control	50.20 (10.24)	48.98 (7.54)	49.96 (10.01)
PA	54.79 (10.72)	53.68 (9.25)	54.26 (10.37)
CPP	55.60 (13.97)	51.80 (10.57)	54.00 (12.68)

Between-groups analysis of the higher-order scales produced no significant differences between the three groups. When the clinical groups were collapsed into a single clinical group, however, a significant difference was observed on the internalising behaviour scale between the combined clinical group and control group ($U(46, 49)= 823.50$, $z= -2.27$, $p= .023$). Furthermore the differences between groups on the externalising behaviour scale and total syndrome scale scores were close to significance (externalising: $U(46, 49)= 868.50$, $z= -1.93$, $p= .053$; total syndrome scale: $U(46, 49)= 874.00$, $z= -1.89$, $p= .059$).

The higher-order scales comparative T-scores were then analysed. Kruskal-Wallis analysis demonstrated no differences between groups on the higher-order internalising,

externalising subscales and the total syndrome scale. After collapsing the clinical groups and comparing scores to the control group, a significant difference was found on the internalising subscale (U(46, 49)= 843.50, z= -2.12, p= .034), the externalising subscale (U(46, 49)= 850.50, z= -2.06, p= .039), and a near significant difference for the total syndrome score (U(46, 49)= 873.50, z= -1.89, p= .059).

Chi-squared analyses were conducted for both syndrome subscales and higher-order scales, to identify whether there was, firstly, an association between pubertal group membership and whether the participant scored more than a standard deviation above the mean, and secondly, between clinical and control participants and whether they scored more than one standard deviation above the mean. Again, calculated T-scores are presented first. A significant association was identified on the higher-order externalising subscale, with a higher proportion of the clinical groups scoring more than one standard deviation above the mean compared to controls ($X^2(1, 95)= 3.87$, p=.049). See Table 21 for the proportion of each group scoring within the normal range, and those scoring greater than one standard deviation above the mean on the externalising behaviour scale.

Table 21: *Proportion of each group scoring within the normal range, and more than one standard deviation above the mean on the externalising behaviour scale of the CBCL, on the Chi-squared analysis of the calculated T-scores*

	Within normal range	>1 SD above mean
Control	94%	6%
Clinical	80%	20%

Chi-squared analyses were then conducted for the comparative T-scores to explore whether each group were more likely to fall in the ‘normal’, ‘borderline’ or ‘clinical’

ranges of the CBCL. Analysis of the anxious-depressed subscale showed a near significant association ($X^2(4, 95)= 9.21$, $p= .056$), as well as a significant association for the aggression subscale ($X^2(4, 90)= 11.29$, $p= .023$). See Table 22 for the proportion of each group scoring within the normal, borderline and clinical ranges, on the anxious-depressed and aggressive behaviour scales.

Table 22: *Proportion of each group scoring within the normal, borderline and clinical on the anxious-depressed and aggressive behaviour scales of the CBCL, on the Chi-squared analysis of the comparative T-scores*

	Anxious-Depressed			Aggression		
	Normal	Borderline	Clinical	Normal	Borderline	Clinical
Control	100%			98%	2%	
PA	85%	9%	6%	100%		
CPP	87%	13%		87%		13%

The clinical groups were then collapsed and analysed as a single clinical group. A significant association was identified for the anxious-depressed subscale ($X^2(2, 95)= 7.09$, $p= .029$), the social problems subscale ($X^2(2, 95)= 6.10$, $p= .047$), and the higher-order externalising subscale ($X^2(2, 95)= 6.15$, $p= .046$). See Table 23 for the proportion of each group scoring within the normal, borderline and clinical ranges, on the anxious-depressed and social problems, and externalising behaviour scales.

Table 23: *Proportion of each group scoring within the normal, borderline and clinical on the anxious-depressed, social problems and externalising behaviour scales of the CBCL, on the Chi-squared analysis of the comparative T-scores*

	Anxious-Depressed			Social Problems			Externalising Behaviour		
	Normal	Borderline	Clinical	Normal	Borderline	Clinical	Normal	Borderline	Clinical
Control	100%			91%	9%		94%	4%	2%
Clinical	86%	10%	4%	82%	6%	12%	76%	10%	14%

- *CBCL Syndrome Scales- Exploratory Analysis.*After hypothesis testing, more exploratory analyses of the data were conducted to explore whether any relationshipsbetween variables existed in the data. When analysing the calculated t-scores of the CBCL syndrome subscale data, significant trends were identified for the anxious-depressed and aggressive behaviour subscales, in which clinical groups scored higher than the control group (anxious-depressed: J= 1753.50, z= 2.64, p= .008; aggression: J= 1672.50, z= 2.06, p= .04). Jonckheere-Terpstra analyses for the comparative t-score data also identified significant trends across the three groups for the anxious-depressed scale, as well as the withdrawn-depressed subscale (anxious-depressed: J= 1662.50, z= 2.02, p= .043; withdrawn-depressed: J= 1731.50, z= 2.50, p= .012), with clinical groups scoring higher than the control group.

Jonckheere-Terpstra analyses of the calculated higher-order scale scores identified a significant trend on the internalising behaviour scale (J= 1665.50, z- 2.00, p= .045), with clinical participants scoring higher than controls. The higher-order scales comparative T-scores were then analysed, and a near significant trend was, again, observed on the internalising subscale (J= 1649.50, z= 1.89, p= .059).

Spearman's correlational analyses between pubertal development and the calculated CBCL subscale scores demonstrated significant relationships for all subscales on the CBCL, bar the rule-breaking subscale. When the analyses were repeated controlling for SES, these relationships were still evident, except for that between pubertal development and thought problems, and again, rule-breaking behaviour. Exploratory curve estimation analyses were conducted to test the predictive value of pubertal development on the CBCL scales. The correlation and regression analyses are summarised in Table 24.

Tanner rating was predictive of several disordered behaviour types, explaining between 7% and 35% of the variance in these T-scores.

Table 24: *Correlation analysis whilst partialling out SES, and regression models derived from curve estimation analysis, for the calculated syndrome and higher-order T-scores on the CBCL*

	Correlation with Tanner Rating		Curve Estimation Analysis – Model Parameters					
	r	Sig.	Regression Model	R^2	Constant	Unstandardised β	F (df)	Sig.
Anxious-Depressed	.39	.000	Linear	.16	38.70	6.61	18.02 (1, 93)	.000
Withdrawn-Depressed	.29	.007	Cubic	.29	-17.04	113.59, -61.55, 10.69	12.57 (3, 91)	.000
Somatic Problems	.38	.000	Cubic	.35	-39.20	144.93, -74.95, 12.49	16.54 (3, 91)	.000
Social Problems	.23	.034	Linear	.07	42.60	4.27	6.48 (1, 88)	.013
Thought Problems	.18	.087	NA					
Attention Problems	.27	.011	Linear	.10	41.23	5.13	10.07 (1, 93)	.002

Rule-breaking Behaviour	.05	.638	NA					
Aggressive Behaviour	.27	.012	Linear	.08	41.90	4.74	8.46 (1, 93)	.005
Internalising Behaviour	.44	.000	Cubic	.33	-12.53	99-82, -52.17, 9.02	15.22 (3, 91)	.000
Externalising Behaviour	.23	.033	Linear	.06	43.00	4.09	6.18 (1, 93)	.015
Total Syndrome Score	.38	.000	Linear	.17	38.59	6.67	18.42 (1, 93)	.000

Correlational analyses of comparative T-scores demonstrated that pubertal development was significantly related to all higher-order scales and syndrome subscales, except for aggression. These significant relationships persisted after controlling for SES, except for the attention problem subscale. Age in months showed no significant relationship with any of the CBCL subscale scores. Exploratory regression analyses identified pubertal development as a significant predictor of the comparative T-scores on several scales of the CBCL, and demonstrated that pubertal development could explain between 4% and 26% of the variance in these scores. See Table 25 for correlational and regression model statistics.

Table 25: *Correlation analysis whilst partialling out SES, and regression models derived from curve estimation analysis, for the comparative syndrome and higher-order T-scores on the CBCL*

	Correlation with Tanner Rating		Curve Estimation Analysis – Model Parameters					
	r	Sig.	Model	R^2	Constant	Unstandardised β	F (df)	Sig.
Anxious-Depressed	0.26	0.015	Linear	0.09	49.38	2.98	9.24 (1, 93)	0.003
Withdrawn-Depressed	0.33	0.002	Linear	0.11	49.04	3.75	11.54 (1, 93)	0.001
Somatic Problems	0.25	0.017	Cubic	0.26	6.3	82.6, -44.82, 7.78	10.69 (3, 91)	0
Social Problems	0.38	0	Cubic	0.26	3.2	84.39, -43.4, 7.3	10.85 (3, 91)	0
Thought Problems	0.23	0.029	Linear	0.07	50.45	2.9	6.86 (7, 88)	0.01
Attention Problems	0.19	0.074	Linear	0.04	52.45	2.32	4.01 (1, 88)	0.048
Rule-breaking	0.26	0.007	Linear	0.11	49.17	3.98	11.44 (1, 93)	0.001

Behaviour								
Aggressive Behaviour	0.11	0.314	NA					
Internalising Behaviour	0.39	0	Cubic	0.23	-12.21	97.91, -47.99, 7.86	9.01 (3, 91)	0
Externalising Behaviour	0.22	0.041	Linear	0.08	44.28	3.99	7.56 (1, 93)	0.007
Total Syndrome Score	0.36	0.001	Linear	0.16	40.14	7.02	17.74 (1, 93)	0

- *TRF Adaptive Behaviour Scales- Hypothesis Testing***.** The TRF data were then analysed using the same analyses as the CBCL. No significant differences in gender, SES or age were identified. The academic competence section of the TRF included items on academic performance, ability to learn and behave appropriately, happiness at school and how hard the child typically works. A higher score denotes greater competence. These items contributed to a total adaptive behaviour T-score. The TRF analysis included both calculated T-scores for the sample, as well as comparative T-scores for the academic performance and total adaptive behaviour scales. The calculated T-scores were analysed first; descriptive statistics are presented in Table 26.

Table 26: *Means and standard deviations for the calculated adaptive behaviour T-scores of the TRF*

	Academic Performance	Working Hard	Appropriate behaviour	Learning Well	Happy at School	Total Adaptive T-score
Control	49.62 (11.60)	50.23 (10.86)	51.10 (9.63)	49.93 (10.01)	51.03 (10.65)	50. 88 (10.09)
PA	49.46 (8.55)	48.05 (9.06)	48.09 (9.86)	47.92 (9.46)	48.55 (9.04)	47.69 (9.49)
CPP	53.52 (6.77)	55.48 (7.96)	51.41 (12.86)	57.23 (9.94)	50.19 (11.18)	53.73 (11.24)

Between-group analyses demonstrated no significant difference between groups on the adaptive behaviour subscales, nor were there significant trends across groups. This was replicated when the clinical groups were collapsed into a single group and compared with the control group. Chi-squared analyses also demonstrated no significant associations between group and the probability of scoring lower than one standard deviation below the mean.

Similarly, no significant differences were identified for the comparative T-scores for the academic performance and total adaptive behaviour scales between the three groups, nor between the collapsed clinical group and control group. Comparative T-scores were not provided for the other subscales. Chi-squared analyses identified no significant associations between group and whether the score fell above borderline or clinical thresholds. See Table 27 for descriptive statistics.

Table 27: *Means and standard deviations for the comparative adaptive behaviour T-scores of the TRF*

	Academic Performance	Total Adaptive T-score
Control	54.00 (9.48)	55.22 (8.23)
PA	54.45 (7.22)	52.45 (7.84)
CPP	57.50 (4.28)	57.00 (9.76)

- *TRF Adaptive Behaviour Scales- Exploratory Analysis*. Exploratory analysis of the TRF adaptive behaviour data was then carried out, to identify whether any interesting relationships existed in the data. Correlational analyses found no relationships between Tanner stage and adaptive subscale T-scores in either calculated or comparative T-scores, although SES was significantly related to working hard ($r = .27$, $p = .047$), appropriateness

of behaviour (r= .32, p= .020), and total adaptive behaviour score (r= .31, p= .023) calculated T-scores.

- *TRF Syndrome Scales- Hypothesis Testing*. The syndrome scales section of the TRF was then analysed. A higher score in this section indicates presence of behavioural problems. The T-scores for each syndrome scale were calculated, and the means and standard deviations for each group are presented in Table 28.

Kruskal-Wallis analyses demonstrated a significant difference between groups and significant trend across groups on the rule-breaking behaviour subscales, where the CPP group scored the highest and the control group the lowest (H(2)= 8.43, p= .015, J= 478.50 , z= 1.96 , p= .049). Post-hoc analyses identified differences between the control and CPP groups (U(27, 6)= 45.50, z= -2.66, p= .008) and PA and CPP groups (U(20, 6)= 35.00, z= -2.22, p= .026). Reanalysis of the data with the two clinical groups combined produced no significant differences between the collapsed clinical group and the control group.

The comparative T-scores were then analysed. Descriptive statistics are given in Table 29. Kruskal-Wallis analyses demonstrated a significant difference between groups (H(2)= 8.43, p= .015) on the rule-breaking behaviour subscale. Post-hoc analyses identified that the significant differences fell between the CPP group and the PA and control groups (CPP-control: U(27, 6)= 45.50, z= -2.66, p= .008; CPP-PA: U(20, 6)= 35.00, z= -2.22, p= .026), although the difference between the CPP and PA groups was not significant after Bonferroni correction.

Table 28: *Means and standard deviations for the calculated syndrome scale T-scores of the TRF*

	Anxious-Depressed	Withdrawn-Depressed	Somatic Problems	Social Problems	Thought Problems	Total Attention Problems	Rule-breaking	Aggressive Behaviour
Control	49.26 (10.03)	48.56 (9.46)	48.38 (5.61)	48.46 (8.05)	50.86 (13.41)	50.66 (12.98)	48.20 (3.59)	48.85 (4.34)
PA	48.77 (7.98)	50.33 (7.98)	49.36 (9.02)	50.18 (6.38)	48.54 (3.38)	49.91 (5.92)	48.55 (4.14)	49.29 (5.95)
CPP	57.44 (12.80)	55.38 (16.93)	59.44 (21.08)	56.36 (22.07)	51.01 (6.90)	47.34 (4.48)	62.91 (26.12)	57.56 (27.01)

Table 29: *Means and standard deviations for the comparative syndrome scale T-scores of the TRF*

	Anxious-Depressed	Withdrawn-Depressed	Somatic Problems	Social Problems	Thought Problems	Total Attention Problems	Rule-breaking	Aggressive Behaviour
Control	53.22 (4.00)	51.67 (3.75)	52.30 (4.52)	51.93 (4.86)	51.70 (5.45)	51.30 (3.60)	50.37 (1.33)	51.70 (2.93)
PA	53.05 (3.58)	52.70 (3.76)	52.55 (5.05)	53.55 (4.57)	51.40 (2.87)	50.90 (1.65)	50.50 (1.54)	51.85 (3.31)
CPP	56.67 (5.39)	54.33 (6.74)	57.17 (9.02)	56.17 (11.36)	53.33 (5.50)	50.50 (1.22)	54.67 (6.98)	53.33 (8.16)

Chi-squared analysis of the calculated T-scores demonstrated significant associations between groups and whether participants scored higher than one standard deviation above the mean for the calculated T-scores on the anxious-depressed subscale ($X^2(2, 53)= 6.95$, $p= .031$) and rule-breaking subscale ($X^2(2, 53)= 8.06$, $p= .018$) (Table 30).

Table 30: *Proportion of each group scoring greater than one standard deviation above the mean on the anxious-depressed and rule-breaking subscales of the TRF, on the Chi-squared analysis of the calculated T-scores*

	Anxious- Depressed		Rule-breaking	
	Normal	>1SD	Normal	>1SD
Control	93%	7%	93%	7%
PA	85%	15%	90%	10%
CPP	50%	50%	50%	50%

Chi-squared analyses of the comparative T-scores identified a significant association on whether groups scored within the normal, borderline, or clinical ranges for the somatic ($X^2(4, 53)= 12.16$, $p= .016$), social problems ($X^2(4, 53)= 9.91$, $p= .042$), rule-breaking ($X^2(2, 53)= 7.98$, $p= .018$) and aggression subscales ($X^2(2, 53)= 7.98$, $p= .018$). For the proportion of each group falling in each category, please see Table 31.

Table 31: *Proportion of each group scoring in the normal, borderline or clinical ranges on the syndrome subscales of the TRF, on the Chi-squared analysis of the comparative T-scores*

	Somatic Problems			Social Problems			Rule-breaking Behaviour			Aggression		
	Normal	Border-line	Clinical	Normal	Border-line	Clinical	Normal	Border-line	Clinical	Normal	Border-line	Clinical
Control	100%			93%	7%		100%			100%		
PA	95%	5%		100%			100%			100%		
CPP	66%	17%	17%	83%		17%	83%		17%	83%		17%

The higher-order scales of the TRF were then calculated by combining the syndrome subscales to produce the internalising behaviour, externalising behaviour and total syndrome scale scores. Descriptive statistics are given in Table 32 for the calculated T-scores, and Table 33 for the comparative T-scores.

Table 32: *Means and standard deviations for the calculated higher-order scale T-scores of the TRF*

	Internalising Behaviour	Externalising Behaviour	Total Syndrome Score
Control	48.60 (8.67)	48.18 (10.05)	49.00 (9.74)
PA	49.22 (7.83)	50.46 (8.56)	49.56 (6.05)
CPP	58.91 (17.54)	56.66 (12.84)	55.98 (18.93)

Table 33: *Means and standard deviations for the comparative higher-order scale T-scores of the TRF*

	Internalising Behaviour	Externalising Behaviour	Total Syndrome Score
Control	45.63 (9.32)	46.41 (5.99)	44.78 (8.02)
PA	47.75 (7.94)	47.25 (5.86)	46.10 (7.29)
CPP	53.50 (11.91)	50.50 (10.52)	49.00 (11.87)

Kruskal-Wallis analyses of the calculated and comparative T-scores demonstrated no significant differences on the higher-order scales, nor when clinical groups were collapsed and analysed as a single group. Chi-squared analyses of the calculated T-scores also identified no significant associations between group and whether participants scored

more than one standard deviation greater than the mean. Chi-squared analyses of the comparative T-scores, however, identified significant associations between groups and whether a participant scored above the borderline or clinical thresholds on both internalising and externalising higher-order scales (internalising: $X^2(2, 53)= 6.48$, $p= .039$; externalising: $X^2(4, 53)= 9.63$, $p= .047$). The percentage of each group falling above each threshold is given in Table 34.

Table 34: *Proportion of each group scoring in the normal, borderline, and clinical ranges on the internalising and externalising behaviour scales of the TRF, on the Chi-squared analysis of the calculated T-scores*

	Internalising Behaviour			Externalising Behaviour		
	Normal	Borderline	Clinical	Normal	Borderline	Clinical
Control	96%		4%	100%		
PA	95%		5%	95%	5%	
CPP	67%		33%	83%		17%

- *TRF Syndrome Scales- Exploratory Analysis*. After hypothesis testing, exploratory analysis of the data was conducted. Jonckheere-Terpstra analysis demonstrated a significant trend on the comparative t-scores of the rule-breaking behaviour subscale ($J= 478.50$, $z= 1.96$, $p= .049$), whereby the control group scored lowest, and the CPP group the highest. Furthermore, Spearman's correlational analysis of the calculated and comparative T-scores demonstrated several significant relationships between mean Tanner rating and the syndrome and higher-order scales of the TRF. However after controlling for SES only the relationships with total syndrome calculated T-score ($r= .33$, $p= .015$), calculated and comparative rule-breaking T-scores (calculated: $r= .29$, $p= .037$; comparative: $r= .32$, $p=$

.022) and comparative externalising behaviour T-score (r= .36, p= .009) remained. When entered into a regression analysis it was found that pubertal development was not predictive of total syndrome T-score, but was weakly predictive of rule-breaking behaviour (calculated rule-breaking: R^2= .12, constant= 54.71, β= -12.42, β^2= 5.32, F(2, 50)= 3.45, p= .040; comparative rule-breaking R^2= .12, constant= 47.73, β= 1.94, F(1, 51)= 6.94, p= .011) and comparative externalising behaviour (R^2= .15, F(1, 51)= 9.12, p= .004).

The calculated T-scores on the CBCL were then compared with those on the TRF to explore whether any behavioural problems could be seen in both home and school environments. None of the subscales on the CBCL correlated with their counterparts on the TRF.

- *Sleep Problems.* The total score for the sleep subscale was calculated for each participant, and T-scores calculated for the sample. Unlike the previous CBCL analyses, the sleep subscale was not included in Achenbach's original factorial structure, and no standardised T-scores or clinical thresholds were available with which to compare the current sample. There is therefore no comparative T-score analysis in this section. Descriptive statistics were analysed for each group, and are given in Table 35.

Table 35: *Means and standard deviation for the total sleep subscale T-scores*

	Mean (SD)
Control	48.72 (9.04)
PA	51.11 (9.94)
CPP	51.41 (12.86)

Kruskal-Wallis analyses demonstrated no significant differences between sleep problem scores for each group, and no significant trends. Similarly, no significant differences were identified between the clinical participants and the controls once clinical groups were combined and analysed with a Mann-Whitney analysis. There were also no significant associations between group membership and scoring more than one standard deviation above the mean, both between the three experimental groups, and between the collapsed clinical group and controls.

Spearman's correlation demonstrated that Tanner rating was significantly positively related to sleep problem T-score ($r= .34$, $p= .001$). Age was not related to sleep problems. Furthermore, a linear regression identified pubertal development as a significant, although weak, predictor of sleep problems, explaining 11% of the variance in the T-scores ($R^2= .11$, constant= 40.65, $\beta= 5.47$, $F(1, 93)= 11.61$, $p= .001$).

Discussion. This study has compared the behaviour, sleep and emotion of children with diagnoses of precocious pubertal development to typically-developing children. Both parent and teacher reports were taken into consideration, to identify whether problems were evident both at home and school. The first observation was that the standard deviations in the CPP group were generally much larger than those of the PA and control groups. This suggests that the data for the CPP group were more widely distributed, and that observations are not indicative of a behavioural profile for this clinical group as a whole, but an increased risk for developing these problems. Furthermore, the sample was considerably smaller in this group, which may have resulted in biases in the data. A second observation is that, where analyses between clinical groups were significant, collapsed clinical group analyses were often not. This would suggest that it is not the general effect of having a diagnosis of early puberty which alters behaviour or emotion, but the

biological or environmental conditions specific to each disorder. Finally, the lack of coherence between parent and teacher reports would imply that at least one of the measures is not assessing the desired construct, or that bias occurred in parents' or teachers' responses. However, parents frequently reported that their children behaved very well at school compared to at home, and this would support the lack of consistency between the two measures.

A further point when considering the T-scores taken from Achenbach's original scoring frame-work is that on the syndrome subscales it is impossible for a child to attain a T-score less than 50, despite this being the mean value. The scoring framework has been designed, so that the presence of any item will score above average, and it is therefore assumed that 50% of the population would score zero. When using these T-scores for analysis, it must be noted that the normal distribution is essentially halved, and the variance in this comparative T-score analysis is therefore considerably lower than when analysing the calculated T-scores for our sample. The T-scores have still been included in the analysis however, for comparison of our sample against the standardised data and recommended clinical thresholds.

- *Competency and Adaptive Behaviour.* Analysis of the CBCL competence scales and TRF adaptive behaviour scales would largely suggest that children with precocious pubertal development are no different to typically-developing children. However those with greater pubertal development scored lower on the school subscale, with 33% of the group falling more than one standard deviation below the mean on the parent measure. This scale included items on academic ability, whether the child was receiving additional support at school, and if they had had to repeat a school year. This would imply that although children with earlier pubertal development appear to be doing well, they may

need more support at school than their peers. Furthermore, it was found that after Tanner stage II a decline in school competence was reported by parents. This implies that children begin to perform less well at school as they enter puberty, or that their parents perceive them to be less successful. This may be related to a lack of motivation to do homework, leading parents to believe that their child is underperforming, or alternatively, a developing interest in peers and socialisation may actually cause a loss of interest in performing well at school. The TRF data, however, suggests that the CPP participants performed better at school than the PA and control groups, despite scoring highest for pubertal development. Although this advantage was not significantly different, it was consistent, and it may therefore be that children present as more capable at school than they do at home, leading parents to think they are under-performing.

- *Problem Behaviour and Psychopathology.* Analysis of the CBCL syndrome scales suggest that both clinical groups demonstrated significantly more symptoms of anxiety and depression than the typically-developing group, with 14% scoring above the typical threshold. However, parents perceived the PA participants to be most anxious, whereas teachers scored the CPP group highest for anxiety and depression. PA and CPP participants were also more likely to be classed above the normal range by their parents, although only participants in the PA group fell into the clinical range. This suggests that although the CPP group may also show elevated signs of anxiety and depression both at home and at school, it is the PA group who demonstrate these behaviours to a clinical degree at home. This is supported both by informal parent observations, and by the teacher-report data, where, although half of the CPP group scored greater than one standard deviation from the mean, none scored above the clinical threshold. Correlational analysis of the parent data also identified a positive relationship between puberty and signs

of anxiety and depression, although this relationship was only weakly predictive, suggesting that although this trait increases with puberty, as seen in the CPP group, there may something specific to adrenarchal development that further increases risk of anxiety and depression, particularly in the home environment.

Differences between clinical participants and typically-developing children were also observed on the comparative T-scores for the withdrawn-depressed subscale, and a significant predictive relationship was identified between pubertal development and scores on this subscale. As there was no significant difference between the two clinical groups on this scale, and because the regression model that best fit the data was cubic, we might assume a general impact of puberty on withdrawn-depressed symptomatology, such as looking different to peers, or biological changes occurring in both disorders. This may be exacerbated by being in puberty at a time asynchronous to peers. The cubic model implies that the initial increase is gradual until around Tanner stage II. At this point there is a gradual decrease before a sharp increase at Tanner stage III. The PA and CPP groups would both score Tanner stage III and above, and would therefore fall on the sharp incline of the regression curve. This would imply that being in puberty is related to increases in withdrawn-depressed symptomatology in these patients, either through biological or environmental factors.

When looking at internalising behaviour problems overall, it was found that there was a significant trend across groups on the calculated T-scores, as well as near significant differences between groups on the comparative T-scores, suggesting that higher levels of pubertal development are related to internalising problem behaviours. This is supported by the correlation and regression analyses, which imply that pubertal development is a significant predictor of internalising behaviour on both sets of T-scores. The best model

for the data was a cubic model, suggesting that internalising behaviours are not constant, but fluctuate over the course of development. The regression model implies that internalising behaviours increase initially at the onset of puberty and then again much more rapidly as the child reaches an average Tanner III before levelling out. This is consistent with the findings of Angold et al. (1998) and Flannery et al. (1993), who reported that the onset of several mental health problems was most common after Tanner stage III or IV. Furthermore, this would be consistent with the mid-level of development shown by those with a diagnosis of PA, suggesting that internalising behaviours begin to increase in this group as they enter adrenarche and remain high in those with CPP who show greater development. This may explain the finding that a third of the CPP group were rated as being in the clinical range by their teachers, as puberty in general may be related to increases in internalising behaviour problems, but this is exacerbated at school by asynchronous timing in the CPP group.

When considering the externalising behaviour subscales, significant differences were observed between groups in reported aggressive behaviour, specifically, the PA group scoring highest and the CPP groups scoring a little lower. When considering the Chi-squared data, however, the CPP group were the only participants to score in the clinical range, both on parent and teacher measures. This may be slightly illogical, as we would therefore expect the mean values for the CPP group to be highest, however, the CPP group had much larger standard deviations, meaning that some individuals score at the more extreme end of the scale but the majority score much lower, bringing the mean value down. The majority of the PA group may score higher than the other groups, but still within the typical range. A subsection of the CPP group may therefore be at an increased risk of clinical levels of aggressive behaviour rather than this being a common problem in

this group, but the PA group may display low-level aggressive behaviour more consistently. Interestingly, there was no significant relationship between pubertal development across groups and aggression. This implies that there is something specific to the two pubertal disorders. Furthermore, teachers reported that half of those in the CPP group displayed rule-breaking behaviour greater than one standard deviation above the mean, compared to less than 10% in the PA and control groups, and 17% of the CPP group scored clinically compared to none of the PA or control participants. This implies that there is something specific to the diagnosis of CPP which is associated with higher scores on the rule-breaking behaviour subscale.

Evidence of another association between puberty and externalising behaviour was found on the social problems subscale, where only 82% of clinical participants scored within the normal range compared to over 90% of controls. Only those diagnosed with CPP scored above the clinical threshold, as reported on both parent and teacher measures. Correlational and regression analyses support this finding, as pubertal development was both significantly related to, and predictive of social problems, predicting as much as 26% of the comparative T-score data. Furthermore, the regression model to best fit the data was, again, cubic, implying that social problems do not increase linearly alongside pubertal development, but increase rapidly at around Tanner stage III before stabilising. This would explain the increased incidence of social problems in the CPP group, the majority of whom score around or above Tanner stage III.

The overall externalising behaviour analysis demonstrated that the clinical groups scored significantly higher than control participants. On both sets of T-scores the clinical participants were more likely to score above the mean, and over borderline and clinical thresholds, with only three quarters of clinical participants scoring within the normal

range, compared to nearly all controls on the parent measure, and with 5% of the PA group scoring in the borderline range, and 17% of the CPP group scoring clinically on the teacher measure. As there was no difference between the two clinical groups' means on this scale, it would seem that an increase in externalising problems is not specific to either of the pubertal disorders. Furthermore, although there was a significant correlation and predictive relationship between pubertal development and externalising behaviour, these relationships were weak at best, suggesting that externalising behaviours do not increase greatly over the course of puberty. However, the chi-squared data would suggest otherwise, and it may therefore be that it is the environmental impact of having a pubertal disorder and being slightly different to peers that is the cause of these externalising behaviour problems. This would explain the greater proportion of the CPP group scoring above the clinical threshold at school.

- *Sleep Problems.* The sleep data analysis demonstrated no differences between groups on the presence of sleep problems, although there was a significant correlation between sleep problems and puberty. This is not surprising as it is commonly reported that adolescents undergo a transition in their sleeping pattern, in that they fall asleep later in the evening and wake up later in the morning (Carskadon et al., 2004; Laberge et al., 2001). Furthermore, increased daytime sleepiness is often observed, possibly because of an incompatibility between the adolescent's preferred sleep pattern and the conventional daily routine. What is surprising, however, is that these sleep problems were not evident in our early puberty groups, despite parents often reporting this. It may therefore be that the newly-constructed sleep scale did not best identify the specific sleep problems in this population, and that other measures may be better suited to this construct. Another alternative may be that due to the smaller sample size of our CPP group, there was not

enough data at the higher end of the pubertal ratings to demonstrate the strength of the relationship with sleep problems.

Summary. The findings of this study support the hypothesis that those with a diagnosis of precocious pubertal development are at an increased risk of psychopathology, specifically anxiety and depression, overall internalising behaviours, aggression, social problems and overall externalising behaviours, as well as rule-breaking at school. Although there was evidence for this across several syndrome subscales, there is less certainty as to whether this increased risk is related to specific hormonal or morphological features of each disorder, changes related to pubertal development in general, or environmental effects, such as looking different or having to have hormone injections. It is likely that these effects differ between individuals and between behavioural disorders and that the only true way to measure the influence of each factor, would be to directly measure or control for them.

One example of an improvement to the study would be the use of hormonal assays. This would have shed some light as to whether the differences between groups may have been linked to hormonal fluctuations, as some problematic behaviours and psychological disorders evident in those with early puberty have been attributed to pubertal hormones (Angold et al., 1999; Cicchetti & Rogosch, 2007; Dorn et al., 1999, 2008; Herbert et al., 1996; Susman et al., 1985; Van Goozen, Matthys, Cohen-Kettenis, Thijssen & van England, 1998). If so, this would imply that these problems would occur across all individuals, irrespective of their pubertal onset. It may be, however, that if these difficulties occur at a time typically expected for puberty, the individual may be better equipped to manage these changes and therefore have a better level of resilience. Support for this theory comes from the findings of Sontag-Padilla and colleagues (2012), who

proposed that participants with a diagnosis of PA who showed greater executive function were less likely to display psychopathology. Other limitations of the study include the unequal sample sizes, and a low return rate of the teacher-reports. Because the effect sizes could potentially be fairly small, it may be that our sample was not big enough to demonstrate the strength of differences between groups. The study does however, make use of between-groups comparisons and variable relationships with pubertal development, providing clarification as to which findings are related to puberty as a general process, and which are related to the specific disorders and the environmental and social changes associated with this.

4.3 Self-Perception: Perceived Age and Self-Esteem

Adolescence is the time of an individual's life when they begin to learn more about themselves and develop preferences about who they socialise with, what they like to look like, and who they will be in the future. Since Erikson's work in the 1960's on adolescent identity formation, this period has been considered crucial in the transition from dependency into becoming one's own person. Through this independence, an individual forms their own identity, which can only come about by being aware of the self and how one compares to others. We might therefore expect those entering adolescence to become more self-aware and observant of differences between themselves and their peers, and conscious of whether others perceive these differences too. This heightened awareness and concern with how one is perceived has been reported in the literature (Harter, 1990; Parker, Rubin, Erath, Wojslawowicz & Buskirk, 2006; Rosenberg & Simmons, 1975; Vartanian, 2000), and has been coupled with neurological development in the medial prefrontal cortex, implicated in self-other evaluations (Pfeifer et al., 2013). This implies that increased self-appraisal combined with improvements in higher level cognitive functioning may lead to more evaluative thinking about the self and one's relationships with others. Adolescents may begin to perceive themselves with less satisfaction and more criticism, and this dissatisfaction may motivate the individual to change their appearance, their attitude and their social group to suit their perceived ideals. However, an inability to meet these perceived ideals could lead to unhappiness and a further decrease in self-esteem.

O'Dea and Abraham (1999) reported that pubertal status, rather than adolescence *per se,* was a key factor in self-esteem. Furthermore, they found that males who had reached puberty had the greatest self-perception, whereas females who had reached

menarche had the poorest self-perception. This suggests that not only does pubertal development affect self-perception in adolescents, but that it produces a dissociation between genders. This gender difference was supported by Simmons et al. (1979), although these authors also reported that pubertal development in combination with environmental changes, such as starting a new school or dating is most likely to cause changes in self-esteem. Cole and colleagues (2001) also found that self-concept became less stable during times of change, and that this was evident across several aspects of self-perception.

When looking at self-perception in relation to physical appearance, it has been found that, during puberty, this aspect of self-perception becomes unstable. For example, Benjet and Hernández-Guzmán (2002) reported that a decrease in females' bodily self-esteem occurs approximately 6 months after the onset of menarche, before which a temporary increase in bodily satisfaction is often observed. Much of the literature suggests that, whilst males become more satisfied with their body shape as they progress through puberty, girls demonstrate a decrease in self-esteem (Duke-Duncan, Ritter, Dornbusch, Gross & Carlsmith, 1985). Furthermore, girls entering puberty earlier who feel more negatively about their body and have lower self-esteem, demonstrate these changes to a greater extent than do peers who enter puberty late or on-time (Alsaker, 1992; Fend, 1994; Ge, Elder, Regnerus & Cox, 2001; Graber et al., 1997; Tobin-Richards & Kavrell, 1984). Blyth, Simmons and Zakin (1985) report that body dissatisfaction relates specifically to weight, and that no changes were observed in satisfaction with attractiveness or overall body image. However, Folk, Pedersen and Cullari (1993) demonstrated that general self-esteem is correlated to satisfaction with several features of physical appearance, and it is therefore likely that many features of the self-concept are interdependent.

It would appear that pubertal timing is related to the level of dissatisfaction experienced, particularly with regards to body image and physical development. This may be even more prevalent in the population of children with precocious pubertal development, as not only are these children experiencing the difficulties typically encountered during puberty, but also at a considerably earlier age than would be expected, and likely in isolation from their peers. This is supported by research by Brack, Orr and Ingersoll (1988) who reported that no relationship existed between pubertal stage and self-esteem, but that there was a difference between early and late-maturing male participants in their sample, suggesting that it is the asynchrony of the pubertal development with the peer group that is problematic, rather than pubertal stage. Furthermore, Blyth et al. (1985) reported that, although they had hypothesised that their early-maturing participants would perceive themselves to be at an advantage over their peers because of their more adult appearance, this was not found to be the case.

Research with children diagnosed with precocious puberty has indeed shown altered self-perception. For example, research by Solyom, Austad, Sherick and Bacon (1980) reported that children with a diagnosis of premature adrenarche (PA) had a negative self-image and were angry about their development. Those with a diagnosis of central precocious puberty (CPP), however, were very self-aware and felt embarrassed over their physical development. Similarly, Xhrouet-Heinrichs and colleagues (1997) found that their participants with a diagnosis of early pubertal development were self-conscious about their physical development and worried about looking different to their peers. This would suggest that experiencing puberty clinically early is indeed detrimental to a child's satisfaction with their physical appearance.

Changes in self-perception are also observed in social interaction. Children become more aware of their position in the social group as they reach adolescence, and begin to make comparisons between themselves and others. This can lead to dissatisfaction when one doesn't measure up to peers or stands out from the crowd. Adolescents begin to spend increasing amounts of time with their peer groups, in some cases twice the amount of time spent with adults (Csikszentmihalyi & Larson, 1986; Larson & Richards, 1991), and are steadily more influenced by others' opinions and behaviours.

However, Fend (1994) has suggested that those who begin pubertal development earlier perceive themselves to be socially accepted to a greater extent than those who develop later. This suggests that early development may give the child an advantage over their peers. Furthermore, when a child begins pubertal development, people may begin to perceive them as more adult, and therefore they may begin to be spoken to and treated differently than they were as a pre-pubertal child (Alsaker, 1995a; in Alsaker 1995b). It is unclear whether this is due to the child behaving differently as they believe that others notice their development, or because others truly are treating them differently. It may be, therefore, that beginning pubertal development at an early age, but still within the range considered 'normal', gives the child a higher status amongst their social group.

For those who begin puberty before the clinical thresholds of 7-8 years in girls and 8-9 years in boys, however, this development may not be of such an advantage. For children of secondary school age, puberty is a process they have learned about and something they are anticipating. For a child of this age to enter puberty before their peers possibly identifies them as the leader, and their peers have a desire to catch up and not to be the last in their group to develop. For those in primary school, puberty is largely unknown and a child who has reached puberty will appear to have something 'wrong' with

them because they look different. Any advantage associated with early puberty that is still within the typical range, may therefore not be evident in children with clinically early pubertal development. This was observed by Xhrouet-Heinrichs et al. (1997) who found that the majority of those with a diagnosis of CPP felt unhappy with their friendships and described themselves as lonely.

The aim of this study was to identify whether children with a diagnosis of PA or CPP would have altered levels of self-perception, in comparison to each other and to a control group. The first area of self-perception under study was perceived age, where exploratory analyses were conducted to assess whether the PA and CPP groups perceived themselves as looking older, and whether they would report higher negative consequences of looking older in comparison to the typically-developing group. This analysis was repeated for the parent-completion version of the scale, and comparisons made between the child and parent responses.

It was also hypothesised that the two clinical groups would score lower on scales of self-esteem, where a high score denotes a positive self-perception, and a low score negative perception. Specifically, it was hypothesised that significant differences would be observed between the patient groups and the typically-developing group in their perception of physical appearance and social acceptance, but not for perception of scholastic or physical competence, behavioural conduct and global self-worth. Exploratory analyses were also conducted to explore whether relationships existed between any of the self-perception variables and pubertal development.

Methodology. For full information on the recruitment procedure and the inclusion criteria, please see chapter 3.2. Demographic information can be found in Table 1. Parents and children both completed a Perceived Age Questionnaire (PAQ), which was newly

constructed for this project. Reliability and validity properties of the scale can be found in Appendix 15. This scale included items on whether the child looked older or bigger than their peers, whether they were treated as older in a negative way, such as people making jokes about their size, or whether being treated as older was a positive thing, perhaps by being given more responsibility or looking 'grown up'. Children also completed a self-perception scale with the help of the experimenter. The self-perception scale was either the Pictorial Scale of Perceived Competence and Social Acceptance for Young Children (PSPCSA: Harter & Pike, 1984) or the Self-perception Profile for Children (SPPC: Harter, 1985). The PSPCSA is designed for children between 4-7 years of age, whereas the SPPC was completed by those aged 8 years and above. The reported reliability and validity statistics can be found in chapter 3.3.1.6. These measures assess self-perception across physical, cognitive and social capacities, as well as satisfaction with behavioural conduct and overall self-worth, where a higher score indicates higher satisfaction. The statements on the PSPCSA are presented alongside pictures for ease of understanding, and are focused on topics such as whether people share toys with them, or whether they are good at climbing. These items fall under four subscales; scholastic competence, physical competence, peer acceptance and family acceptance, whereas the SPPC consists of scholastic competence, physical competence, physical appearance, peer acceptance, and satisfaction with behavioural conduct and with global self-worth.

Results. No significant differences were identified between groups for age, gender, socioeconomic status (SES) or ethnicity. Tests of distribution and variance were carried out prior to analysis. Where these tests were significant, non-parametric analyses were conducted.

- *PAQ.* The means and standard deviations for each subscale on both the parent and child measures are presented in Table 36.

Table 36: *Means and standard deviations for both the child and parent PAQ subscales*

	Parent			Child		
	Looking Bigger/ Older	Negatives	Positives	Looking Bigger/ Older	Negatives	Positives
Control	.62 (.62)	.18 (.31)	.63 (.50)	.88 (.49)	.27 (.27)	.98 (.49)
PA	1.10 (.59)	.36 (.43)	.81 (.63)	1.07 (.45)	.29 (.27)	.93 (.57)
CPP	1.19 (.52)	.42 (.56)	.72 (.50)	1.06 (.42)	.33 (.26)	.90 (.57)

Non-parametric between-group analyses identified a significant difference between the three groups on the parental report of their child looking bigger/ older than their peers ($H(98)= 15.85$, $p< .001$) where the CPP group scored highest, and the control group lowest. Mann-Whitney post-hoc tests with a Bonferroni correction identified that significant differences lay between the control group and the two clinical groups (Control-PA: $U(46, 35)= 460.00$, $z= -3.30$, $p= .001$; Control-CPP: $U(46, 15)= 154.50$, $z= -3.21$, $p= .001$). Furthermore, significant trends were identified for the looking bigger/ older on both the parent ($J= 1969.50$, $z= 3.87$, $p< .001$) and child scales ($J= 1753.50$, $z= 1.97$, $p= .049$), and the perceived negatives of looking older on the parent scale ($J= 1676.0$, $z= 2.03$, $p= .042$). Reanalysis with the two clinical groups collapsed into one clinical group identified significant differences between this group and the controls on both parent and child measures of the child looking bigger/ older (parent: $U(46, 50)= 614.50$, $z= -3.94$, $p< .001$; child: $U(48, 50)= 898.50$, $z= -2.15$, $p= .031$), and the parent negatives of looking older

scale (U(46,50)= 886.50, z= -2.15, z= .031), with the clinical group scoring higher across all three scales.

Spearman's correlational analyses demonstrated relationships, firstly between child age and all three subscales on the parent measure, and secondly, between pubertal development and all subscales on both parent and child measures, except for the positive consequences subscale. After controlling for the effects of age, the relationships between puberty and the subscales remained significant. These are presented in Table 37.

Table 37: *Spearman's correlations between the parent and child PAQ subscales and age, pubertal development, and pubertal development controlling for age*

	Parent PAQ			Child PAQ		
	Looking Bigger	Negatives	Positives	Looking Bigger	Negatives	Positives
Age	.24*	.33**	.28**	.18	-.02	-.15
Tanner Stage	.55***	.34**	.31**	.36***	.22*	.04
Tanner Stage (controlling for age)	.46***	.30**	.25*	.32**	.30**	.15

Note *p< .05 ** p< .01 ***p< .001

Curve estimation analysis of the predictive value of pubertal stage identified it as a significant predictor of all three subscales on the parent measure and the looking bigger/ older and negative consequences variables of the child measure. The output for this is presented in Table 38.

- *PSPCSA and SPPC- Hypothesis Testing.* Where the PSPCSA and SPPC had congruent subscales, these were combined and analysed as one group for the self-perception analysis. Descriptive statistics are given in Table 39.

Between-groups ANOVA analyses identified no significant differences on any of the PSPCSA or SPPC subscales. Similarly, there were no significant differences between control participants and the PA and CPP groups combined into a single patient group.

- *PSPCSA and SPPC- Exploratory Analysis.* Exploratory analysis of relationships between variables in the data was then conducted. Correlational analyses for all participants demonstrated a significant relationship between the scholastic competence subscale and pubertal development ($r = -.30$, $p = .003$), as well as relationships between age and the scholastic competence ($r = -.48$, $p < .001$) and athletic competence subscales ($r = -.49$, $p < .001$), and SES with global self-worth ($r = .30$, $p = .018$). The correlational analyses were repeated controlling for SES and age, and demonstrated that pubertal development and scholastic competence were now more strongly correlated, where increased pubertal development was related to decreased perceived scholastic competence ($r = -.33$, $p = .011$).

Table 38: *Regression models derived from curve estimation analysis, for the parent and child PAQ subscales*

	Dependent	Model	R^2	Constant	Unstandardised β	F (df)	Sig.
Parent	Looking Bigger/ Older	Quadratic	.31	-1.02	1.68, -.29	20.82 (2, 93)	.000
	Negative Consequences	Linear	.12	-.11	.23	13.30 (1, 94)	.000
	Positive Consequences	Linear	.11	.22	.29	11.05 (1, 94)	.001
Child	Looking Bigger/ Older	Linear	.13	.51	.27	13.75 (1, 95)	.000
	Negative Consequences	Linear	.06	.10	.11	6.30 (1, 95)	.014

Table 39: *Means and standard deviations for the PSPCSA and SPPC subscales*

	Scholastic Competence	Athletic Competence	Physical Appearance	Social Acceptance	Behavioural Conduct	Global Self-Worth	Family Acceptance
Control	3.12 (.57)	3.06 (.61)	3.00 (.55)	3.06 (.69)	3.32 (.43)	3.35 (.56)	3.02 (.50)
PA	3.02 (.65)	2.87 (.70)	3.12 (.68)	3.01 (.79)	3.19 (.54)	3.64 (.49)	2.95 (.61)
CPP	2.98 (.81)	2.87 (.63)	2.65 (.66)	3.03 (.86)	3.48 (.56)	3.50 (.67)	2.53 (.40)

Backward regression modelling suggested that whilst puberty was a significant predictor of perceived scholastic competence, this was superseded by age (See Table 40). Both variables were retained in the model however, suggesting that puberty does have significant predictive value.

Table 40: *Linear regression model for scholastic competence, with puberty and age as predictors*

Dependent	Predictors	Std. β	t	Sig.	R^2	F	Sig.
Scholastic Competence	Constant		13.83	.000	.24	14.63	.000
	Age	-.40	-4.15	.000			
	Mean Tanner Stage	-.17	-1.77	.081			

The perceived age and self-perception variables were entered into a Spearman's correlation, to identify whether those that perceived themselves as older also perceived themselves differently across other domains. Age was controlled for; findings are presented in Table 41. It was observed that participants who perceived themselves to have lower academic ability, reported more negative consequences of looking older. This was also evident in their parents' reports of negative consequences. Children who perceived their physical appearance negatively also reported more negative consequences of looking older, and this was also evident on the parent scales. Those who perceived themselves as looking bigger or older however, reported being more popular. Participants who perceived their behaviour positively, reported more negative consequences to looking older, and finally, those with greater self-worth perceived themselves as looking older, but having more positive consequences.

Table 41: *Correlation analyses controlling for age, between the PAQ subscales and SPPC and PSPCSA subscales*

		Scholastic Competence	Physical Competence	Physical Appearance	Peer Acceptance	Behavioural Conduct	Global Self-Worth
Parent	Looking Bigger/ Older	-.03	.10	-.18	.11	.01	.15
	Negatives	-.36**	-.16	-.49***	-.23	.10	-.12
	Positives	.05	.13	.90	.12	.05	.18
Child	Looking Bigger/ Older	-.07	.16	-.08	.25*	.05	.30*
	Negatives	-.31*	-.11	-.29*	-.15	.26*	.01
	Positives	-.18	.15	-.01	.09	.03	.27*

Note *p< .05 **p< .01 ***p< .001

Discussion. This study aimed to investigate the way in which children with precocious pubertal development perceive themselves in comparison with their peers. Another aim was to investigate whether children perceived themselves as looking older or bigger than their peers, and if so, whether they had encountered positive or negative consequences for looking older. This study was carried out to validate many of the other hypotheses in the thesis, as it is easy to assume that children are aware that they are different to their peers, and that looking several years older than their chronological age is experienced negatively, whereas this may not necessarily be the case.

The findings suggest that parents of the clinical groups do notice that their child looks older or bigger than their peers, and perceive others to treat them as such. Furthermore, parents were sensitive to the extent of this difference in perceived age, as those with a diagnosis of CPP were perceived as looking older than those with PA. In the analysis of the children's data, there was no difference between the two clinical groups, although the combined clinical group did report looking significantly bigger or older than did the control group. Furthermore, parents showed a greater awareness of the negative consequences of looking older than did the children, and parents of the two clinical groups reported these negative consequences to occur more than did parents of the control group. It was also observed that, as the child's pubertal development increased, both the child and parent perceptions of the child's perceived age increased. Furthermore, as pubertal development increased, so did both parent and child reports of the negative consequences of looking older, and parent reports of the positive consequences of looking older. It is interesting that there were no differences in the reports of positive consequences of being perceived as older in the child data. When looking at the descriptive statistics, all groups reported similarly high levels of these positive consequences, suggesting that all children

perceived growing up to be positive, but that the children and parents in the patient groups were also aware of the negative consequences.

Pubertal development was also found to be predictive of the PAQ scores. Although the majority of the regression models presented in the results are linear, in a number of cases the data also fit quadratic or cubic models just as well. Although one generally chooses the simplest model to fit the data when there is little difference in the standard error or predicted variance, it may be that when considering the real-life application of the findings one might consider a non-linear relationship to be more representative of the actual trajectory. For example, the perception of looking older or bigger than peers is more likely to level out over time as others catch up in their development, and therefore a relationship that may be linear as a child progresses through puberty will level off towards the end of development. It would have been interesting to continue the research with those who had completed pubertal development to observe how the regression trajectory changed after puberty.

The findings of this study do not suggest that having a diagnosis of early pubertal development has any effect on self-perception, and perception of oneself in relation to their family and peers. One explanation for this may be that children are not necessarily aware that they are different to their peers, and therefore have similar levels of satisfaction in their self-perception as they would have if they did not have the condition. In this way, any pubertal development would not impact on their self-perception, because it is not a part of their self-perception. The results of the PAQ, however, imply that children are aware that they look bigger or older than their peers and that people treat them accordingly, and this explanation is therefore unlikely. Despite being aware of physical differences, however, they may be less aware of their differences in mood and behaviour,

or that their social integration is different to the way it was before the onset of their disorder. An alternative explanation is that the clinical participants are aware of the differences but this has not altered their perception of their abilities or social acceptance. Children may see the disorder as separate to the aspects of self-perception being studied here, and therefore do not consider themselves to be any different in these areas. A further suggestion is that clinically early pubertal development actually does not impact on any of these areas and their resulting self-perception, in which case we would not expect to see any differences. For example, if a child does not develop social difficulties as a result of their pubertal development, then we would not observe changes in their perception of this.

The correlational and regression data suggests that being older and of earlier pubertal development was related to lower perceived scholastic competence. This relationship was of moderate strength, and persisted when SES was taken into consideration. One suggestion for this is that as children grow older and progress through puberty they lose confidence in their academic ability and perceive themselves as having lower scholastic competence, or that they develop increased modesty and therefore report their perceived ability as lower than they actually believe it to be. Research by Eccles and colleagues has suggested that this decrease in perceived competence at school is related to the transition from primary school to secondary school, and becoming the youngest and most inferior pupils in their new school compared to previously being the most superior (Eccles et al., 1989; Wigfield, Eccles, MacIver, Reuman & Midgley, 1991). This transition also happens to occur at a time close in proximity to the onset of pubertal development, and it may therefore be that these papers have made an oversight by not controlling for this variable in their analyses. In our sample, however, all participants were of primary school age, and therefore the transition between schools cannot be responsible for the change in

perceived scholastic competence. A final possibility is that there actually is a change in academic ability as children enter puberty, perhaps in concentration or memory (See 4.4). Either way, it does appear that the children in our sample who were progressing into adolescence, both chronologically and biologically, believed themselves to be less competent in their studies than those who remained prepubertal.

The main limitations of this study are that children may have demonstrated a social desirability bias and not responded truthfully to the questions. For example, children may not want to admit to being unhappy with their self-perception, and therefore respond more positively than they would if answering honestly. As none of the scales included items to detect social desirability, it was not possible to determine if this was the case. Another limitation is that children may not have fully understood what was asked of them. This was not evident during data collection, as children appeared happy to ask for clarification and considered their answers carefully before responding. A further limitation is that the measures employed in this project perhaps did not assess the specific areas of self-perception affected by pubertal development, and that questions more specific to weight and body shape, or peers' comments may have been a better measure.

In an article by Biro, Striegel-Moore, Franko, Padgett & Bean (2006) there is some discussion as to whether self-esteem is a stable trait, or whether self-perception fluctuates in response to the current environment and changes over the life-span. The authors suggest that there is evidence for both, and that while a child's self-esteem may alter in response to changes in their BMI or transition to a new school, there is likely stability in their self-perception over time. If this is indeed the case, clinical participants' self-perception may vary over the course of their disorder. For example, a child at the onset of early puberty may have lower self-esteem than a child who has been receiving hormone therapy for

several months, or a child who is at the onset of puberty but at the age where this is typically expected. An improvement on this project therefore, would be to either test the participants on measures of self-perception at several time-points during the disorder, or to add time since pubertal onset as a variable and to analyse fluctuations in self-perception against this.

Summary. Overall, the study has demonstrated that children with a diagnosis of PA and of CPP do look older or bigger on both self- and parent-reports. Furthermore, there appear to be positive consequences to looking older, but also negative consequences that the clinical groups experience, that typically-developing children do not. Although aware of the negative side of developing early, children with precocious pubertal development do not seem to develop a particularly negative perception of themselves across any domain, although pubertal development does appear to be related to a perceived lower efficacy in scholastic competence.

These findings have application for medical professionals, who may find this useful when providing families with information about the benefits of undergoing hormone treatment, or preparing families for what to expect as their child progresses through puberty. Furthermore, teachers may find these results useful when trying to understand children's motivation at school. Children may need extra reassurance to increase their confidence in their academic ability, to prevent them becoming disheartened and losing interest in school. In summary, it is important to consider how a child perceives themselves, as well as how others perceive them, in order to fully understand the ways in which they may be affected by their earlier pubertal development.

4.4 Intellectual Ability

Puberty and adolescence is a period of development, in both neurological structure and function. It has been well-documented that structural changes include general increases in white matter and corpus callosum volume, and non-linear changes in grey matter volume, as well as changes in density and quality of neuronal structures (Barnea-Goraly et al., 2005; Giedd et al., 1999; Jernigan, Trauner, Hesselink & Tallal, 1991; Lenroot et al., 2007; Paus et al., 1999; Pfefferbaum et al., 1994; Sowell et al., 1999; Thompson et al., 2000). Giedd et al. (1999) found that these changes in volume peak at 11 years in females and 12 years in males, which they argue is related to the average onset of gonadarche, although no direct measures of pubertal development were taken. This conclusion has been supported by several research groups (Gogtay et al. 2004; Lenroot et al., 2007). For example, De Bellis and colleagues (2001) studied the relationship between structural changes and age in children and adolescents, as well as with markers of pubertal development, and found that there was a significant interaction between pubertal development and age, when measuring neurological changes in the corpus callosum as well as white and grey matter volumes. The authors concluded that there is a significant endocrinological impact on neurological structure during puberty. Furthermore, van Soelen et al. (2012) found that girls who were further through pubertal development had a lower density of grey matter than girls who were prepubertal, and recent work by Blanton and colleagues (2012) demonstrated that puberty was associated with bilateral volume of the hippocampus and amygdala. This supports the evidence for a hormonal basis for neurological changes during the pubertal period.

More specific structural changes reported during adolescence include an increase in the volume of the amygdala and a decrease in the hippocampus. For example, Bramen et

al. (2011) found an interaction between gender and Tanner stage on the volume of the hippocampus, amygdala and cortical grey matter, whereby males with greater pubertal development demonstrated a greater volume in neurological structures compared to smaller volumes in more developed girls, suggesting a dissociation between genders. These findings were consistent even after controlling for chronological age. Similarly, Neufang and colleagues (2009) found a significant relationship between neurological changes in the amygdala and hippocampus, and pubertal markers across genders. They also demonstrated a predictive relationship between gonadal steroids and sex-specific structural changes, suggesting that testosterone in males could be linked to amygdala volume, whereas oestrogen in females is likely related to volume of grey matter in the parahippocampal gyrus. Other research has found sex steroids to have an effect on structure, excitability and maintenance of the hippocampus, and therefore hypothesised that they may be influential on memory and emotion (Cahill, 2006; MacLusky, Hajszan, Prange-Kiel & Leranth, 2006; Parducz, et al., 2006). Research into the role of other hormones present during puberty has reported that secretion of Luteinizing Hormone (LH) is predictive of white matter growth, whilst estradiol is related to changes in grey matter (Van Soelen et al., 2012). Furthermore, there is evidence for the impact of sex steroids on the hypothalamus (Blakemore, Burnett & Dahl, 2010), visual cortex (Nuñez, Huppenbauer, McAbee, Juraska & DonCarlos, 2002) and pathways associated with reward (Sato, Schulz, Sisk & Wood, 2008). This suggests that puberty is a time of sexual dimorphism, and that the change in hormone levels present during puberty has an organisational role in brain development, which differs between males and females. Animal studies have supported the hypothesis that hormonal changes during puberty

impact on neurological structure and function, however this process is less well understood in humans (Cahill, 2006; Petanjek et al., 2011; Sisk & Foster, 2004).

Researchers trying to amalgamate the evidence for neurological development, circulation of sex steroids and changes in cognitive functioning differ greatly in their conclusions. Yurgelun-Todd, Killgore and Cintron (2003) presented regression data in which larger amygdala volume was predictive of spelling, reading, number, verbal IQ and total IQ in males, and increased hippocampal volume was predictive of reading. In females, amygdala volume was positively predictive of spelling, verbal IQ and total IQ, but hippocampal volume was negatively predictive of spelling, number and delayed recall. These findings were corrected for total brain volume, but not for age. However, in her discussion of the literature on the relationship between spatial ability and sex steroids, Liben et al. (2002) concluded that evidence for an influence of hormones present during puberty on spatial skills is complex and controversial at best. Liben's own research assessed changes in spatial ability in patients who were receiving treatment for delayed puberty, and again found no significant effect of treatment, suggesting no direct effect of sex hormones on spatial cognition. Furthermore, in a study investigating intellectual ability in women with a diagnosis of congenital adrenal hyperplasia, Malouf and colleagues found androgen exposure to have no impact on cognitive abilities (Malouf, Migeon, Carson, Petrucci & Wisniewski, 2006). The overwhelming evidence suggests that there is a negligible relationship between sex steroids and cognition during puberty, yet sex differences on a variety of cognitive tasks continue to be published. However, Herlitz and colleagues point out that age has not been consistently controlled for in many studies, and that any conclusions should be accepted with caution (Herlitz, Reuterskiöld, Lovén, Thilers & Rehnman, 2013).

Early research on the cognitive abilities of children with altered pubertal timing focused on evidence for altered lateralisation of language and spatial abilities. Waber (1977) presented some of the earliest data on this clinical group, and suggested that fast maturation or earlier puberty prevented hemispheric lateralisation, resulting in reduced spatial ability. Others have tried to replicate her work using a variety of tasks, such as dichotic listening, dichhaptic shape manipulation, mental rotation, and tachistoscopic trials with varied results (Newcombe & Bandura, 1983; Newcombe, Dubas & Baenninger, 1989; Rovet, 1983). More recently, Mul and colleagues (2007) investigated intellectual functioning in adopted children being treated for central precocious puberty (CPP), as CPP has been shown to occur more frequently in the adopted population (Baron, Battin, David & Limal, 2000; Krstevska-Konstantinova et al., 2001; Mason, Narad, Jester & Parks, 2000; Proos, Hofvander & Tuvemo, 1991; Soriano-Guillen et al., 2010; Teilmann, Pedersen, Skakkebæk & Jensen, 2006). Children with CPP performed better on subscales of verbal ability than performance subscales, although both of these scores were comparable to the typical population. The authors however, also reported that children decreased in IQ by nearly 10 points over the course of hormonal treatment. They argue that this may be due to a reduction in the impact of sex steroids on the left hemisphere, and a resulting drop in IQ to the level typically expected for their age. A series of studies by Dorn and colleagues has investigated several psychological variables in a group of girls with a diagnosis of premature adrenarche (PA) (Dorn et al., 1999, 2008; Dorn, Susman & Ponirakis, 2003; Tissot et al., 2012). In their 2012 paper, the authors reported no significant differences in neuropsychological functioning between their patient group and controls, but observed a general advantage for the control group over patients. This suggests that the adrenal hormones circulating during adrenarche may not be as influential

on neurological systems as the sex steroids released during gonadarche. Similarly, Nass, Baker, Sadler and Sidtis (1990) found their participants with a diagnosis of PA to score no differently from others of their age on tests of IQ, although they noted that participants who were older and had been diagnosed with PA several years previously, had lower scores than both the younger participants in the PA group and also the typical population. However, the authors still suggest that early androgen exposure affects neural organisation, in particular the lateralisation of language, although this conclusion is heavily based on differences in dichotic listening tasks in a small sample.

The aim of the current study was to investigate the suggested relationship between pubertal development and cognitive functioning, by comparing performance on the British Ability Scales III (BAS III) between three groups; children with a diagnosis of CPP, children diagnosed with PA, and typically-developing children. It was hypothesised that children in the PA groups would score significantly lower on the verbal subscale than participants in the other two groups. In addition, differences between males and females were studied through exploratory analyses, as well as relationships between Tanner staging and scores on the BAS III tasks. Observation of significant differences between the PA and CPP groups would suggest an endocrine basis due to the differing hormonal profile of these two disorders. If the two clinical groups were significantly different to the control group but not to each other, it may be that the environmental and social impact of having a pubertal disorder has had an effect on cognitive functioning.

Method. Information on recruitment and the inclusion criteria is given in 3.2, and details on the BAS III are given in 3.3.1.7. Children completed the BAS III with the lead investigator, and parents completed demographic and pubertal rating questionnaires.

Results. Tests of distribution, variance and covariance were conducted for each set dataset. Non-parametric statistics were used for the analysis, as in virtually all scales at least one group was of a non-normal distribution, and in some cases inhomogeneous variance was also evident. No significant differences were observed between the three groups, for age or socioeconomic status (SES).

- *Hypothesis Testing.* Differences between the three groups on the core subscales were analysed first. The core subscales included tasks such as design recognition, pattern construction and quantitative reasoning (See Table 42 for descriptive statistics). Kruskal-Wallis analyses demonstrated that groups performed similarly except for on the word definition subscale ($H(2, 97)= 7.62$, $p= .022$). Post-hoc Mann-Whitney analyses with a Bonferroni correction demonstrated that the significant difference lay between the control and CPP groups ($U(47,15)= 204.00$, $z= -2.44$, $p= .015$). After combining the PA and CPP groups into a single group to identify whether the difference between clinical participants and controls remained, significant differences were observed again on the word definition task ($U(48, 50)= 835.00$, $z= -2.46$, $p= .014$).

($U(48, 50)= 910.50$, $z= -2.06$, p- .039), but also on the delayed spatial recall task ($U(48, 50)= 895.00$, $z= -2.17$, $p= .030$).

Table 42: *Means and standard deviations on each of the core subscales of the BAS III*

	Design Recognition	Word Definition	Pattern Construction	Matrices	Verbal Similarities	Quantitative Reasoning
Control	48.45 (8.50)	52.55 (10.02)	52.48 (10.01)	47.21 (10.30)	56.11 (8.14)	52.32 (10.36)
PA	48.06 (8.18)	49.06 (8.15)	52.17 (6.99)	46.57 (10.40)	54.89 (8.85)	52.26 (8.86)
CPP	48.67 (8.16)	45.67 (8.03)	53.20 (9.37)	49.73 (10.98)	55.27 (7.58)	51.33 (10.27)

Table 43: *Means and standard deviations on each of the diagnostic subscales of the BAS III*

	Immediate		Delayed		Forward Digit Recall	Speed of Information Processing	Picture Recognition	Backward Digit Recall
	Verbal Recall	Spatial Recall	Verbal Recall	Spatial Recall				
Control	52.56 (11.05)	49.10 (11.49)	51.98 (11.09)	50.04 (8.90)	50.71 (6.50)	55.23 (8.85)	48.26 (10.31)	54.19 (7.41)
PA	50.06 (11.15)	46.57 (8.53)	53.83 (11.63)	46.83 (7.77)	49.40 (8.65)	59.34 (10.85)	51.69 (8.93)	54.77 (8.08)
CPP	49.87 (12.03)	45.47 (8.40)	48.33 (9.07)	47.00 (8.15)	53.87 (13.76)	63.20 (10.32)	50.00 (9.74)	57.87 (10.31)

The diagnostic subscales of the BAS III were then analysed using the Kruskal-Wallis method. These subscales included digit recall tasks and speed of processing (See Table 43 for descriptive statistics). No significant differences were identified between the three groups, except for a near significant difference on the speed of information processing task ($H(2, 98)= 5.96$, $p= .051$). Post-hoc analyses demonstrated that the significant difference lay between the control and CPP groups ($U(48, 15)= 208.00$, $z= -2.46$, $p= .014$).When comparing the combined clinical groups to the control group, significant differences were again observed on the speed of information processing tasks

The ability scales of the BAS III, which consisted of reading, spelling and number skills, were then analysed (Descriptive statistics are given in Table 44). No significant differences were observed between the three groups, nor when the clinical groups were combined and compared with the control group.

Table 44: *Means and standard deviations on each of the ability subscales of the BAS III*

	Number Skills	Spelling	Reading
Control	106.32 (11.94)	103.05 (13.67)	104.15 (12.49)
PA	104.91 (13.08)	108.06 (13.14)	108.17 (12.03)
CPP	108.27 (12.19)	107.27 (10.72)	104.07 (10.56)

Finally, the total scale values of the BAS III were analysed for differences between the three groups. Descriptive statistics are given in Table 45. No significant differences were identified on the Kruskal-Wallis analysis, but when the two clinical groups were combined and compared to the control group, a significant difference was observed on the total verbal scale scores ($U(48, 50)= 887.50$. $z= -2.22$, $p= .026$).

Table 45: *Means and standard deviations on each of the total scales of the BAS III*

	Total Verbal	Total Nonverbal	Total Spatial	GCA
Control	106.92 (13.11)	98.27 (14.86)	100.92 (14.90)	102.63 (14.42)
PA	102.83 (12.56)	98.20 (13.46)	100.11 (10.65)	100.43 (12.12)
CPP	100.00 (11.36)	100.13 (14.85)	102.00 (13.63)	100.73 (14.35)

- *Exploratory Analysis.* Exploratory analysis of the data was then carried out in order to assess the strength of any relationships between variables in the data. Jonckheere-Terpstra analyses identified a significant trend across the three groups on the word defimition task, where the control group scored highest and the CPP group the lowest (J= 1029.50, z= -2.79, p= .005). Furthermore, trend analysis identified significant trends across groups on the speed of information processing task, where the CPP group scored highest and the control participants scored the lowest (J= 1807.50, z= 2.33, p= .02), and on the total verbal scale scores (J= 1114.50, z= -2.35, p= .019), whereby the control group scored the highest, and the CPP group the lowest. Spearman's correlational analyses showed a significant relationship between pubertal development and performance on several tasks. However, after controlling for SES, pubertal development was only significantly related to speed of information processing (r= .25, p= .016). Regression analyses identified a linear model, in which pubertal development was a significant predictor of performance on the speed of information processing scale (R^2= .04, constant= 52.00, β= 3.34, F(1, 95)= 4.22, p= .043).

Due to the commonly reported gender differences in intellectual domains, differences on the subscale and total scale scores on the BAS III were analysed between males (n= 18, mean age= 101.33, SD= 21.39) and females (n= 83, mean age= 103.14, SD=

17.38) across all three groups. The descriptive statistics are given in Tables 46-48. Kruskal-Wallis analyses identified significant differences between male and female scores on the subscales of delayed verbal recall ($U(81, 17)= 404.50$, $z= -2.67$, $p= .008$) and picture recognition ($U(81, 16)= 440.00$, $z= -2.02$, $p= .043$). No significant differences were observed on any of the other subscales or total scale scores.

Table 46: *Means and standard deviations for males and females on the core subscales of the BAS III*

	Design Recognition	Word Definition	Pattern Construction	Matrices	Verbal Similarities	Quantitative Reasoning
Males	49.13 (7.87)	52.88 (10.29)	52.82 (10.81)	45.71 (10.91)	56.25 (6.95)	54.06 (11.53)
Females	48.19 (8.36)	49.70 (9.13)	52.41 (8.47)	47.72 (10.30)	55.40 (8.52)	51.77 (9.37)

Table 47: *Means and standard deviations for males and females on the diagnostic subscales of the BAS III*

	Immediate Verbal Recall	Immediate Spatial Recall	Delayed Verbal Recall	Delayed Spatial Recall	Forward Digit Recall	Speed of Information Processing	Picture Recognition	Backward Digit Recall
Males	53.76 (11.61)	50.12 (13.03)	57.82 (10.58)	49.29 (9.71)	53.12 (5.05)	55.82 (10.57)	45.75 (8.41)	56.63 (8.12)
Females	50.73 (11.11)	47.12 (9.38)	50.88 (10.83)	48.25 (8.24)	50.22 (9.26)	58.36 (10.08)	50.56 (9.87)	54.64 (8.18)

Table 48: *Means and standard deviations for males and females on the ability and total scales of the BAS III*

	Number	Spelling	Reading	Total Verbal	Total Nonverbal	Total Spatial	GCA
Males	106.75 (13.36)	107.44 (13.61)	107.00 (12.31)	97.65 (10.68)	97.65 (14.68)	101.71 (15.73)	103.06 (14.35)
Females	105.99 (12.19)	105.12 (13.13)	105.31 (12.07)	103.65 (12.87)	98.72 (14.24)	100.60 (12.73)	101.23 (13.42)

Discussion. The current chapter aimed to identify whether children with precocious pubertal development performed any differently to typically-developing children on measures of intellectual ability. It was found that the participants with a diagnosis of CPP performed poorer than did control participants on the word definition task. Furthermore, a trend could be observed on this task, as well as on the total verbal scale scores, where those in the CPP groups scored lowest, the control group highest with the PA group falling in between. This would suggest that over the course of puberty children become less competent in their verbal ability, particularly in their knowledge of word definitions. This may be related to having a poorer knowledge of words, lower confidence, or a lower ability to explain the word's definition, although why this would occur as children pass through pubertal development is unclear.

Differences in verbal ability according to pubertal development, have been reported in the literature, typically with the trend that those with greater pubertal development show improved verbal ability (Galatzer, Beth-Halachmi, Kauli & Laron, 1984; Mul et al., 2007). The data presented here, however, appear to show that children with advanced puberty have poorer verbal ability than their typically-developing peers, and that this develops as pubertal development progresses. Although this deficit is not clinically significant, it is still noteworthy. One explanation for this may be that patients commonly have to miss school for hospital appointments or to receive their hormone treatment. As the hormone treatment can be very uncomfortable, many families report that the entire day is taken off from school. However, we might expect other intellectual abilities to also be affected by this absenteeism, and it is unlikely that this would explain the reduced ability specifically in word definition knowledge. It is more likely that this deficit is related to puberty as a typical process, rather than a deficit that is specific to the clinical groups. It is unclear

however, why this was not evident in the correlational analysis. It may therefore be the case that other factors, such as SES, age, or motivation to peform well at school, also contribute to verbal ability over the course of puberty.

A second finding is that the clinical groups were able to process information more quickly than controls. This ability was assessed using a task in which the participant had to find the largest number or biggest quantity in a series of arrays, whilst being timed. This finding was significant when the clinical groups were collapsed into one group, as well as when they were analysed individually. Performance was highest for those in the CPP group and lowest for the control group, with the PA group in the middle. This finding would imply that the clinical participants are able to process information more quickly than the control group. However, pubertal development was also found to be predictive of performance on the speed of processing task, which supports the conclusion that children process information faster as they progress through puberty, rather than this being an advantage specifically in the clinical groups.

Surprisingly, no differences in intellectual ability between genders were observed. There were, however, differences on subscales of delayed verbal recall, where boys outperformed girls, and picture recognition, where girls outperformed boys. It is unclear why these two subscales would demonstrate these sex differences, and it is possible that this finding has been confounded by the considerably smaller sample size in the male group. It is also worth noting, that these two subscales formed part of the diagnostic battery, and therefore do not contribute to the total scale scores of verbal, non-verbal and spatial ability. As the aim of this study was not to identify differences between the two genders, and because PA and CPP is considerably more prevalent in girls, the lack of differences between genders on the core subscales, ability scales and total scale scores is

reassuring, as it would suggest that analysing males and females together in each of the experimental groups does not confound the analysis.

Summary. Overall, it would seem that children with a diagnosis of PA or CPP perform no differently to typically-developing children on assessments of intellectual ability, and are functioning well within the average range expected for their age. There is some evidence that the CPP group performed less well in comparison to the control group on verbal tasks, but better on information processing tasks. However, it is crucial to acknowledge that the sample size was small in this group, and that this limitation will have affected the strength of the findings presented here. Furthermore, it would appear that the findings are more representative of changes that occur over the course of pubertal development, rather than a phenomenon that occurs specifically in the patient groups. However as this study was cross-sectional, rather than longitudinal, this can only be inferred from the data, and has not been explicitly tested.

Patients with a diagnosis of early puberty can tell us a great deal about the impact of sex steroids and other hormones on neurological development and cognitive functioning. Further research should also consider the use of hormonal assays, or even MRI data, to complement the pubertal rating scales, and aim for a larger sample in order to compare between genders as well as diagnostic groups. Practically, however, these findings would suggest that care should be taken when working with children with early puberty in education, to ensure that they fully understand the information they are given. Furthermore, these children may benefit from extra support on tasks that require verbal skills, in order to ensure that they achieve their full potential.

4.5 Family Environment and Parental Stress

The family environment is important in shaping a child's psychological and physical development, but similarly problematic child development can influence the stability of the family environment. In addition to the anxiety over the health of their child, parents may have the stress of balancing working hours with medical appointments, or having to ensure that the child's condition is managed at school or when they visit friends. Furthermore, it may be difficult to explain the diagnosis to their child or to others, and to console the child if they become upset about their diagnosis. We might therefore expect families of children with a medical diagnosis to have higher stress levels than they would otherwise.

For parents of children with precocious pubertal development there are several additional difficulties we may expect parents to find stressful. The nature of the diagnosis itself is sensitive, and parents may find it difficult to find a suitable explanation of the diagnosis for young children to understand. Similarly, parents may not feel comfortable with others knowing about the condition, and struggle to hide this from the school or from the child's peers. Parents may feel uncomfortable that their child is developing adult sexual characteristics, and find this hard to come to terms with. This is in addition to having to take the child for invasive medical examinations and tests for serious conditions, such as neurological abnormalities or disorders of the reproductive or adrenal systems. In summary, there are likely to be concerns faced by parents of children with precocious pubertal development. Furthermore, previous research has reported that parents of children with a diagnosis of clinically early puberty showed embarrassment and denial over the child's disorder, and harboured fears that their child would behave sexually earlier than they should (Officioso et al., 2004; Solyom et al., 1980).

There is an evolutionary argument for a relationship between the child's home environment and pubertal processes from an evolutionary basis, in that for children entering adolescence, puberty may be advantageous in coping with, or adapting to, potentially difficult situations. For example, Flinn and colleagues suggest that during adrenarche, changes occur over the hypothalamic-pituitary-adrenal axis, which function to increase a child's resiliency and social behaviour to manage the physical, emotional and social changes which occur during this time in a child's life (Flinn, Nepomnaschy, Muehlenbein & Ponzi, 2011). Campbell (2006) suggests that these changes occur out of three localised influences of dehydroepiandrosterone (DHEA), which is an adrenal steroid implicated in adrenarche. The first of these three influences is a feeling of increased security and confidence in novel environments as a result of DHEA action on the amygdala. This facilitates social interaction with peers and development of new social contacts. The second influence is heightened memory retention in the hippocampus, and therefore more effective learning in new social environments. Finally, DHEA acts as a GABA receptor allosteric antagonist, meaning that neural plasticity remains throughout late childhood and adolescence for further social learning. However, Campbell suggests these changes function to increase socialisation, rather than resiliency, as proposed by Flinn and colleagues. In both cases however, it is suggested that humans have evolved to be better able to cope with stressful social environments as they reach puberty.

Whereas in earlier stages of evolution, early puberty was considered to give a longer reproductive window in which to produce offspring, early puberty is now considered disadvantageous, with younger individuals not having the resources or experience to raise children of their own. Evolutionary and developmental models of pubertal timing have frequently implicated stress and suitability of the living environment

on reproductive capacity. One example of this is that proposed by Gluckman and Hanson (2006) on pre- and post-natal nutrition from a life history perspective. They suggest that a poor prenatal environment is likely to result in a child being born small for gestational age (SGA) and that the timing of the child's developmental trajectory will be adjusted in the assumption that this level of poor nutrition will continue. The authors suggest that this results in earlier independence through accelerated pubertal onset, and the resulting ability to travel to a more adaptive environment and reproduce, but results in a trade-off with future growth. The authors further suggest that children living in a postnatal environment where nutrition is poor are more likely to be delayed in their pubertal onset. This is because of the reproductive cost on the individual and the low probability of survival in either the individual or their offspring. In comparison, an individual living in a fruitful environment is likely to become capable of reproduction earlier, as the environment is more stable and the reproductive costs are less. Another study has proposed a less linear relationship, in which not only those in difficult environments will have delayed pubertal development, but also those who are raised in ideal situations, as these individuals are likely to have pubertal development delayed until they have passed juvenility, in the confidence that this level of stability will continue. In contrast, those in between, who are raised in a moderately low stress environment, are likely to reach pubertal onset earlier, whilst conditions are still favourable enough for reproduction and chances of survival are fairly high, in the assumption that this level of security may not continue (Hochberg & Belsky, 2013).

Further evidence for environmental hypotheses can be seen by comparing pubertal onset in different socioeconomic (SES) groups across society. Those of a lower SES are likely to reach reproductive maturity earlier (Braithwaite et al., 2009; James-Todd,

Tehranifar, Rich-Edwards, Titievsky & Terry , 2010), and it may be that this is due to greater stress levels in this group, due to financial difficulties or family illness. Furthermore, those of a lower SES group are more likely to have a diet which is of lower nutritional value than those of a higher SES, and these individuals are therefore at a greater risk of gaining weight (Darmon & Drewnowski, 2008; Parsons, Power, Logan & Summerbell, 1999), which is a further predictor for earlier pubertal development (Adair & Gordon-Larsen, 2001; Anderson et al., 2003; De Simone et al., 1995; Harlan et al., 1980; Himes et al., 2004; Lee et al., 2007). If we assume these findings to be true, those of a lower SES are therefore likely to experience both the negative pre- and postnatal environments, proposed by Gluckman and Hanson (2006), as well as an increased risk of obesity, explaining the lower age of pubertal onset in this population.

Another group of individuals who have allowed us to test these hypotheses are those who have been adopted due to poor home conditions. These individuals are likely to have come from a high stress and poor nutritional environment, and move into a low stress and high nutrition environment, and we may therefore expect these individuals to begin puberty earlier than their peers. In particular, we may expect those at greatest risk to be children who have been adopted from a developing country where food is less available, into a more affluent country, where standard of nutrition is typically higher, thus signalling suitability of the environment for reproduction. These children are also likely to have experienced stressful situations; for example difficulties at home, the stress of leaving their family and having to live with strangers, and moving to a foreign country. This does indeed appear to be the case, and these children are in fact 10-25 times more likely to develop precocious puberty than the typically-developing population (Soriano-Guillen et al., 2010; Teilmann, et al., 2006), particularly for those being adopted from developing

countries into American or Western European families (Baron et al., 2000; Krstevska-Konstantinova et al., 2001; Mason et al., 2000; Proos et al., 1991). This supports the theories that stress and nutrition have an influence on the onset of pubertal development.

Other researchers have suggested that more common stressors, such as parental divorce, presence of a non-relative care-giver and disruptive family relationships may also affect the timing of puberty (Ellis & Garber, 2000; Ellis, McFadyen-Ketchum, Dodge, Pettit & Bates, 1999; Graber, Brooks-Gunn & Warren, 1995; Moffitt, Caspi, Belsky & Silva, 1992; Romans, Martin, Gendall & Herbison, 2003; Tremblay & Frigon, 2005; Wierson, Long & Forehand, 1993). Belsky, Houts and Fearon (2010) studied the prevalence of early puberty in adolescents who had previously been classified as having a secure or insecure attachement at 15 months of age. They found that those who had been insecure as toddlers were more likely to attain pubertal development earlier. Furthermore, researchers measuring hormone levels in children who have suffered abuse have reported higher levels of DHEA and its sulphate in these children compared with control participants (Cicchetti & Rogosch, 2007; Romans et al., 2003). It appears evident that there is a role of stress in pubertal timing, whereby an environment associated with heightened stress levels elicits earlier puberty. Belsky, Steinberg and Draper (1991) discussed these observations from an evolutionary perspective, and proposed that stressors, such as parental conflict or financial difficulties, cause an unstable home environment, and it is therefore adaptive for a young female to seek out a suitable mate for reproduction and to create a more stable environment in which to live. In this way it is adaptive for those reared in a high stress environment to achieve reproductive capacity earlier.

The aim of the current project was to investigate the relationship between three measures of family stress and pubertal development in the child. It was hypothesised that

families of those with a diagnosis of premature adrenarche (PA) or central precocious puberty (CPP) would score higher on measures of stress, would report more life events, and would be less likely to live in a traditional family structure, i.e. with both biological parents. Exploratory analyses were also conducted to identify whether any relationships existed in the data between family stress variables, demographic variables and pubertal development.

Methodology. See Table 1 for participants' demographic information. For a full description of the recruitment process, please see 3.2. Parents or guardians completed a questionnaire taking demographic details, as well as three questionnaires on family stress (See 3.3.1.8 for details on each measure). The first of these questionnaires was the Perceived Stress Scale (PSS; Cohen,Kamarck & Mermelstein, 1983), which assessed the extent to which parents felt they were currently able to manage stressors in their lives. The second questionnaire was a modified version of the Life Events Questionnaire (LEQ; Norbeck, 1984; adapted from the Life Events Scale; Sarason, Johnson & Siegel, 1978), which measured the type of stressors that families had experienced, as well as the impact of each of these events and whether they were positive or negative. Finally parents in the two clinical groups completed the Impact of Event Scale (IES; Horowitz, Wilner & Alvarez, 1979), which assessed both the intrusion of their child's diagnosis on the parents' lives, and the extent to which parents tried to avoid thoughts of the diagnosis.

Results. Tests of distribution and variance were calculated prior to each analysis. As at least one group demonstrated a non-normal distribution in each analysis, non-parametric tests were employed. No statistically significant differences were observed between the three groups for age, SES and ethnicity.

- *Care-giver status and SES- HypothesisTesting.* When looking at the structure of the families taking part in this project, it was evident from the Chi-squared analyses of a significant association between participant group and care-giver status ($X^2(6, 96)= 13.15$, $p= .041$), where the CPP group were the only group to include participants who were living with non-biological care-givers (See Table 49).

Table 49: *Proportion of each group living with both biological parents, one biological parent and a step-parent, a single parent, or non-biological care-givers*

	Two Biological Parents	Biological Parent and one Step-parent	Single Biological Parent	Legal Guardian/s
Control	74%	9%	17%	
PA	68%	6%	27%	
CPP	67%		20%	13%

When care-giver status was analysed using a Kruskal-Wallis method, several significant differences were identified between groups. Descriptive statistics are given in Table 50. The first was a significant difference in the mean Tanner rating ($H(3, 96)= 10.90$, $p= .012$), where those with both biological care-givers had the least progression through pubertal development, and those with legal guardians as their care-givers had developed the most. There were no significant differences between care-giver groups for age of child. Mann-Whitney analyses identified that differences in pubertal development lay between children with both parents living in the same household and those with legal guardians ($U(68, 2)= 8.50$, $z= -2.11$, $p= .035$), and with a single parent ($U(68, 20)= 417.50$, $z= -2.63$, $p= .009$). However, these findings were not significant after Bonferroni correction for multiple comparisons, and a new alpha level of .008.

Table 50: *Means and standard deviations for the SES, number of siblings and pubertal stage for each care-giver status group*

	SES	Number of Siblings	Pubertal Development
Two Biological Parents	3.99 (1.19)	1.47 (.91)	1.59 (.54)
Biological Parent and One Step-Parent	4.00 (.89)	2.33 (1.21)	1.63 (.37)
Biological Single Parent	2.55 (1.19)	.85 (.88)	2.04 (.73)
Non-Biological Care-Giver	2.00 (1.41)	1.50 (.71)	2.55 (.21)

- *Care-giver status and SES- Exploratory Analysis.*Spearman's correlation analysis demonstrated a near significant relationship between SES and pubertal development ($r = -.20$, $p = .057$), and a significant association was identified between SES band and care-giver status ($X^2(12, 96) = 38.4$, $p < .001$), in that families with a higher income were most likely to live in a household with both biological parents (See Table 51).

Table 51: *Proportion of families in each SES group consisting of both biological parents, one biological parent and a step-parent, a single parent, or non-biological care-givers*

	Two Biological Parents	Biological Parent and one Step-parent	Single Biological Parent	Legal Guardian/s
<£14,000	56%	0%	33%	11%
£14,000-£24,999	25%	0%	75%	0%
£25,000-£34,999	56%	13%	25%	6%
£35,000-£49,999	85%	7.5%	7.5%	0%
>£50,000	88%	6%	6%	0%

- *Parental Stress- Hypothesis Testing.* Parental stress levels were analysed using a Kruskal-Wallis analysis, but yielded no significant differences or significant trends between the two clinical groups or the control group. Similarly, there were no significant differences in the number or type of life events experienced by families in each group. Finally, there were no significant differences between the PA and CPP groups in neither the total scores, nor the intrusion subscale or avoidance subscale scores of the IES. See Table 52 for descriptive statistics.

Table 52: *Means and standard deviations for each group on the three parental stress measures*

	Impact of Event			Perceived Stress	Life Events		
	Intrusion Subscale	Avoidance Subscale	Total		Good	Bad	Total
Control	NA	NA	NA	19.6 (8.9)	6.4 (5.5)	6.4 (6.0)	12.8 (9.3)
PA	.9 (.7)	.7 (.8)	11.5 (10.3)	20.7 (7.2)	8.3 (5.9)	7.8 (7.7)	16.1 (9.8)
CPP	1.3 (.9)	1.1 (.9)	17.9 (12.7)	24.0 (8.7)	7.4 (9.2)	10.4 (10.6)	17.7 (12.7)

- *Parental Stress- Exploratory Analysis.* Spearman's correlational analyses for the complete dataset suggest several significant relationships in the data, although the majority of these were weak in strength (See Table 53). However, the overall view of the data suggests that greater pubertal development in the child is related to higher scores on multiple measures of parental stress. The relationship between puberty and measures of stress appears to be stronger than the relationship between stress and SES, as Tanner rating was related to a greater number of stress variables, and with higher r-values than that of SES. However, higher SES was negatively correlated with life events, particularly negative events, as well as with intrusion scores for impact of the child's diagnosis. Pubertal development was significantly related to total impact of the child's diagnosis, as well as intrusion of the diagnosis in the two patient groups. Puberty was also positively related to both negative and total life events, and parents' perceived stress. Child's age was only weakly related to negative life events.

When combining the demographic variables with the measures of family stress, significant differences were found between care-giver groups on intrusion scores of the IES ($H(3, 49)= 11.38$, $p= .010$). Mann-Whitney analyses identified that these significant differences lay between children with both biological parents, and those with a single parent for number of siblings ($U(68, 20)= 420.50$, $z= -2.80$, $p= .005$), SES ($U(68, 20)= 279.00$, $z= -4.14$, $p< .001$) and IES intrusion scores ($U(33, 12)=77.5$, $z= -3.11$, $p= .002$); and between single-parent families and families with a step-parent for number of siblings ($U(20, 6)= 19.50$, $z= -2.58$, $p= .010$) and SES ($U(20, 6)= 20.00$, $z= -2.51$, $p= .012$), although these were non-significant after Bonferroni correction.

Table 53: *Spearman's correlations between the three parental stress measures, pubertal development and demographic variables*

	Impact of Event			Perceived Stress	Life Events - Good	Life Events - Bad	Life Events - Total
	Intrusion	Avoidance	Total				
Age (months)	-.01	-.04	-.01	.1	-.09	.21*	.12
SES	-.43**	-.1	-.28	-.19	-.06	-.23*	-.22*
Mean Tanner	.39**	.22	.33*	.22*	.15	.33**	.35**

Note *p< .05 **p< .01 ***p< .001

Linear regression analyses were conducted to explore the relationships between variables (See Table 54). SES and mean Tanner score were both entered into a stepwise regression as independent predictors of total IES score, intrusion and avoidance subscale scores and perceived stress score. The regression analyses supported the correlational data for the intrusion subscale of the IES, as both pubertal rating and SES were significant predictors, with SES contributing negatively and pubertal development positively to intrusion scores. For the total IES score and IES avoidance subscale however, pubertal development was the only significant predictor and was responsible for 10-17% of the variance. The regression analyses for perceived stress scores found that both puberty and SES were significant predictors, with SES contributing negatively and pubertal development positively to the equation. Again, this model only predicted a small amount of variance in the scores. When SES and the good, bad and total life events scores were entered into the model to predict pubertal development ratings, only the negative life events score was revealed as a significant predictor, explaining 12% of the variance in pubertal development.

Table 54: *Linear regression models, where SES and pubertal development have been entered as predictors for the three stress scales, and SES and the three stress scales as predictors of pubertal development*

Dependent	Predictors	Std. β	t	Sig.	R^2	F	Sig.
IES Intrusion	Constant		1.19	.242	.36	13.0	.000
	Mean Tanner	.40	3.39	.001			
	SES	-.39	-3.22	.002			
IES Avoidance	Constant		-.36	.724	.10	4.91	.032
	Mean Tanner	.31	2.22	.032			
IES Total	Constant		-.82	.414	.17	9.73	.003
	Mean Tanner	.41	3.12	.003			
Perceived Stress	Constant		5.57	.000	.10	5.35	.006
	SES	-.21	-2.12	.038			
	Mean Tanner	.21	2.06	.042			
Mean Tanner	Constant		18.37	.000	.12	12.76	.001
	Life Events - Bad	.35	3.57	.001			

Correlations were then carried out for each group to see whether the relationships observed in the complete dataset were maintained when studied individually (See Table 55). The data for the control group suggested that greater pubertal development was related to a greater frequency of negative life events, and total number of life events. These data were consistent with those seen in the complete dataset, however this relationship was

not evident for the two clinical groups. In the PA group, greater pubertal development was related to higher scores on the impact of event scale, particularly scores on the intrusion subscale. Being of a higher SES however, was related to a lower impact of the child's diagnosis on the parent, and appeared to be protective from intrusion of the child's disorder on the parents' lives. There were no significant relationships between the demographic variables, pubertal development and any of the stress scales in the CPP group. Furthermore, child age was not related to any measures of parental stress, and SES was only negatively associated with intrusion and total scores on the IES scale in the PA group.

Table 55: *Spearman's correlations between pubertal development, SES and age, against the three parental stress measures*

		Impact of Event			Perceived Stress	Life Events		
		Intrusion	Avoidance	Total		Good	Bad	Total
Control	Age (months)	NA	NA	NA	.12	-.11	.15	.03
	SES	NA	NA	NA	-.12	-.02	-.20	-.13
	Mean Tanner	NA	NA	NA	.15	.14	.39**	.31*
PA	Age (months)	-.12	-.18	-.17	.17	-.1	.15	.03
	SES	-.54**	-.17	-.40*	-.21	-.17	-.25	-.30
	Mean Tanner	.43*	.23	.38*	.09	.07	.25	.18
CPP	Age (months)	-.08	-.02	-.01	.08	.11	.43	.48
	SES	-.28	.10	-.10	-.31	.39	-.21	-.13
	Mean Tanner	.16	-.06	.06	.40	-.02	.41	.39

Note *p< .05 ** p< .01 ***p< .001

Discussion. The findings suggest several interesting features about the families and the home environments of children with a disorder of early pubertal development. The first finding was that although the majority of participants in each group lived with both biological parents, children in the two clinical groups were more likely to live in a single parent household or under the care of a legal guardian. This is consistent with the literature on altered family dynamics, where events such as parental conflict and father absence have been suggested as influential factors in pubertal timing (Ellis et al., 1999; Ellis & Garber, 2000; Graber et al., 1995; Moffitt et al., 1992; Tremblay & Frigon, 2005; Wierson et al., 1993).

The data is also consistent with studies suggesting that there is an increased prevalence of early puberty in children who have been adopted (Baron et al., 2000; Krstevska-Konstantinova et al., 2001; Mason et al., 2000; Proos et al., 1991; Soriano-Guillen et al., 2010; Teilmann et al., 2006). Of our CPP group, 13% were adopted into a new family or under the care of a non-parent legal guardian. The CPP sample was small and it could therefore be argued that this finding was due to chance; however, considering that this high proportion of adopted children were in the smaller CPP group and not the larger PA or control groups it would seem unlikely. Furthermore, when analysing differences between care-giver groups, it was found that children with a single parent or who had been adopted had greater mean Tanner ratings than those with two biological parents, irrespective of diagnostic group. This implies that children who have experienced parental divorce or family break-down may begin puberty earlier. However, children whose parents were of a higher SES were also more likely to be living with both biological parents. It may therefore be that these variables are related, and that those from single-parent families have a lower income than families with two wage-earners, meaning that

single-parent families may be subject to higher stress levels because of a reduced income, and that this is related to early puberty in the child. Similarly, those who had been adopted may have come from high stress backgrounds which would necessitate their need for adoption. In this way, children with these care-giver structures may be at an increased risk of environmental stress and therefore early pubertal development.

Analysis of family stress between the clinical groups and the control group identified no significant differences between them. Furthermore, there were no differences in life events or impact of the child's diagnosis. This suggests that parents of children with a diagnosis of precocious puberty were not necessarily more stressed than those of typically-developing children, experienced no more or fewer life events, and the impact of the diagnosis was equivalent across the two patient groups. When looking at the relationship between pubertal development and these measures across all of the groups, however, it would appear that parents' stress levels increased alongside their child progression through puberty. Parents also reported more negative and total life events as their child progressed through puberty, but not as their child got older. When groups were analysed separately, significant relationships between pubertal development, and negative and total life events were no longer observed in the two clinical groups. It is unclear why this is the case, however, it may be that in the families of typically-developing children, negative life events cause a stressful home environment, and this stress is related to earlier puberty. The occurrence of early puberty in the clinical groups may not be related to stress in the family, but a biological factor. Alternatively, it may be that there is not enough variance in the pubertal development ratings of the pubertal development groups for a correlational relationship to be observed. A final suggestion, is that families in the clinical groups may have experienced greater stress at the onset of the child's disorder, and that

because in some cases this may have been several years prior to this research, their stress levels and reported life events may be now considerably less. This is a key limitation of the project. Regression analyses for the complete dataset however, suggested that pubertal development was a significant predictor of both IES and perceived stress scores, and in some instances, to a greater extent than SES. Furthermore, negative life events were significantly predictive of pubertal development. This supports the literature, positing that stress, stressful events and pubertal development are inter-related, and pubertal onset may not only be caused by stress, but may also cause stress in the family.

The correlation and regression analyses suggest that higher income was negatively related to intrusion subscale scores in the patient groups. This implies that a higher income is a protective factor against intrusion of the child's diagnosis into the parents' thoughts, meaning that they worry about the disorder less. Individual group analysis, however, showed that this was only true for the PA group, implying that there was no protective factor of high SES in the CPP group. This may mean that parents were similarly affected by their child's diagnosis in the CPP group irrespective of SES, or that they were not affected by their child's diagnosis in the first place. This second hypothesis is inconsistent with the mean subscale scores for each group (Table 52), and it is therefore likely that being of a higher SES was of no benefit to parents of children in the CPP group when coping with their child's disorder. Possible explanations for this may be that parents of the CPP group worry more because of impending menarche, that the disorder is more difficult to hide, or because their child requires treatment, which is not the case for the PA group

Summary. The findings seem to suggest a relationship between stress, the family environment and a child's pubertal development. This is particularly salient when considering family structure and SES. Although it would intuitively seem that family

structure and SES must influence pubertal development, if stress from these factors is implicated in pubertal onset, one must question why precocious puberty is not more common. When considering the vast number of families living in a lower SES bracket or as single-parent families, one would expect the prevalence of precocious puberty to be far higher. Furthermore, we do not necessarily observe early puberty in siblings, which would be expected if the home environment was a key contributor to the onset of puberty. We must therefore take into consideration that other factors are likely to be implicated, such as diet, body composition, ethnicity, or hormonal variations. Parents of children with precocious pubertal development do not appear to have higher stress levels than those of typically-developing children, and we may therefore assume that parents do not find parenting a child with early puberty particularly stressful. It must be considered, however, that parents may have been considerably more anxious at the onset of the disorder, or that the scale employed here simply did not measure the right aspects of stress experienced by these families.

In conclusion, the findings provide an interesting insight into the relationships between stressful life events, the home environment and pubertal development. Although the sample was of unequal group sizes, and the life events scale was completed by parents somewhat retrospectively and not necessarily for the time of their child's pubertal onset, there are still key findings presented here which are unlikely to be due to chance. From these findings, we may be able to identify those children who are at risk of entering puberty earlier, and provide adoptive parents and schools in lower SES areas with more information on the disorder, as they are most likely to encounter children with this condition. These findings also add to the literature on the relationship between biological stress processes and puberty.

5. QUALITATIVE DATA ANALYSIS

5.1 The Experience of Parenting a Child with Precocious Pubertal Development: An Interpretative Phenomenological Analysis

The transition between childhood and adulthood is a period of physical and emotional change (Blondell, Foster & Dave, 1999; Pinyerd & Zipf, 2005). Puberty is a high-risk time for the onset of several mental health difficulties (Angold & Costello, 2006; Graber, Lewinsohn, Seeley & Brooks-Gunn, 1997; Kaltiala-Heino, Martunnen, Rantanen & Rimpela, 2003; Mendle, Harden, Brooks-Gunn & Graber, 2010), and a stage at which changes occur in the individual's relationships, particularly with parents (Csikszentmihalyi & Larson, 1986; Larson & Richards, 1991). For children entering puberty at a younger age, these changes may be even more difficult to manage (For a review, please see 2.1). At a young age, children may not understand the changes that are happening to their bodies, and find it more difficult to control their emotions. There may be a greater discrepancy between their physical appearance and that of their peers. Parents are likely to find it hard to manage changes in temperament and behaviour, and may be unsure how to explain puberty to their young child, or how to manage pubertal changes at school.

Despite the problems we might expect them to encounter, children with precocious pubertal development have received little attention in the psychological literature, particularly with regards to their experiences, worries and perceptions. Some research using clinical interviews reported that children find some aspects of the disorder difficult to manage, such as physical examinations, hiding breast development and increased shyness (Solyom, Austad, Sherick & Bacon, 1980; Xhrouet-Heinrichs et al., 1997). Furthermore, children with a diagnosis of central precocious puberty (CPP) were described as self-conscious and uncomfortable when talking about their diagnosis, whereas children

with a diagnosis of premature adrenarche (PA) were found to feel negatively about their appearance, and presented aggressively (Solyom et al., 1980). Xhrouet-Heinrichs et al. (1997) reported that participants with CPP had concerns about looking different to their typically-developing peers, particularly those with breast development. The authors also reported that three quarters of their participants with CPP admitted to feeling lonely, suggesting social problems at school.

There has only been a single paper employing exploratory qualitative methods in the precocious pubertal development population (Liao, Missenden, Hallam & Conway, 2005). Liao and colleagues interviewed six adult women, who had been diagnosed with precocious puberty during childhood, and applied thematic and discursive analytical methods to their data, in order to understand the women's experiences during this stage of their development. The findings suggested that participants found their pubertal development to be problematic, and something that they felt very strongly about, even into adulthood. The first theme described negative experiences that participants had encountered due to appearing physically different to their peers, and the way that this feeling of being different persisted in their self-perception after these differences had long disappeared. The second theme was of being simultaneously both an adult and a child, derived from feelings of a lost childhood and an inability to combine feeling child-like and similar to peers, whilst looking older and being different. Other subthemes under the adult-child theme included ambiguity in the perceived boundary between childhood and adulthood, and the sexual connotations associated with advanced physical development. The authors argue in their discursive analysis, that the data point to broader issues of gender and sexuality in society, and the way in which early pubertal development altered the participants' sense of femininity and positioned them as objects of sexuality. The

overall impression of the paper was that participants' early pubertal development was inconsistent with the cultural norms of what is expected of a child, and was somehow wrong and something to be ashamed of.

Although Liao and colleagues (2005) describe the experiences of the individuals, there has been no systematic research into the experiences of their parents. Findings taken from clinical interviews include concern that the child may experience early sexual relationships, embarrassment, denial of the condition, and fears of other medical disorders, such as cancer or polycystic ovarian syndrome (Officioso et al., 2004; Solyom et al., 1980; Xhrouet-Heinrichs et al., 1997). However, there is no information on how parents feel about their child's diagnosis, the parent-child relationship or their experiences of parenting a child with early pubertal development.

Recruitment. The project was conducted to study how a diagnosis of precocious pubertal development affects a child and their family, with the aim of understanding the ways in which families' experiences were perceived, managed and understood. Families were recruited as part of the empirical project, and later asked if they would like to participate in a qualitative follow-up (For full recruitment process, see 3.2 and 3.3.2). From those that demonstrated an interest, six families were selected for quantitative data collection, based on the ease with which they had talked about the condition during home visits.

Table 56: *Demographic information for the children whose families completed the qualitative interviews*

	Participant Pseudonym	Diagnosis	Age	Ethnicity	Pseudonyms of Relatives Interviewed
P2	Amy	CPP	9 years 10 months	Black	Angela (Grandmother)
P3	Becky	PA	8 years 4 months	White	Beth (Mother)
P4	Charlotte	CPP	8 years 9 months	Indian	Carol (Mother)
P5	Daisy	PA	8 years 6 months	White	Deb (Mother) and Danny (Father)
P6	Ellie	CPP	9 years 6 months	White	Eve (Mother) and Edward (Father)

Unfortunately one family (P1) withdrew due to unforeseen circumstances. Therefore, the parents or carers of five children participated in the interviews. The children had all received a diagnosis of CPP or PA from the Birmingham Children's Hospital or the University Hospital of Coventry and Warwick, and were aged between 8 and 10 years (See Table 56 for demographic information). The family members interviewed were mainly the children's mothers, although some fathers took part, as well as one child's grandmother, who was her legal guardian. Parents and guardians all had a good standard of English and gave fully informed consent to participate in the project.

Data Collection. Interviews were based on topics that had arisen during family support groups and clinical observations during outpatient appointments. The research questions derived from these observations were: 1) What are the most salient experiences of precocious pubertal development? 2) How do families understand and respond to precocious pubertal development? 3) How do families feel about precocious pubertal development? Research questions were chosen to elicit discussion of topics, such as family relationships, and experiences with schools and hospitals (See Appendix 19 for interview questions). Interviews were semi-structured, and questions were open-ended to allow families to expand on the points that they felt most important. Parents or carers were also given the opportunity to add anything that they felt had not been covered during the interview. Interviews lasted between 30 to 60 minutes. Families were given the option to not discuss any topics with which they were uncomfortable, and were aware that they could withdraw their data from the study at any point. Interviews were conducted by a Masters student from the research team, who is referred to as '*J*' throughout the transcripts, and whose speech is provided in italics. See acknowledgements for further details.

Data Analysis. The interview data were transcribed verbatim from Dictaphone recordings by the Masters student, and then given to the lead experimenter for anonymisation. All further handling of the data was carried out by the lead experimenter. Each transcript was divided into cohesive phrases for coding, and codes were generated on the content of each excerpt. Coding was conducted from an interpretative phenomenological standpoint (IPA: Smith, Flowers & Larkin, 2009). IPA is a qualitative approach to studying idiographic data, which aims to explore how individuals understand significant life experiences. This is achieved by allowing participants to reflect on their experiences and discuss whatever is most salient to them. The researcher attempts to interpret the data without being led by preconceived ideas, but being directed by what appears to be most important to the individual. This in-depth analysis provides the researcher with an insight into how the experience is made sense of, and the convergence and divergence of experiences across and between cases. The aim of IPA, however, is not to generalise findings to other populations, but to provide case-by-case interpretations of an experience, which elicit insights that would normally be overlooked in nomothetic research. This perspective was decided upon, as the aim of the project was to explore the perceptions and emotions elicited by the shared experience of early puberty.

The codes created from each excerpt consisted of a brief summary of the content and any indication of experience or meaning which could be inferred from the text (See Appendix 21 for example codes). These codes were regularly reviewed and revised with colleagues who had greater experience in qualitative analysis. This coding method allowed for understanding of what the most important and salient aspects of participants' experiences were. Excerpts and their codes were organised by putting text of a similar content together to construct themes for each each case individually, and then repeated

across all cases. Similarities and differences between transcripts were then identified, allowing for the development and re-development of a structure to conceptualise the data.

Table 57: *Overview of themes and subthemes derived from the interview data*

Theme	Subthemes
1. "This diagnosis isn't so bad"	1.1 "It's a relief"
	1.2 "I understand what is happening now"
	1.3 "It's not as bad as I expected"
	1.4 "It could be much worse"
	1.5 "It's only temporary"
2. "I'm not sure how big a problem this is"	2.1 "My child seems to be at an advantage"
	2.2 "But then again perhaps not"
	2.3 "My child is really no different"
	2.4 "As far as anyone else needs to know, my child is no different"
3. "So why don't I feel comfortable with this?"	3.1 "Will this affect her in the future?"
	3.2 "I feel sorry for her"
	3.3 "I still don't really don't know what to expect"
	3.4 "It's just wrong"
	3.5 "I don't understand where it has come from"
	3.6 "I don't know how to manage this"
4. "What are we going to do?"	4.1 "Let's get support"
	4.2 "We aren't being given enough support"
	4.3 "We need to support our daughter"
	4.4 "We're going to get on with it"

Results. After coding and organisation of the excerpts, it was found that the majority of the data fell under four main themes. Strikingly similar experiences were observed across families, yet each family understood or reasoned with their experiences in very different ways. Each theme is discussed along with more specific subthemes, with the aim of demonstrating to the reader how the data was understood and how the conclusions were drawn. An overview is given in Table 57.

Theme 1 "This diagnosis isn't so bad". The first theme describes how families felt that the diagnosis was manageable, and, in some ways, positive. Although one might assume that having a child receive any medical diagnosis is a negative event, there was evidence of families feeling positively about the naming and meaning of the disorder. Furthermore, there were several reasons for this optimistic perception, each of which is described in the following subthemes.

Emergent Theme 1.1 "This diagnosis isn't so bad: It's a relief". The first subtheme was that the diagnosis was a relief. The secondary sexual characteristics which develop during precocious pubertal development can be indicative of other medical conditions, such as neurological abnormalities, or tumours of the gonads or adrenal glands. Children presenting with early puberty therefore undergo a battery of tests to exclude more pervasive disorders. Parents feel concerned that a serious medical condition may be diagnosed when the results of these tests are revealed, and when the child is diagnosed as entering puberty early without signs of other disorders, parents are therefore relieved.

The excerpt below demonstrates how one parent, Carol, was warned of the other conditions associated with signs of early puberty, and how these conditions, 'could have been worse'.

> CAROL: […] It could have been worse. […] The, erm, Adrenal Hyperplasia, that's what, when the doctor said she, […] doesn't have it, the tests are negative. I was like, 'Oh thank God, if that's not it I can deal with anything else,' […] so I always look at it that way.

There appears to be a period of concern when the medical tests are processed, and parents fear that they may not be able to manage more serious conditions if they are diagnosed. Once Carol was informed that the results were negative, she was thankful and relieved that her child did not have a more chronic illness. She also states that she feels that she 'can deal with anything else', and that the diagnosis of early puberty was manageable in comparison to what might have been. Furthermore, there seems to be an active decision in how to perceive the disorder, in that Carol states that she 'always look(s) at it that way', and tries to think positively about the disorder. This sense of relief allows the parent to feel in control.

Theme 1.2 "This diagnosis isn't so bad: I understand what is happening now".

The diagnosis was also a relief for families, because it removed ambiguity. Families knew little of the disorder prior to visiting the doctor and disliked facing something unknown. Furthermore, families were reassured that their child would be monitored regularly, and that they were not facing the problem alone. The diagnosis therefore provided security for families because this was something that others had experienced and had knowledge of, and they were not the only ones going through the process.

One excerpt from Beth demonstrates that even though having a diagnostic label isn't what one would hope for their child, it is reassuring to know that the condition is recognised and that something can be done. The fear that even medical professionals may not know what the problem is, or how to manage it, is removed once a diagnosis is

reached. Furthermore, a diagnosis gives the problem a name and identity, and therefore makes it less elusive and more predictable. There seems to be security in knowing what is going to happen.

> BETH: Because the minute somebody puts a label on it, and tells you exactly what it is, and this is going to happen, and that's going to happen. It sort of, straight away, it makes you feel better, erm. Even if I say, […] a diagnosis wasn't that good if you know what I mean. You want […] to know what it is, what caused it.

Danny also states that when the diagnosis is known and understood by medical professionals, it suggests that the disorder is seen frequently. It is reassuring that others have been through the same problems, and that the family is not alone in this experience.

> DANNY: Once it's been diagnosed, you sort of feel, more secure, more confidence, because you know there have been other people that, sort of, trod that path, […] they know sort of what might happen.

Theme 1.3 "This diagnosis isn't so bad: It's not as bad as I expected". Families talked about fears that their child would be more severely affected by the condition, and that symptoms such as strong mood swings or reduced growth would develop, particularly those who had sought further information on the internet. A need for knowledge about the disorder was often described. However, information on the internet was found to be exaggerated or inaccurate and this scared families even more. Families commonly expected the worst and worried that the disorder would have a greater impact on their child, in some cases into adulthood. Families were therefore pleased when the difficulties others had reported on the internet were not the case for their own child.

Excerpts from Carol and Deb demonstrate how they had both expected the disorder to have a greater impact. The fact that these fears were not realised, is therefore seen positively, and families are pleased that the effect of the disorder is not as severe as they first thought.

> *J: You said you were expecting it to be harder, what were you expecting?*
>
> CAROL: Erm… because I've heard a lot, erm, that can go wrong with this, once, you know, the injections starts, and… the hormones trying to cope, all of that, I've heard it can be really hard on the parents and the children, but, erm. I think we've done extremely well, yeah. And I think it's a blessing, that's all.
>
> DEB: I was glad when her bone age was not that advanced, because, I thought it would be.

Theme 1.4 "This diagnosis isn't so bad: It could be much worse." Several families compared their situation to that of others, and felt fortunate in comparison. They acknowledged that some families have to manage terrible medical conditions, and felt that their child's diagnosis was minor in relation. It was interesting that families perceived themselves as lucky and felt that the disorder was something to be thankful for, rather than feeling angry that their child had developed the condition, as one might first assume.

The quotes below demonstrate this fortunate attitude, as both Carol and Eve compare their daughters' conditions to disorders that other children may suffer, and conclude that they are lucky in comparison.

> CAROL: Is it thyroid? Or is it… what is it? But then again, if it was thyroid, my friend's daughter, […] back in India, she's been diagnosed with

> hypothyroidism right from birth, so I'm like well compared to so many things, we're better off.
>
> EVE: You know, just the normal, 'Why, why, why?' really. Erm, although I would never wish it upon anybody else, […] what, she's gone through, it's nothing compared to what a lot of kids have gone through.

This suggests that families are able to reason about, and maintain perspective of, the magnitude of the disorder. An alternative perspective, however, may be that this grateful attitude is a coping mechanism, where comparison with those who are worse off makes families feel better about their situation. In this way, families are trying to reason away any negative emotions they are experiencing about the condition, and motivate themselves to move on.

Theme 1.5 "This diagnosis isn't so bad: It's only temporary". Another subtheme was that the condition was transient, and therefore not so bad. Families talked about what would happen in the immediate future, for example, Deb describes how Daisy's peers are "all getting older", and are "all going to start developing", to catch up with Daisy. Other families reported that their child would soon attend a different school and consequently socialise with older, more developed pupils. Furthermore, families only needed to attend medical appointments for a short period of time, after which the child would be considered 'normal'. This sense of the diagnosis only being an issue for a short period of time was a positive feature for families.

> ANGELA: School, she doesn't seem to have any problems with that now. I think everybody has sort of caught up in her class, so, we're not having no issues or no feedback in that department. […] She's a, she's a lot happier in herself, a lot happier.

In summary, the first theme demonstrates that families harbour several positive perceptions of their child's diagnosis, and shows the various ways in which parents and carers reasoned about the ways in which the disorder could have been much worse. This reasoning helped them to perceive the diagnosis as something that they could manage, and a problem that was not as bad as it could have been.

Theme 2 "I'm not sure how big a problem this is…" The second theme focused around families' attempts to understand the condition and how it would affect their child. Families appeared to simultaneously hold multiple perceptions of their child. In some instances they understood the child to be no different to their peers, but in others, their condition desperately needed to be kept hidden. Similarly, the child was presented as confident, mature and resilient, but also as a victim of bullying and low self-esteem. It would seem that parents and carers were unsure whether to perceive the child's diagnosis as a variant of 'normal', or as a medical condition which posed a problem, and constantly fluctuated from one perspective to another.

Theme 2.1 "I'm not sure how big a problem this is…: My child seems to be at an advantage." When addressing the first perspective, it appeared that families felt that the condition had given their child an advantage. For example, children were described as intelligent, mature, and capable of understanding concepts beyond their years. Parents described interactions with their child as akin to speaking to a much older child, and felt confident discussing the diagnosis with them, as they were sure the child would understand. Other families described how well the child was doing at school. There was evidence of a positive consequence of the disorder in every transcript, with children described as resilient, confident and self-assured.

An example of this is given in Beths' interview, where she talks about her daughter being 'old-headed', and like an older child in her behaviour and understanding. Beth feels that this maturity has helped the family to manage the diagnosis, as Beckys' older attitude and level of understanding has helped communication between them, and therefore she perceived this positively.

> BETH: […] because she's so old-headed, it's like talking to an older child, a much older child, so, she, she's very easy to talk to, and she takes everything in straight away really, so it's not a problem.

Carol talks of her daughter's disorder as making her 'tougher', and how the disorder will give Charlotte experiences that she can learn from and use to build empathy for others who may be worse off than herself. Furthermore, Charlotte aspires to become a doctor, and to help other children. Carol therefore believes that Charlotte has benefitted in some ways from having the disorder.

> CAROL: Yeah, things like that [CHARLOTTE's ambition to be a doctor], […] sometimes it just touches my heart you know, just things she says. […]. In a way, I'm thinking all this has made her even more tougher, and umm… As she grows up, […] she will know she's been through this, and there are many more children who'll have to, […] go through much more than what she's done in her childhood, so she can do everything to help them. I always keep thinking like that, so that, that's how we look at her [sigh].

Finally, Eve describes how her daughter has changed over the course of the disorder, progressing from a child into a 'little young lady' due to a growth in her maturity.

> EVE: You can see this… little girl, going into a little young lady. But not a young lady as in a teenager, you can see there is a transition going on, of her maturity.

Eve's overall perception of Ellie is that she is still child-like, shown by the words, 'little' and 'young', but her demeanour is similar to somebody much older. In this sense, the disorder is perceived to have increased Ellie's maturity and she has begun to behave in a more adult manner.

Theme 2.2.1 "I'm not sure how big a problem this is…: My child seems to be at an advantage, but then again, perhaps not. They are physically different." Families also talked about their child being at a disadvantage as a result of their diagnosis. One difficult aspect which was frequently mentioned was the secondary sexual development observed in their child. For some families, breast development was the most prevalent concern, whereas for others it was pubic hair growth or body odour. In some instances there was physical discomfort associated with the development, whereas in other cases it was the child's embarrassment which was upsetting. Across all cases, others had made comments about the child's development, which served to draw attention to the differences between them and their peers.

Angela described incidences in which Amy's cousins had asked about her breast development, drawing Amy's attention to it. She then became fixated on this, and began comparing herself with others. Angela reported that Amy felt sad and embarrassed about her breast development, despite reassurance from her grandmother. Similarly, Beth reported that Beckys' classmates would notice an aspect of her pubertal development and question her about it.

> ANGELA: Like her cousin, she's got cousins of the same age as her, […] 'You've got titties, I haven't got titties. How come your titties are so big?' She'd be embarrassed, and looking around, and she'd start looking at other people's [chest areas] then, and you'd see it all the time, she'd be looking at everything on girls, and everything else. And I'd be like, you know, 'Don't worry about that'. She looked a bit sad about that.
>
> BETH: But occasionally there will be times when they [others at school] pick up that she's bigger than them. You know, she's gotten breast development or something and so they'll pick on one particular thing and, you know, 'Why is this bigger than...? Why is yours bigger than...?'

Neither case is described in a way that would suggest the children are being bullied, but their attention is repeatedly drawn to their physical development. This may make the difference between them and their peers something that they begin to worry over, and in this way their pubertal development may become a source of upset.

Eve described the different stages of development that Ellie had progressed through as unexpected. This suggests that the child's physical development is also difficult for families, as the development comes as a shock, and something that they aren't prepared for.

> EVE: Erm… then she started to develop hair, erm, down below. Her boobs have obviously got bigger. Her spots have continued to be, erm… more pronounced. Erm, and hair under the arms… so over the past four years, although it's been very slow, it does seem to be going a bit quicker again at the moment. You know, things are popping up, where you don't expect them to go.

Theme 2.2.2 "I'm not sure how big a problem this is…: My child seems to be at an advantage, but then again, perhaps not. They are bigger." Several families mentioned that their child's weight was a concern, and something that caused tension in the household. Other families described their child as being 'bigger' in both height and weight, and that this was sometimes the subject of teasing from other children at school. Trying to manage the child's weight was something families found to be a daily battle, as families wanted to encourage weight management without making it an issue for the child.

This was most evident in Charlotte's family, where Carol described how Charlotte is kept on a healthy diet and is encouraged to exercise regularly. Carol described her frustration at not being able to reduce Charlotte's weight, despite her best efforts. Furthermore, although Charlotte's father tries to encourage her to dress appropriately for her size, Carol is concerned that Charlotte will begin to take issue with her size, and become upset over it. There appears to be a conflict between managing the problem, without emphasising the problem to the child.

> CAROL: And then the way it just seemed to, just start escalating, and I was like, we're non-meat eaters, […] we're vegetarians, […] she don't eat that much junk food, you know, like crisps, and she eats quite healthy food. And I'm like, 'Where is the weight coming from?'
>
> CAROL: Yeah, so um my husband's, I tell him, 'Don't do that, don't you know, don't.' At first, he used to do a lot, like you know, 'CHARLOTTE don't put these dresses, they're very tight,' and […] 'Don't put this, you've grown out of this,' […]. But I tell him, 'Don't,' I said sometimes, you know, 'If you keep on mentioning about her weight now, and then, and then she's going to think, 'Why am I...?'

Similarly, Beth feels that her daughter already has concerns over her weight, and is very conscious of others' comments. She describes how Becky 'pulls herself to pieces', and is very critical of herself and her weight.

> BETH: I know now BECKY will internalise something […]. So even her weight, she's very conscious of her weight, so if somebody in the family says she's big, you know, BECKY stands in front of the mirror and says, 'I'm fat', and pulls herself to pieces, even now.

Theme 2.2.3 "I'm not sure how big a problem this is…: My child seems to be at an advantage, but then again, perhaps not. They are socially different." Another way in which families reported their child being at a disadvantage was socially. Each family reported that classmates had commented on their child's weight or pubertal development. The children were teased for being different, and in some cases this upset them considerably or caused them to lose interest in school. Other families talked about comments made by other family members, and the negative impact this had on the child's self-esteem. In some instances, the child's mood swings had affected their ability to maintain friendships and put them at a disadvantage.

One example of this was described by Angela. She spoke of how Amy was bullied by the other children because of her pubertal development and her emotional outbursts, and she was left on her own at playtimes. Angela describes this as a difficult time, for both herself and Amy, and reported that Amy stopped wanting to go to school.

> *J: So when she's had those problems at school, coming home crying, how does that make you feel?* ANGELA: Awful, but you know, it's more for her, […] I've left school, I'm grown up, it's all for her, it's just horrible for her, it's horrible for any child to go to school and not want to go to school and then,

> you know, everybody else is playing and you're being picked on, and people calling you names and things like, it was a lot, it wasn't just one thing, it was lots of various… nobody to play with her at playtime, nobody to sit with at dinnertime. It was affecting everything, but I'm glad to say everything's changed.

Beth also describes how Beckys' classmates teased her about the size of her clothes, and that this was something that she had to 'cope with'. The use of the word 'cope' suggests that this was something that was difficult for her. Similarly, Deb recalled a time where Daisy's friends had noticed her pubic hair and had laughed about it. Deb talks about how she was hurt that Daisy's friends had behaved in that manner, and it therefore seems to be hard for both parents and children to cope with others' comments.

> BETH: So because of her size, […] they tease her that BECKY's erm, pinafore dresses are this big, say, when a lot of the little girls are very slight little things, so they're a lot smaller. BECKY's cycle shorts for PE, a couple of them teased her that her cycle shorts were this big, whereas the other girls' ones were tiny, you know. So, it's things like that, that erm… she has to cope with at school.
>
> DEB: It [the disorder] obviously must have impacted her in some way, […] it did impact her when [DAISY's friend] saw her [pubic hair], and [DAISY's friends] giggling, that really hurt me, that did.

Theme 2.2.4 "I'm not sure how big a problem this is…: My child seems to be at an advantage, but then again, perhaps not. They are emotionally different." Families also reported their child being at a disadvantage in their emotional control. All families described their child as having mood swings or tantrums. In some instances this emotional turbulence was considered part of normal childhood, but in others, parents felt it

had changed people's interactions with the child, and the child's ability to cope at school. Emotional changes appeared to be the most prevalent difficulty that families faced. Furthermore, two families had concerns over their child's mental health because of their inability to manage their emotions; one of whom was receiving psychological support as a result.

Much of Angela's interview was concerned with Amy's emotions, and she mentioned how this had impacted on almost all aspects of her life; school in particular. Angela stated that it was 'saddening', 'hard' and 'difficult' for the family during this time. There was also a sense of injustice, as Amy was experiencing such 'stress' and 'pent-up rage' 'after what she'd been through', suggesting that she had already been through enough difficulty, without also having to manage her earlier pubertal development.

> *J: […] have there been any situations that have been particularly difficult because of her early puberty?* ANGELA: Schooling, like I said, the things at school was difficult because, not keeping her friends and coming home crying and things like that, […] and the mood swings.

Beth described Becky's emotions as being incredibly intense, and with no basis or rationale. She also describes how Becky can voice how she feels, but doesn't understand why she feels this way. Beth finds it difficult to manage these emotions in her daughter, and there is a sense of feeling helpless at not being able to do anything to make Becky feel better.

> BETH: It's very difficult sometimes in that respect. And because the emotions are so strong as well. […] on her birthday she got up and she was very weepy, and you know she's a happy child, she's a happy girl, and I say to her, you know, 'What's wrong?' and she'll say, 'I don't know', so I give her a cuddle,

> and then she'll come to me a little while later and she'll say, 'Mum I feel like screaming' and she'll, she'll put her fist like that [balls up her fists] but she doesn't

Daisy's parents describe how surprised they were by her mood swings, as her behaviour was more similar to an adolescent than a young child. This lack of coherence with expected behaviour is likely to be confusing for parents.

> DEB: Moody. DANNY: Moody, very moody. That's one of the things that I think has shocked us both, is that, you know, she will just get emotional, which is, which is not what you expect from a 7 or 8 year old. […] DEB: It's like having a teenager sometimes.

Finally, Ellie's parents describe how her emotional changes meant that she struggled to manage her problems at school. This resulted in her releasing her frustration on her parents once she had returned home, making parent-child relationships difficult during this time.

> EVE: So I think with all the rubbish that was going on at school for her […] it was getting her angry and frustrated and build ti– time, of the month in with that just escalated it even more so. Where do you go with all of that anger? It would be to us. EDWARD: Oh, when she used to come out of school, one of us used to get it, [to EVE] didn't we? EVE: Yeah. And she was vile at times, there's no question about it. She was vile.

Theme 2.3 "I'm not sure how big a problem this is…: My child is really no different." Another view families held of their child was that they were neither advantaged nor disadvantaged by the disorder, and were actually no different to their

peers. This subtheme was concerned with puberty being an entirely normal process, which had simply occurred at the wrong time, rather than being part of a medical condition. This served to play down the problem, and portray the child's development as simply being at the earlier end of typical variation. Other quotes were concerned with the efficacy of hormone treatment, and how this had removed any differences between the child and their peers, and meant that they were now 'normal'.

Angela describes how Amy is coming to the age where she would be expected to attain puberty, and that it is therefore not 'a big thing'. Similarly, Eve describes how she reassured Ellie that everyone goes through puberty at different rates, and that it is normal for people not to be exactly the same as each other. Furthermore, Edward points out that puberty is a 'normal' 'part of life', albeit having occurred at a younger age. In each of these instances, the parents and carers perceive puberty as a natural process, and their child is therefore no different to their peers for entering puberty a little earlier than usual.

> ANGELA: The puberty, […] she's coming to that age. A lot of people it's just, she hit puberty, not to worry. So, it's not, a big thing.

> EVE: Yeah, I told her that she's not different in any, in ANY WAY. She's just older in her body. Some people… erm… develop boobies quicker than others, some don't develop boobies at all, you know, this is how we talked about it.

Deb however, reiterated throughout the interview that Daisy is 'normal', which implied that this was either a habitual response that Deb had given to other parents or teachers at Daisys' school, or an attempt to reassure herself, or Daisy, that nothing was wrong.

> DEB: Just that she's really normal, she's such a normal little girl, and she's had this thing on us too early, and it's… a strange thing, and it's, probably… it's… it's just, she's so normal, she really is.

Theme 2.4 "I'm not sure how big a problem this is…: As far as anyone else needs to know, my child is no different." In order to uphold this appearance of the child being 'normal', families described ways in which they endeavoured to keep the condition a secret. This was evident in all cases, despite many families having previously insisted that their child was 'normal', 'average' or 'no different' to their peers. This almost presented as trying to convince others, or perhaps themselves, that there was nothing wrong. Maintaining privacy was most problematic in situations such as changing for swimming, or staying at friends' houses. In some cases, the children appeared to have developed their own methods of hiding their bodies; however families were still concerned with the consequences of other children seeing their child's physical development, and expressed a wish that the child change alone. In some instances families' desire for privacy drove them to not disclose the diagnosis to the child's school at all, which then led to difficulties when the child was absent to attend hospital appointments.

Theme 2.4.1 "I'm not sure how big a problem this is…: As far as anyone else needs to know, my child is no different." - Keeping things hidden. Examples of trying to keep the disorder hidden were given by Beth and Deb, both of whom had concerns over their daughters' classmates noticing their physical development when changing. Beth described how Becky hides herself when she is changing, because she is aware of the possible consequences of others noticing her pubic hair. In Deb's case, Daisy's friends had noticed her pubic hair when she stayed at a friend's house, and their mother had felt guilty for not encouraging Daisy to change separately. Deb had assured her friend that she wasn't

upset, although she admitted that she was privately disappointed, as she had previously made great efforts to hide Daisy's development from others. It seems that both parents and children are therefore concerned that the child's peers will see their physical development.

> BETH: […] when they go swimming, they, the boys get changed in a communal changing room, and the girls get changed in a communal changing room. Well, because when they get changed for PE, they obviously don't take their underwear off, their knickers, but when they get changed for swimming, they get, you know, they completely strip off don't they, so because of the pubic hair, I've asked her if she wants to be segregated again there, but she's figured out a way of doing it by wrapping the towel round her, so that she doesn't have to be segregated, and she can do it without anyone seeing, because she's worried that if people do see, that she will be teased.
>
> DEB: My friend, [DAISY's friend's mum], was devastated, 'DEB, I should have, I should have told her to go in the bathroom and get changed on her own', and, I said, 'No, it doesn't matter, she didn't know,' but it was a very difficult situation, because I had always been very careful.

Theme 2.4.2 "I'm not sure how big a problem this is…: As far as anyone else needs to know, my child is no different." – Keeping things private. Several families had decided to keep their child's diagnosis from other adults, such as school teachers or other relatives. In some cases, this was because of a desire to prevent rumours from circulating, and in others to protect people who may be upset with the diagnosis.

Charlotte's parents had decided not to tell the school of the diagnosis, as they wanted to maintain as much privacy as possible. The use of the word, 'debating' suggests that this was not an easy decision, and had been discussed at great length. It may be that

Charlotte's parents believe she will be treated differently if people know of her diagnosis, or that she will be the focus of gossip and rumours. Alternatively, the family may be embarrassed about the diagnosis, and not want to have to explain it to others, or simply believe that it is not others' business.

> CAROL: I was debating about it [telling school about CHARLOTTE's diagnosis], and then my husband and I, […] I was like…. […] you can talk to the headmistress, ok fine, but she will be changing in like two years, she will be changing to different classes. There'll be a class teacher and a classroom assistant. I don't want everybody knowing about her, you know?

Eve also talks about keeping the diagnosis a secret, as she doesn't want Ellie to be perceived as 'different'. She states how important this is, which suggests that there is something very negative about being perceived as 'different'.

> EVE: What I don't want, more than anything, is for ELLIE to be different. And I think that is really important.

Some families had decided to keep their child's condition a secret from other family members, such as grandparents or aunties and uncles. This was typically to prevent the child from being treated with pity. In other cases this was because of a perceived inability of other family members to understand the condition, or an expected lack of support.

Carol didn't believe that Charlotte's grandparents would be able to deal with her diagnosis, and had therefore chosen not to tell them. Similarly, Deb's mother had been told about Daisy's condition, but had struggled to come to terms with this to the point that she would no longer help her grand-daughter have a bath. This left a great deal of

responsibility with the children's parents, as their grand-parents were not a source of support.

> CAROL: Back home, we haven't [told people about CHARLOTTE's diagnosis], except you know, people who can take it, because you know the old parents, they can't take it, they can't deal with that.

> DEB: My mum struggled with the physical side of the change, you know, because not everybody can cope with how her body's developed, which isn't similar to other eight year olds, which is something that we both handle and do recognise, but my mum can't bath DAISY. She just, she, she just finds it a bit odd that she's different.

Eve also had chosen not to tell Ellie's grandparents of her diagnosis, although this was because she believed that Ellie's grandparents would feel sorry for her, and therefore treat her differently. Eve did not want Ellie to be pitied for her pubertal development, and there seemed to be a level of frustration in her use of the phrase, 'it's a bloody period, that's all'. This suggests that families feel defensive when people show pity towards their children.

> EVE: Yeah, and just… and so we haven't [told ELLIE's grandparents about her pubertal development] yeah, I suppose we're just protecting her really. I don't want people to feel sorry for her. You know? It's a bloody period, that's all, you know.

Theme 2.4.3 "I'm not sure how big a problem this is…: As far as anyone else needs to know, my child is no different."- Keeping things 'normal'. The desire to hide the child's disorder conflicted with the need not to segregate the child or inadvertently

emphasise that they are different. Parents appeared to find it difficult to strike a balance between protecting their child and encouraging normality and independence.

Beth discussed the way in which she has managed this problem, by offering to ask Becky's school if she can change alone but whilst keeping the conversation light-hearted. She describes not wanting 'to make a big thing of it' for Becky.

> BETH: I mean, I try not to, I don't want to make a big thing of it, because if I do, then obviously, it will make a big thing of it for her, […] like the question about asking her if she wants to get changed separately. It's not a deep and meaningful conversation, it's a, you know, 'Do you want me to go into school and see if you can get changed on your own?' and she knows exactly why I'm asking, and she'll say yes or no, and you know, that's it. Conversation's done.

Carol describes conflict between protecting Charlotte and being overprotective, and how it was 'tough' to know what was best for their daughter. It therefore seems difficult for parents to balance this desire to protect their child, whilst still encouraging their independence and not emphasising the problem.

> *J: That sounds like a hard line to draw sometimes [between being protective and being overprotective]*. CAROL: Oh it is very tough, believe me, you don't know whether you're doing the right thing or the wrong thing, and you have to, you know, sit and think. Are you doing this, is this the right thing for her?

Deb felt that she had no choice when it came to letting the school protect her child. Although the family had decided that it would be best for Daisy to change alone, there were still concerns that her peers would ask questions, and that she would still be

identified as different. Deb admitted to worrying about her daughter, and it would appear that this is something that families feel considerably anxious about.

> DEB: It's just… I've just got to trust them [school]. That they'll… they've been great, they've said she'll go in her own cubicle. But then there's the whole question of all the girls will ask well why is she going in a cubicle? You know, it's… yeah, so it'll be… it'll be fine, I just worry about her, that's all.

The second theme therefore suggests that parents' positive perceptions of the diagnosis described in theme one, are not necessarily stable. In fact, families maintain several simultaneous perceptions of how the diagnosis has affected their child, and move between these perceptions regularly.

Theme 3: "So why don't I feel comfortable with this?" The third theme was of families experiencing negative emotions surrounding the child's condition, despite in other instances perceiving the condition as a relief and reporting that their child was still, in fact, 'normal'. The negative experiences reported by families were varied; from feeling sorry for their child to fearing what the future might hold. There was a great deal of cross-over across cases, and it would appear that these sources of discomfort and emotional reactions are common in this group.

Theme 3.1 "So why don't I feel comfortable with this? Will this affect her in the future?" The most prevalent negative emotion was fear of what would happen to the child in the future, predominantly regarding physical and mental health problems. Families who had researched the disorder on the internet appeared to harbour more concerns than those who accepted the medical opinion of their consultant. Some families also reported mistrust in their consultant and were not comforted despite their reassurance.

Beth reported several concerns, one of which was that there were underlying neurological problems that had been missed in the initial examinations, that would present themselves later in Becky's life. Beth does not seem to have been satisfied by her consultant's reassurance, and still has lots of questions about the condition.

> BETH: I have had the worry occasionally, 'Is she going to be taken away from me early?' […] I mean I know it's a stupid, I mean, when they were looking for the brain tumour initially, I think the doctor I spoke to at the time said that if it's not there now, it can't be there in the future, and I, I hope that's right, but that has been a worry sometimes, that something more sinister has been going on with her health that, you know, is going to appear at some point.

Eve's concerns were related to her daughter's fertility, and fears that because she had begun to menstruate earlier she may also reach menopause earlier. Eve discussed this at great length, although she recognised that she should not worry about it as much as she does.

> EVE: […] Will she stop having her period, you know? A woman only produces so many eggs. Will she be thirty? […] But, she, she might go through the change early. I do, I know I shouldn't run away with it, but there are times when it think, could she, could it all come to an end at such a young age as well, because she's starting at a young age?

A lack of predictability and sense of hopelessness at being unable to help seemed to be prevalent across all cases, and families seemed resigned to the fact there was nothing that could be done, other than wait to see what the future would bring.

Theme 3.2 "So why don't I feel comfortable with this? I feel sorry for her." Another negative experience was feeling pity for their child. Families were upset that their child had to manage such difficult emotions, and felt that it was a shame for them to have to cope with using different toilets at school or other children making comments about their physical development. This was a sad thing for families to see, and the excerpts suggested that families felt a sense of helplessness.

J: How did you feel when you first noticed these changes? ANGELA: I felt… upset for her.

CAROL: But I feel very sorry for her sometimes, because I'm like, why does she have to face all this at this age, now? And she's only 9 now.

DEB: I felt sorry for DAISY because she was different.

EVE: Only in the sense of puberty, what a shame. She's too young.

Theme 3.3 "So why don't I feel comfortable with this? I still don't really know what to expect." Parents also felt unable to predict how the disorder would progress. This was particularly salient when discussing the impending onset of menarche, and the ways in which they tried to prepare for this. The use of words such as 'at risk' or 'fear', strongly imply that menarche is a negative event, and is perceived as the marker of reaching adolescence, and the final sign that their daughter is no longer a child.

Both Eve and Deb discuss how they have prepared for their daughters to have their first period; Deb by informing Daisy's school and Eve by teaching Ellie how to use sanitary products. Families seemed resigned to the fact that their daughters will reach menarche early, but frustrated with the inability to predict when this might be. Eve states that they will 'just have to take it as it comes' and that there is nothing that can be done.

Similarly, Deb has been informed that Daisy will not be eligible for treatment to delay her menstrual cycle once she is nine years of age, and again, that there is nothing that can be done.

> DEB: But she is at risk of going into early menarche, so... if that happens then, we want people to be aware, because there's nothing we can do to stop it, and Dr S says she don't want to stop it, and she's not going to treat it if it's after nine, and she's nine in March, […]

Eve summarises the situation in the excerpt below, by explaining how situations are manageable when you have control over them, but that because the family have no control over their child's pubertal development it becomes harder to cope with. This was evident across all transcripts.

> EVE: I think that's the worst of every life situation. When you're in control of something, it is easy. When you've got no control over something, that's when it becomes a problem. And we've got no control over what's going to happen.

Theme 3.4 "So why don't I feel comfortable with this? It's just wrong." Another observation was that the early development was perceived as 'wrong', and caused families considerable discomfort. This seemed to be most related to fertility, and the connotations associated with pubertal development. The knowledge that their child was now capable of reproduction was something that families found difficult to come to terms with. For other families, some of the physical signs of development were not consistent with what was expected for a child their age, and this was upsetting.

Theme 3.4.1 "So why don't I feel comfortable with this? It's just wrong; it's not how children should look." Quotes from Carol and Deb's interviews demonstrate

how their daughters' pubertal development was not consistent with what was expected for their ages. The physical changes were described as, 'not right', 'frightening' and 'strange', and seemed alien to parents.

> CAROL: And umm… yeah, the breast changes, that's when I thought, no, this is, this is not the right age to have all this. [Quieter voice] And of course the pubic hair.

> DEB: Before it was frightening… initially it was, 'Oh no, we've got a little girl and she's six and she's got a body of a twelve year old,' you know, and she was still not seven, and it was just… weird, strange, to see it really.

Eve was most uncomfortable with the knowledge that her daughter was now biologically able to reproduce, and found it very difficult to accept this.

> EVE: I think for me, and I know this is going to sound really stupid, but when, when… when the doctor or consultant, professor or whatever says to you, when your daughter's eight… 'They can have a baby,' it kinda puts a different perspective.

There is a direct contradiction to the way in which we perceive children and the expectations we have of them, compared to the changes that occur during pubertal development, as it is this development that typically marks the end of their childhood. This conflict is hard for parents to accommodate.

Theme 3.4.2 "So why don't I feel comfortable with this? It's just wrong; it's not how children should behave." Other aspects of the disorder that families considered to be 'wrong' were the child's adult-like thoughts, emotions and behaviour. Families compared their child to a much older individual, and found these adolescent or adult

characteristics to be strange for their age. In the excerpts below, three main issues are described; behaviour, thoughts and emotions.

Angela describes how Amy's behaviour is more similar to a teenager than a child, and that this felt 'wrong' from a carer's perspective. Nearly all families likened their child to a teenager at some point during the interview, and it seems that this is a common perception.

> ANGELA: Oh. It's like a teenager already at a young age which is wrong, in all areas.

Beth describes how her daughter thinks about things that are not normal for a child. Becky is reported to talk about mortality and 'profound' topics that a child her age should not be aware of. This concerns Beth greatly, and she worries that this may become problematic for her later in life. Beth also voices concerns about the emotions that Becky is experiencing as a result of her pubertal development, and feels that it is wrong for such a young child to have to manage these emotions. There are therefore several behavioural and emotional aspects of puberty that parents feel uncomfortable about in their young child.

> BETH: But she comes out with some odd things sometimes. I mean, I've had to say to her recently, she'll wake up and say, 'Mum I could die today' and I, I say to her, 'Why on earth are you saying that', and she just laughs it off, but it's just… profound things like that coming out of an eight year old, well, now nine, nine year old's mouth, you know, it just doesn't seem right.

Theme 3.5 "So why don't I feel comfortable with this? I don't understand where it has come from." Families expressed a strong need to know what the cause of the disorder was in their child, and one case wondered openly if difficulties the family had

experienced had caused their child's early pubertal development. Other families knew the cause of the disorder, whilst others had their own ideas as to what had triggered puberty in their child. This need to understand why the disorder had come about was evident across all cases.

Both Becky and Daisy's families had wondered about different factors that may have caused the early puberty. Whilst Daisy's family pondered whether there was a genetic basis for the disorder, Beth feared that difficulties the family had had in the past had caused her condition. Beth reported feeling guilty that this may be the case, and had researched other factors in the media, such as diet and computer use.

> DANNY: It's not genetic though is it? DEB: Yeah, that was it, I thought, 'Where has this come from?' You know? So that was the worry, particularly when we first sort of, found out.

> BETH: So that's the only thing, like I said. It's a factor in BECKY's life that there was a quite stressful period going on at that time. So has it got anything to do with the fact that she… erm… started developing early?

Eve, however, took a more 'karmic' perspective, and questioned why this had happened to them, and what the family had done to deserve it.

> *J: Could you tell me more about, sort of, how you felt, and what was going through your mind at that time?* EVE: Why her? You know, why, what, why should it be… what have we ever done?

All families wondered why the disorder had come about, and wanted to understand more about it.

Theme 3.6 "So why don't I feel comfortable with this? I don't know how to manage this." The final negative point concerned the various sources of difficulty families had experienced when trying to manage the disorder in their child. This covered several areas, from emotional outbursts, to relationships with school or other family members.

Theme 3.6.1 "So why don't I feel comfortable with this? I don't know how to manage this sudden change." Families appeared to face a difficult period towards the onset of the disorder, and they struggled to come to terms with this shift in their everyday lives. This initial shock was daunting for both the child and the parents or carer, and took a period of time to become accustomed to, as these two quotes demonstrate.

> CAROL: It was difficult, the first, I think, I think the first month was really tough, and the first injection was [sigh] was really tough.

> *J: […] Could you, explain to me a bit more about how you felt before that diagnosis?* DEB: Frightened. Frightened. DANNY: Yeah. DEB: Very frightened.

Theme 3.6.2 "So why don't I feel comfortable with this? I don't know how to manage these physical changes." Another aspect that families found difficult was managing the physical aspect of the disorder, such as improving hygiene or finding suitable clothing for the child's size. Children were reportedly unhappy with some of the new changes that they had no choice but to manage, and this unhappiness in the child was distressing for families.

Beth, in particular, talked about how Becky's body odour was not helped by deodorant or regular washing, and how her pubic hair growth had affected her hygiene,

meaning that Beth had to ensure this was well-managed. Becky was upset by this, despite Beth giving her as much choice over the matter as possible. Again, there was a sense of helplessness and not knowing what to do for the best. Similarly, Carol described her struggles in getting items of clothing to fit Charlotte because of her larger size. It is evident that the physical side of puberty is difficult for both parents and children.

> BETH: Erm, I, we deal with her sweating, her body odour, […] it's a lot stronger, […] because the body odour is a lot stronger than mine's ever been, or that I ever remember mine being, so I don't know whether it is stronger in them for some reason? You know, so I have to deal with that, because I can, we can wash her arm pits four or five times and it can still smell, it's that strong.
>
> BETH: Erm, then she doesn't like the fact that, erm, she's having to let me trim her [pubic] hairs, but I ask her what she wants to do, what does she want me to do about it, give her the options that she's got, and then she picks what she wants to do, and that's what we do. You know, so, trying to make her feel like she's got a bit of control over it, […] but just something silly like that can cause a lot of upset, because she does get quite upset over it, so, and then if she gets sore, that upsets her as well, so it can sort of spiral, if you, if you're not careful, […]
>
> CAROL: And then again, […] When I have to go to get her clothes, she's 9, but I have to look for 12 to 13, and sometimes even the 12 to 13 doesn't just, go around her waist.

Theme 3.6.3 "So why don't I feel comfortable with this? I don't know how to manage these emotional changes." As previously mentioned, managing the child's

emotions was another challenge for parents. Families reported that their child's emotional outbursts affected their ability to communicate as a family, and that they struggled to reason with the child during these periods. Some cases reported that they felt unable to control their child's behaviour, and one parent reported feeling a failure because of this.

This example from Beth's interview, demonstrates how intense Becky's emotions are, and how difficult it can be to break through these emotions to speak to her rationally. Beth mentions that she sometimes wonders what she has done wrong when she is unable to help Becky out of her emotional state, and that she sometimes feels like a 'failure'.

> *J: What's the most difficult bit for you*? BETH: Getting her to… to listen to what you're saying, […] and actually taking it in, because sometimes she's so…. How can I say it, she's so stuck on how she's feeling, that her emotions are so strong, that she won't take on board anything that you're trying to say to her to help her.

Eve also describes how hurt she felt seeing her daughter struggling with these negative emotions, and it therefore seems to be difficult not only from a behaviour control perspective, but because of the emotions elicited in the parent when seeing their child so distressed. The parent or carer must therefore not only manage their child's emotions but also their own.

> EVE: So not only emotionally has she had a lot to deal with, which is pain-which hurts, it hurts me as a mum.

Carol also describes how she has had to regulate her emotion during Charlotte's outbursts, but in having to manage her temper rather than feeling empathy for Charlotte. Carol reminds herself of the reasons behind Charlotte's emotional behaviour in order to

remain patient. Eve also describes how she has to demonstrate restraint when Ellie speaks to her rudely. It therefore seems to be incredibly challenging for parents to manage these emotional difficulties in their children as they progress through early puberty.

> CAROL: Now um… I'm more patient, I'm like, I know I have to calm myself. She's doing this just because there's something going on in her body. Otherwise she's normally very good. I keep telling that to myself before I start screaming [laugh].
>
> EVE: You know, 'You can't talk to me like that, I know you're going through a really tough time at the minute, but that doesn't allow you to give me, for you to speak to me in that way. Erm, let's find another way of getting the frustration out, ok?'

Theme 3.6.4 "So why don't I feel comfortable with this? I don't know how to manage these changes at school." A further difficulty experienced by families was managing the child's condition at school. In some instances this was due to the hormone treatment, which resulted in the child taking days off. In some cases, keeping the disorder private from school was the most problematic, but in other families it was ensuring that sanitary facilities would be provided or monitoring peer interactions and changing situations to prevent teasing.

Carol's main concern was keeping Charlotte's diagnosis from the school. This meant that Carol had to lie about the reason for Charlotte taking time off school when she was absent for treatment. Carol admitted feeling uncomfortable about this, and it appears that there is a conflict between wanting to protect Charlotte and not wanting to deceive the school.

> CAROL: […] I don't want to tell them why she, and anyway, I just say she's had an appointment and it's been late, or that she's sick that's all. So that is the hard, I mean, I don't like to lie to the school, so that's the only hard thing that I face.

Deb and Eve also felt anxious with regards to their daughters' schools, however, their concern was surrounding changing facilities. Families felt uncomfortable at the possibility that their children's peers may see signs of their pubertal development whilst changing for swimming or sports, and this caused them considerable concern.

> *J: […] do you feel sort of anxious when she's having to change?* DEB: Oh yeah, If I'm not there, yeah, I'm very anxious, yeah.
>
> *J: Are there any situations that you have found particularly difficult because of the precocious puberty?* EVE: At school, undressing really. I found that quite difficult.

Theme three therefore encompasses the various sources of difficulty that families have encountered due to their child's disorder, as well as the negative emotions that they have experienced since the diagnosis.

Theme 4: "What are we going to do?" The final theme focused on the ways in which families managed the difficulties encountered as a result of their child's disorder. This theme could be divided, firstly, into the ways in which families sought support from professional and non-professional sources, and secondly, into the ways in which families provided their child with support to aid their management of the disorder.

Theme 4.1.1 "What are we going to do? Let's get support from medical professionals." One of the main sources of support was from medical professionals. This

was highly valued by families, and all cases considered themselves fortunate to have received such help. Several families questioned what would have happened if this support had not been available, or if the support offered had not been as efficient. The excerpts below demonstrate this feeling of gratitude towards medical staff and the level of satisfaction with the care received. Furthermore, Eve talked about how they felt valued by the hospital staff, and that even though the doctors were likely to see children with far more serious conditions, they still treated their daughter with the same level of concern and dedication.

> ANGELA: […] if I didn't have the support from the doctors, and the hospital, and the timescale, […] if I didn't have the speed that we had, [sigh] it doesn't bear thinking about, but everything just flew nicely, and everybody understood everything, and the help was there, and she's getting the help now, and she doesn't need it for much longer. I mean, it sounds stupid, but it's all worked out lovely.
>
> EVE: She was ours, she was going through it, this is devastating, this is massive, and they respected that, [to EDWARD] didn't they? EDWARD: Yeah.

Families also felt anxious about what would happen when this support was removed and the child was discharged. Consultants' reassurance and regular monitoring of the child's health was very important to families in helping them to feel secure and able to cope with the disorder, and the loss of this consistent contact caused apprehension, even though there would be nothing further medically wrong with the child after a certain age. This was particularly salient in Daisy's family, who felt it would be 'a bit frightening'.

Theme 4.1.2 "What are we going to do? Let's seek medical treatment." Families of the children who were eligible for hormone treatment found this to be valuable, and were surprised but pleased with its efficacy. They reported that they had observed improvements in their child, both physically and emotionally. The treatment appeared to reduce the extent of the difference between the child and their peers, and the concerns and implications associated with this difference.

Angela describes how the hormone treatment has made improvements on several areas of Angela's life, as well as Amy's. Angela feels that the treatment has not only improved the physical side of Amys' diagnosis, but also that she is happier and better able to communicate.

> ANGELA: She is having treatment at the [hospital], she's having […] artificial hormone replacement thing. It is actually working. It's slowed down the growth side of things, and you can see she's not as argumentative, or stressed out, or het up about things as she was before. So, I'd say she's less like a moody teenager, and more like a happy person, a happier child, so I can see the difference, definitely.
>
> *J: Has that [the hormone treatment] made it easier for you?* ANGELA: Yeah, in lots of things. 'Cause I'm understanding it better, and she's calming down, so we can talk better. It's just easier in the whole house, you know, it's not just her, she's got like three siblings as well, so it's a lot easier. I can, 'cause, you know, every little thing can have a knock-on effect, and it, all it is it's easier for us now.

Theme 4.1.3 "What are we going to do? Let's seek psychological support." In Angela's case, the family was also receiving support from mental health services for

Amy's emotional problems. Angela felt very positively about this, and reported that it was beneficial for the entire family. There was a sense of the psychological help being necessary for Amy to be able to cope with secondary school, and the family's support not being sufficient in this, however, Angela was keen to work alongside professionals to support Amy.

> ANGELA: I'm going to have an appointment with her [CAMHS] worker […] So, I'll know exactly where I can help AMY, if I can help AMY, you know if can work alongside in that area, and see exactly where AMY is. So yeah, I'm looking forward to that, but I can already see that she's a lot happier, and we're all happier.

Theme 4.1.4 "What are we going to do? Let's ask the school for support." Although not all families had informed the school of the child's diagnosis, most of those that had felt well-supported by school staff. Families trusted them, and felt confident that they would care for the child. Furthermore, teachers balanced not appearing to favour the child, but still caring for them if they became emotional, and ensuring that their classmates did not tease them for their physical development. There was a sense of not having any choice but to trust the school to protect their child, however, most families were pleased with the care that their child received.

Angela, in particular, was pleased with the school's support, because they had provided extra tuition for Amy and someone for her to confide in when she was feeling emotional. Furthermore, the school had worked to support Angela and work together as a team to ensure that she was not managing the problem alone.

> *J: How did you feel when she was going through that quite… erm… quite volatile period?* ANGELA: Yeah, I'd say that was a good word. Upset. A bit

> stre-, well no… I'd say stressed definitely, but with the school, we used to have meetings with the school, with social workers as well. And once everything was sort of identified, teachers were saying, 'Well we can work with this'. She had another person who could come in and do some extra reading with her. And then she had, there's another lady at the school who, anytime she's got some problems, she can go to her. So, you know, between all the meetings and all that support, everything has knitted together. Like I said, in every area I don't know what we would have done without each and every one of them.

Beth and Eve were also pleased with the support that the school were giving to their daughters; Beth because they were understanding of Becky's behaviour and were considerate of this when dealing with peer disputes, and Eve because Ellie's teacher was open with her about sanitary products and allowed her extra time to visit the bathroom. Both families felt that the school staff understood and took their daughters' conditions seriously.

> *J: […] how did you explain, erm, her diagnosis to her teachers, if you did explain it to her teachers?* BETH: Yeah, I did. I went in and I've spoken to them. Again, they're a really good, good school, good bunch of teachers, just told them exactly, you know, what the diagnosis was, and they've been very understanding, and with that in mind they do look after BECKY as well. If there's anything going on, they always take that into account as well.
>
> EVE: So this teacher, I mean, she is good, she goes, 'ELLIE if you need, I've got my own in the cupboard, if you run out, I've got some in there, if I run out,

> I know you've got some in your bag,' so they've got a really good relationship. She's absolutely brilliant. She's a really nice teacher.

Theme 4.1.5 "What are we going to do? Let's seek support in our religion." Carol talked about feeling supported in her religious beliefs, and how praying and having faith that their God was watching over them, gave them confidence that they could manage Charlotte's disorder.

> CAROL: I think again, the talking, the talking and erm… I have to tell you, I don't know what you're going to think, but we pray a lot, so it's just, 'God please help us,' you know? So that has helped, really really helped.

Theme 4.1.6 "What are we going to do? Let's seek support from family and friends." Other families sought support from friends and relatives, although this appeared less valuable than medical support, and more of a bonus than a necessity. Some families had chosen not to divulge the child's condition to other family members, and needing support from these individuals was in no way consistent across cases. Those who had confided in friends and relatives, however, found this to be helpful and were comforted by the support. Beth also sought advice from her sisters, and felt that this had helped her to manage Becky's emotional behaviour.

> CAROL: You don't know where that support comes from. It's just, people have been supportive. Not many know, but whoever knows they've been really really nice about it, yeah.

> DEB: I mean, it's been here for the last three years, so. They [DEB's friends] were with me when it was diagnosed, and you know, DAISY goes to the hospital, and they're very supportive really.

Theme 4.2 "What are we going to do? We aren't being given enough support."

There was, however, evidence of families not feeling as though their concerns were taken seriously when seeking support. Parents were frustrated by others' lack of concern and found this hard to accept. This did not appease families, however, and all cases continued to search for support, and fought to ensure that their child was receiving all of the care they were entitled to.

In some cases this lack of support was from their local doctor, as was the case for Beth, who reported insisting for nearly a year that something was wrong with Becky before being taken seriously. In other cases, families did not feel supported by their child's school. Eve in particular was unhappy with the lack of suitable facilities for Ellie, and she had had to argue for things she believed should have been provided as standard.

> BETH: But it was very frustrating here, trying to get any help. It wasn't until I actually went to [name of second hospital] that I got any decent kind of help with her.
>
> EVE: It's only been her year four teacher that, I duno, and now her year five teacher, the head teacher knows, and the deputy head knows of the puberty, but that is because we've had to put things in place, like a sanitary box, silly things like that that they hadn't got in place, for, you know, nine year old girls, for goodness sake. They should be having these things in plain sight.

These situations were frustrating for families, and added further anxiety on to an already stressful situation

Theme 4.3 "What are we going to do? We need to support our daughter."

Families talked a great deal of how they supported their child through their disorder. This

support took several forms, but all with the aim of ensuring the child felt comfortable with their development and didn't feel any different to their peers.

Theme 4.3.1: "What are we going to do? We need to support our daughter by reassuring her." The first way in which parents provided support was through love and reassurance. Examples of this included physical affection, or verbally comforting the child. The quotes below demonstrate the way in which this comforting approach by parents was used to console their daughters when they were unhappy.

> ANGELA: People erm, double-taking [at AMY's breast development], and, you could see their double-take, and you could see her look, and the arms go up and things like that, so you know she's a bit embarrassed. Just, call her over, or give her a hug and come out the room and say like, 'If you don't want to be here you don't have to.'

> BETH: And she knows it's hard to deal with at times, because she gets quite cross about it sometimes. You know, she'll have a little shout and, you know, say, 'Why should I? Why have I got this that makes me feel like this?' And I just tell her it's because she's special.

Theme 4.3.2: "What are we going to do? We need to support our daughter by protecting her." Another method of support was to protect the child, for example by physically helping the child to hide their body whilst changing their clothes, through speaking to teachers to ensure they are not teased by their peers, or protecting them from finding out too much about the processes of puberty and sexual reproduction, and therefore protecting their innocence. All families described ways in which they needed to protect their child as a result of the diagnosis, which implies that families perceived the disorder as something they needed to be protected against.

There was a sense that knowledge of puberty and reproduction could be harmful to their children's innocence. Parents and carers did not seem confident in how much their child was capable of understanding, and didn't want to give them more information than they could manage. An example of this comes from Deb, who has been careful to only provide Daisy with as much information as she felt necessary

> DEB: That's why I haven't gone into the birds and the bees and about reproduction and periods at eight, because I don't want her, I wouldn't normally do that for an eight year old and I don't think she needs it, so we have treated her like an eight year old, so… when she's older, when she's in year six, […] they will start explaining things, and she knows about the difference in… I just want her to be normal, and I think she is.

Other parents were concerned with protecting their child socially, and ensuring that they would not be teased as a result of their condition. All parents were determined that their child would not stand out as being different. Eve describes the desire to protect her child as a 'mother's instinct', and something that she will 'continue to do'.

Furthermore, Beth was adamant that she would not let Becky stand out as being different, and for Deb, whose daughter's development was noticed by her peers, this had become even more of an issue and she admitted to having become even more protective afterwards.

> BETH: You're thinking about that, and whether she's going to get teased, or, you know, what's going to happen, but, you know, we'll deal with it at the time. So, I won't, I won't let it happen basically. You know, I've already said I'm not going to let her get bullied in any way shape or form for being different, so, we'll just have to deal with it.

DEB: And I have always been very protective, and now it's made me even more protective, and I don't want anybody to see her in that situation again.

There was a sense of determination from the parents and care-givers, and a strong feeling that their children had become vulnerable and in need of protection as a result of their condition.

Theme 4.3.3 "What are we going to do? We need to support our daughter by preparing her." Families provided further support by promoting understanding of the disorder and preparing the child for what might come. This was in contrast to trying to protect the child, as discussed previously. Families valued the ability to communicate with their child and felt that this helped them to cope with the associated physical and emotional changes. Some families reported being completely honest with their child about puberty, and encouraged them to fully understand what was involved so that they knew what to expect. These families had confidence in their child's understanding, and reported having a more open and honest relationship with their child. This seemed to be strongly related to the perceived increase in the child's maturity and resilience discussed in theme two. The excerpts below demonstrate this point.

CAROL: […] we talk to her a lot and all the talking helped I think. Because she understands, she really understands, and I don't know if, how the other children are, […] because she understands, it makes things a lot easier, even when explaining.

J: Ok. And how did you go about explaining that to her? EVE: I told her the truth. I told her this is how it is.

Theme 4.4 "What are we going to do? We're going to get on with it." Through these methods of support, families felt able to manage their child's disorder. Several excerpts contained phrases relating to getting on with things, and carrying on as normal. In this sense, the disorder seems to be something that is problematic, but not impossible to cope with.

> ANGELA: It was, just, you know, things are just a shock at the time, but once you start dealing with them, you can get through them.
>
> CAROL: Now looking back, it's been… how many months now? 10 months? Yeah. And like, she's finished four injections now, yeah. So it's um… I think we've handled it really well, to be very modest.
>
> DEB: We've learned to live with it and not brush it under the carpet, but… DANNY: Try and put it at the back of your mind, and… DEB: Yeah. And that's it really, there's nothing really… it's just one of those really.

The fourth theme incorporates the various actions that families took when managing their child's diagnosis. In some cases this was seeking help from elsewhere, and in others, providing support for the child. Through these mechanisms families appeared to feel as though the disorder was something that they could cope with.

Structure of the Data. Analysis of the transcripts identified several commonalities in how families had experienced their child's early puberty. As can be seen from the above excerpts, families held mixed feelings about the disorder, and used different perspectives to understand and manage the changes appearing in their child. The most striking feature of the data was the lack of linearity with which the diagnosis was understood. Families appeared to move through transitional stages in the way one might

expect when identifying a problem, seeking medical help, being given a diagnosis and then subsequently being offered treatment; however, families' understanding of the implications of their child's diagnosis and how they felt about it was far more cyclical. Families appeared to hold concerns about the future and how they would cope, yet also report feeling confident and able to continue life as normal. Similarly, families seemed to have several perceptions of their child; perceiving them as mature and resilient, yet vulnerable and in need of protection simultaneously. The diagnosis of clinically early pubertal development therefore seems to elicit multiple conflicts in the perception of the child, and families move back and forth between these perceptions over time. Figure 1 attempts to present this observation more clearly.

Figure 1: *Proposed structure of emergent themes and subthemes*

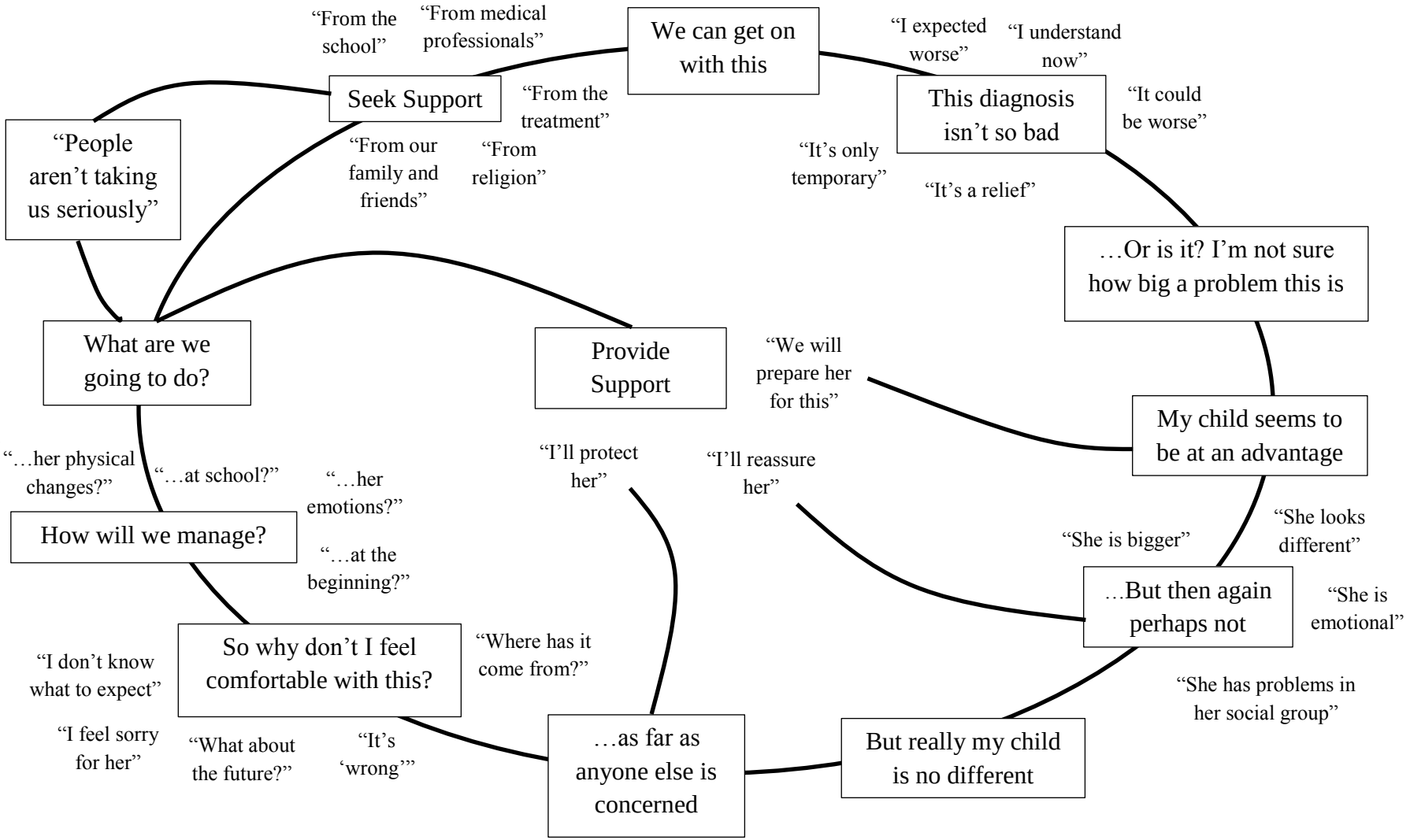

Figure 1 shows how each of the main emergent themes are positioned around the circular structure, with the subthemes distributed around them. Each family talked about how the diagnosis was positive, in that their child could have had a more debilitating condition, or could have been worse affected by the disorder. Furthermore, the diagnosis removed ambiguity caused by the child's physical development, and families felt secure in knowing what they were facing. As we move around the structure, however, we can see that this positivity is not the complete picture. Families reported feeling uncertain as to how much their child was affected by the disorder, whether this be positively or negatively. Families were also keen to normalise the disorder as much as possible, either through hiding the disorder from others, or focusing on the child's similarities to their peers. Families also described situations where they felt negatively about the disorder, in the form of pity for their child, fear of the future, or uncertainty with how best to manage the problems encountered. Lastly, families talked about how they went about dealing with the disorder. Firstly, through ensuring that they were supporting their child, and secondly, through seeking external support. This could be in the form of tangible support, such as medical care, or emotional support from friends and relatives. When this support was not available or unsatisfactory, families expressed frustration and anger at this lack of help. However, when support was effective, families felt reassured, valued, and fortunate to have had this assistance. This helped families to feel confident in their ability to continue as normal.

Discussion. The current study has sought to explore the experiences of families who have a child with a diagnosis of precocious pubertal development, with the aim of finding out what aspects of the process have been most impactful on families, and the ways in which they perceive, understand and manage the disorder. The interview analysis

has produced some interesting and novel findings. When children have a medical diagnosis, it is often they who are the focus when studying the extent of the impact of the condition. However, being the parent or carer of a child with a medical disorder is likely to have its own troubles, as there is a need to manage both the child's emotions, as well as their own. Furthermore, families have to ensure the child's condition is properly managed outside of the home, and do their best to understand medical terminology and adjust to their child having something that sets them apart from their peers. Therefore it is important to gain a parent's insight into the implications of a child's medical diagnosis, particularly when the diagnosis is as sensitive a topic as early pubertal development.

The overall sense of the data suggested that there was little linearity to the way in which families thought of the condition. Although one might expect there to be initial shock, followed by decreasing levels of anxiety as families become accustomed to the diagnosis, the data presented here suggested that families move back and forth between being comfortable and confident in managing the disorder, to feeling worried for their child and having fears about their future. Families held multiple perceptions of their child in different contexts, and would further respond to each of these perceptions in a different way. For example, families experienced relief that the disorder had not been something worse, and were reassured by the fact that the condition was only temporary. Families therefore felt that this was something that they could manage, now that they knew what they were facing. Their children were perceived as mature, resilient, and able to manage their disorder and any effects it may have had on peer relationships or emotional stability. When in this mind-set, families reported feeling positively about the early puberty and able to 'get on with it'.

This positivity did not appear to be enduring however, as families described at length the physical, emotional and behavioural difficulties that they had encountered since the onset of the child's pubertal development. Some of these problems put the child at a perceived disadvantage in comparison to peers, and these difficulties posed a problem for the parent as they required some form of coping strategy or management. Parents and carers spoke of protecting their child, although not from the effects of the disorder, but from the implications of the disorder and being different. Parents desperately wanted the child to feel 'normal', or to prevent others from noticing their development and treating them differently as a result. Families therefore went to great lengths to keep the diagnosis a secret, most predominantly from the child's classmates, and in some cases from family members and the child's school.

Families also repeatedly used terminology such as, 'wrong' or 'not right', suggesting that puberty and sexual development was something that did not fit with their expectations of what a child should be. This was consistent with the findings of Liao and colleagues (2005) who suggested that their participants felt like an 'adult-child' and an object of unwanted sexual attention. In the current project, sexually-related concerns were not explicitly reported in any of the cases, and this may be something that arises as the children grow older. Alternatively, it may be that the participants in Liao's paper were keen to take part in the research because they harboured these concerns and wished to talk about them. In this case there may have been a selection bias, and concerns over early sexuality may not be the case for all patients with clinically early puberty. However, the authors made no claims of generalisability and their findings are interesting whether the experiences reported are for all cases, or a select few.

Families not only reported the disorder as being ‘wrong’, but also described feeling sorry for their child, feeling unable to predict what would happen and when, and feeling powerless to help. Furthermore, families harboured strong fears of how the disorder would affect the child’s future health, and were not reassured by their consultant’s opinion. Families therefore had a great deal of negative emotions to deal with, as a result of their child’s diagnosis.

However, this discomfort with their child’s disorder and desire to protect the child, may be what leads many families to state that their child is ‘normal’ and ‘no different’, despite having given multiple examples as to why this is not the case. This may be a way of coping, by trying to remind or convince themselves that they can manage the problem, and that although the child may have difficulties, these could be much worse. A final suggestion is that the parent has invested so much energy into protecting the child and preventing others from finding out, that this has become embedded in their view of the disorder, and almost a force of habit rather than a perception that the parent actually holds. Either way, it is unlikely that the family actually perceives the child as completely ‘normal’, but more “normal despite their differences”.

A further observation was the variety of ways in which families coped with difficulties which arose from the disorder. Families were keen to seek support from medical professionals, the child’s school and family and friends. The medical support was by far the most valued, and families reported feeling reassured by this regular monitoring and support. Support gained from the school was less stable, and families felt that they had little choice but to trust the school to manage the child’s disorder in their absence. School was also a source of difficulty, with regards to a lack of changing provisions and sanitary facilities. Furthermore, the majority of children’s peer interactions took place at school,

and therefore this was where they experienced the most social problems. Support from friends and relatives was even less consistent, with most families deciding to keep the disorder within the immediate family. This was because individuals, such as grandparents were perceived as not being able to cope with their grandchild's disorder, or that they would treat the child differently and be overly sympathetic. This increased attention would not be consistent with the families' desire to not emphasise that the child was in any way different.

The parents' methods of supporting their child reflected the multiple perceptions that they held. For example, in some instances, families felt that they needed to protect their child, through hiding their body from others, intervening against bullying, and withholding information that they felt the child would not be capable of understanding. This need to protect the child could be related to the disadvantages the parents perceived the child to have as a result of their disorder, and the need to maintain normality. In other instances, parents talked about placating their child with love and reassurance, which linked to the idea of the disorder being manageable and something that they could cope with as a family. A final method of supporting the child, was through promoting understanding and preparing the child for puberty. Here, parents were honest with their child, and trusted that, with their help, they would be able to understand and prepare for the pubertal development. Parents spoke positively about this way of supporting the child, and felt that it had improved their communication and ability to be open with each other. This type of support also appeared to alleviate anxiety in the parent or caregiver, as they felt at ease that the child was comfortable with what they had discussed.

There are several strengths of this project, the key advantage being that it is the only study to use such rich data collection techniques within this population. The findings

demonstrate that, whilst it is accepted that there will likely be difficulties faced by children with precocious pubertal development, there are also several challenges to be faced by parents. This research may therefore inform medical staff working in paediatrics of the ways in which they can support these families, and could also reassure parents of newly diagnosed patients that their anxieties are not unusual. The main limitation of the project is that it only provides half of the story, and combining the parent accounts with the child's experiences would give a more complete impression of how families truly experience the diagnosis. This was, however, beyond the scope of this project. Furthermore, the interviews were conducted by a different member of the research team, and arguably this could have limited the emotional interpretation during the analysis. However, the families had been visited several times as part of a previous project, and were well-known to the lead investigator. Furthermore, Dictaphone recordings of the transcripts were available when clarification was required.

Summary. Overall, parenting a child with a diagnosis of clinically precocious pubertal development seems to be a turbulent process. Families have the unenviable job of trying to support their child through an emotional time, protect them from others' judgement, manage difficulties at school, whilst trying to come to terms with something that they may not fully understand themselves. Parents also have to balance reassuring the child, without drawing attention to the fact that they are different to their peers; and similarly protect the child from others discovering the diagnosis without segregating them. Although there are periods where families feel able to cope with the disorder and its challenges, there is an undertone of discomfort and unease. Families seem to be faced with multiple dilemmas, for example; is my child still 'normal'? How do I protect my child without segregating them? How much of a secret should this be? Although families do not

necessarily face the exact same challenges, they all appear to experience similar emotions, and a cyclical process of understanding and reasoning about their child's disorder.

Although this project simply provided a glimpse into the experiences of parenting a child with precocious pubertal development, it has provided us with valuable insights as to the difficulties families may face in this population. When looking at these findings, we are able to put together a more detailed picture of the experiences of families in this population, and ways in which, healthcare professionals and educational establishments could help others going through the same process in the future.

6. GENERAL DISCUSSION

The aim of the current thesis was to study the psychological profile of children with a diagnosis of Premature Adrenarche (PA) or Central Precocious Puberty (CPP), in order to assess whether these children showed any differences in functioning, in comparison to typically-developing children. Furthermore, stress within the family was assessed, to identify whether this could be affected by the child's disorder, or in some way be causal to the child's early development. Data collection employed the use of varied psychometrics, as well as qualitative analysis of interview data, in order to assess the children and their families across several domains. The hypotheses driving the analyses were derived, firstly, from previous empirical findings reported in the literature, and secondly, from exploring the data to identify any further interesting relationships or differences not previously considered by other authors. A variety of statistical analyses were conducted, in order to best identify where observed differences in performance were related to changes brought about by typical pubertal processes, when performance was altered specifically due to the disorder of either PA or CPP, or when differences were due to atypical timing in general. This meant that psychological profiles for each specific disorder could be explored, as well as the more general effects of being different to peers, or being in puberty, whether early or on-time.

6.1 Body Composition, Body Image, and Eating Behaviour. The first empirical chapter reported findings on body composition, eating behaviour and body image satisfaction. In accordance with previous findings, it was observed that those in the patient groups were taller and heavier than those in the control group, and correlational and trend analyses suggest that as children progress through puberty they grow in height, weight and body mass index (BMI). This would seem logical, as children tend to experience a growth

spurt at the onset of puberty, and grow until they reach their adult size. It could be seen, however, that the clinical groups gained more weight than was proportionate to their height, and were therefore significantly more likely to be classed as overweight. Furthermore, it was observed that those in the PA group gained considerably more weight than the CPP and control groups as they progressed through puberty, whereas the CPP group appeared to be taller and heavier earlier on in their development.

The observation that the two patient groups were of a larger BMI in comparison to the typically-developing group, and therefore at an increased risk of being overweight, corresponds with the available literature (Biro et al., 2001; Charkaluk, Trivin & Brauner, 2004; de Ferran, Paiva, Garcia, Gama & Guimaraes, 2011; Golub et al., 2008; Ibáñez et al., 2003; Kaplowitz, Slora, Wasserman, Pedlow & Herman-Giddens, 2001; Midyett, Moore & Jacobson, 2003). Furthermore, the observed nature of weight gain in the two patient groups is supported by Utriainen, Voutilainen and Jääskeläinen (2009), who reported that growth in height precedes weight gain, suggesting that onset of adrenarche and corresponding skeletal growth, typically occurs before increases in BMI and gonadal puberty. This would support the current observation that those with a diagnosis of PA were gaining height as expected over the course of their adrenarchal development; however, they were also increasing in weight too, and to an extent disproportionate to their height. Conversely, those with a diagnosis of CPP had gained in height and weight earlier on, or prior to, their pubertal development, and although they had the highest values of body composition, they were no longer gaining in height and weight. These findings suggest that although changes in body composition are typical during puberty, changes in weight and BMI are altered in those with a diagnosis of PA and CPP, resulting in them gaining weight to a greater extent than their typically-developing peers, and, in some cases,

becoming overweight. This could have serious health implications long into adulthood, and puts these individuals at an increased risk of multiple health conditions (Prentice & Viner, 2013; Widen et al., 2012), as well as psychological health problems, such as dissatisfaction with body image or disordered eating behaviour (Decaluwé, Braet & Fairburn, 2002; Gualdi-Russo et al., 2008; Lamerz et al., 2005; Vander Wal & Thelan, 2000).

Analysis of the body image data demonstrated that those of a higher weight and BMI, irrespective of diagnostic group, perceived themselves as more similar to the larger figures on the body image scale. This suggests that the scale is a valid and useful tool in exploring body image perceptions in participants of this age group. However, despite a greater proportion of participants in the two clinical groups being overweight, they did not rate themselves as being significantly bigger than the control group. The intervals between figures on the rating scale may therefore not have allowed sufficient variability in participants' responses, in order to distinguish between those who perceived themselves as overweight, and those that did not. It may be that inclusion of a greater number of figures from which to choose may have improved the specificity of the scale. The two clinical groups, however, were twice as likely to rate themselves as bigger than their preferred figure, suggesting that they were more likely to be dissatisfied with their body image and would prefer to be thinner, particularly those in the PA group. Furthermore, when correlating BMI with the discrepancy between perceived own body image, and that of friends' or preferred figures, the PA group showed strong positive correlations, implying that those of a high BMI in the PA group also perceived a larger difference between their body image and how they perceive their friends and their ideal figure. This may put the PA group at increased risk for disordered eating behaviours and body dissatisfaction,

particularly when considering this population's risk of becoming overweight in combination with their heightened perception of differences between their body size and that of their friends'.

Analysis of the eating behaviour data identified no significant differences between the three groups, except for the satiety subscale of the Child Eating Behaviour Questionnaire (CEBQ). Here it was observed that those in the PA group showed lower levels of satiety than the CPP and control groups. This decreased awareness of feelings of satiety may be related to the increased BMI in this patient group, but equally may be encountered during adrenarche in the general population, as this stage typically marks the onset of skeletal growth and therefore a need for increased dietary intake. When the two clinical groups were analysed together and compared to the typically-developing group, it could be seen that the clinical groups also displayed higher levels of restrictive eating, which does not seem to fit with the greater BMI in these groups. It may be, however, that these children demonstrate restrictive eating behaviours in response to their increased body size, in an attempt to alter their body shape. It has also been suggested that girls entering puberty earlier may mistake pubertal growth for weight gain (Ge, Elder, Regnerus & Cox, 2001). As the two clinical groups develop considerably earlier than their peers, it may also be that they perceive themselves as bigger, whether they are overweight or not, and an increase in restrictive eating may be in response to this.

Analysis of the relationships between pubertal development and eating behaviour across all groups, suggested that more advanced puberty was related to increases in emotional and restrictive eating behaviour and reduced satiety. This reduced satiety may reflect biological motivation to increase food intake in preparation for impending pubertal growth spurt. Furthermore, increases in emotional instability frequently observed in

adolescence (Dahl, 2004; Dahl & Gunnar, 2009; Larson, Moneta, Richards & Wilson, 2002) may heighten the desire to eat in response to negative emotions. Additionally, a more acute awareness of one's own body in comparison to peers' may explain the increases in restrictive eating behaviour as children progress into puberty. The combination of these observations, however, suggests a conflicting scenario occurs, in which children feel a biological and emotional need to eat more, but a societal pressure to eat less. The effects of this conflict may cause children going through puberty to develop difficult relationships with food and their bodies, and to experience guilt surrounding dietary intake. For children with earlier pubertal development, this problem may be even more pertinent, as they are at an increased risk of becoming overweight and are developing at a time when their peers are yet to begin their growth spurt.

When looking at each group individually, it was found that those with a diagnosis of CPP became fussier with their food as they progressed through puberty, whereas control participants enjoyed their food to a greater extent and became more susceptible to external and emotional cues. When considering this increased fussiness, in combination with the greater weight and BMI seen in the CPP group, it may be that these children are developing an awareness of their body shape and become increasingly fussy with their intake in order to control weight gain. Typically-developing children, however, may eat more in response to the coming growth spurt, and therefore be more sensitive to cues to increase their dietary intake. In contrast, those in the PA group were more responsive to external and emotional cues, and showed reduced responsiveness to satiety cues as they progressed through puberty. This may explain the observed greater extent of weight gain than would typically be expected in the transition through puberty, as although these

children experience the same drive to increase their food intake, they do not experience the satiety cues signalling them when to stop.

When analysing the relationships between BMI and eating behaviour, it was found that those of a higher BMI demonstrated greater levels of all disordered eating behaviours, except for desire to drink, emotional under-eating and fussiness surrounding food. This would conceptually appear accurate, as the other eating behaviours would be considered to be related to increases in weight. When looking at the relationships between individual groups, however, it could be seen that BMI was not related to any of the eating behaviour profiles in the CPP group apart from reduced satiety. Although this would suggest that the high BMI values in this group could be attributed to a reduced awareness of satiety, when looking at the descriptive statistics, it can be seen that the CPP group showed no difference in satiety scores compared to the control group. This observation, and the lack of any further evidence of disordered eating behaviour in this group, would imply that there is an underlying biological factor which is implicated in the increased weight and BMI in this group. This underlying factor may also mediate pubertal timing in this group. For example, Remsberg and colleagues (2005) found that girls who began menarche earlier were at a significantly higher risk of increased adiposity, high blood pressure, high lipid concentrations and hyperinsulinism throughout pubertal development. As hyperinsulinism is strongly related to weight gain, it is likely that obesity, insulin levels and pubertal development are interrelated. Furthermore, Frontini, Srinivasan and Berenson (2003) investigated metabolic syndrome risk factors in girls who reached menarche either before or after the age of 12 years, and found that the early menarche group showed high levels of fasting insulin and glucose, insulin resistance and a higher BMI and skinfold thickness. They also observed ‘clustering’ of several of these variables in the early menarche group,

suggesting these children are at a greater risk of developing metabolic problems. It may be, therefore, that biological factors mediate the relationship between BMI and pubertal timing in the CPP group.

In both the PA and control groups however, BMI was related to nearly all eating behaviours, and the strongest relationships between these variables were seen in the PA group. This implies that eating behaviour may play more of a role in weight gain in these groups, and to an even greater extent than normal in those with a diagnosis of PA. There is evidence that those with a diagnosis of PA may also be at risk of an altered metabolic profile, which could result in greater weight gain in response to food than in the typically-developing population. For example, dyslipidaemia, hyperinsulinaemia and insulin resistance have all been reportedly observed in those with a diagnosis of PA (de Ferran et al., 2011; Denburg et al., 2002; Evliyaoğlu, Berberoğlu, Adiyaman, Aycan & Öcal, 2007; Ibáñez et al., 1997b ; Ibáñez, Castell, Tresserras & Potau, 1999b*;* Ibáñez, Potau, Chacon, Pascual & Carrascosa, 1998b; Ibáñez, Potau, Francois & de Zegher, 1998a; Oppenheimer, Linder & DiMartino-Nardi, 1995; Potau et al., 2003). It may be that those with a diagnosis of PA are therefore not only more likely to demonstrate disordered eating behaviours, but may also respond to any intake of food differently, putting them at an increased risk of weight gain.

One criticism of this chapter is that the two patient groups were slightly chronologically older than the typically-developing participants, although not significantly so. This may have inflated the differences in body composition between the three groups, as some of the typically-developing participants may have been smaller and lighter simply because they were younger. However, this confound would not necessarily have influenced the group differences in BMI, as it was evident that the patient groups were

heavier for their height than were the typically-developing participants. This is unlikely to have occurred as a result of the participants being slightly older. A further limitation is that although assumptions have been made about the trajectory of variables such as body composition and eating behaviour over the course of puberty, these conclusions are derived from cross-sectional data, rather than longitudinal. This limits the generalisability of these findings. It would have been preferable to measure eating behaviour or body composition variables alongside hormonal assays over a period of time, in order to better understand the relationships between puberty, metabolism, eating behaviour and weight gain. However, this design was not feasible within the constraints of the current thesis. Another improvement on the methodology would have been to assess a child's body image satisfaction over the course of pubertal development, or to compare those beginning puberty at an age considerably younger than expected to those reaching puberty perhaps one year earlier than their peers. This would have better identified what aspects of puberty are influential of the way in which a child perceives their body image.

Overall, however, this chapter has provided compelling evidence that children with a diagnosis of PA or CPP should be monitored, firstly, for any signs of weight gain and the associated health implications, and secondly, for psychological distress with regards to their body image or eating behaviours. Furthermore, the data have provided support for the literature on body composition in those with disorders of early puberty, and also posited possible mechanisms for why these observations of increase BMI may occur. Finally, suggestions have been made for methodological improvements and directions for future research into why children with diagnoses of precocious pubertal development may demonstrate these changes in body composition, eating behaviour and body image satisfaction.

6.2 Behaviour, Emotion and Sleep. The second empirical chapter presented data from the Child Behavior Checklist (CBCL) and Teacher Report Form (TRF) which measured competencies and adaptive behaviours, as well as problem behaviour and psychopathology at home and at school. The first finding was that children in the CPP group were more likely to score below the mean on measures of school competence, although they were not more likely to score below the borderline or clinical thresholds. To some extent, this finding could be explained by progression through puberty, as pubertal development was also found to be negatively predictive of performance at school. This corresponds with findings in the self-perception chapter, where a relationship between greater pubertal development, and perceived lower scholastic competence was observed. This finding, however, was not observed in the TRF data, and descriptive statistics actually implied that the CPP group performed the best at school. This is consistent with parent reports of the child behaving well at school, but being very different at home. This may have also coloured parents' ratings of their performance at school, and also children's perceived competence in their ability.

The syndrome scales were then analysed, and demonstrated that several signs of psychopathology increase over the course of pubertal development, including withdrawn-depressed symptoms, internalising behaviours, and also sleep problems. Although the patient groups scored slightly higher than control participants on these subscales, their performance could be better explained by the predictive value of pubertal development in the regression models. Therefore clinical groups would show these traits to a greater extent due to their further progression through puberty, rather than their diagnosis. This finding is still pertinent in the care of those with a diagnosis of PA or CPP, however, as parents and teachers may be unsure how to manage these difficulties in a younger child. Furthermore,

the child may be less able to cope with experiencing these emotions at a younger age, and therefore show signs of psychological distress.

In addition to the problems encountered as a result of puberty as a typical process, those with a diagnosis of PA or CPP were found to present with more signs of anxiety and depression. However, those with a diagnosis of PA demonstrated this to a greater extent at home, whereas those in the CPP group were rated as of the highest risk at school, although not clinically so. These symptoms did appear to increase with pubertal development across groups, and may typically increase as children progress through puberty; however, the PA group were the only participants to score above the clinical threshold. This suggests that this group is at a heightened risk to that typically encountered during puberty, and that this may be something specific to biological or psychological processes active during PA. For example, it may be that the hormones active during adrenarche impact on levels of anxiety in these children. Alternatively, research has suggested a relationship between stress and levels of dehydroepiandrosterone (DHEA) and its respective sulphate (DHEAS), which are implicated in adrenarchal development (Cicchetti & Rogosch, 2007), suggesting that it may be anxiety which has caused the premature onset of adrenarche. In either case, the data presented here suggest that there is something particular to the diagnosis of PA that is related to heightened levels of anxiety.

Evidence for differing behavioural profiles in the two patient groups was also observed in the aggression subscale analysis, where it was found that those in the PA group showed slightly elevated levels of aggression, whereas a subsection of the CPP group were more likely to score above the clinical threshold for this behaviour. Additionally, the patients with a diagnosis of CPP were more likely to display social problems at a clinical level, both at home and at school, as well as clinical levels of rule-

breaking behaviour at school. These observations of behavioural and psychological problems at a clinical level in the CPP group would imply that there is something specific to the diagnosis which influences this behaviour. It may also be that these observations in the CPP group are related, for example difficulties in peer interaction because of bullying over breast development or weight gain may result in a child displaying social problems and aggression towards the individuals involved, and rule-breaking behaviour such as shouting or fighting at school. These behaviours may also be brought home with the child, who is upset and frustrated because of the difficulties they are having at school. Alternatively, hormonal influences on emotion and the ability to control ones behavioural outbursts may result in higher scores on these subscales. Although the current study cannot determine which specific environmental or biological factors are influencing scores on these subscales in the CPP group, it is still important to consider these findings when working with children with this diagnosis.

A further observation was that both PA and CPP groups scored higher on measures of externalising behaviour as rated by parents, which may again suggest that increased externalising behaviour is related to more advanced pubertal development at any age; however, performance on this scale was less well predicted by the pubertal development variable in the regression analyses. This would imply that the social or environmental consequences of having a pubertal disorder may be related to the increase in externalising behaviours, such as appearing different to peers or having to manage hormonal and morphological changes. These changes are not specific to either diagnosis, and would explain the lack of difference in presentation between these clinical groups. The TRF data, however, would suggest that those in the CPP group are more likely to display clinical levels of externalising behaviour, as well as internalising behaviour. This may be because

features of CPP, such as breast development or skin blemishes, are perhaps more easily observable than features of PA, such as pubic or underarm hair growth. These observable features of CPP may therefore be the subject of teasing at school, and increase the risk of these behaviour problems at school in the CPP patients. It is therefore important to consider, not only possible hormonal bases for changes in behaviour and psychological wellbeing in children with early puberty, but also whether their diagnosis has any impact on their environment or social group, and the potential repercussions this could have on their behaviour and psychological health.

Analysis of the the newly-constructed sleep scale identified no significant differences between the clinical groups or the control group for sleep problems. This was unexpected, as several parents of children in the two patient groups reported having noticed a change in their child's sleeping patterns, and it was these observations that provided the rationale for analysing the sleep data. However, the analysis did identify a significant relationship between pubertal development and increased sleep problems. This suggests that sleep changes increase over typical pubertal development, independent of age, and that although sleep problems are likely to be encountered earlier in the two patient groups, the extent of the sleep problems is no greater than would be expected during typical pubertal development. Nevertheless, it would be helpful for parents of children with a diagnosis of precocious pubertal development to be aware that these possible changes to a child's sleeping pattern may occur earlier than expected, to ensure that tiredness does not affect them at school or in their daily routine.

Despite there being several key findings in this chapter, there are also some limitations that should be acknowledged. The first limitation is that there may have been some recruitment bias in the two patient groups. When considering the rates of attrition,

and the nature of the families who withdrew or declined to participate in the study, it was commonly those who felt that their child may not be able to cope with participation. This would suggest that the children, who were experiencing the greatest levels of behavioural or psychological difficulties, such that they would be distressed by taking part, were not included in the sample. Although this bias in recruitment could not have been helped, as both parents and children were required to consent to participation, it does raise the issue that the sample may not be representative of the patient population, and that one end of the population distribution may not have been included. However, the findings have still clearly demonstrated that children with a diagnosis of PA or CPP may be at an increased risk several types of psychopathology, despite the estimates derived from the present data perhaps being an underestimation of the difficulties present in the population as a whole.

A further limitation of the data presented in this chapter, is that the levels of behavioural and psychological problems were assumed to be a stable construct. It may be, however, that for those in the patient groups, these traits may have changed over the course of the diagnosis. This may be particularly relevant for those in the CPP group who were receiving hormonal treatment with GnRH analogues, as it is generally considered to improve behavioural and psychosocial features of CPP, although no empirical evidence for this as a long-term outcome has been reported (Heger, Sippell & Partsch, 2005). Therefore, a cross-sectional design may not have captured the nature of the behavioural and psychological problems experienced in this population.

It would have been interesting to conduct further analysis into whether scores on the measures of problem behaviour and psychopathology were related to other variables, such as sleep problems, executive functioning, hormonal correlates or family demographics. For example, Sontag-Padilla et al. (2012) suggested that higher levels of

executive functioning were protective against symptoms of psychopathology in children with a diagnosis of PA, and sleep problems have been shown to influence the experience of negative emotions (Ireland & Culpin, 2006). By looking at the relationships between variables, it may have been possible to better identify which children are most at risk of developing psychological and behavioural problems, however, this was beyond the scope of the present thesis.

Despite some limitations, this chapter has identified increases in problem behaviour associated with pubertal development across groups, as well as distinguishing behavioural problems specific to each diagnostic group. This corroborates previous findings in the literature (Dorn et al., 2008; Dorn, Hitt & Rotenstein, 1999; Sonis et al., 1985), and provides further novel observations that have not been previously reported. It was found that children with a diagnosis of PA are more likely to demonstrate greater anxiety and depression, aggression and internalising behaviours at home, whereas those with CPP show anxiety and depression and clinical aggression at home, and aggression, rule-breaking behaviour, social problems and both internalising and externalising behaviour at school. The findings strongly support the conclusion that those with diagnoses of precocious puberty are at an increased risk of problem behaviour and psychopathology, and that they should be routinely monitored for early signs of such. Additionally, behaviours observed differed considerably between home and school. Parents therefore need to be in communication with teachers to ensure that any problem behaviour does not impact on the child's capacity for learning.

6.3 Self-Perception: Perceived Age and Self-Esteem. The primary aim of the self-perception chapter was to identify whether children with a diagnosis of early pubertal development were perceived as older and whether there were any consequences associated

with this, such as having to wear clothes for older children, being spoken to as older, and being given more responsibilities. The second aim of the chapter was to identify whether the clinical groups differed on measures of self-esteem across several domains, including scholastic competence and peer acceptance. It was therefore important to establish, at first, whether or not children in the clinical groups were actually perceived as older. Throughout the thesis, any significant differences observed in the clinical groups could theoretically be attributed to being physically different to peers, and the social and emotional consequences of this. However, it may be that the children in the two clinical groups are not perceived as looking older, or that children do not notice these differences between themselves and their peers, in which case, the conclusion that this difference might be influential in predicting performance on psychometric measures would be invalid.

It was found that participants in the two clinical groups were perceived as looking older to a greater extent than typically-developing children, both by the children and their parents. This demonstrates that children in the two patient groups are noticeably more developed than children in the typically-developing group, and therefore, it would be reasonable to hypothesise that there may be social consequences to having a diagnosis of precocious pubertal development. Furthermore, because the data showed that the children in the patient groups were aware that they appeared older than their peers, it would be fair to assume that there may be some response to this difference, be this positive or negative. It may be therefore, that some of the findings reported in other chapters, such as changes in eating behaviour, or evidence of aggressive or rule-breaking behaviour, could be explained by the child feeling dissatisfied or upset at the differences between them and their peers.

Furthermore, parents of children in the two patient groups reported greater negative consequences to their child looking older, such as people making jokes about their child's

size, or their child expressing a wish to look more like their friends. This suggests that looking older was not necessarily considered advantageous, which could have implications on the child's self-esteem and psychological well-being. Pubertal development across groups was related to higher parent- and child-reports of negative consequences, but reports of positive consequences only increased alongside pubertal development on the parent measure. This suggests that growing up is generally perceived positively in the child group, whatever the level of pubertal development, whilst the positive consequences only become apparent to parents as the child develops. Negative consequences to growing older, however, become apparent to both parents and children as the child progresses through puberty.

In the analysis of the self-esteem measures, it was observed that participants who were further through puberty perceived their scholastic competence to be lower than participants earlier on in development. No other differences in self-perception were found between the clinical groups and the control group. This suggests that as children develop, they either become less competent in their academic ability, or become less satisfied in their ability. This would seem hard to explain, as we would not expect pubertal development to have a negative effect on cognitive functioning. Furthermore, when considering the findings from the intellectual ability chapter, there would be no corroborating evidence for a decline in cognitive functioning as a child progresses through puberty. It may be therefore, that a child's focus on school achievement alters at the onset of puberty, such that they become more interested in peer interaction rather than academic progress. The implications of this are that if children begin to lose confidence or interest in their scholastic competence, they may also lose motivation in their schoolwork and be less likely to achieve their full potential. This therefore should be the focus of future research

and possible intervention. Educational professionals may be able to use these findings to better monitor those going through puberty, to ensure that they receive the support or praise needed to retain their academic confidence and the motivation to do their best.

In other areas of self-perception, such as peer acceptance or satisfaction with behavioural conduct, there appeared to be no influence of pubertal development. This is a positive finding, as research has suggested that children, who experience pubertal development at a time asynchronous to their peers, can show a decrease in their level of self-esteem, particularly regarding their physical appearance (Solyom, Austad, Sherick & Bacon, 1980; Xhrouet-Heinrichs et al., 1997). Furthermore, there was no evidence of lower bodily self-esteem in the two patient groups compared to the typically-developing participants. Although the analysis of the body image scale data (Chapter 4.1) demonstrated that those in the two patient groups reported greater discrepancies between their own figure and their preferred figure or friends' figures, and therefore were assumed to experience a greater level of bodily dissatisfaction, this was not observed in the self-perception data analysis. It may be that although those with precocious pubertal development were more different to their ideal figure, this did not impact on their self-esteem. Furthermore, although there was a greater incidence of behavioural problems in the PA and CPP groups when analysed in chapter 4.2, there was no evidence of dissatisfaction with behavioural conduct in the self-perception chapter. These findings would suggest that despite children with a diagnosis of early puberty being at a greater risk of multiple difficulties, this does not appear to have any bearing on their self-perception or self-esteem.

The findings of this chapter help to tease apart the different influences that early pubertal development may have on a child, by validating the assumption that children in

the patient groups are indeed physically different to their typically-developing peers, and are indeed perceived as such. Overall, the findings demonstrate that whilst children from the clinical groups are aware of looking older or bigger, and report negative experiences as a result of this, their self-perception is generally at no disadvantage in comparison to typically-developing children. This is a positive finding, and suggests that having a diagnosis of precocious pubertal development does not affect a child's self-esteem, or the way in which they perceive themselves in comparison to others. It does appear that children are perceived as older, and that they experience negative and positive consequences to this. This should be therefore be acknowledged and controlled for in future research.

One limitation of this chapter is that the measures used to assess self-esteem were not designed specifically for the clinical population in this project, and therefore the particular aspects of self-esteem which could be affected by precocious pubertal development may not have been identified in the analysis. For example, items designed to assess peer acceptance focused on whether children perceived themselves as having many or few friends, whereas for children with precocious pubertal development, the pertinent feature of peer acceptance may be whether or not their friends make comments about their physical development. The items may therefore not have measured the specific nature of self-esteem affected by having a diagnosis of precocious pubertal development, and the two patient groups did not score higher than the control group as a result of this.

Overall, the chapter has confirmed that children in the PA and CPP groups do perceive themselves as being physical different to their typically-developing peers, and that there are consequences to this difference which could impact on their psychological and emotional well-being. The evidence presented here, however, suggests that despite

these differences, the patient groups have shown no detriment in their levels of self-esteem.

6.4 Intellectual Ability. The fourth empirical chapter provides findings on the intellectual ability data. The aim of the chapter was to identify whether children with disorders of early puberty demonstrated any differences on tasks of intellectual functioning, possibly as a result of early neurological maturation, the effect of sex steroids on brain structures or functioning, or increased environmental demands associated with being treated as older. Here, it was found that the two clinical groups scored fairly consistently with the control group, and all groups scored within the normal range. It was observed, however, that the clinical groups performed worse on verbal tasks, but better on speed of processing tasks, particularly in the CPP group. When considering the correlation and regression data, it is likely that this observation can largely be explained in terms of changes over the course of pubertal development rather than traits specific to the clinical groups. This finding is positive, in that children with a diagnosis of clinically early pubertal development do not appear to require additional support at school, although teachers may need to bear in mind that those progressing through puberty may benefit from extra support on verbal tasks, or more simple written instructions.

The observed decrease in verbal ability may be related to the self-perception data in chapter 4.3, or the school competence data in chapter 4.2, where perceived scholastic competence and parental ratings of school performance decreased alongside increases in pubertal development. Furthermore, the speed of information processing data suggests that children in puberty may finish classroom tasks faster than their pre-pubertal classmates. It may be that children who are further through puberty process information more quickly because of a more general advantage in cognitive speed, but at the cost of other academic

achievements, such as verbal performance. Alternatively, there may be a mediating factor, such as a hormone active during pubertal development, which influences both verbal ability and speed of information processing, although the research supporting the effects of sex steroids on cognition is inconsistent (Herlitz, Reuterskiöld, Lovén, Thilers & Rehnman, 2013).

Although the findings reported in this chapter seem difficult to explain, the fact that they appear to correspond with the reported findings of the other chapters suggests that there is some basis for these observations. However, when looking more closely at the descriptive statistics, it can be seen that the CPP group mean for the verbal ability subscale is 100, which is also the standardised mean for the general population. It may be, therefore, that it is actually the control participants in the sample, who are performing better than predicted, rather than the clinical groups scoring worse than predicted. This may be due to an error in administration of the measure, or possibly a sampling bias. It may be that the nature of the families most likely to respond to recruitment requests through their child's school, may be families for whom their child's academic achievement is very important, or who take an interest in research. The control group may therefore not be representative of the typically-developing population. For the speed of information processing subscale, however, it can be seen that the control participants are scoring closer to the standardised mean, and that the clinical groups are scoring considerably above this mean value. This would therefore suggest that there is a change in cognitive processing speed over the course of pubertal development and into adulthood, which would seem a logical concept.

One limitation of the intellectual ability chapter is that the sample size may not have been large enough to demonstrate any differences between groups. It is likely that any changes occurring as a result of, or in relation to, precocious pubertal development

would be of a small effect size, and therefore the small sample size may have limited the statistical power required to identify these differences. Furthermore, the fact that there were so few male participants in the two patient groups restricted the analysis of gender differences. This is an important limitation when considering the sexually dimorphic nature of pubertal development and neurological lateralisation (Blakemore, Burnett & Dahl, 2010; Giedd et al., 1999; Lenroot et al., 2007; Neufang et al., 2009; Nuñez, Huppenbauer, McAbee, Juraska & DonCarlos, 2002). A larger and more homogenous sample would have enabled a greater level of sensitivity in the data analysis, and better detection of any influences of pubertal processes on intellectual ability.

A final limitation is that the effects of circulating hormones were not considered in the analysis. For example, it may be that participants in the CPP group, who were receiving hormone treatment, may have been exposed to different levels of sex steroids to those in the PA and control groups. Similarly, it may be that participants who were diagnosed with a disorder of precocious puberty at a later age may have shown less of an effect of circulating sex steroids in comparison to those who were diagnosed at a younger age. This is because any effects of sex steroids active during puberty may have a stronger impact on a younger brain, or have a cumulative impact over time. Therefore the duration of exposure to circulating sex steroids, and the nature of the sex steroids itself, may have influenced performance on measures of intellectual functioning. This was not controlled for in the present chapter.

Despite the limitations described above, the findings of the intellectual ability chapter still provide evidence for changes in intellectual ability in relation to pubertal development. It is reassuring to note that there seems to be no observable disadvantage to

participants in the PA and CPP groups, although further research should seek to validate these findings with a more representative sample and use of hormonal measures.

6.5 Family Environment and Parental Stress. The final empirical chapter focused on the family environment and parental stress levels. The aim was to identify whether certain demographic variables posed risk factors for children developing early puberty. Measures of parental stress and the occurrence of life events were also conducted, firstly, to test whether having a child with a diagnosis of precocious pubertal development caused increased stress levels in the parents, or whether increased stress or the occurrence of life events may have been related to the onset of early puberty.

When looking at the structure of the families participating in the project, it was found that those in the control group were most likely to be living with both biological parents, whereas a larger proportion of the clinical groups were living with a single parent or a non-biological care-giver. Furthermore, when discounting diagnostic group, children living in non-biological care-giver or single-parent households had significantly greater pubertal development. This would imply a relationship exists between family structure and pubertal development, and suggests that family breakdown may be a contributing factor to the onset of puberty.

This observation is concordant with several findings in the literature. For example, Tither and Ellis (2008) demonstrated that younger sisters, who had spent more time being raised in a household with an absent father, experienced menarche 3-4 months earlier than their older sister who had spent less time in this environment. This was especially evident in girls who were exposed to more severe levels of dysfunctional paternal conduct, such as substance abuse or violent behaviour. However, Deardorff and colleagues (2011) found that father absence was only associated with earlier development in higher income

families, specifically early breast development in white families, and pubarche in African-American families. These findings seem difficult to explain, and Deardorff and colleagues suggested other factors contribute to early development, such as longer maternal working hours, smaller social support networks in single-parent families, and exposure to technology and beauty products in higher-income families. Rates of family breakdown in the population, however, are fairly high. We might therefore expect the incidence of disorders of precocious puberty to be fair higher than is actually observed, and to be present in all siblings within the family household. The relationship between family structure and disorders of early puberty is likely not to be straightforward.

The present chapter measured parental stress, as well as family structure and socioeconomic status (SES). Families with both biological parents were more likely to be of a higher SES, and therefore, it is assumed, lower stress levels. Those with a single parent may therefore be at a greater risk of financial problems and as a result, increased levels of stress. Analysis of the families' stress levels demonstrated that no significant differences in the reported stress levels between groups, however, correlational data demonstrated that in the control group, family life events, particularly negative events, were related to a child's pubertal development. Furthermore, regression data suggested that negative life events were the best predictor of pubertal development out of all stress and demographic measures. This suggests that family stress, or negative events occurring in the family, is related to earlier onset of puberty, but not necessarily causing onset to occur before the clinical cut-off. This would imply that stressful events may advance pubertal onset in typically-developing children.

In the PA or CPP groups, however, pubertal onset does not appear to be related to parental stress or family life events, as there was a lack of significant differences between

the control group and clinical groups on measures of parents' stress. If onset of precocious pubertal development was related to stressful life events, we would expect the clinical groups to score significantly higher than the control group on measures of this variable. This does not seem to support the finding that the patient groups were more likely to live in single-parent households. Stress in the family however, is not necessarily a stable construct, and it is possible that if families in the two patient groups had completed the same measures immediately prior to the onset of their child's pubertal disorder, they may have scored far higher. This is a limitation of the present study.

Further analysis of the stress variables in relation to family demographic variables demonstrated that being of a higher SES appeared to be protective against the impact of the diagnosis in the PA group. This meant that families in the PA group who were of a higher SES bracket, showed lower scores on the impact of event scale, than did those of a lower SES. However, the same analysis demonstrated no beneficial effect of SES in the CPP group. This implies that receiving a diagnosis of CPP has a greater impact on families, than does a diagnosis of PA, such that it was not mediated by SES in any way. This was particularly evident on the intrusion subscale, suggesting that thoughts of the child's diagnosis were more disturbing for parents in the CPP group.

This observation differs from the findings of Solyom, Austad, Sherick & Bacon (1980), who reported that parents of children with a diagnosis of PA or premature thelarche showed greater difficulty in coping with their child's disorder, than did parents of children with a diagnosis of CPP. The authors concluded that because the development of a child with a diagnosis of CPP is comparable to how the body would change during typical pubertal development, it suggests to parents that the child's body is functioning as it should, despite developing slightly earlier than expected. For parents of children with a

diagnosis of PA, however, the child's development may signal that something is wrong. This conclusion was not supported by the current sample.

Taken together, these findings not only identify which children are at greatest risk for developing puberty early, but also demonstrate that parenting a child progressing through puberty is likely to be stressful for parents. Furthermore, families with a child diagnosed with CPP or PA, particularly those of a lower SES, may require additional support from medical professionals, to ensure that the family are coping with the diagnosis. This has implications for individuals, such as school nurses, who may be best able to identify a child at risk of early puberty, and for paediatricians or endocrinologists, who can monitor the family for signs of high stress. Additionally, the findings contribute to the life-history literature and evolutionary-development theories, which commonly implicate the role of family functioning on pubertal timing.

There are several limitations to the research presented in this chapter. The first is that the measures of parental stress were not administered at the onset of the pubertal disorder in the patient groups. This limits the extent to which we can associate family stress or life events to be associated with the onset of a precocious pubertal development, and the effect that parenting a child with a diagnosis of early puberty has on parental stress. Measuring parental stress and family life events longitudinally would have been an improvement on the methodology described here.

A further improvement could be to compare rates of parental divorce across groups, and, in those where parental separation has occurred, assess temporal proximity of the divorce to the child's pubertal onset. Although the data presented here suggest that those residing with a single-parent or non-biological relative are more likely to have earlier pubertal development, this has not been demonstrated experimentally, and may have been

caused by a mediating variable. For example, other authors have suggested that a genetic factor influencing parental relationships may also mediate the child's pubertal onset. For example, Comings, Muhleman, Johnson and MacMurray (2002), suggested that an X-linked androgen receptor gene may not only incline fathers to aggression, impulsivity and more sexual relationships, but also may be passed on to their daughters, resulting in earlier pubertal development. It could be that this genetic predisposition in fathers causes them to behave more sexually, resulting in them being absent from the family home, as well as influencing pubertal onset in their daughters. Future research could therefore measure rates of sexual impulsivity and parental age in relation to child's pubertal onset, to explore the existence of relationships between variables. The majority of the available research literature focuses on the experience of the child, and behavioural or emotional changes that occur as a result of their diagnosis. This chapter, therefore, provides a new perspective on precocious pubertal development, and the ways in which the diagnoses impact on the lives of all family members.

It is clear that there is much uncertainty surrounding the relationship between stress in the family environment and pubertal onset. Future research should look to identify more specifically what aspects of the family environment influence pubertal timing, and whether these factors are also accountable for the development of a pubertal disorder of precocious development.

6.6 Qualitative Analysis. The final chapter of the thesis summarised the qualitative analysis of interview data, from five families recruited from the patient groups. The findings derived from this analysis provide the reader with a greater insight of families' experiences when parenting a child with a diagnosis of early pubertal development. One key observation from this analysis is that it is not only children who

have to adjust become accustomed to this transformation, but also parents and carers, and that in some ways this is more difficult for families to cope with, than the child. The majority of the available research literature focuses on the experience of the child, and behavioural or emotional changes that occur as a result of their diagnosis. This chapter, therefore, provides a new perspective on precocious pubertal development, and the ways in which the diagnoses impact on the lives of all family members.

One interesting observation finding taken from the data is the evident instability of families' feelings towards the diagnosis and its consequences. Families fluctuate from feeling confident in the diagnosis being manageable, to feeling overwhelmed with the problems that they now face. This left the family feeling unsure as to how much of an impact the diagnosis had actually had on their lives, and families spoke at length of how they reasoned around and tried understand what the diagnosis actually meant to them and their child.

When investigating the impact of medical diagnoses, it is often assumed that any experiences or changes occurring as a result of a disorder are stable constructs. The findings of chapter 4.6, however, suggest that families' experiences of parenting a child with a diagnosis of a pubertal disorder fluctuate considerably, and are incredibly dynamic. Experiences are not observed to simply change gradually over time, in the way one might expect an individual to adjust to a difficult event, but can vary even within the same conversation. This may reflect the way in which parents reason about their child's disorder, in that they hold fears about the diagnosis, as well as feeling relief that the diagnosis was not something more serious. Balancing these conflicting perceptions may be confusing for parents, as they may be unsure as to how they really feel about the diagnosis.

The findings presented in this chapter, therefore, suggested a more cyclical structure to the data, rather than the linear structure expected initially.

In addition to highlighting some interesting observations in the way that parents and carers understand the meaning of their child's diagnosis, the qualitative data facilitates understanding of the results of the quantitative analyses. It may be that the inconsistencies observed in the research literature, and even within the data from the current sample, can be explained by the variability in parents' feeling about their child's diagnosis. If parents hold multiple perceptions about their child and the diagnosis, it is unclear which of these perceptions will be measured at any one time-point. This poses a problem for research in this population, and indeed, other populations of parents caring for a child with a medical diagnosis.

One limitation of the research presented here is that there was limited opportunity to relay the findings of the analysis back to families, for verification of the conclusions drawn from the data. There was therefore no respondent validity process employed in the process. When discussing the analysis and conclusions with other researchers, and medical professionals working with families, however, the findings were met with agreement, and appeared sufficient with respect to face validity. Furthermore, although Interpretative Phenomenological Analysis (IPA) methods often recommend use of a reflexive diary, in order to explore the role of the researcher's prior experiences and internal processes on data interpretation, this practise was not employed in the current project. Instead, regular supervision was made use of, to discuss dynamics between the researcher and the data, to ensure that any biases in interpretation were acknowledged and accounted for. Furthermore, the use of different researchers for interview data collection and data analysis facilitated objectivity. This is because the researcher conducting the interviews was not

guided by any hopes or expectations of what would transpire in the analysis, and the researcher conducting the data analysis was not able to change their interviewing style in accordance with the findings from analysis of the prior interview. An improvement on the methodology may have been to include a reflexive account, describing more explicitly any values or assumptions that could have influenced interpretation of the data at each stage of the analysis.

6.7 Strengths, Limitations, and Future Directions. There were several strengths to the current thesis. The first strength being that functioning was measured across multiple domains. This allowed for a more thorough investigation of the behaviour and mental health of children with early pubertal development, as well as any potential impact of this on their parents, and therefore a better understanding of the ways in which families may benefit from further support. Furthermore, this level of data collection provided the opportunity to explore the relationships between a greater number of variables, and whether any of these variables were observed to cluster in certain participants. This provides a more detailed picture of how a child with a diagnosis of early pubertal development may present, and the different features which may increase a child's risk of altered psychological wellbeing.

A further strength of the study was that not only were the clinical groups compared with typically-developing children, but also with each other, enabling the study of specific profiles for each diagnosis. This enables for a clearer understanding of which areas of development occur as a result of the hormone-specific changes particular to each diagnosis, or which can be attributed to morphological or social consequences of having a diagnosis of either PA or CPP. Additionally, the more exploratory analysis of pubertal development as a continuous variable against performance on psychometric measures

allowed identification of traits that likely develop over the course of puberty, independent of time of onset. Although this variety of analyses is useful in exploring the different factors which may influence a parent or child's experience of precocious pubertal development, the vast amount of data and complex combination of analyses may have presented a confusing picture. Furthermore, combining the findings from each chapter into a cohesive and comprehensible account poses a challenge, particularly as some of the findings appear to be contradictory.

Despite the variety of findings throughout the thesis presenting a somewhat complex interpretation of the impact of PA or CPP on a child and their family, several of the observations can be used to inform other chapters. For example, it was observed in chapter 4.3, that the children in the two patient groups were perceived as older than their peers, as hypothesised. The observation of increased internalising and externalising behaviour at school in the CPP group may therefore be in response to a social consequence of the disorder, such as peers' reaction to the child looking older. Furthermore, the reduced scores on verbal ability scales as children progress through puberty may be related to the decreased scholastic competence observed on the measures of self-perception. In this manner, each chapter of the thesis contributes to the understanding and interpretation of the findings of other chapters. Although it would have been interesting to study the relationships between all variables across the dataset, this was unfortunately beyond the scope of this current thesis. However, this large-scale analysis would have identified whether certain features of a child's behaviour or psychological well-being could be observed to cluster together. Clustering of behavioural or emotional variables would suggest that some children are at risk of multiple difficulties as a result of their diagnosis, or alternatively, clustering of several risk factors, such as lower SES or obesity, would

identify children at greatest risk for developing a disorder or precocious pubertal development. Future research should therefore seek to explore relationships between multiple variables, in order to better understand the difficulties faced by this population, or the factors which may predict development of a disorder of puberty.

Another important observation made from the chapters of the present thesis, was the lack of consistency observed in families' perception of the diagnosis in the qualitative chapter. This instability in how families felt about the disorder may go some way to explaining the mixed findings of the empirical analyses. Whether the families were visited on a positive day or a negative day could have influenced the way in which they completed the psychometric measures. If we assume parents of children in the two patient groups to move continually between multiple perspectives of their child's disorder, we may expect their responses to the quantitative questionnaires to vary depending on which perspective they hold at the time of completing the measure. It may be that test-retest reliability analyses would show little consistency of parents' perceptions across time-points, as their experiences vary from day to day. It may be, therefore, that longitudinal analysis of the constructs being measured would have better demonstrated the way in which the diagnosis of precocious pubertal development has impacted on both the child and the parents. This is a methodological flaw in the quantitative section of the thesis, as it was based on the assumption that the traits being measured are stable, and persist throughout the duration of the disorder. It may actually be that the various constructs being assessed were more prevalent at the onset of the disorder, before hormone treatment commenced, or at certain times during the month. Furthermore, parents' perceptions of these traits may vary over time as they become accustomed to managing them. The findings of the qualitative data

analysis are therefore incredibly important in understanding how families are affected by a disorder of PA or CPP, and how best to measure this impact.

During the qualitative interviews, an observation of note is that parents and carers typically began the interviews quite positively, and it wasn't until they were encouraged to give further information, that they admitted to the difficulties they had experienced and became more honest and openly emotional about their experiences. In this sense, psychometric measures may not have captured this more honest data. Families may also have wished to appear as though they were coping well with the child's diagnosis, and that there was little of concern. This may have led to a social desirability bias, and potentially, the larger standard deviations observed in the CPP group. Returning to families to seek respondent validity for the conclusions drawn from the data, may have better clarified whether this was indeed the case, or whether there was actually a large amount of variability in the presentation of children in this population.

When considering the existing research literature exploring the features of precocious pubertal development and its impact on the family, it is evident that the area of study is vast and complex, with few conclusions being undisputedly agreed upon. As a result, the literature review sections of the thesis presented here were considerably longer and more convoluted than initially intended. The basis for the literature review sections were that it was considered important to provide a comprehensive overview of previous research publications for the reader, and to demonstrate the reasons for incorporating such a variety of variables and analyses in the thesis. Because the existing research literature on puberty and pubertal disorders extends across medical, psychological and evolutionary fields, and varies further within each domain, selecting which aspects of the research to present in the thesis posed a challenge. It may have been preferable to select fewer features

of the diagnoses to discuss, and subsequently explore empirically, to provide a more concise and elegant research thesis.

However, the research presented in this thesis has several applications across medical, educational and family settings. Firstly, risk factors of early puberty have been identified, such as family structure, life events and body composition. This information could be used by school nurses or GP's to identify those most at risk of early puberty, and aid referral to endocrinologists at an earlier stage of development. Furthermore, it has been shown that those with a diagnosis of early puberty are at an increased risk of several psychological problems, such as symptoms of anxiety and depression, aggression and body image dissatisfaction. Awareness of this increased risk by medical staff may improve identification of children that require extra support, or even psychological input, and allows practitioners to prepare parents for the types of behaviours they may face as a result of their child's diagnosis. In addition, the findings add to several areas of the research literature, including evolutionary and life history theories, paediatric and adolescent psychology, family and social studies and behavioural sciences.

A limitation of the research as a whole is the lack of consistency between group sizes. The smaller sample size in one group limited the statistical analyses that could be used, and therefore the sensitivity to more subtle differences between groups due to a loss of statistical power. Furthermore, the lack of normality in the distribution and homogeneity of variance of all of the groups determined the use of non-parametric statistics in the majority of analyses. This may have increased the risk of a Type I error, and over-confirmation of hypotheses. Unfortunately, recruitment of unequal group sizes is a common hazard when conducted research with clinical populations, and it was found that the CPP group in particular were difficult to recruit. This may simply be because of the

nature of the diagnosis; in fact several families mentioned that their child was embarrassed by the diagnosis and was therefore uncomfortable with participating in anything in connection to this. Other families withdrew from the project or declined because they felt that their child would not be able to cope with taking part. This attrition has likely meant that those who would have scored at the more extreme end of the psychometric scoring distribution were not prepared to participate, therefore bringing the group performance closer to the overall mean.

Further research could incorporate measures, such as hormone assays, in the methodology, to observe any direct influence of hormonal levels on constructs such as emotion or behaviour, or relationships between the two. This may also have functioned to validate the pubertal development measure upon which all correlational and regression analyses were based. For example, Cance, Ennett, Morgan-Lopez and Foshee (2012) suggest that there is error in cross-sectional measures of pubertal development, and that puberty is better coded as early, on-time or late using multiple data-points over time. It would also have been preferable to recruit patients at the onset of the disorder, in order to assess them before any hormone treatment, and whilst families still felt most concern over their child's condition. Families could then have been visited on multiple occasions over time to identify whether changes in behaviour may correlate with changes in hormone levels, or families became less stressed as they grew accustomed to their child's diagnosis. It would also have been informative to include older children who were at a similar pubertal stage to the patient groups. In this way it could have been observed whether or not the clinical groups' behaviour was similar to those progressing through puberty at the age typically expected, or whether behaviours were specific to the two diagnoses due to asynchronous timing of pubertal development.

In conclusion, the current thesis has given an overview of the psychological and behavioural problems observed in children with diagnoses of precocious pubertal development, as well as identifying areas where these children are no different to their peers. Furthermore, the impact of a diagnosis on the family environment, and the influence that the family environment has on the diagnosis, has also been explored. It would appear that although the clinical groups are at an increased risk in some areas, they are no different to their peers in others. It is therefore advisable that these individuals are monitored for psychological and behavioural problems, both at home and at school, to ensure that signs of poor wellbeing or psychopathology are identified early and the child and their family are referred for extra support.

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LIST OF APPENDICES

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APPENDIX 1: PUBERTAL DEVELOPMENT QUESTIONNAIRES

Please complete the questionnaire by marking a circle around the options which are most applicable to your child's stage of pubertal development.

Testicular Development	No development	Small amount of growth	Moderate amount of growth	Almost fully developed	Fully developed
Genital Development	No development	Scrotum slightly pigmented	Lengthening of the penis	Increase in length and thickening of the penis	Adult genitalia; appears fully developed
Pubic Hair Development	No hair growth	Few long, downy hairs, mostly around the base of the penis	Increase in amount & distribution of pubic hair. Hair becomes darker in colour	Hair is adult in colour and texture, but extending over less of body	Adult amount & distribution of pubic hair
Facial Hair	No growth	Fine hairs under the nose and on the chin	Few longer hairs on the face. Hair near the ears is becoming distinctive	Occasional need to shave	Adult growth and frequent need to shave
Body Hair	No real hair on arms and legs	Fine hairs on arms and legs	Noticeable hair on arms and legs. Under-arm hair begins to develop	Arm and leg hair becomes darker and more wiry. Thicker under-arm hair	Adult-like growth
Skin Changes	No changes	Occasional blemishes	Frequent breakouts of blemishes	Fewer breakouts of blemishes compared to younger years	Occasional blemishes when compared to younger years
Growth	Steady and usual growth in height or weight	Beginning to increase more quickly in height, and gaining 1-2 inches a year	Experiencing a growth spurt, and gaining several inches a year	Growth is beginning to slow	No change in height for over a year
Body Shape	Child-like body shape	Child-like body shape but with larger hands and feet	Limbs begin to grow out of proportion to body	Body proportions similar to an adult, but with narrower shoulders	Adult body shape
Voice Changes	No change in voice	Voice-box becoming more noticeable	Breaking or squeaking of the voice	Deepening of the voice, with occasional breaking	Voice is consistently deeper.

APPENDIX 1: PUBERTAL DEVELOPMENT QUESTIONNAIRES

Please complete the questionnaire by marking a circle around the options which are most applicable to your child's stage of pubertal development.

Breast Development	No development	Breast buds form	Breast contour growth	Growth and darkening in colour of the nipples.	Breast is adult in appearance
Pubic Hair Development	No hair growth	Sparse longer, downy hairs, close to genitalia	Hair is darker, curly and more widespread	Adult type over less of body	Adult amount & distribution of pubic hair
Body Hair	No real hair on arms and legs	Fine hairs on arms and legs	More noticeable hair on arms and legs. Under-arm hair begins to develop	Hair on arms and legs becomes darker and thicker. Occasional need to remove hair	Adult-like growth on arms, legs, and under-arms
Skin Changes	No changes	Occasional blemishes	Frequent breakouts of blemishes	Less frequent breakouts of blemishes compared to younger years	Occasional blemishes when compared to younger years
Growth	Steady and usual growth in height or weight	Beginning to increase more quickly in height, and gaining 1-2 inches a year	Experiencing a growth spurt, and gaining several inches a year	Growth is beginning to slow	No change in height for over a year
Body Shape	Child-like body shape	Slight increase in weight	Increase in length of legs and size of feet. Breast development is more noticeable	Widening of the hips and thighs. Development of typical female form	Adult female body shape
Age at which your child first started menarche (periods)			years		months

APPENDIX 2: PARTICIPANT INFORMATION PACK – CONTROL GROUP

UNIVERSITY OF BIRMINGHAM

Frankland Building: School of Psychology
University of Birmingham
Edgbaston
Birmingham
B15 2TT

Covering Letter

Dear Parents and Guardians,

My name is Emma Clarkson, and I am a researcher working at both the University of Birmingham and Birmingham Children's Hospital. I am currently carrying out a study funded by the Child Growth Foundation, and am writing to you as you have demonstrated an interest in taking part. The study is looking at the behaviour of children diagnosed with disorders of puberty, and would involve you and your child simply answering a few questionnaires, and your child taking part in a few assessments, including IQ and self-esteem. None of the assessments are invasive or distressing, and your child may even find them quite fun! The study would require approximately three hours of your time, can be spread over two sessions and would be arranged entirely at your convenience.

You would not directly benefit from the study; however your participation will have a huge impact on the families of children with puberty disorders, as there is currently very little information available. We aim to raise awareness of these disorders amongst healthcare professionals, so that families can access the correct treatment and support more quickly.

In this information pack you will find the following:

- A 'Participant Information Sheet' for parents. This contains the contact details of the research team, and general information about the project.
- An 'Interviewing Schedule' with further information on the different tasks involved in each stage of the project.
- A 'Participant Information Sheet' for the child taking part.
- A prepaid envelope containing questionnaires, to be returned to the research team once they have been completed.

Please do not hesitate to get in contact if you have any questions.

Many thanks for your time,

Emma Clarkson
Doctoral Researcher

Investigating psychological and behavioural differences in children diagnosed with atypical adrenarche and central precocious puberty, compared to children of typical pubertal development.

Participant Information Sheet for Parents/ Guardians of Typically Developing Children

Background Information

Early puberty is becoming more common in recent years, yet there is very little information available to families with a child diagnosed with a puberty disorder. Atypical adrenarche is where children develop particular signs of puberty earlier than would typically be expected, such as pubic and under-arm hair growth, skin problems and body odour. Children diagnosed with central precocious puberty also show these bodily changes in addition to breast and genital development. These children also experiences changes in their emotions and behaviour, as well as in their physical appearance. Research has suggested that children with a puberty disorder are more likely to show signs of depression, anxiety, aggressive behaviour and poor self-esteem. Other research has suggested that children who develop early may have higher levels of stress in their family. The aim of this research is therefore to study these problems in detail, in order to provide information for families and evidence for healthcare professionals that children with this diagnosis, and their families, may require psychological support. This research has been funded by the Child Growth Foundation, and has therefore been deemed useful and indeed necessary to improve the lives of children diagnosed with these disorders.

Procedure

The procedure for the study consists of several parts. These are listed below.

1. Questionnaires and consent forms have been sent to you. These questionnaires ask for demographic information, contact details, and basic information regarding your family. A form to determine your child's stage of pubertal development has also been included, to ensure that your child is eligible to participate. This stage will take approximately 30 minutes.

2. A meeting will be arranged, during which a measure of your child's IQ and BMI (body mass index) will be carried out. You will be asked to complete questionnaires on your child's eating behaviours. This meeting will take approximately two hours.

3. A second meeting will be arranged, during which your child's view of their body image will be measured, and an assessment of their self-perception and self-esteem will be carried out. You will also be asked to complete questionnaires on some of your child's behaviours, as well as questionnaires looking at family stress levels and events that have occurred recently in your family which may have affected this. This will take approximately two hours.

4. Questionnaires will be sent to your child's school teacher, to assess their behaviour at school. This will then be returned directly to the research team.

Further Information and Ethical Declarations

What we ask of you

All meetings and questionnaires will be completed when it is most convenient, and will not require you to undertake any form of travel. We will call you to arrange the best time to visit you in your home. None of the assessments are at all intrusive, and will simply require yourself or your child to answer simple questions, or indicate one choice out of several options. We will take no more than five hours of your time overall. If your child shows any sign of becoming distressed or bored then the assessment will be stopped immediately.

What you can expect from us

We will maintain full confidentiality by taking several precautions. All paper and digital data gained through the study will remain secure, by being kept in locked filing cabinets or on password-protected USB drives. This data will remain at the University for a period of three years after the project has finished, after which all paper and digital forms of data will be destroyed. This will take place in accordance with the University of Birmingham's data protection and confidential waste policies. No data published in the final article will contain any identifiable information, and your details will be known only to the researchers. If any causes for concern arise regarding child protection, I am under duty to discuss this with my supervisor, and may have to report the concern further.

What if you change your mind?

If at any point during the course of the research yourself or your child express a wish to withdraw from the study, this wish will be respected with no reason required, up to the point of data analysis. If your child becomes frustrated or bored during any one of the assessments, the assessment can be postponed to another date if this would be more suitable.

How this will benefit you

Your family will not directly benefit from taking part in the project, however we will give a book voucher to your child's school or make a charitable gift donation of your child's choice, as a token of gratitude for your participation. The study will be highly beneficial to children diagnosed with disorders of puberty in the future, and your participation will contribute massively to improving the information available to parents, teachers and healthcare professionals. This will raise awareness of the disorder and encourage provision of psychological and behavioural support for children and their families.

What if I have any questions?

If you have any questions or concerns at any point during the study, please do not hesitate to contact the main researcher, Emma Clarkson, whose contact details are on the covering letter included in this pack. Alternatively, you may contact Dr Gill Harris, Consultant Paediatric Clinical Psychologist, or Dr Jeremy Kirk, Consultant Paediatric Endocrinologist, whose contact details may be found below.

Dr Gill Harris
Frankland Building, School of Psychology
University of Birmingham
Edgbaston
Birmingham

Dr Jeremy Kirk
Paediatric Endocrinology and Diabetes
Birmingham Children's Hospital
Steelhouse Lane
Birmingham

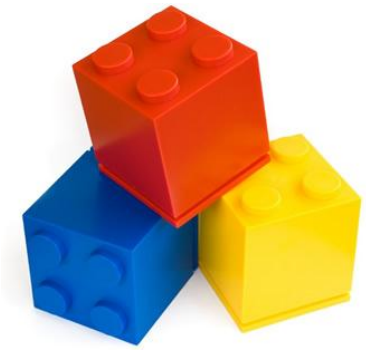
Investigating psychological and behavioural differences in children diagnosed with atypical adrenarche and central precocious puberty, compared to children of typical pubertal development.

Participant Information Sheet to be Read to the Participating Child (Typical Development)

Dear Parents/ Guardians,

Please read the information below to your child before agreeing to take part in the study. It is important that parents, guardians and children understand what is involved in the project, and are happy to participate. Thank you.

Not all children are exactly the same, and some children look older than other children in their class. Sometimes these children have to go to the doctor a lot. Sometimes they feel like they don't fit in with their friends, and their parents and doctors don't know how to help.



Our family can help these other children if we want to, by doing some easy tasks and answering some questions, a bit like at school. These tasks will have lots of questions, to see things like how many words you know, whether your friends are the same or different, and what sort of things you think about. These questions won't be too difficult and it might even be fun!

Someone would bring the tasks to our house, and we would spend some time doing them together. You would do some questions, and we (mum/dad etc.) would do some questions too. If we don't want to do the tasks, then we can just say, 'I want to stop'.

Our answers on the tasks will help to show why some children are different and how we can make them feel better. Would you like to help the children by doing the tasks?

If you would like to take part, this is what you can expect to happen.

Mum and Dad will fill in some questionnaires at home.

Then someone will come to our house. They will measure your height and weight and ask you to do some tasks whilst mum and dad answer some more questions.

On a different day we will see them again to do some more tasks. These will be things like seeing if you think you look the same as your friends, and whether you are a confident or a shy person. Mum and dad will finish off any more questions.

Taking part in the study won't change things for you, but will help other children in the future. Would you like to take part? Do you have any questions?

Interviewing Schedule

Project Title: Psychology and Behaviour in Children Diagnosed with Atypical Adrenarche and Central Precocious Puberty

If you have expressed an interest in taking part in the project you will have been sent a full information pack through the post. This will contain a full protocol for both parents and children, a summary of the ethical considerations being taken, consent forms and demographic questionnaires. There will also be a questionnaire to assess your child's pubertal status to confirm they are suitable for the study.

Fourteen days after we have sent the information pack out to you, you will receive a telephone call to ensure that you have received the pack and to answer any questions you may have. Once packs have been returned to the research team, you will receive another telephone call to arrange a meeting, either in the Children's Hospital or your home.

During the first meeting, your child will be weighed and measured in order to calculate their BMI. They will also complete the British Ability Scales to give scores of their intelligence.

During the first meeting, you will be asked to complete questionnaires on your child's eating behaviours. These are a modified version of the Dutch Eating Behaviour Questionnaire for Children (DEBQ-C: van Strien and Oosterveld, 2007; Clarkson and Harris, in prep) and the Child Eating Behaviour Questionnaire (CEBQ: Wardle, Guthrie, Sanderson and Rapoport, 2001).

During the second meeting, you child will complete a scale to assess their body image, and will also complete a questionnaire to measure the extent to which they are perceived older than their chronological age. Finally they will have an assessment of self-perception called the Harter's Pictorial Scale of Perceived Self Confidence and Social Acceptance for Young Children (PSPSCSA; Harter and Pike, 1984) or the Self Perception Profile for Children (SPPC; Harter, 1982), dependant on your child's age.

You will be asked to complete a parent version of the perceived age questionnaire, and a questionnaire looking at your child's behaviour called the Achenbach Child Behavior Checklist (CBCL: 1991). You will also be asked to complete questionnaires of recent family life events and levels of parental stress. These are called the Life Events Questionnaire (adapted from Norbeck, 1984), the Perceived Stress Scale (Cohen, Kamarck and Mermelstein, 1983) and the Impact of Event Scale (Horowitz, Wilner and Avarez, 1979).

Your child's school teacher will also be asked to complete a teacher's questionnaire to look at your child's behaviour at school.

On completion of the project, you will be sent a summary of the project findings through the post, along with a letter of thanks for your participation.

APPENDIX 2: PARTICIPANT INFORMATION PACK – CLINICAL GROUPS

UNIVERSITY OF BIRMINGHAM

Frankland Building: School of Psychology
University of Birmingham
Edgbaston
Birmingham
B15 2TT

Covering Letter

Dear Parents and Guardians,

My name is Emma Clarkson, and I am a researcher working at both the University of Birmingham and Birmingham Children's Hospital. I am currently carrying out a study funded by the Child Growth Foundation, and am writing to you as you have demonstrated an interest in taking part. The study is looking at the behaviour of children diagnosed with disorders of puberty, and would involve you and your child simply answering a few questionnaires, and your child taking part in a few assessments, including IQ and self-esteem. None of the assessments are invasive or distressing, and your child may even find them quite fun! The study would require approximately three hours of your time, can be spread over two sessions and would be arranged entirely at your convenience.

You would not directly benefit from the study; however your participation will have a huge impact on the families of children with puberty disorders, as there is currently very little information available. We aim to raise awareness of these disorders amongst healthcare professionals, so that families can access the correct treatment and support more quickly.

In this information pack you will find the following:

- A 'Participant Information Sheet' for parents. This contains the contact details of the research team, and general information about the project.
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- A 'Participant Information Sheet' for the child taking part.
- A prepaid envelope containing questionnaires, to be returned to the research team once they have been completed.

Please do not hesitate to get in contact if you have any questions.

Many thanks for your time,

Emma Clarkson
Doctoral Researcher

Investigating psychological and behavioural differences in children diagnosed with atypical adrenarche and central precocious puberty, compared to children of typical pubertal development.

Participant Information Sheet for Parents/ Guardians of Children Diagnosed with PP or AA

Background Information

Early puberty has become more common in recent years, yet there is very little information available to families with a child diagnosed with a puberty disorder. Children diagnosed with a puberty disorder may be likely to experience changes in their emotions and behaviour, as well as in their physical appearance, and research has suggested that children with a puberty disorder are more likely to show signs of low self-esteem and difficulties interacting with other children. Other research has suggested that children who develop early may have higher levels of stress in their family. Therefore the aim of this research is to study these problems in detail, in order to provide more detailed information for families, and evidence for healthcare professionals that children with these diagnoses should be offered psychological support or extra help in school. This research has been funded by the Child Growth Foundation, and has therefore been deemed useful and indeed necessary to improve the lives of children diagnosed with these disorders.

Procedure

The procedure for the study consists of several parts. These are listed below.

1. Questionnaires and consent forms have been sent to you. These questionnaires ask for demographic information, contact details, and basic information regarding your family. A form to determine your child's stage of pubertal development has also been included, to ensure that your child is eligible to participate. This stage will take approximately 30 minutes.

2. A meeting will be arranged, during which a measure of your child's IQ and BMI (body mass index) will be carried out. You will be asked to complete questionnaires on your child's eating behaviours. This meeting will take approximately two hours.

3. A second meeting will be arranged, during which your child's view of their body image will be measured, and an assessment of their self-perception and self-esteem will be carried out. You will also be asked to complete questionnaires on some of your child's behaviours, as well as questionnaires looking at family stress levels and events that have occurred recently in your family which may have affected this. This will take approximately two hours.

4. Questionnaires will be sent to your child's school teacher, to assess their behaviour at school. This will then be returned directly to the research team.

5. You will also be given the opportunity to take part in an additional visit where either parents or children can give more detailed information on what it is like to live with a diagnosis of atypical puberty in the family. This will be discussed with you at a later date.

Further Information and Ethical Declarations

What we ask of you

All meetings and questionnaires will be completed when it is most convenient, and will not require you to undertake any form of travel. We will call you to arrange the best time to visit you in your home or to coincide with an appointment at the Birmingham Children's Hospital. None of the assessments are at all intrusive, and will simply require yourself or your child to answer simple questions, or indicate one choice out of several options. We will take no more than four hours of your time overall. If your child shows any sign of becoming distressed or bored then the assessment will be stopped immediately.

What you can expect from us

We will maintain full confidentiality by taking several precautions. All paper and digital data gained through the study will remain secure, by being kept in locked filing cabinets or on password-protected USB drives. Any voice recordings taken on a dictaphone will be kept in the same manner. This data will remain at the University for a period of three years after the project has finished, after which all paper and digital forms of data will be destroyed. This will take place in accordance with the University of Birmingham's data protection and confidential waste policies. No data published in the final article will contain any identifiable information, and your details will be known only to the researchers. If any causes for concern arise regarding child protection, I am under duty to discuss this with my supervisor, and may have to report the concern further.

What if you change your mind?

If at any point during the course of the research yourself or your child express a wish to withdraw from the study, this wish will be respected with no reason required, up to the point of data analysis. If your child becomes frustrated or bored during any one of the assessments, the assessment can be postponed to another date if this would be more suitable.

How this will benefit you

Your family will not directly benefit from taking part in the project, however the study will be highly beneficial to children diagnosed with disorders of puberty in the future, and your participation will contribute massively to improving the information available to parents, teachers and healthcare professionals. This will raise awareness of the disorder and encourage provision of psychological and behavioural support for children and their families.

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Dr Gill Harris
Frankland Building, School of Psychology
University of Birmingham
Edgbaston
Birmingham
B15 2TT

Dr Jeremy Kirk
Paediatric Endocrinology and Diabetes
Birmingham Children's Hospital
Steelhouse Lane
Birmingham
B4 6NH

Looking at how children with puberty problems behave, compared to children with normal puberty.

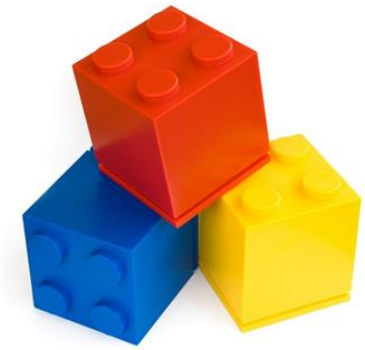
Participant Information Sheet to be Read to the Participating Child Diagnosed with PP or AA

Dear Parents/ Guardians,

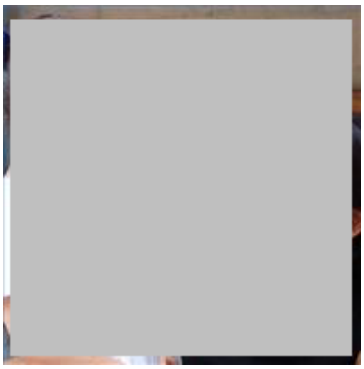
Please read the information below to your child before agreeing to take part in the study. It is important that parents, guardians and children understand what is involved in the project, and are happy to participate. Thank you.

Not all children are exactly the same, and some children look older than other children in their class. Sometimes these children have to go to the doctor or to the hospital, like you do.

Sometimes they feel unhappy and their parents and doctors don't know how to help them.



Our family can help if we want to, by doing some easy tasks and answering some questions, a bit like at school. These tasks will have different questions, to see things like how many words you know, whether you and your friends are the same or different, and what sort of things you think about yourself. These questions won't be too difficult and it might even be fun!

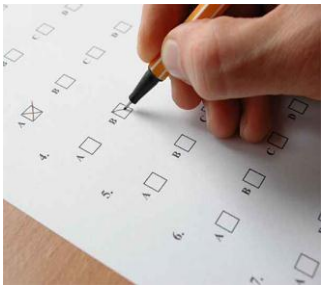


Someone would meet us at the hospital when we next go to see the doctor, or they might bring the tasks to our house, and we would spend some time doing them together. You would do some questions, and we (mum/dad etc.) would do some questions too. If we don't want to do the tasks, then we can just say, 'I want to stop'. We don't have to take part if we don't want to.

Our answers on the tasks will show why some children are different and how we can make them feel better. Would you like to help the children by doing the tasks?

If you would like to take part, this is what you can expect to happen.

Mum and Dad will fill in some questionnaires at home, or when we are next at the hospital.



Then someone will come to our house or meet us at the hospital. They will measure your height and weight and ask you to do some tasks whilst mum and dad answer some more questions.

On a different day we will see them again to do some more tasks. These will be things like seeing if you think you look the same as your friends, and whether you are a confident or a shy person. Mum and dad will finish off any more questions.

Taking part in the study won't change things for you, but will help other children in the future. Would you like to take part? Do you have any questions?

Interviewing Schedule

Project Title: Psychology and Behaviour in Children Diagnosed with Atypical Adrenarche and Central Precocious Puberty

You will have been sent a full information pack through the post. This will contain a full protocol for parents and children, a summary of the ethical considerations being taken, consent forms and demographic questionnaires. There will also be a questionnaire to assess your child's pubertal status to confirm they are suitable for the study.

Once the documents in the information pack have been returned to the research team, you will receive telephone call to answer any questions you may have, and to arrange a meeting, either in the Birmingham Children's Hospital or your home.

During the session, your child will be weighed and measured in order to calculate their BMI. They will also complete the British Ability Scales to see what their strengths and weaknesses are at school. Your child will also complete a scale to assess their body image, and a questionnaire to measure the extent to which they are perceived older than their chronological age. Finally they will have an assessment of self-perception called the Harter's Pictorial Scale of Perceived Self Confidence and Social Acceptance for Young Children (PSPSCSA; Harter and Pike, 1984) or the Self Perception Profile for Children (SPPC; Harter, 1982), dependant on your child's age.

During the session, you will be asked to complete a few questionnaires. These are a modified version of the Dutch Eating Behaviour Questionnaire for Children (DEBQ-C: van Strien and Oosterveld, 2007; Clarkson and Harris, in prep) and the Child Eating Behaviour Questionnaire (CEBQ: Wardle, Guthrie, Sanderson and Rapoport, 2001), a parent version of the perceived age questionnaire, and a questionnaire looking at your child's behaviour called the Achenbach Child Behavior Checklist (CBCL: 1991). You will also be asked to complete questionnaires of recent family life events and levels of parental stress. These are called the Life Events Questionnaire (adapted from Norbeck, 1984), the Perceived Stress Scale (Cohen, Kamarck and Mermelstein, 1983) and the Impact of Event Scale

Your child's school teacher will also be asked to complete a teacher's questionnaire to look at your child's behaviour at school.

Once the main project has been completed, your child will be asked if they would like to take part in an additional qualitative data analysis. This will involve an additional visit from the experimenter during which conversations will take place regarding your child and their friends, their disorder, their appearance, their family and their worries. These conversations will be facilitated with props and games.

Once the main project has been completed, you will be asked if you would like to take part in an additional qualitative data analysis. This will involve an additional visit from a Masters student who will facilitate discussions surrounding your views of your child and how the disorder has affected them, how the disorder has affected you as parents, whether your relationship with your child has changed, and whether other people's perceptions of your child has changed.

On completion of the project, you will be sent a summary of the project findings through the post, along with a letter of thanks for your participation.

APPENDIX 3: DEMOGRAPHIC QUESTIONNAIRE PACK

Demographic Questionnaire

Please give the details of the child who is participating in the research project.

Name	
Date of Birth	
Gender	
Ethnicity	
Does your child have any medical problems, excluding signs of early puberty, which have required multiple hospital or GP appointments? If so please give brief details.	
Is your child taking any medication, or have they been on any form of medication for a considerable amount of time, excluding medication prescribed for signs of early puberty? If so please give details.	
Please give the address of your child's school and the name of your child's teacher, as we will approach them to also complete a questionnaire.	
Please give details of any other agencies or services that have had dealings with your child eg. Social Services, CAMHS (Child and Adolescent Mental Health Services), Birmingham Children's Hospital	

Please give details of parents/ guardians living in the same household as the child participating in the research project.

	First Parent/ Guardian	Second Parent/ Guardian (if applicable)
Name		
Date of Birth		
Gender		
Ethnicity		
Relationship to Child eg. Biological Mother		
If the child is not a biological relative, please indicate how long the child has been in your care.		
Highest Level of Education eg. O Levels		
Occupation		
Approximate Annual Household Income		

Please give details of any other children or young adults living in the same household as the child participating in the research project. Eg. Siblings, half-siblings

Name		
Date of Birth		
Gender		
Ethnicity		
Relationship to Child eg. Biological Sister		

Name		
Date of Birth		
Gender		
Ethnicity		
Relationship to Child eg. Biological Sister		

Name		
Date of Birth		
Gender		
Ethnicity		
Relationship to Child eg. Biological Sister		

Thank you for completing this questionnaire.

Contact Details

Please complete the forms below with details of the best way in which to contact you.

	First Parent/ Guardian	Second Parent/ Guardian (if applicable)
Name		
Home Telephone Number		
Mobile Telephone Number		
Work Telephone Number		
Email Address		
Postal Address		
Home Address (if different to postal address)		
Preferred method of contact		
Preferred time of contact		

Parental Consent

Please read the information sheet and ensure your child is happy to participate before signing the consent form.

Project Title: Investigating the Psychology and Behaviour of Children Diagnosed with Atypical Adrenarche and Central Precocious Puberty.		
Child's Name		
Parent/ Guardian's Name		
I confirm that I have read and understood the project information provided. I have had the opportunity to consider the information, ask questions and have had adequate answers to my questions.	Yes	No
I give consent for the above named child to participate in the proposed research study.	Yes	No
I fully understand that I, or my child, can choose to withdraw from the study at any time, and that participation is entirely voluntary.	Yes	No
I have been given full contact details of the chief investigator, and know that any concerns I have at any time during the study will be immediately addressed.	Yes	No
I consent for the acquired data to be used in a publishable article, and am aware that no identifying information will be published alongside this data or elsewhere in the article.	Yes	No
I understand that any reasons for concern of child safety will be reported, according to child protection guidelines.	Yes	No
Signed	Date	

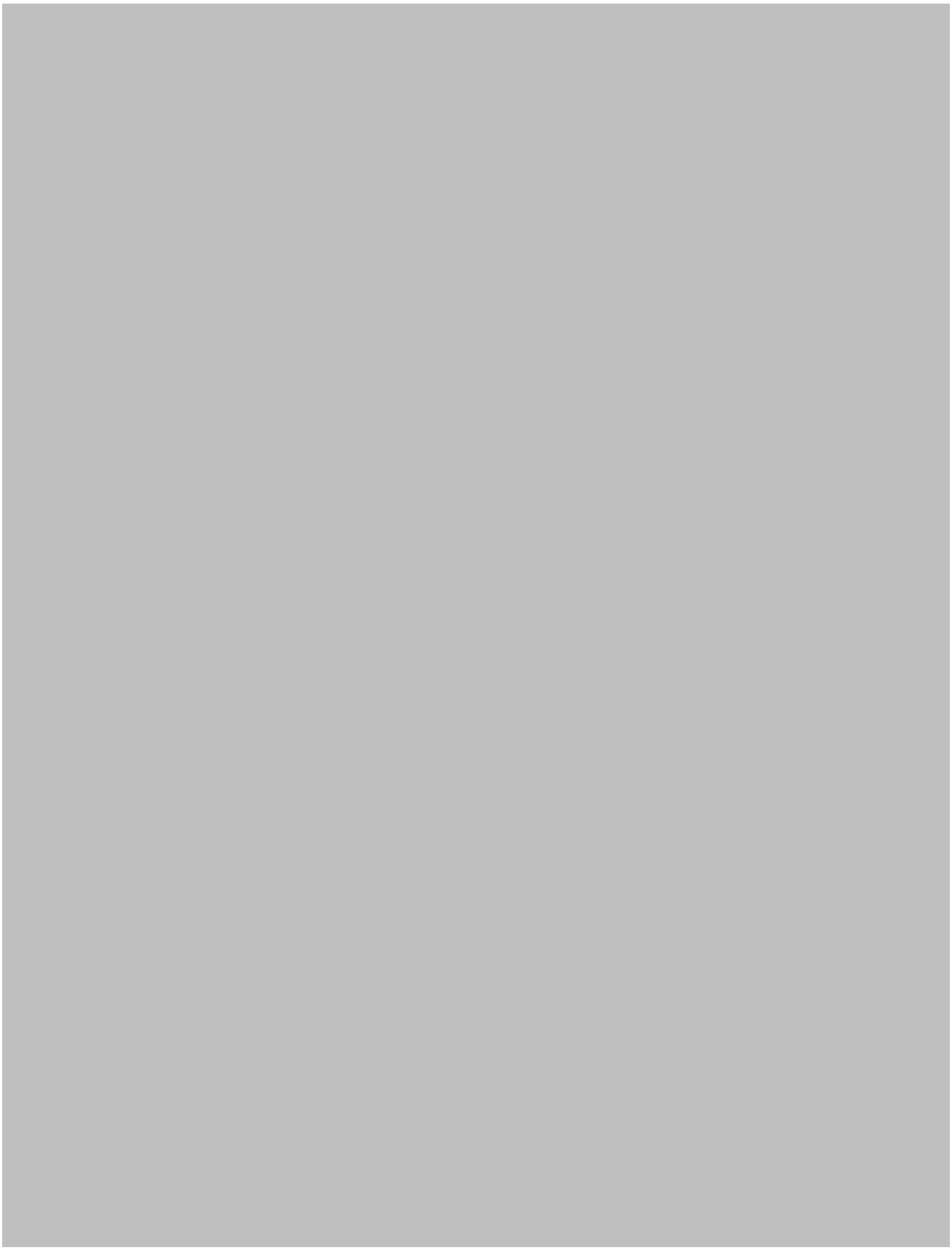
Thank you for agreeing to participate in this study. Please place the completed documents into the stamped addressed envelope provided and send it back to the research team.

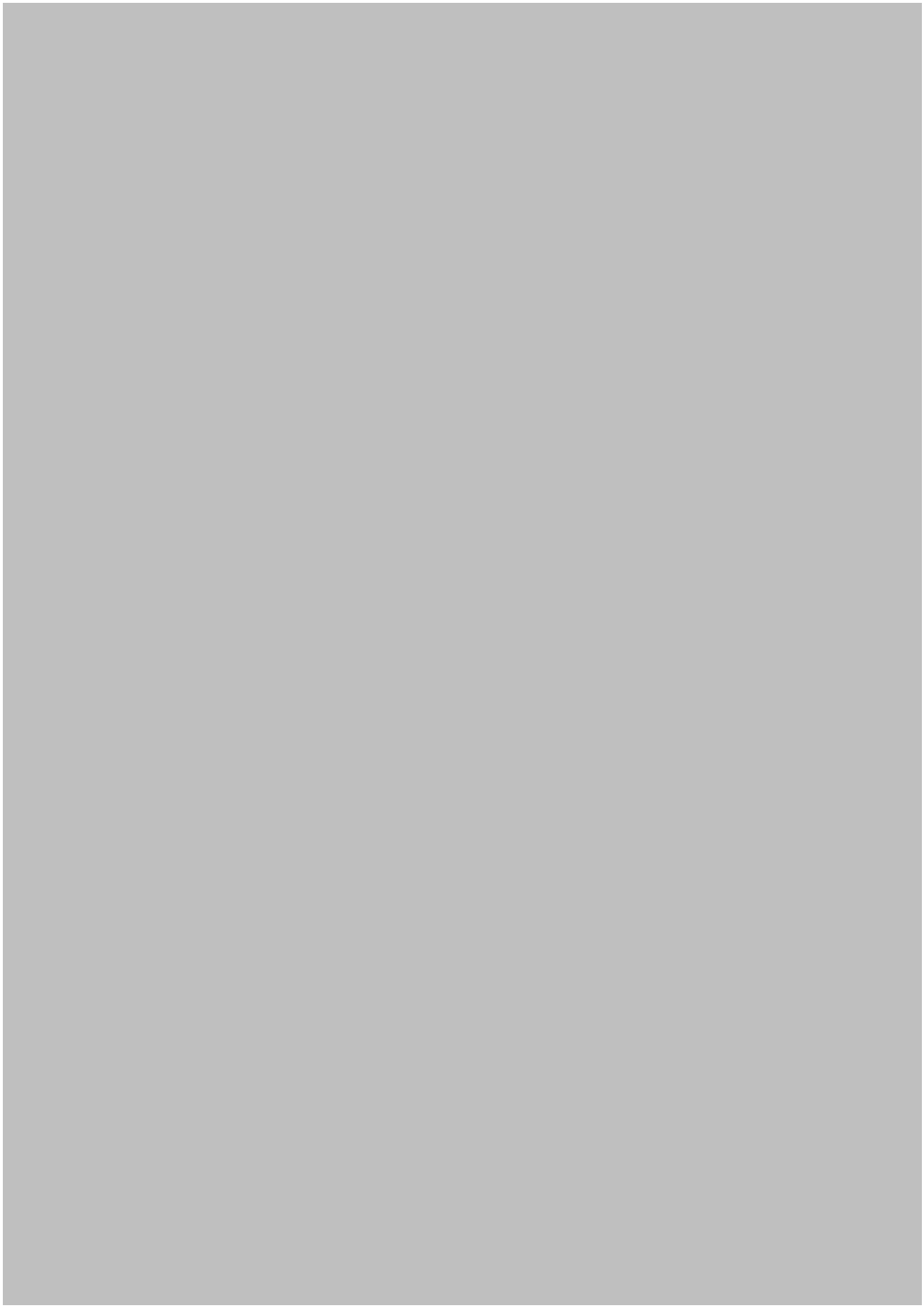
Participating Child's Assent

Please read the child's information sheet to your son or daughter before completing this form with them.

Project Title: Investigating the Psychology and Behaviour of Children Diagnosed with Atypical Adrenarche and Central Precocious Puberty.		
Child's Name		
Parent/ Guardian's Name		
Has somebody explained the project to you?	Yes	No
Do you understand what the project is about?	Yes	No
Have you asked all the questions that you wanted to ask?	Yes	No
Did you understand the answers to your questions?	Yes	No
Do you understand that it is ok to stop taking part at any time?	Yes	No
Do you want to take part?	Yes	No
If you want to take part, please write your name or draw an 'X' below		
Signed	Date	
Parent/ Guardian's Signature	Date	

APPENDIX 4: CONSULTANT LETTER OF INVITATION





APPENDIX 6: DEBQ-C PILOT STUDY

For the purpose of the current thesis it was necessary to assess the validity of the parent-completion version of the DEBQ-C for use with younger children aged between 4 and 10 years from the UK. It was hypothesised that data would fall into the same three factors proposed in the original DEBQ-C; emotional, external and restrictive eating behaviours.

Sample and Recruitment. Those participating in the study were the parents of 140 children between the ages of 4 and 10 years (mean age = 85.46 months; SD = 24.22 months). Sixty-five of the children were male and 75 female. Groups were well-matched for age, height, weight and BMI. Participants were approached through local mainstream primary schools in average socioeconomic areas, which served families from a variety of cultural and ethnic backgrounds. All participants had a good understanding of English. A participant information sheet, consent forms and a copy of the original questionnaire were sent home with the children to be returned to the school and collected by the research team, or parents were approached in the playground whilst waiting to collect their children after school. Responses were collected and entered into IBM SPSS (Version 20).

Measure. The original DEBQ-C was modified from a 20 item self-report questionnaire to a parent-report format. This was done by taking the existing statements and rewording them accordingly, for example, ‘If you feel lonely do you get a desire for food?’ was replaced with, ‘If your child is lonely do they seek or ask for food?’ The content and order of the statements were not altered from the original DEBQ-C. Participants were given the same response options as in the original questionnaire; ‘No’, ‘Sometimes’ and ‘Yes’.

Analysis. Reliability analyses produced an initial total scale Cronbach's value of .82, and values of .75, .77 and .46 for the external, emotional and restrictive subscales respectively. Closer inspection of the items in the restrictive subscale suggested that these items would be better classified into two groups; restrictive eating behaviour items and awareness of health items. The items related to awareness of health were removed, as well as the weaker items in the external and emotional subscales. Reliability analyses of the remaining items resulted in a total scale reliability value of .83, and values of .76, .79 and .84 for the external, emotional and restrictive subscales respectively.

Principal Components Analysis using Varimax rotation with factor extraction fixed at three, demonstrated that the items loaded sufficiently on to the original subscales of external eating, emotional eating and restrictive eating. Items loadings ranged from .86 to .48, and the model accounted for 58% of the variance. Kesier-Meyer-Olkin measure of sampling adequacy was .79. The item factor loadings are given in Table A1.

Table A1: *Item loadings for the modified DEBQ-C*

Item	Emotional	External	Restrictive
When your child is worried do they seek or ask for food?	.832		
Does your child ever seek or ask for food if things have gone wrong?	.791		
If your child is upset do they seek or ask for food?	.657		
If your child is nervous or afraid do they seek or ask for food?	.576		
If your child is sorry do they seek or ask for food?	.511		
If your child sees someone else preparing food, do they show a desire to eat?		.818	
Does your child show a desire to eat if they observe others eating?		.733	
Does your child find it difficult to stay away from tasty food?		.680	
Does your child feel like eating whenever they can see or smell food?		.601	
Does your child ever ask to stop in fast food shops as you walk past?		.541	
If your child is lonely do they seek or ask for food?	.482	.539	
Does your child avoid eating between meals to change their weight or appearance?			.864
Does your child ever intentionally eat less to change their weight or their appearance?			.859
Does your child ever avoid eating after their evening meal to change their weight or appearance?			.841

Discussion. The aim of this pilot study was to determine whether the DEBQ-C was suitable in a parent-completion format for identifying disordered eating behaviour in young children aged 4-10 years. The findings suggest that the modified DEBQ-C is just as suitable for assessing external and emotional eating behaviours as the original measure, but items on the restrictive eating behaviour scale fell into two subscales of altered eating behaviour relating to health awareness, and restrictive eating to alter appearance. The items relating to disordered eating to manipulate appearance were retained in this current project, as this most closely reflected the subscale proposed by the authors in the original measure. The final measure consisted of fourteen items; five emotional subscale items, six external subscale items, and three restrictive subscale items (Appendix 7). The scale was shown to have high internal reliability and sufficient factor loadings.

There are several limitations to this pilot study. The first is that data on ethnicity was not collected, but as the aim of the study was to identify whether the original factorial structure was suitable for a different cultural groups, it was considered unnecessary. A further limitation may have been social desirability bias, in that parents may not have wanted their child to score highly on certain subscales, particularly for restrictive eating. Some parents also noted on their completed questionnaires that they thought some of the restrictive items were not applicable, although in a non-clinical sample this may well have been true. Another concern is that parents may not be aware of certain eating behaviours in their children, and their answers may therefore have not been representative of their child's true eating behaviour. However, this is unlikely in such young children, whose parents provide them with most of their meals. In summary, the modified version of the DEBQ-C was considered suitable for use in the main thesis.

APPENDIX 6: ORIGINAL DEBQ-C – PILOT QUESTIONNAIRE

Please complete the following information as accurately as you can.

Child's Current Weight	
Child's Current Height	
BMI	
Has your child's weight been constant over the past six months?	Yes
	No, gain of (please give weight)
	No, loss of (please give weight)
Has your child ever had an episode of eating an amount of food that others would consider unusual?	No
	Yes, unusually large
	Yes, unusually small

Please read the following questions and indicate the answer which you feel best reflects the behaviour of your child.

Does your child feel like eating whenever they can see or smell food?	**No**	**Sometimes**	**Yes**
If your child is upset do they seek or ask for food?	**No**	**Sometimes**	**Yes**
If your child is lonely do they seek or ask for food?	**No**	**Sometimes**	**Yes**
Is your child very aware of what they eat?	**No**	**Sometimes**	**Yes**
Does your child ever ask to stop in sweet shops as they walk past?	**No**	**Sometimes**	**Yes**
Does your child intentionally try to eat healthy foods?	**No**	**Sometimes**	**Yes**
Does your child show a desire to eat if they observe others eating?	**No**	**Sometimes**	**Yes**

If your child has eaten a great deal one day, will they eat less than usual the following day?	**No**	**Sometimes**	**Yes**
When your child is worried do they seek or ask for food?	**No**	**Sometimes**	**Yes**
Does your child find it difficult to stay away from tasty food?	**No**	**Sometimes**	**Yes**
Does your child ever intentionally eat less to change their weight or their appearance?	**No**	**Sometimes**	**Yes**
Does your child ever seek or ask for food if things have gone wrong?	**No**	**Sometimes**	**Yes**
Does your child ever ask to stop in fast food shops as you walk past?	**No**	**Sometimes**	**Yes**
Does your child avoid eating between meals to change their weight or appearance?	**No**	**Sometimes**	**Yes**
If your child is fidgety or restless do they seek or ask for food?	**No**	**Sometimes**	**Yes**
Does your child ever avoid eating after their evening meal to change their weight or appearance?	**No**	**Sometimes**	**Yes**
If your child is nervous or afraid do they seek or ask for food?	**No**	**Sometimes**	**Yes**
Does your child ever show an interest in what foods are fattening or unhealthy?	**No**	**Sometimes**	**Yes**
If your child is sorry do they seek or ask for food?	**No**	**Sometimes**	**Yes**
If your child sees someone else preparing food, do they show a desire to eat?	**No**	**Sometimes**	**Yes**

You have now completed the questionnaire. If you have any questions please do not hesitate to ask, using the contact information on the information letter. Thank you for taking the time to complete this questionnaire.

APPENDIX 7: MODIFIED DEBQ-C

Please read the following questions and tick the answer which you feel best reflects the behaviour of your child.

Has your child's weight been constant over the past six months?	**Yes**	**No, gain of . . . . . kg**	**No, loss of . . . . . .kg**
Has your child ever had an episode of eating an amount of food that others would consider unusual?	**No**	**Yes, unusually large**	**Yes, unusually small**

Does your child feel like eating whenever they can see or smell food?	**No**	**Sometimes**	**Yes**
If your child is upset do they seek or ask for food?	**No**	**Sometimes**	**Yes**
If your child is lonely do they seek or ask for food?	**No**	**Sometimes**	**Yes**
Does your child ever ask to stop in sweet shops as they walk past?	**No**	**Sometimes**	**Yes**
Does your child show a desire to eat if they observe others eating?	**No**	**Sometimes**	**Yes**
When your child is worried do they seek or ask for food?	**No**	**Sometimes**	**Yes**
Does your child find it difficult to stay away from tasty food?	**No**	**Sometimes**	**Yes**
Does your child ever intentionally eat less to change their weight or their appearance?	**No**	**Sometimes**	**Yes**
Does your child ever seek or ask for food if things have gone wrong?	**No**	**Sometimes**	**Yes**
Does your child ever ask to stop in fast food shops as you walk past?	**No**	**Sometimes**	**Yes**
Does your child avoid eating between meals to change their weight or appearance?	**No**	**Sometimes**	**Yes**
Does your child ever avoid eating after their evening meal to change their weight or appearance?	**No**	**Sometimes**	**Yes**
If your child is nervous or afraid do they seek or ask for food?	**No**	**Sometimes**	**Yes**
If your child is sorry do they seek or ask for food?	**No**	**Sometimes**	**Yes**
If your child sees someone else preparing food, do they show a desire to eat?	**No**	**Sometimes**	**Yes**

APPENDIX 8: CEBQ

Please read the following statements and tick the boxes with the response most relevant to your child's eating behaviours.

1. My child loves food	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**
2. My child eats more when worried	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**
3. My child has a big appetite	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**
4. My child finishes their meal quickly	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**
5. My child is interested in food	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**
6. My child is always asking for a drink	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**
7. My child refuses new foods at first	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**
8. My child eats slowly	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**
9. My child eats less when angry	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**
10. My child enjoys tasting new foods	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**
11. My child eats less when they are tired	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**
12. My child is always asking for food	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**
13. My child eats more when annoyed	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**
14. If allowed to, my child would eat too much	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**
15. My child eats more when anxious	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**
16. My child enjoys a wide variety of foods	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**

17. My child leaves food on their plate at the end of a meal	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**
18. My child takes more than 30 minutes to finish a meal	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**
19. Given the choice, my child would eat most of the time	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**
20. My child looks forward to mealtimes	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**
21. My child gets full before their meal is finished	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**
22. My child enjoys eating	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**
23. My child eats more when they are happy	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**
24. My child is difficult to please with meals	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**
25. My child eats less when upset	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**
26. My child gets full up easily	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**
27. My child eats more when they have nothing else to do	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**
28. Even if my child is full up, they find room to eat their favourite food	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**
29. If given the chance, my child would drink continuously throughout the day	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**
30. My child cannot eat a meal if they have had a snack just before	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**
31. If given the chance, my child would always be having a drink	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**
32. My child is interested in tasting food they haven't tasted before	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**
33. My child decides they don't like food before even tasting it	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**
34. If given the chance, my child would always have food in their mouth	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**

35. My child eats more and more slowly during the course of a meal	**Never**	**Rarely**	**Sometimes**	**Often**	**Always**

APPENDIX 9: BODY IMAGE SCALES PILOT STUDY

Pictorial body image scales were designed for use in the current project. A scale of five figures was deemed suitable due to the age range of the children, as too many figures would have complicated the task. Furthermore, Gardner and Brown describe the findings of two studies employing 8-10 figure scales, in which 85% to 90% of all participant responses were of the same three or four items (Brodie, Bagley & Slade, 1994; Gardner et al., 1999), suggesting that a greater range of figures does not necessarily improve the validity of the scale. Figures were presented in ascending order from left to right, despite there being a reported bias when typically preferred images items are presented to the left-hand side of the scale (Nicholls et al., 2006). This was to ensure younger participants could identify the subtle differences between the figures, which were made clearer by presenting them in order. Figures were presented as black silhouettes to prevent participants being influenced by other features such as hair or clothing, and to ensure the scale could be generalised across ethnic groups.

The aim of the pilot study was to testa newly constructed set of pictorial body image scales for use in the current thesis, to assess participants' self-perception and satisfaction with their body image.

Sample and Recruitment. 300 children took part in the study, 156 males and 144 females (See Table A2; mean age 7.26 years; SD 1.75). Children were aged between 4-10 years and recruited from local primary schools using an opt-out recruitment method. Children were excluded if the opt-out form was returned by their parents, or their teacher considered them to be unable to complete the task due to language barriers or difficulties in understanding. The scales were administered at school. Children were asked if they

would be happy to answer a few short questions on body shapes, and informed that they did not have to take part if they did not wish to.

Measure and Procedure. The first scale was similar to the body image scales previously described, with figures ranging from a low body weight to a high body weight. The second scale depicted figures ranging from a child-like body shape to an adult-like body shape. Male and female versions of each scale were constructed. Figures were manipulated to all be of the same height, with a straight line positioned above and below the figures to make this easy to identify. The figures' age and ethnicity was indeterminate. Children were asked to choose which of the figures they currently perceived themselves to be most similar to, which figure most resembled their friends, and which figure they preferred. Figures were assigned a number from one to five for recording and analysis. The difference between the chosen figures indicated the level of dissatisfaction with the participant's bodily perceptions.

Children were shown the first scale appropriate for their gender and asked to look carefully at the figures. It was explained that all the figures were the same height but different body shapes, and children were asked if they could see that this was true. Children were then asked three questions; which figure looked the most like them, which figure looked the most like their closest friend, and which figure they would like to look like the most. If children chose more than one figure for a single question, a mean was calculated from the numbers assigned to the chosen figures. Children then moved on to the second scale, and the process was repeated.

Table A2: *Participant Demographics for the Pictorial Body Image Scales Pilot Study*

Year Group	Gender			
	Male		Female	
	n	Mean age in months (SD)	n	Mean age in months (SD)
Reception	24	57.50 (4.98)	27	57.33 (5.038)
1	25	67.68 (5.88)	23	69.39 (5.06)
2	25	81.12 (5.23)	24	81.50 (4.98)
3	24	93.50 (4.98)	27	93.78 (4.75)
4	26	103.38 (5.95)	28	106.29 (4.28)
5	32	116.63 (5.48)	15	117.60 (4.97)

Analysis. Means and standard deviations were calculated for each figure rating; self, friends' and preferred. These were calculated for each age group. Descriptive statistics are given in Tables A3 and A4.

Table A3: *Means and standard deviations for each age group's figure ratings on the physical development scale*

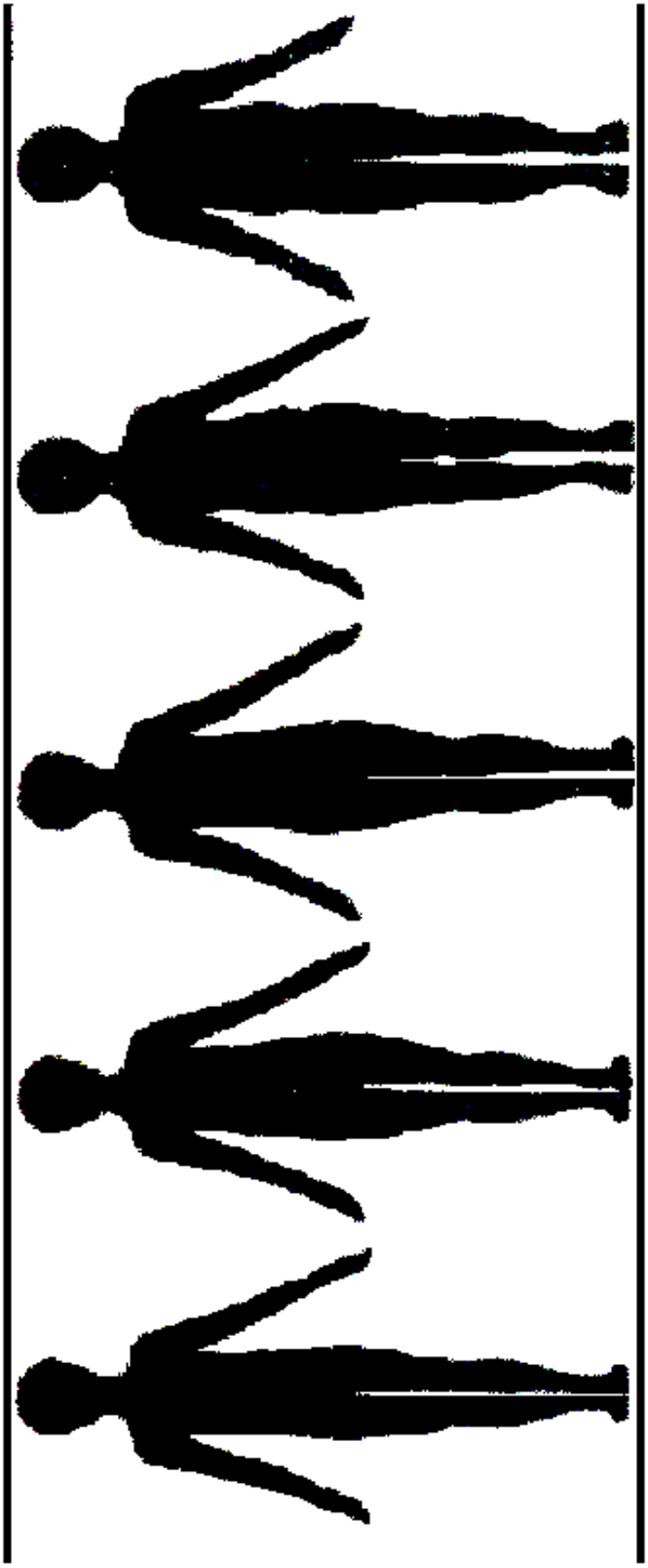
Year Group	Gender					
	Male			Female		
	Self	Friends	Preferred	Self	Friends	Preferred
Reception	3.25 (1.48)	3.15 (1.61)	3.08 (1.67)	2.96 (1.48)	3.87 (1.17)	2.78 (1.31)
1	3.04 (1.57)	2.80 (1.31)	3.52 (1.36)	2.39 (1.37)	3.27 (1.28)	3.26 (1.71)
2	3.04 (1.54)	2.69 (1.31)	3.08 (1.61)	3.25 (1.22)	3.21 (1.32)	2.92 (1.44)
3	2.67 (1.37)	2.72 (1.15)	2.92 (1.56)	2.54 (1.23)	3.02 (1.27)	3.41 (1.72)
4	2.92 (1.26)	2.96 (1.12)	3.12 (1.37)	2.89 (.99)	2.91 (1.00)	2.71 (1.41)
5	2.39 (1.14)	2.41 (.91)	2.42 (1.16)	2.73 (1.16)	2.50 (1.48)	2.80 (1.42)

Table A4: *Means and standard deviations for each age group's figure ratings on the weight scale*

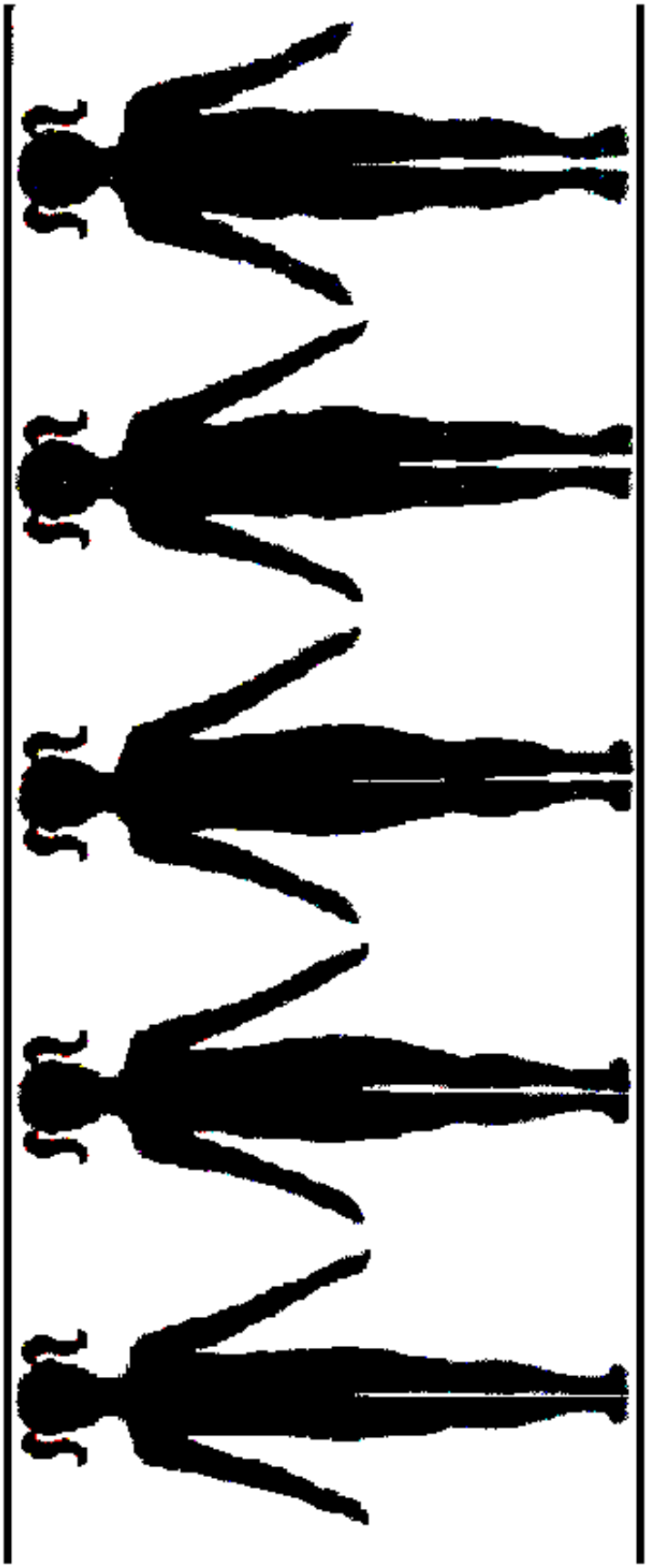
Year Group	Gender					
	Male			Female		
	Self	Friends	Preferred	Self	Friends	Preferred
Reception	2.92 (1.64)	2.54 (1.41)	2.69 (1.28)	3.04 (1.60)	3.52 (1.14)	2.37 (1.33)
1	3.58 (1.61)	3.29 (1.07)	2.24 (1.71)	3.00 (1.51)	3.62 (1.33)	2.22 (1.57)
2	2.88 (1.45)	2.92 (1.29)	2.88 (1.45)	2.50 (1.18)	3.71 (1.26)	2.46 (1.50)
3	3.02 (1.15)	3.20 (1.17)	2.83 (1.27)	2.91 (1.23)	2.98 (1.18)	1.85 (1.03)
4	3.19 (1.27)	2.90 (1.22)	2.54 (1.17)	2.89 (1.17)	2.59 (.97)	2.11 (.99)
5	2.86 (1.18)	2.50 (.87)	2.41 (1.01)	2.93 (1.16)	1.97 (.77)	2.00 (.93)

Discussion. The aim of the project was to assess whether young children could use the pictorial body image scales to make comparisons about their perceived body image with that of others', and to identify whether children could form a preference between different body shapes. It was observed that children were able to use the scales easily, and had clear opinions on different body shapes. This implies that the pictorial body image scales are useful in exploring children's perceptions of body image with regards to both weight, and the age that somebody appears to be. When looking at the descriptive statistics, all ratings were fairly centrally distributed and none of the standard deviations were particularly high, suggesting that children across age groups performed fairly consistently across tasks. It was noted, however, that the standard deviations tended to be higher in the younger groups, which could be interpreted as the younger children being less accurate in their judgements, or older children displaying social desirability bias and choosing figures towards the centre of the scale as a result. Future research using this scale should therefore control for any effects of age during analysis.

There were several limitations to the study, the first being that no reliability statistics could be calculated, due to data being collected at a single time-point for each participant, and no physical measures conducted alongside the pictorial scales with which to compare them. However, the main aim of the project was to assess the ease with which children completed the tasks, and to determine whether the wording of the task and the scale images were suitable. Children had no difficulties completing the task and were able to perceive the differences between the figures without complaint. The data would support the use of pictorial image scales in studying children's perceptions of body image, when looking at weight as well as other differences in bodily composition.



Male Weight



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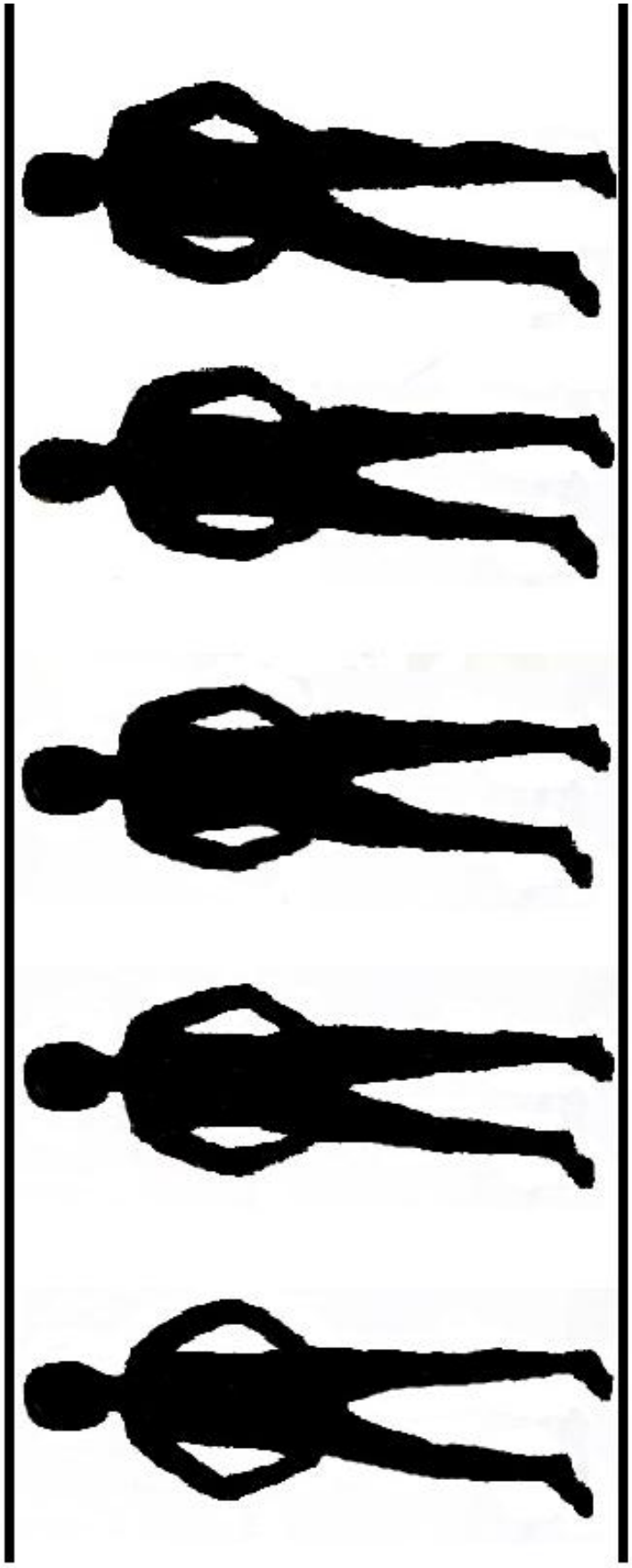
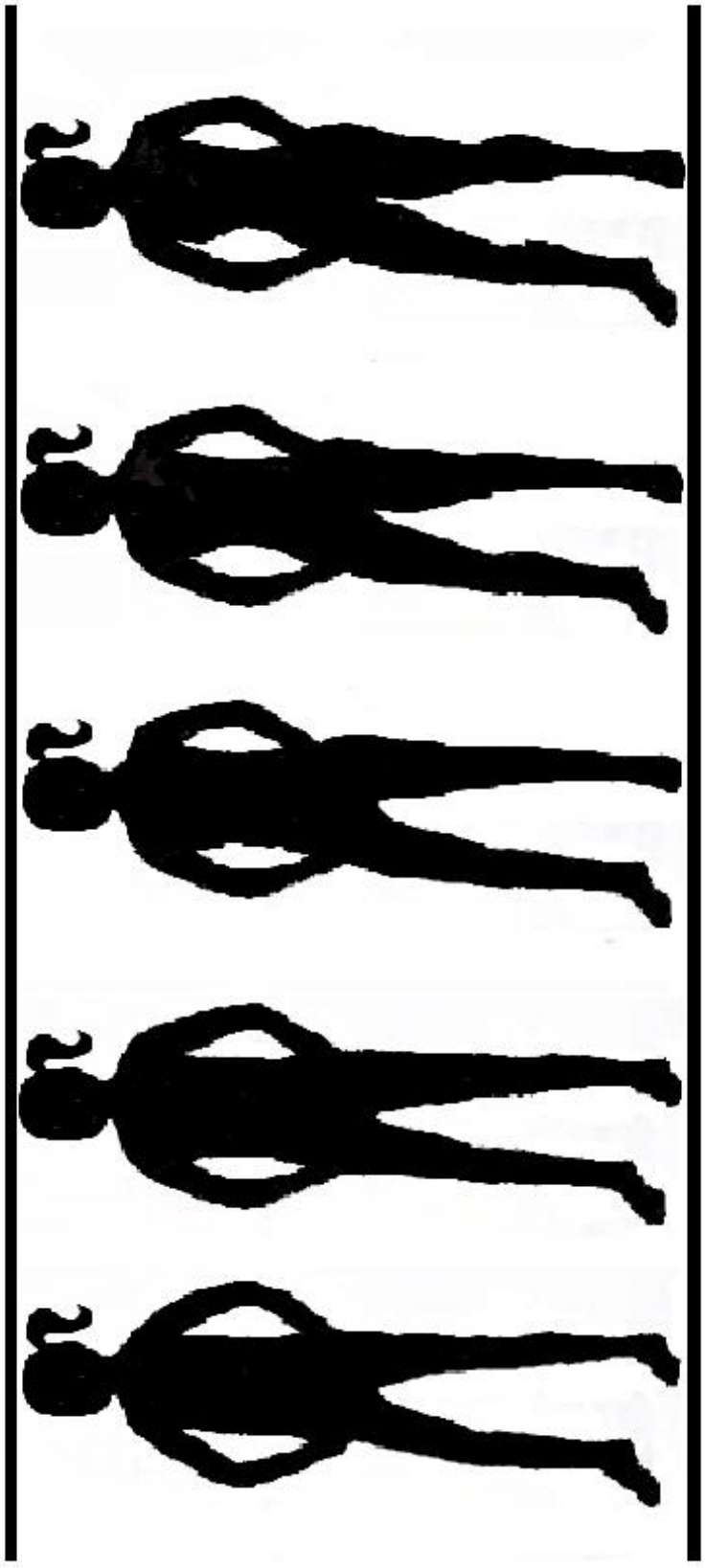
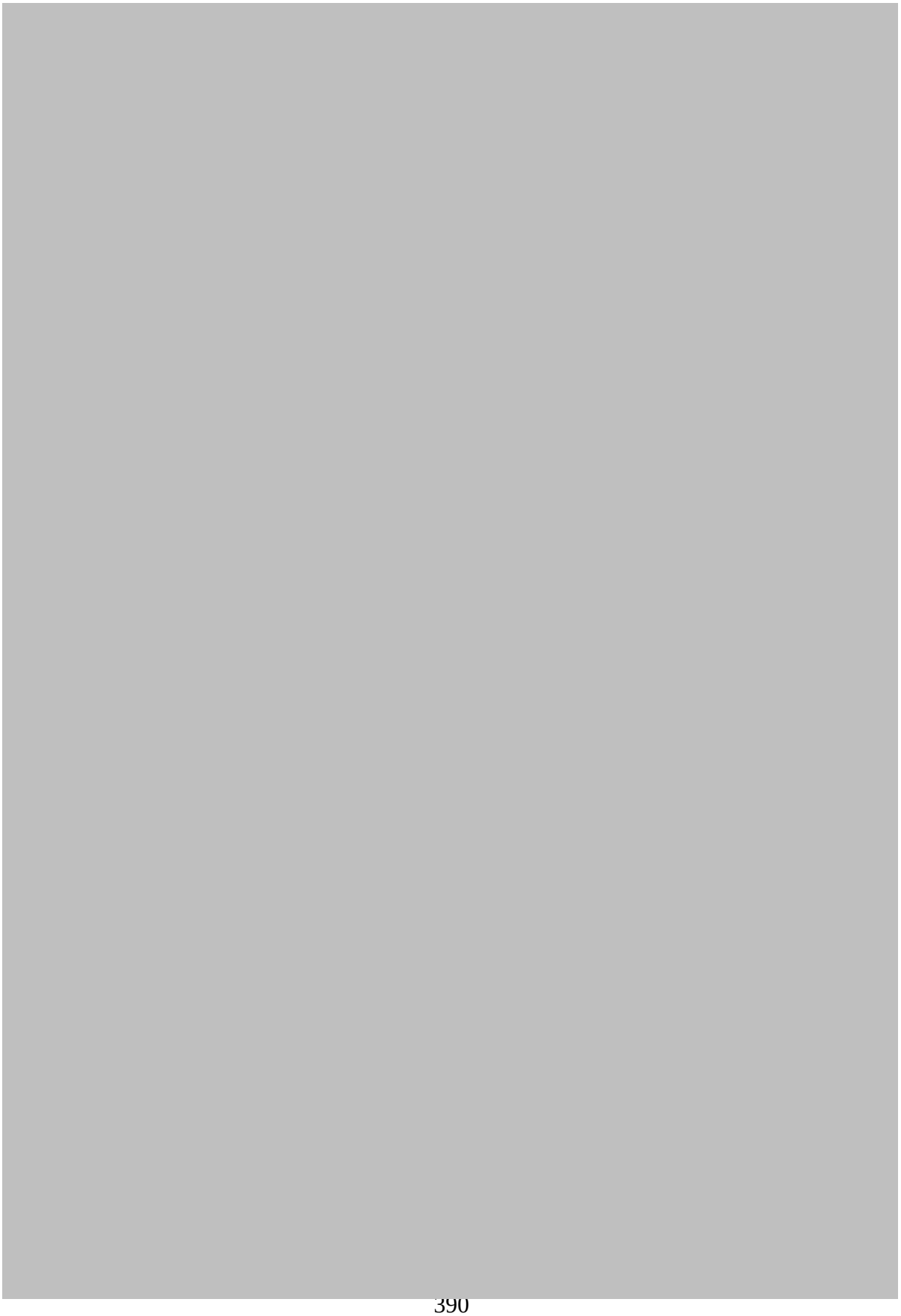


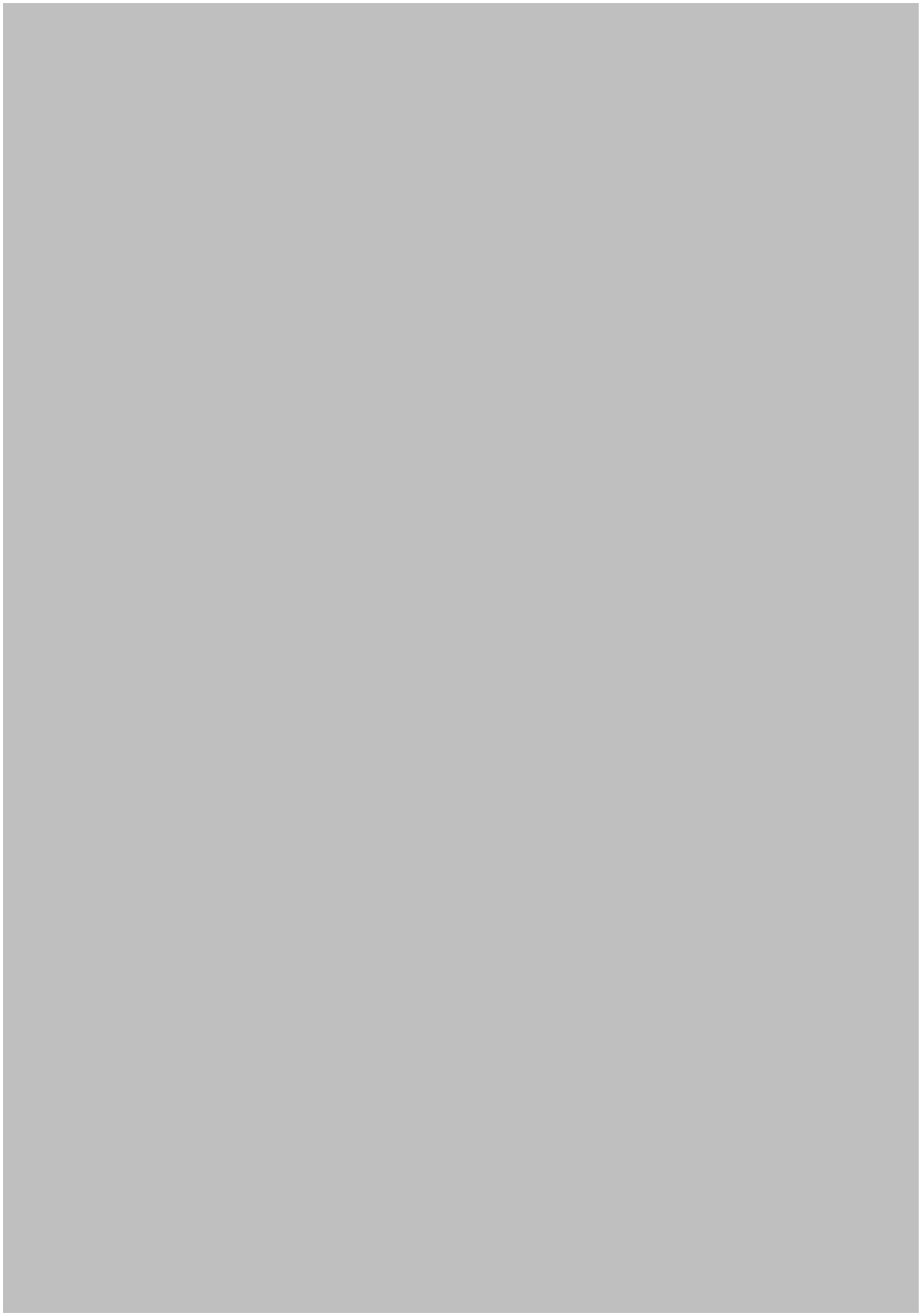
Male Development



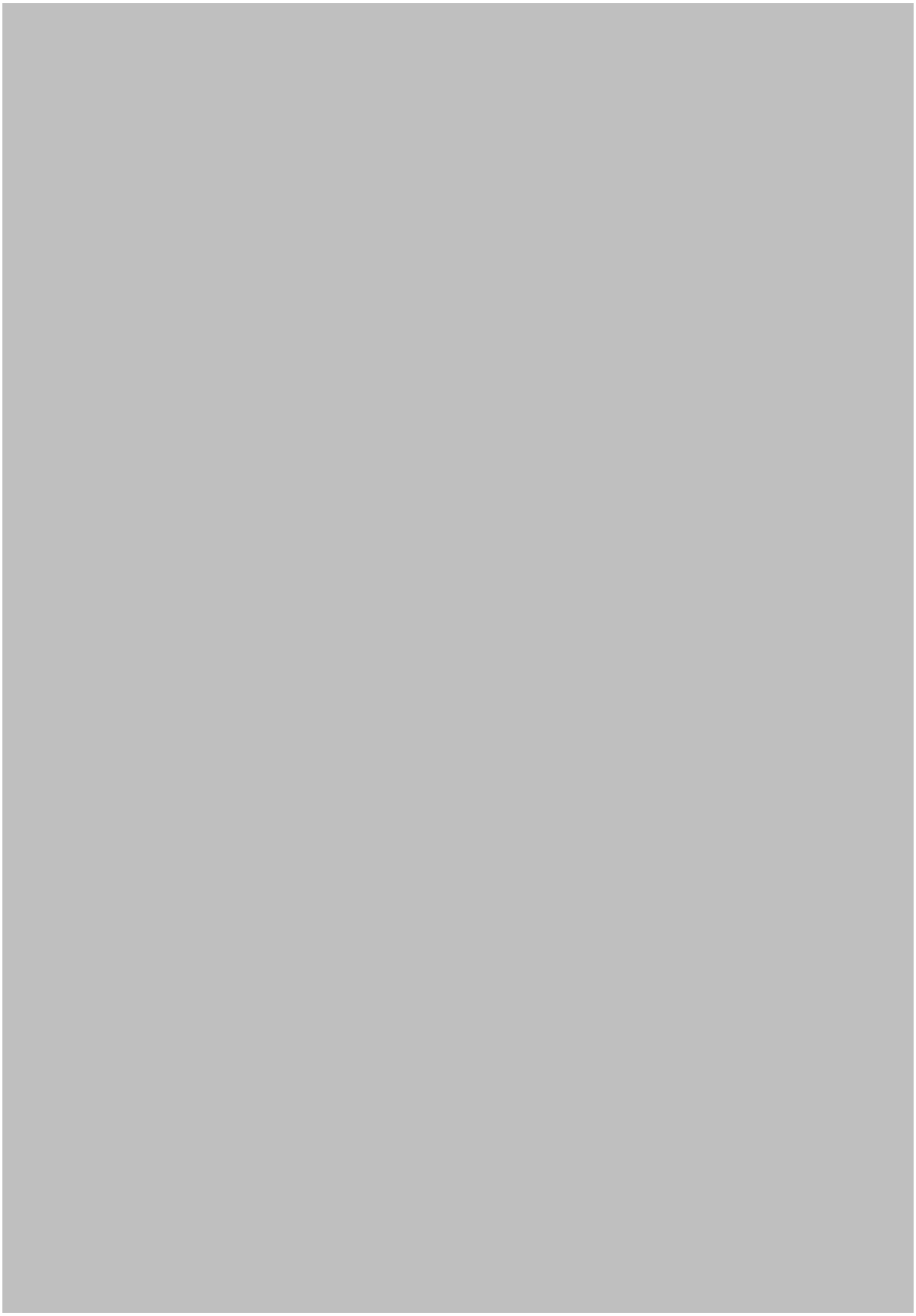
Female Development

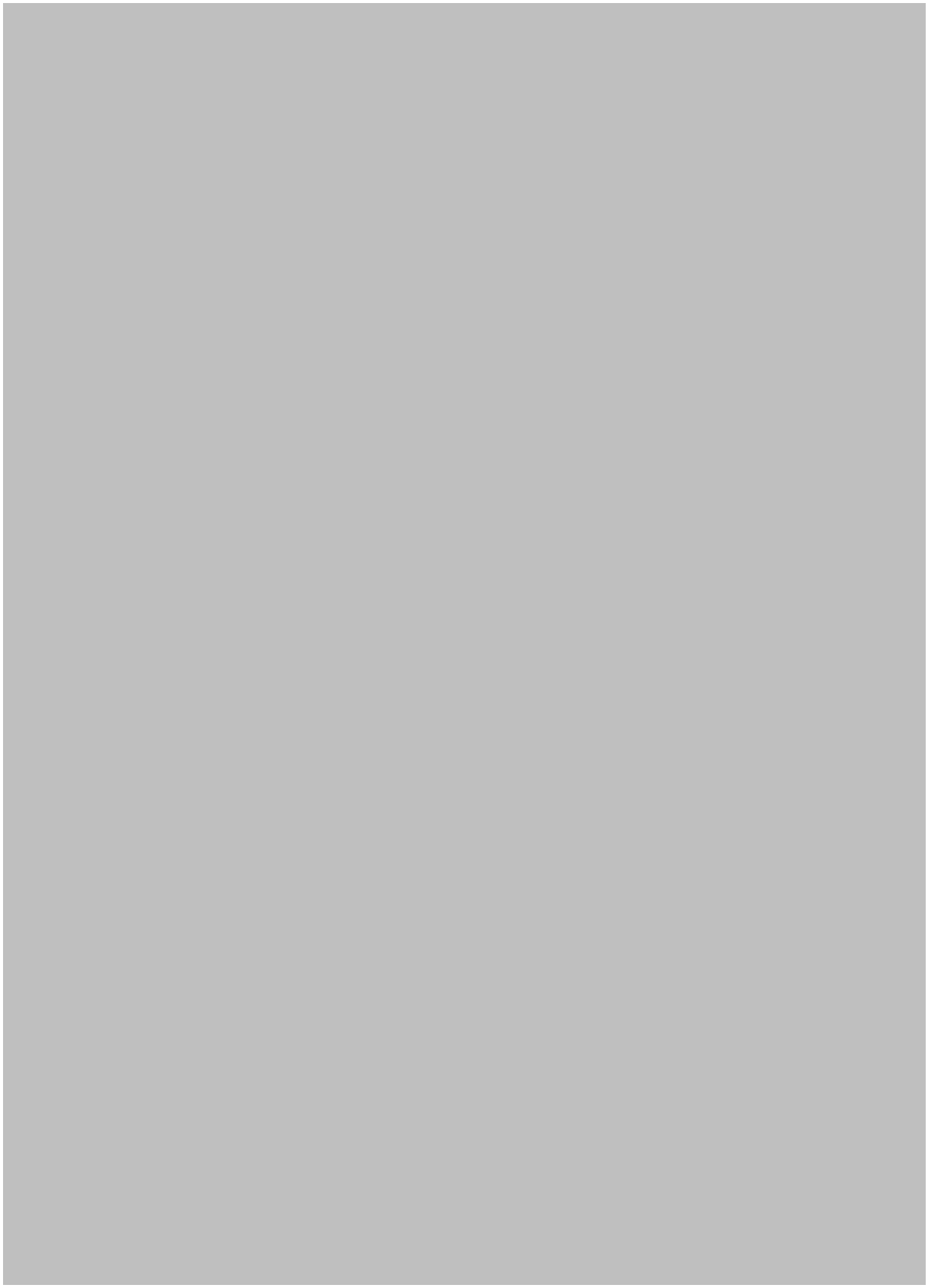
APPENDIX 10: CHILD BEHAVIOR CHECKLIST – 1-5 YEARS

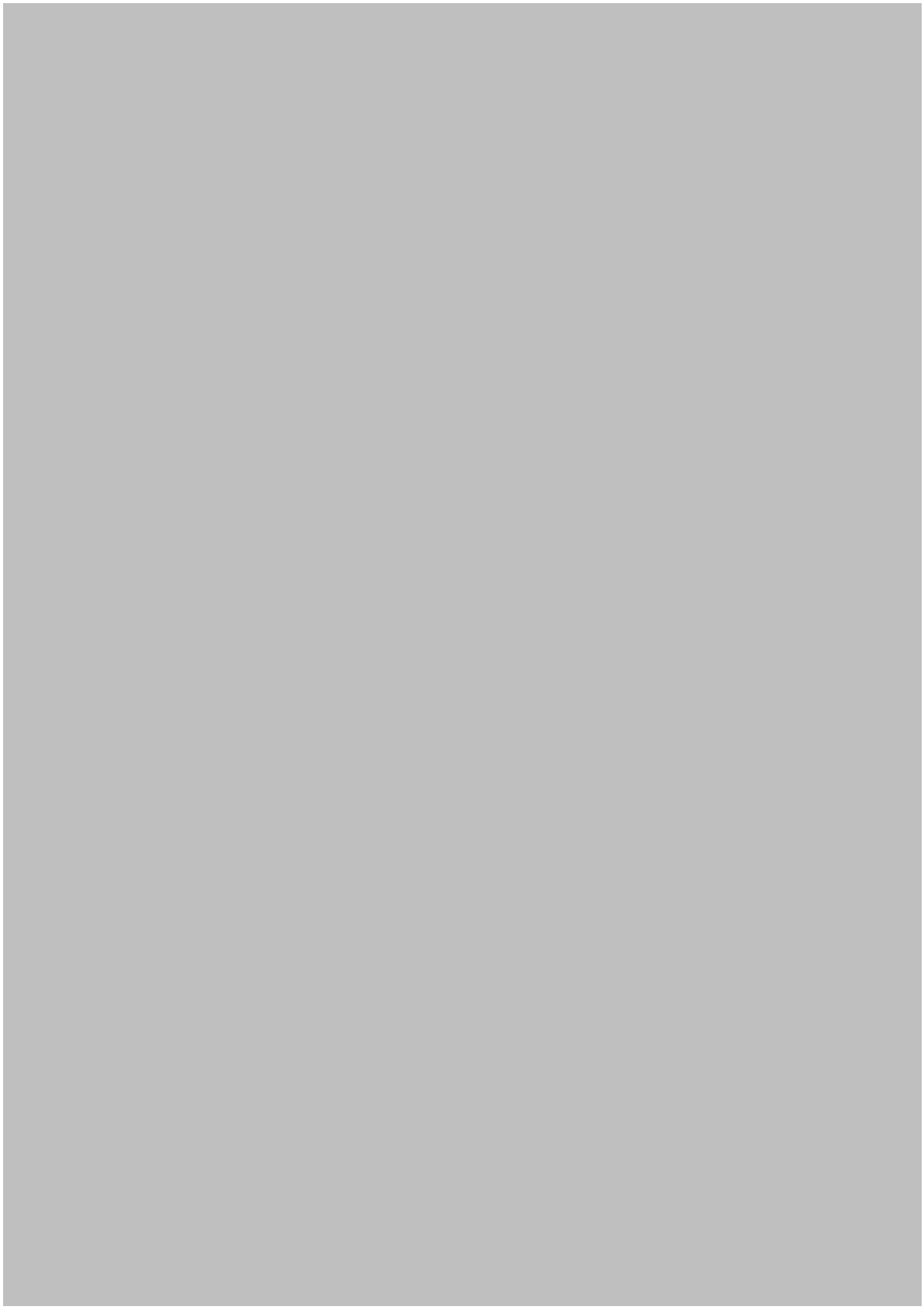


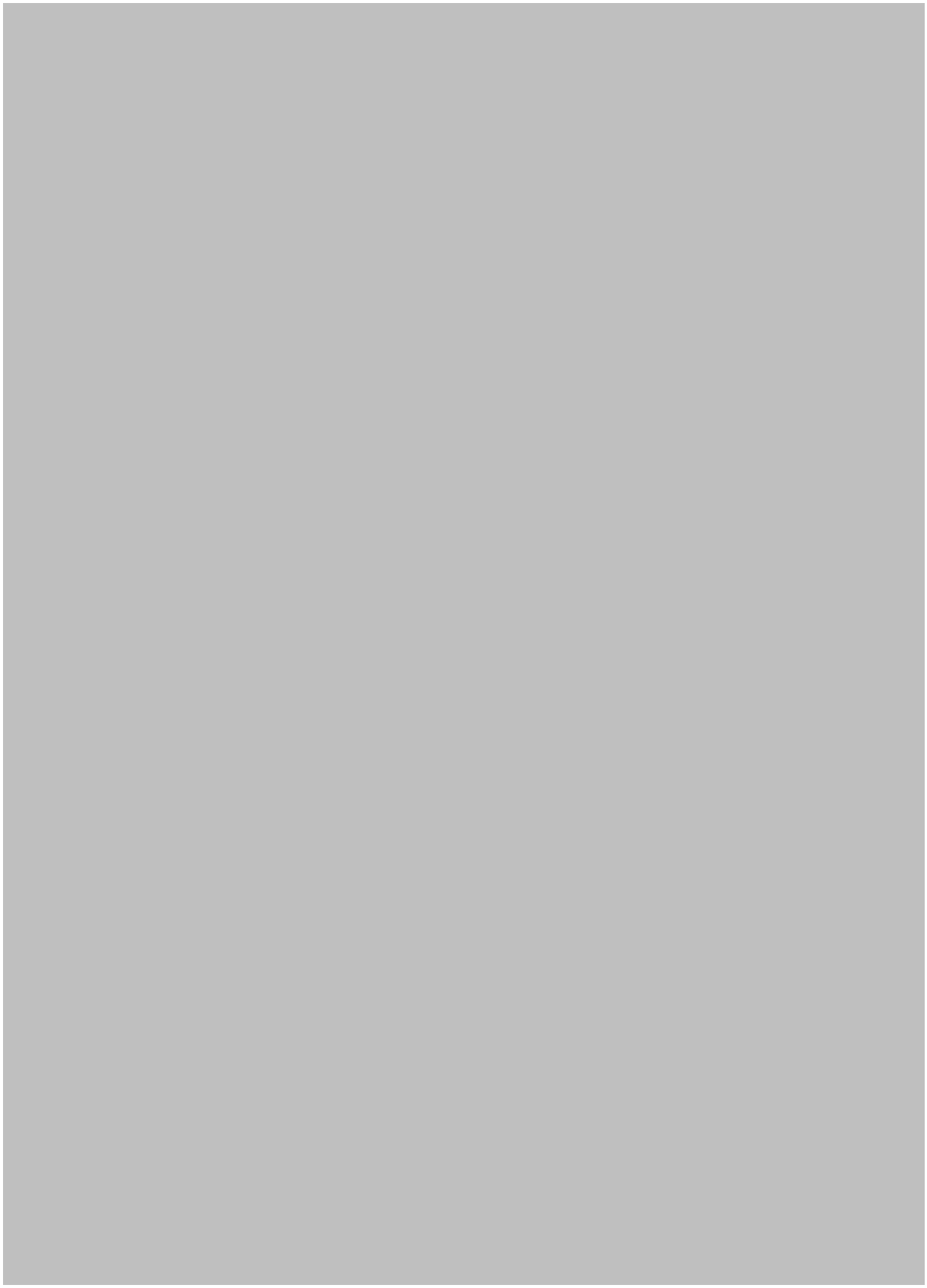


APPENDIX 10: CHILD BEHAVIOR CHECKLIST – 6-18 YEARS

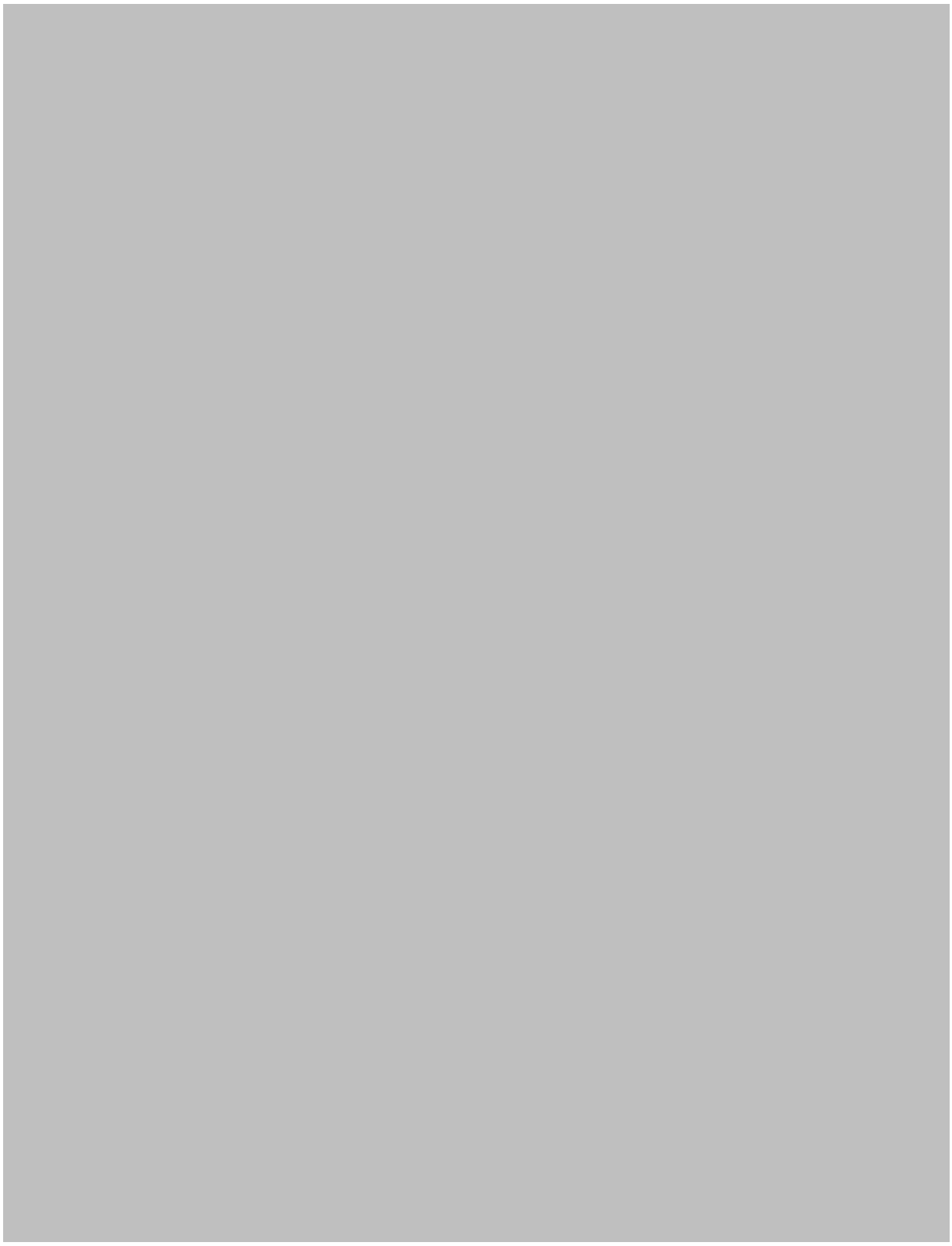


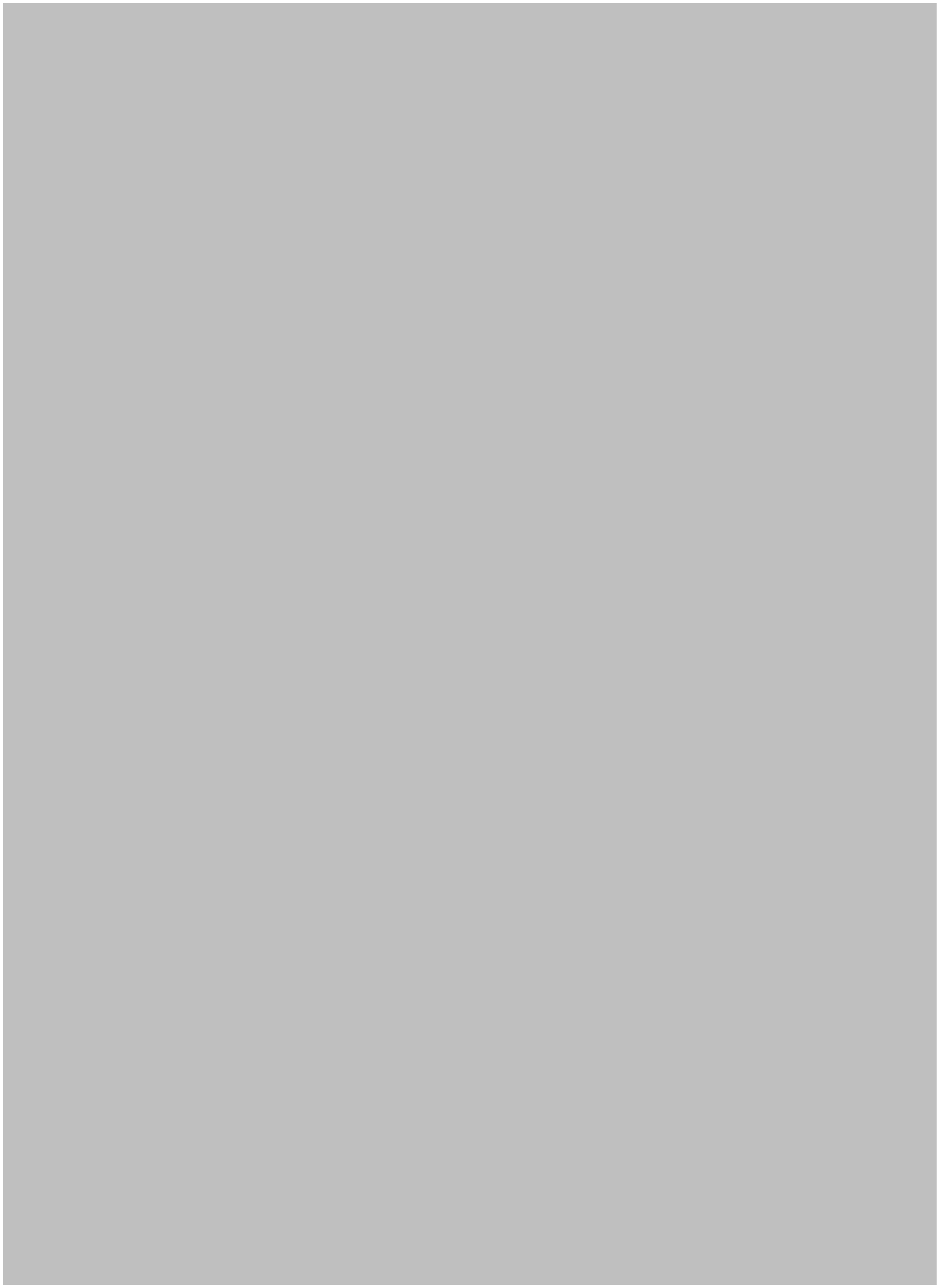


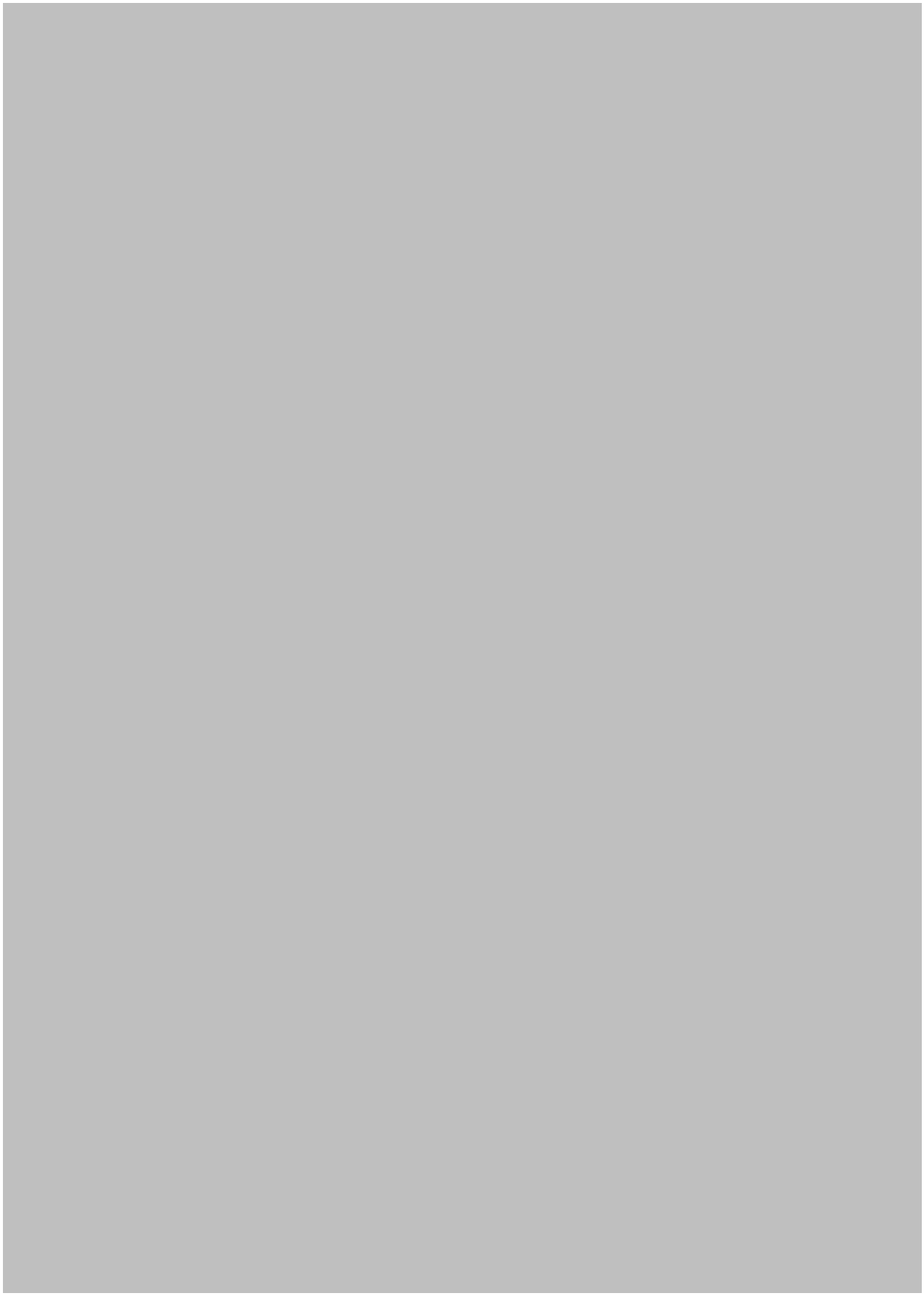


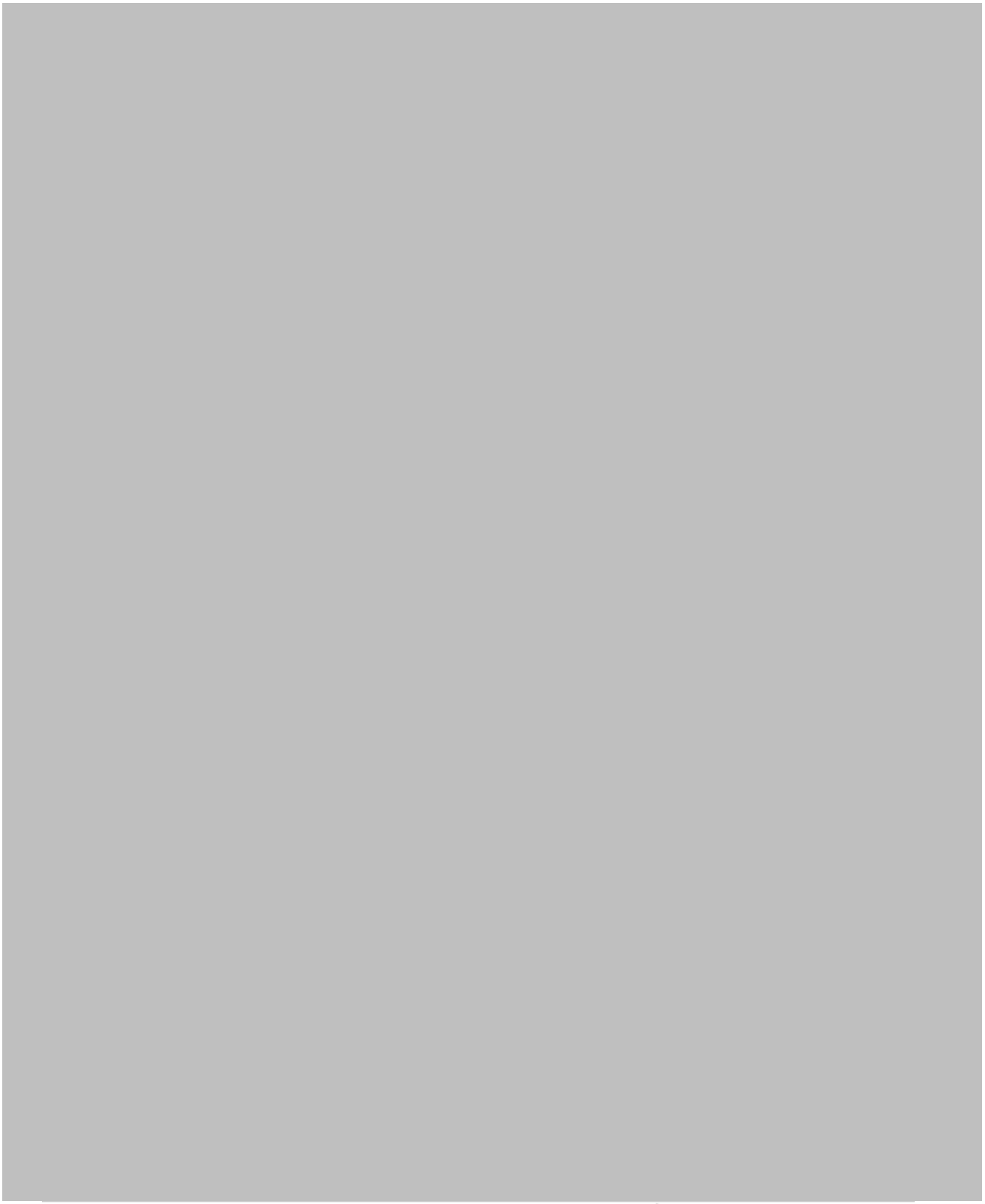


APPENDIX 11: TEACHER REPORT FORM – 6-18 YEARS









APPENDIX 12: SLEEP PROBLEM SUBSCALE ANALYSIS

Several authors in the literature have made use of the items related to sleep problems in the CBCL, and used them to devise a sleep subscale in order to measure the prevalence of sleep problems in their sample (Friedman, Corley, Hewitt & Wright, 2009; Goodnight, Bates, Staples, Pettit & Dodge, 2007; Gregory, Van der Ende, Willis & Verhulst, 2008;). It was decided that as this data would already be collected as part of the emotional and behavioural problems chapter, it may be useful to analyse these sleep items separately in order to identify whether problems with sleep were evident in those with early puberty.

The aim of this analysis was to identify which of the sleep items on the school-age CBCL could be used to form a sleep problem subscale. As the younger version of the CBCL had an existing sleep subscale, a new subscale was therefore only constructed for the school-age version of the CBCL.

Sample and Recruitment. 90 children took part in the study, 15 males and 75 females (Table A5). Children were aged between 6-10 years and recruited as part of the main thesis. The items were administered as part of the CBCL, and given to parents to complete during visit to the families' homes, or during an appointment at the BCH.

Table A5: *Participant Demographics for the Sleep Problem Scale Pilot Study*

N (m:f)	Mean Age in months (SD)	Ethnicity		SES	
90 (15:75)	104.01 (15.90)	White	59%	<£14,000	8%
		Mixed	17%	£14,000-£24,999	12%
		Asian	15%	£25,000-£34,999	17%
		Black	9%	£35,000-£49,999	29%
				>£50,000	34%

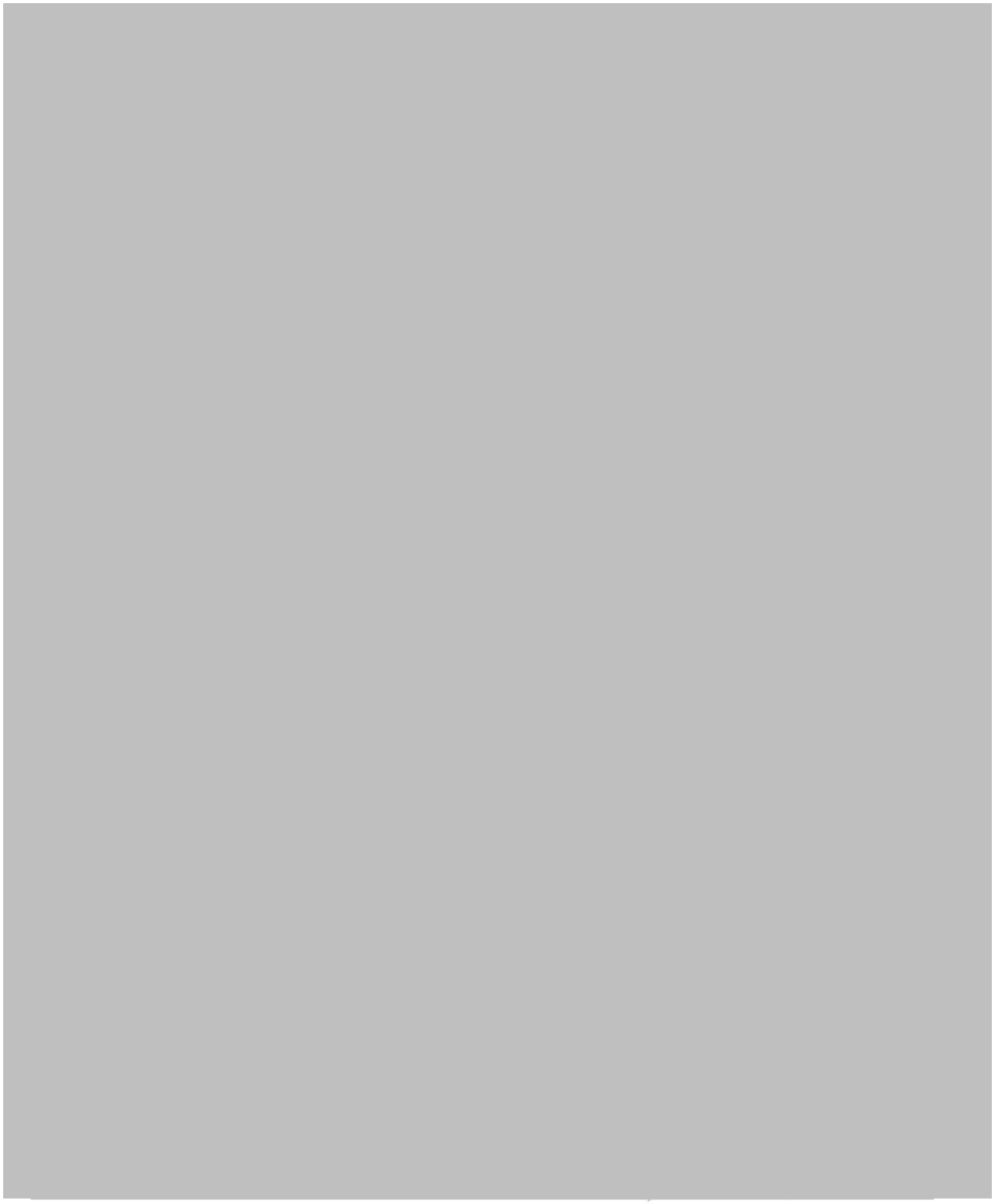
Measure and Procedure. Initially, all items in the CBCL that were potentially related to tiredness or sleep problems were included, for example, 'sleeps less than other children', 'has nightmares' and 'talks or walks in sleep'. These were coded as zero, one or two, where zero indicates an absence of behaviours and two indicates a frequent occurrence of behaviours.

Analysis. Internal reliability analyses were conducted, and items were removed until the highest possible Cronbach's alpha was reached. The resulting subscale consisted of seven items; poor concentration, daydreaming, nightmares, sleeps less than other children, stares, talks in sleep, and has trouble sleeping. The final alpha was .76, suggesting high internal reliability.

Discussion. The aim of this analysis was to assess the value of the existing sleep items in the CBCL when constructing a sleep problem scale. It would appear that the final seven items show adequate reliability when combined into a single scale, and it was

therefore concluded that the sleep problem scale was suitable for inclusion in the main thesis.

APPENDIX 13: PSPCSA – EXAMPLE ITEM



APPENDIX 14: SPPC

	Really Like Me	Sort of Like Me				Sort of Like Me	Really Like Me
A			Some children would rather play outdoors in their spare time	BUT	Other children would rather watch TV		
1			Some children feel that they are very good at their school work	BUT	Other children worry about whether they can do their school work		
2			Some children find it hard to make friends	BUT	Other children find it pretty easy to make friends		
3			Some children are happy with the way they look	BUT	Other children are not happy with the way they look		
4			Some children often do not like the way they behave	BUT	Other children usually like the way they behave		
5			Some children are often unhappy with themselves	BUT	Other children are pretty pleased with themselves		
6			Some children feel like they are just as smart as other children their age	BUT	Other children aren't sure and wonder if they are as smart		
7			Some children have a lot of friends	BUT	Other children don't have very many friends		
8			Some children wish they could be a lot better at sports	BUT	Other children feel they are good enough at sports		
9			Some children do very well at all kinds of sports	BUT	Other children don't feel that they are very good at any sports		
10			Some children are happy with their height and weight	BUT	Other children often wish their height and weight were different		
11			Some children usually do the right thing	BUT	Other children often don't do the right thing		
12			Some children don't like the way they are leading their life	BUT	Other children do like the way they are leading their life		
13			Some children are pretty slow at finishing their school work	BUT	Other children can do their school work quickly		

	Really Like Me	Sort of Like Me				Really Like Me	Sort of Like Me
14			Some children would like to have a lot more friends	BUT	Other children have as many friends as they want		
15			Some children think they can do well at any sport they haven't tried before	BUT	Other children are afraid they might not do well at sports they haven't tried		
16			Some children wish their body was different	BUT	Other children like their body the way it is		
17			Some children usually act the way they know they are supposed to	BUT	Other children often don't act the way they know they are supposed to		
18			Some children are happy with themselves as a person	BUT	Other children are often not happy with themselves		
19			Some children often forget what they learn	BUT	Other children can remember things really easily		
20			Some children are always doing things with lots of other children	BUT	Other children usually do things by themselves		
21			Some children feel that they are better than other children their age at sports	BUT	Other children don't feel that they can play as well		
22			Some children wish that they looked different	BUT	Other children like the way they look		
23			Some children usually get in trouble because of things they do	BUT	Other children don't usually do things that get them into trouble		
24			Some children like the kind of person they are	BUT	Other children often wish they were someone else		
25			Some children do very well with their class-work	BUT	Other children don't do very well with their class-work		
26			Some children wish that more people their age like them	BUT	Other children feel that most people their age do like them		
27			In games and sports some children usually watch instead of play	BUT	Other children usually play rather than just watch		

	Really Like Me	Sort of Like Me				Really Like Me	Sort of Like Me
28			Some children wish something about their face or hair looked different	BUT	Other children like their face and hair the way they are		
29			Some children do things they know they shouldn't do	BUT	Other children hardly ever do things they know they shouldn't do		
30			Some children are very happy being the way they are	BUT	Other children wish they were different		
31			Some children have trouble figuring out the answers in school	BUT	Other children almost always can figure out the answers		
32			Some children are popular with other children their age	BUT	Other children are not very popular		
33			Some children don't do well at new outdoor games	BUT	Other children are good at new games right away		
34			Some children think that they are good looking	BUT	Other children think that they are not very good looking		
35			Some children behave themselves very well	BUT	Other children often find it hard to behave themselves		
36			Some children are not very happy with the way they do a lot of things	BUT	Other children think they way they do things is fine		

APPENDIX 15: PAQ PILOT STUDY

Perceived Age Questionnaire Pilot Study. The newly constructed perceived age questionnaires (PAQ) were piloted with local families recruited through primary schools around Birmingham for internal reliability, subscale structure and ease of understanding. These questionnaires assessed whether the child looked older than their peers, whether they were bigger than their peers, and whether they experienced any positive or negative consequences of looking older. It was hypothesised that the items would fall into these four factors.

Sample and Recruitment. Both child and parent or carer versions of the questionnaire were distributed through local primary schools (Appendix 15). Copies of the parent or carer questionnaire were sent home with the pupils of the school and completed forms returned to the school to be collected by the research team. Parents and carers were also approached in the playground after school, and asked to complete the questionnaire whilst waiting for their child. Participant questionnaires were completed at school during the school day, with the help of the experimenter. Opt-out letters had been sent home (Appendix 16), and children whose parents had returned these letters were not included in the data collection. Furthermore, children whose level of English was not considered to be of standard were excluded, as they may not have had full understanding of what was asked of them. Schools were recruited from the centre of Birmingham, and therefore enrolled pupils of a wide range of ethnicities and socioeconomic groups. Ninety-five completed parent and carer questionnaires and 349 child participant questionnaires were collected. Forty-six of these were completed by parents and children from the same family. Two hundred and four of the child participants were female and 194 were male. The child

participants were aged between 48-130 months, with a mean age of 88.5 months and a standard deviation of 21.3 months. Demographic data of the parents was not collected.

Measure. The PAQ was constructed as a result of concerns mentioned during consultations at the BCH and family meetings at the CGF. Examples of these concerns were that children couldn't fit into clothes suitable for their age, were treated as older, and were considerably taller than their peers. Items were designed to assess the prevalence of these situations, and whether they were also present in the typically-developing population.

Analysis. Principal components analysis of the parent data using a Varimax rotation initially produced a four factor model, however, two of these factors overlapped considerably when looking at the item constructs of each factor. A forced three-factor model was then applied to the data, which explained 60% of the data variance. All factor loadings were still above.5 and the model was therefore deemed a good fit of the data. Furthermore, Cronbach's alpha values for the three factors were .92, .70 and .66 respectively, with a total scale value of .73. This implies that the scale has good internal reliability. Table 6 gives the items within each factor with their relevant factor loadings.

The child participant data was then analysed. Principal components analysis of the child data using a Varimax rotation initially produced a five-factor model; however, one factor contained just a single item. Reanalysis with a forced four component extraction loaded the item on to one factor, but with a loading of .33. Reliability analysis of the factor demonstrated that the alpha value would be greater after removal of the item, and it was therefore discounted. It was also observed that two of the factors overlapped considerably in the four-factor model. A forced three-factor model was therefore applied to the data. This model was found to explain 43% of the data variance, and the majority of factor loadings were above .5. Cronbach's alpha values for the three factors were .74, .50 and .45

respectively, with a total scale value of .76. This implies that the scale has adequate internal reliability.

Table A6: *Item loadings for the pilot parent data of the PAQ*

	Looking Older/ Bigger	Negative Consequences	Positive Consequences
People say that my child is big for their age	.85		
Adults think my child is older than their actual age	.84		
Other children in my child's class are much smaller than they are	.83		
My child is taller than the other children in their class	.81		
Other children think my child is older than their actual age	.80		
My child looks more grown up than their friends	.78		
Other children in my child's class look younger than they do	.73		
My child wears clothes that are really for older children	.67		
People think my child is older than their older siblings or cousins	.54		
My child wishes they looked more like their friends		.80	
My child feels unhappy if they look different to their friends		.78	
My child wishes they were shorter		.68	

Other children make jokes about my child's size	.66	
Older children think my child is the same age as they are		.77
My child likes looking different to their friends		.65
People talk to my child as if they are older than their actual age		.64
Adults sometimes give my child more responsibilities than other children		.55

A7: *Item loadings for the pilot child data of the PAQ*

	Looking Older/ Bigger	Negative Consequences	Positive Consequences
Do you think you are taller than the other children in your class?	.734		
Are the other children in your class much smaller than you?	.702		
Do grown-ups ever think you are older than your actual age?	.612		
Do you wear clothes that are really for older children?	.569		
Do people say that you are big for your age?	.563		
Do other children think that you are older than you really are?	.516		
Do children in your class look a lot younger than you?	.377		
Do you think you look more grown up than your friends?	.372		
Do you wish you were shorter?		.722	
Do people think that you are older than your brothers and sisters?		.543	
Do other children make jokes about your size?		.518	
Do you ever wish that you looked more like your friends?		.489	

Do older children think that you are the same age as they are?	.433	
Do you like looking different to your friends?		.723
Does it make you happy if people think you are older?		.701

3.3.1.6.4.4 Discussion. The findings demonstrate the suitability of the PAQ for use in measuring whether a child is perceived as looking bigger or older than their friends, and whether they experience any of the possible positive or negative consequences of looking older than their peers. The parent or carer measure was found to be highly reliable with a stable factorial structure. Three factors were identified; with nine items concerned with whether the parent perceived their child as looking bigger or older than their peers, four items assessing whether they observed negative consequences associated with looking older in their child, and another four items focussed on whether they observed positive consequences associated with their child looking older.

The same three factors were identified in the child participant data; however this model was not such a good fit, and demonstrated a less reliable internal consistency. The model was still considered suitable for use in a patient population, however, it may have been that using a control population for the pilot group did not demonstrate full use of the measure because of a lack of variance. We might assume that few participants from the general population would demonstrate the characteristics being measured in the questionnaire, and therefore all would score fairly low. This may mean that it is more difficult to distinguish between the scale components. However, we would also expect this same lack of variance in the parent questionnaires, so this explanation may not be correct. An alternative explanation may be that younger children are less aware of how others perceive them, and are not attentive to the way others respond to them with regards to whether this is age appropriate. We might therefore expect these items to fall more randomly across factors in the younger groups. Reanalysis of the data by age group however, demonstrated that the younger age groups did not consistently show the least stability in their scores. We would expect the youngest children to show lower internal

consistency alpha values, and a lower explained variance in the factor analysis but this was not the case.

One observation that was made in the analysis, was the overlap between looking bigger and looking older in both the parent and child measures. In the original questionnaire design, items specifically focused on size were included, as well as separate items related to looking older. It was anticipated that these items would load on to different factors, but that these factors would likely be correlated. It was considered that the child data would likely confuse the items related to looking bigger and the items related to looking older and that these items may load loosely onto both factors, but it was not expected of the parent data. In the analysis, however, these items loaded on to the same factor for both child and parent measures, suggesting that both adults and children perceive bigger children to be older, rather than using other features such as body shape, behaviour, intelligence or facial features. It may also be, however, that these two factors would become more discrete if a patient population was included in the analysis. This is because these children may look older than their chronological age because of breast development or changes to their body shape, as opposed to simply being bigger. It was therefore decided that the PAQ would be useful in the main project, when studying the perceived age of children with disorders of puberty.

There are however several limitations to this project. It would have been ideal to have parents and children from the same families complete the PAQ, and to combine these data with measures of the child's pubertal development and height. This would have enabled testing of the validity of the perceived age measures, and comparison of the accuracy of the child's perceptions of their appeared age to the age predicted by their height and pubertal development. Furthermore, comparisons between the parental

perception and the child's perception of the child's appeared age could have been made, in order to identify any existing differences. Unfortunately however, due to time constraints and a low return rate of the parent questionnaires this amalgamation of data was not possible. Therefore, only parent and child understanding of the questionnaires were tested, alongside whether the factorial structure was adequate for use in research.

APPENDIX 15: PAQ

Age:		Male	Female	
1	Do you think you are taller than the other children in your class?	**Not really**	**Taller than some children**	**Taller than most children**
2	Do grown-ups ever think you are older than your actual age?	**Not really**	**Sometimes**	**A lot**
3	Does it make you happy if people think you are older?	**Not really**	**A little bit**	**A big bit**
4	Do you think you look more grown up than your friends?	**Not really**	**More grown up than some**	**More grown up than most**
5	Do other children think that you are older than you really are?	**Not really**	**Sometimes**	**A lot**
6	Do people say that you are big for your age?	**Not really**	**Sometimes**	**A lot**
7	Do you like looking different to your friends?	**Not really**	**A little bit**	**A big bit**
8	Do older children think that you are the same age as they are?	**Not really**	**Sometimes**	**A lot**
9	Are the other children in your class much smaller than you?	**Not really**	**Some are smaller**	**Lots are smaller**
10	Do you ever wish that you looked more like your friends?	**Not really**	**A little bit**	**A big bit**
11	Do children in your class look a lot younger than you?	**Not really**	**Some children**	**Lots of children**
12	Do people think that you are older than your brothers and sisters?	**Not really**	**Sometimes**	**A lot**
13	Do you wear clothes that are really for older children?	**Not really**	**Some of my clothes**	**Most of my clothes**
14	Do you wish you were shorter?	**Not really**	**Sometimes**	**A lot**
15	Do other children make jokes about your size?	**Not really**	**Sometimes**	**A lot**
16	Does looking different to your friends ever make you unhappy?	**Not really**	**Sometimes**	**A lot**

APPENDIX 16: LEQ

Listed below are a number of events which may bring about changes in the lives of those who experience them. Please circle the numbers corresponding to the events that have occurred in your life during the last year, and mark whether these were 'Good' or 'Bad'. Then show how much each event has affected your life by making a mark in the column which corresponds with the most appropriate statement. If the event has not occurred during the past year please leave it blank.

Event		**Positive/ Negative Effect**		**Impact of the Event on your Life**			
Health		Good	Bad	No Impact	Some Impact	Medium Impact	Great Impact
1	Major personal illness or injury						
2	Major change in eating habits						
3	Major change in sleeping habits						
4	Major change in normal type/ amount of recreation						
5	Major dental work						
6	Pregnancy (females)						
7	Miscarriage or abortion (females)						
8	Start of menopause (females)						
9	Major difficulties with method of birth control						
Work		Good	Bad	No Effect	Some Effect	Medium Effect	Great Effect
10	Difficulty in finding a job						
11	Beginning work outside the home						
12	Starting a new job						
13	Changing to a new type of work						
14	Change in work hours or conditions						
15	Change in work responsibilities						
16	Troubles with employer or colleagues						
17	Major business readjustment						
18	Being fired or laid off						
19	Retirement						
20	Studying at home in addition to work						
Education		Good	Bad	No Effect	Some Effect	Medium Effect	Great Effect
21	Starting or finishing study						
22	Change in study location eg. Moving university						
23	Change in career goal or subject of study						
24	Problem in study eg. Poor grade						
Residence		Good	Bad	No Effect	Some Effect	Medium Effect	Great Effect
25	Difficulty finding housing						
26	Changing residence within same area						
27	Moving to a new area						
28	Change in living conditions eg. Home improvements or repairs						
29	Conflicts with neighbours						

Relationships		Good	Bad	No Effect	Some Effect	Medium Effect	Great Effect
30	Beginning a new relationship						
31	Engagement						
32	Relationship problems						
33	Break-up of relationship						
34	Partner's pregnancy						
35	Partner's miscarriage or abortion						
36	Partner's menopause						
37	Moving in with partner						
38	Marriage						
39	Changes in closeness with partner						
40	Sexual difficulties						
41	Infidelity						
42	Trouble with in-laws						
43	Separation with partner due to conflict						
44	Separation with partner due to travel or work						
45	Reconciliation with partner						
46	Divorce						
47	Change in partner's work						
Family and Close Friends		Good	Bad	No Effect	Some Effect	Medium Effect	Great Effect
48	New family member in the household eg. Adoption, grandparent moving in						
49	Family member leaving home						
50	Major change in health or behaviour of family member or close friend (excluding early onset puberty in offspring)						
51	Conflict with relative or close friend						
52	Death of partner						
53	Death of child						
54	Death of other family member or close friend						
55	Birth of grandchild						
56	Change in parents' marital status						
Parenting		Good	Bad	No Effect	Some Effect	Medium Effect	Great Effect
57	Change in childcare arrangements						
58	Conflicts with partner regarding parenting						
59	Conflicts with other family members regarding parenting						
60	Regular conflicts with child						
61	Becoming a single parent						
62	Custody battles with former partner						
63	Child performing poorly at school						
64	Child being bullied/ unhappy at school						
65	Change in child's sleeping or eating patterns						
66	Change in child's temperament						
67	Change in child's behaviour						

Personal or Social		Good	Bad	No Effect	Some Effect	Medium Effect	Great Effect
68	Major personal achievement						
69	Major decision regarding immediate future						
70	Change in personal habits or life-style						
71	Change in religious beliefs						
72	Change in political beliefs						
73	Loss or damage of personal property						
74	Vacation						
75	Trip other than vacation						
76	Change in family get-togethers						
77	Change in social activities						
78	Gained new friends						
79	Loss of friends						
80	Gain or lose a pet						
Financial		Good	Bad	No Effect	Some Effect	Medium Effect	Great Effect
81	Major change in finances						
82	Moderate purchase eg. New car						
83	Major purchase eg. New home						
84	Foreclosure of mortgage or loan						
85	Credit rating difficulties						
Crime and Legal Matters		Good	Bad	No Effect	Some Effect	Medium Effect	Great Effect
86	Victim of robbery or identity theft						
87	Victim of violent act eg. Assault						
88	Involved in an accident						
89	Involved in a law suit						
90	Involved in minor violation of the law eg. Speeding ticket						
91	Legal troubles resulting in arrest						
Other- Other experiences during this time, which have had an impact on your life		Good	Bad	No Effect	Some Effect	Medium Effect	Great Effect
92							
93							
94							

APPENDIX 17: PSS

The questions in this scale ask you about your feelings and thoughts during the last month. Please indicate how often you felt or thought a certain way. Although some questions are similar, please treat each one as a separate question. Please do not spend too long thinking about each question but simply give a reasonable estimate. For each question please give a number between 0 and 4.

0= never
1= almost never
2= sometimes
3= fairly often
4= very often

1. How often were you upset because of something that happened unexpectedly?	
2. How often did you feel unable to control the important things in your life?	
3. How often did you feel nervous and stressed?	
4. How often did you feel able to deal successfully with life hassles?	
5. How often did you fell that you were effectively coping with important changes that were occurring in your life?	
6. How often did you feel confident that you were able to handle your personal problems?	
7. How often did you feel that things were going your way?	
8. How often did you feel that you could not cope with all of the things you had to do?	
9. How often did you feel able to control the irritations in your life?	
10. How often did you feel that you were on top of things?	
11. How often did you feel angry because of things that were outside of your control?	
12. How often did you think of the things you had to accomplish?	
13. How often did you feel you were in control of how you spent your time?	
14. How often did you feel that difficulties were becoming more than you could overcome?	

APPENDIX 18: IES

Below is a list of comments, which are commonly experienced after a difficult life event. Please check each item indicating how frequently these comments were true for you during the time of your child's diagnosis.

	Not at all	Rarely	Sometimes	Often
1. I had strong waves of feelings about it.				
2. Other things kept making me think about it.				
3. I was aware that I still had a lot of feelings about it, but didn't deal with them.				
4. I thought about it when I didn't mean to.				
5. I avoided letting myself get upset when I was reminded of it.				
6. I tried to remove it from memory.				
7. Images about it kept coming into my mind.				
8. I tried not to talk about it.				
9. Any reminder brought back feelings about it.				
10. I had trouble falling asleep, because of thoughts that came into my mind.				
11. My feelings about it were numb.				
12. I felt as if it were not actually real.				
13. I stayed away from reminders of it.				
14. I tried not to think about it.				
15. I had dreams about it.				

APPENDIX 19: QUALITATIVE INTERVIEW QUESTIONS

Interview Schedule

Tell me a bit about your daughter

Has your perception of your daughter changed?

Tell me about when you first noticed any changes and the process of getting a diagnosis.

What does your daughter understand about their diagnosis?

How do you think the disorder has affected your daughter?

How do you think your daughter has been affected at school?

How did you explain your daughter‘s diagnosis to their teachers?

How has your relationship been with your daughter’s school and their teachers?

How has the disorder affected you as parents?

Are there any experiences you have had regarding your daughter’s diagnosis, which you feel were particularly difficult?

How has information from doctors changed your view of your daughter?

When you found out about your daughter’s condition, what did you think would happen?

How do you think it has affected the relationship between you and your daughter?

How would life be different for you if your daughter did not have the condition?

How do you think it has affected other family members if at all?

How do you think others would understand your daughter’s diagnosis?

How do you think the way other people behave towards your child has changed, if at all?

What do you think other adults notice about your daughter?

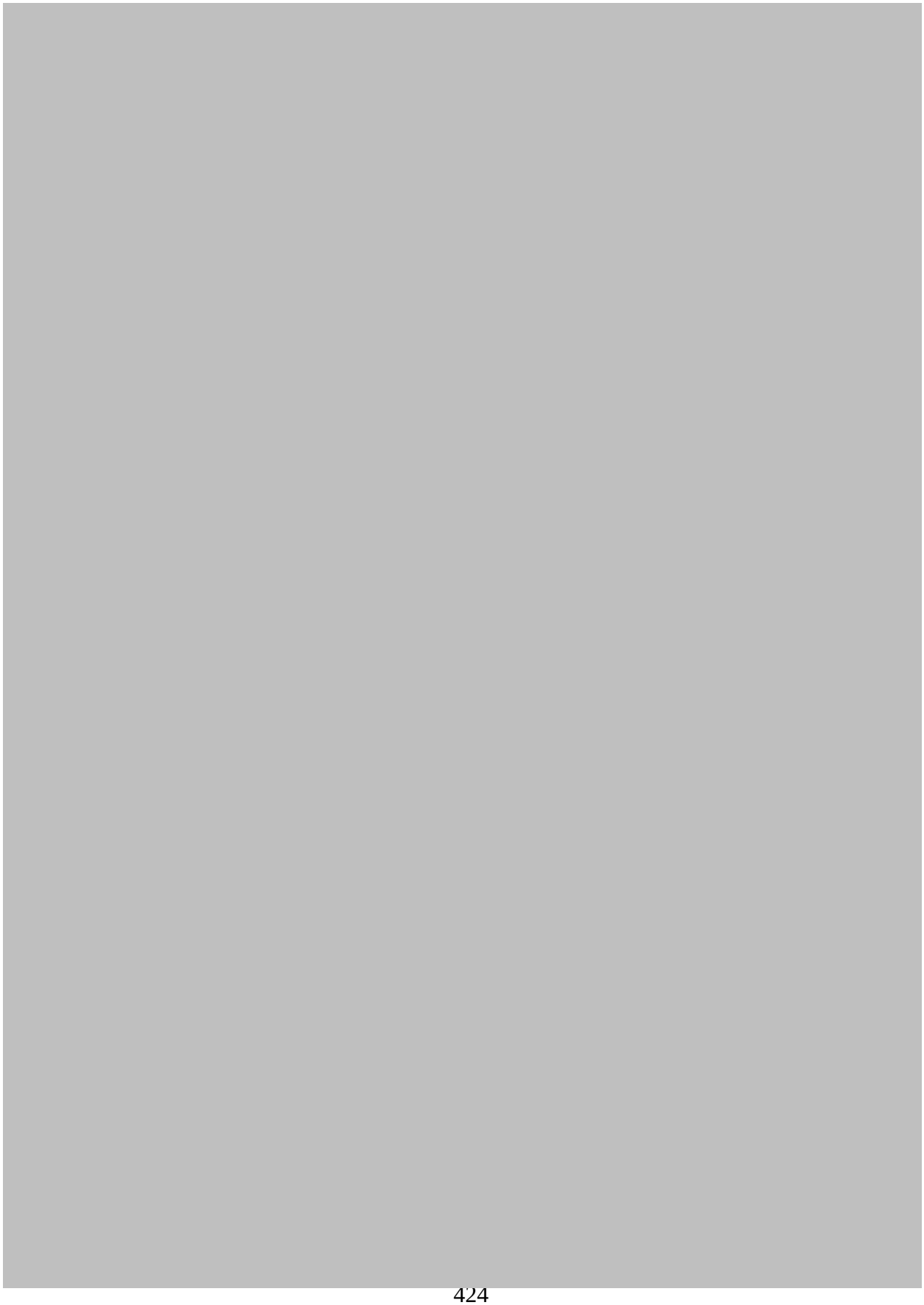
How do you think other children perceive your daughter?

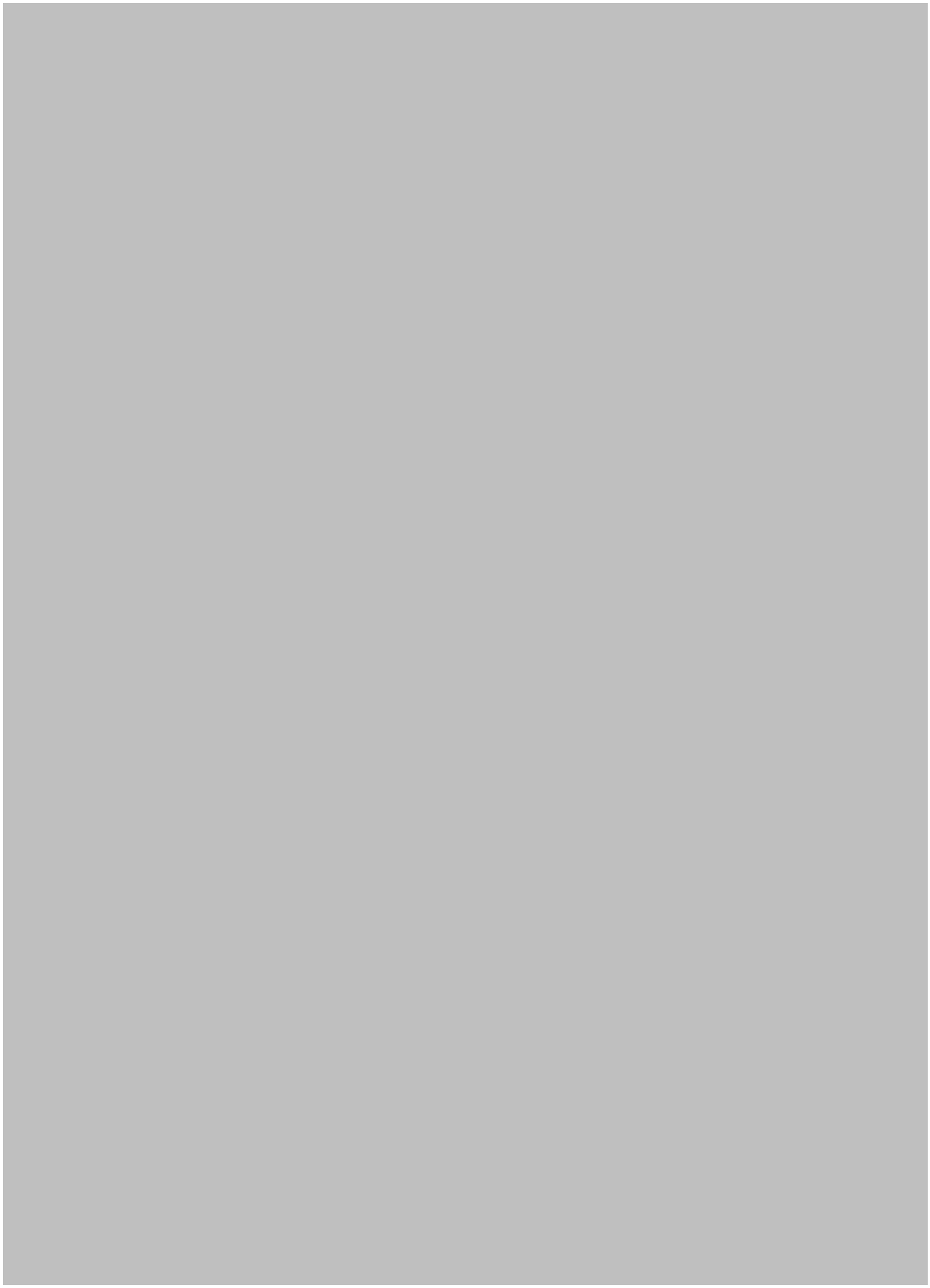
Are there any situations you find particularly difficult?

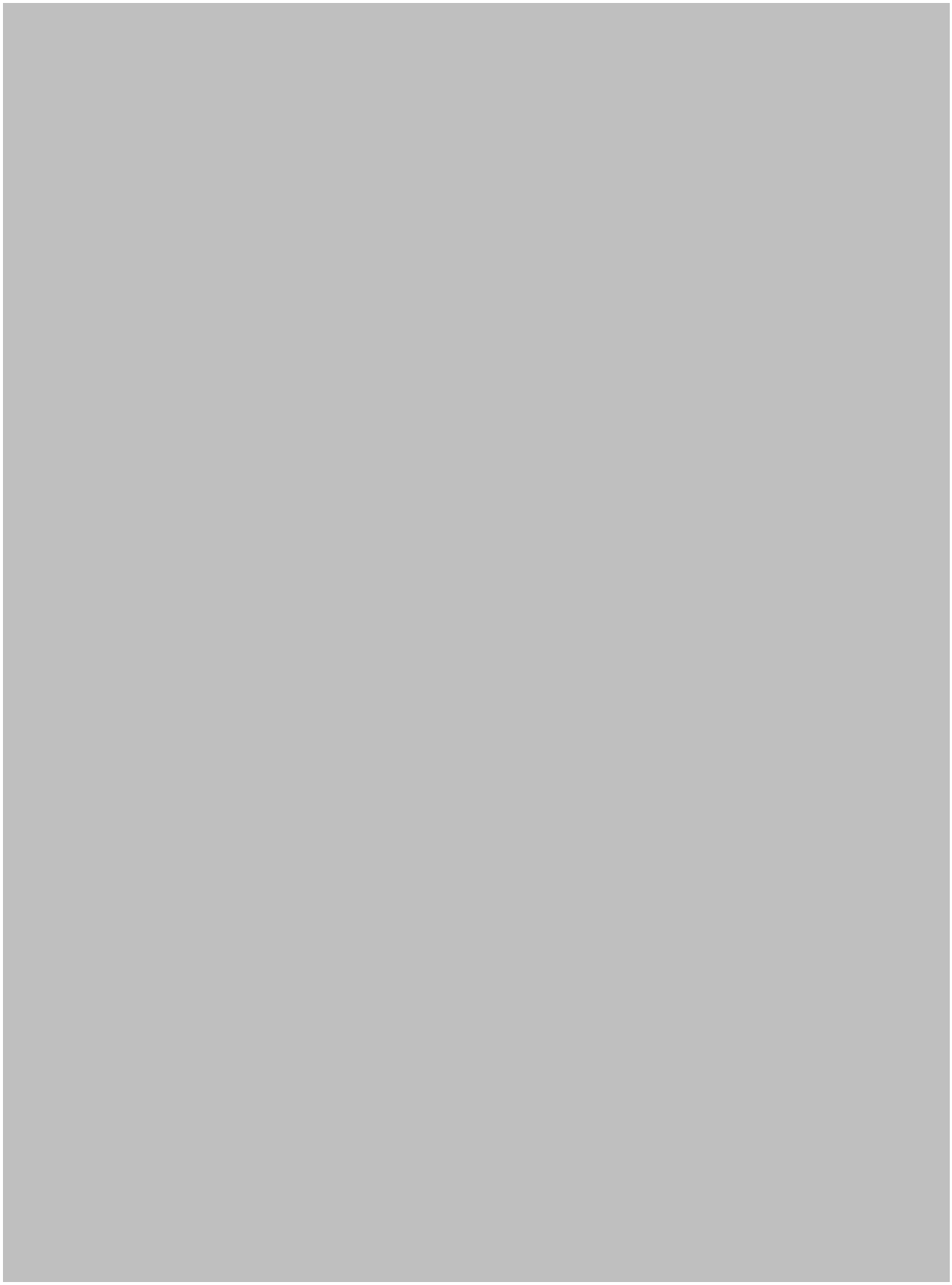
What are your hopes for the future?

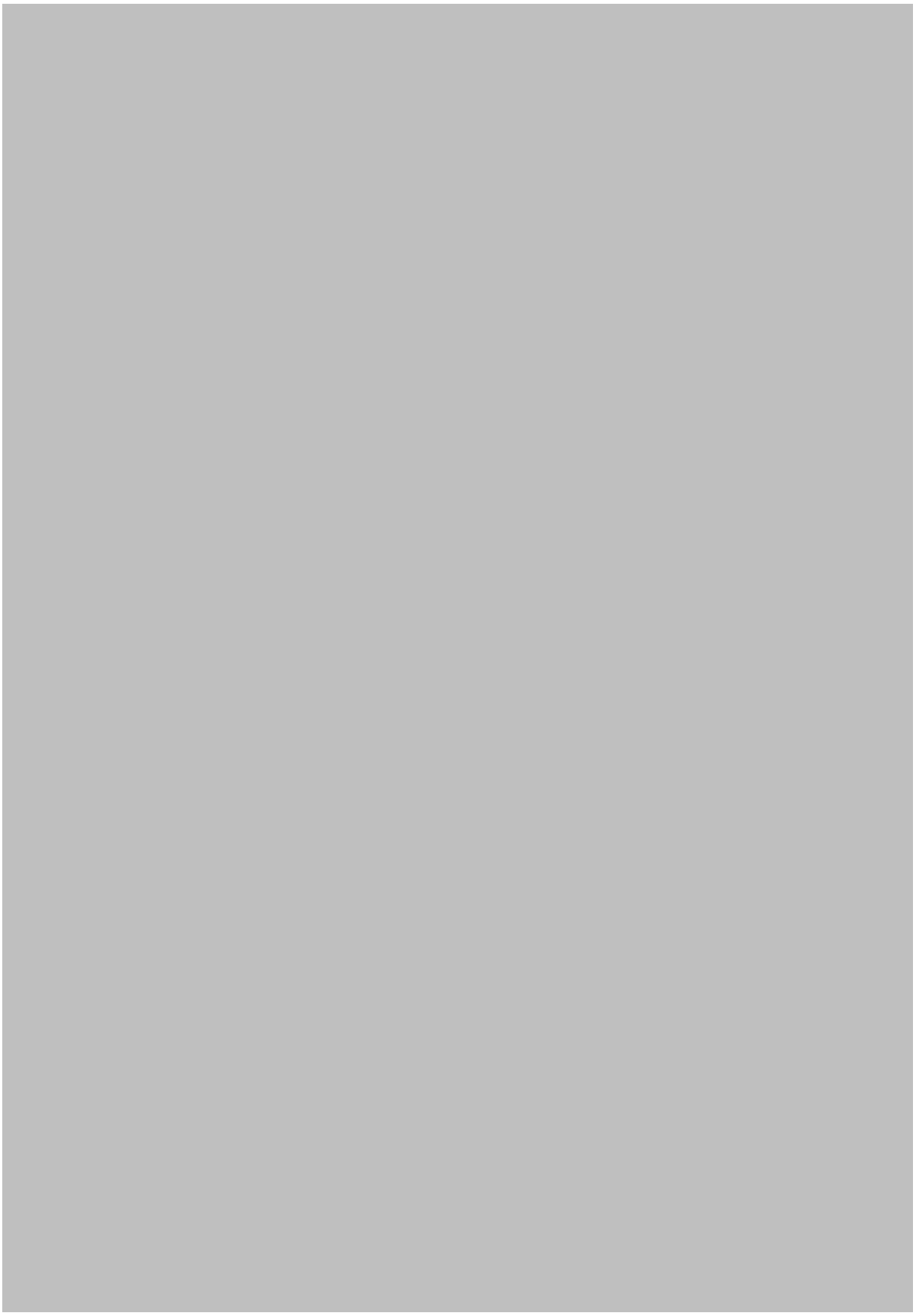
Is there anything else you would like to add?

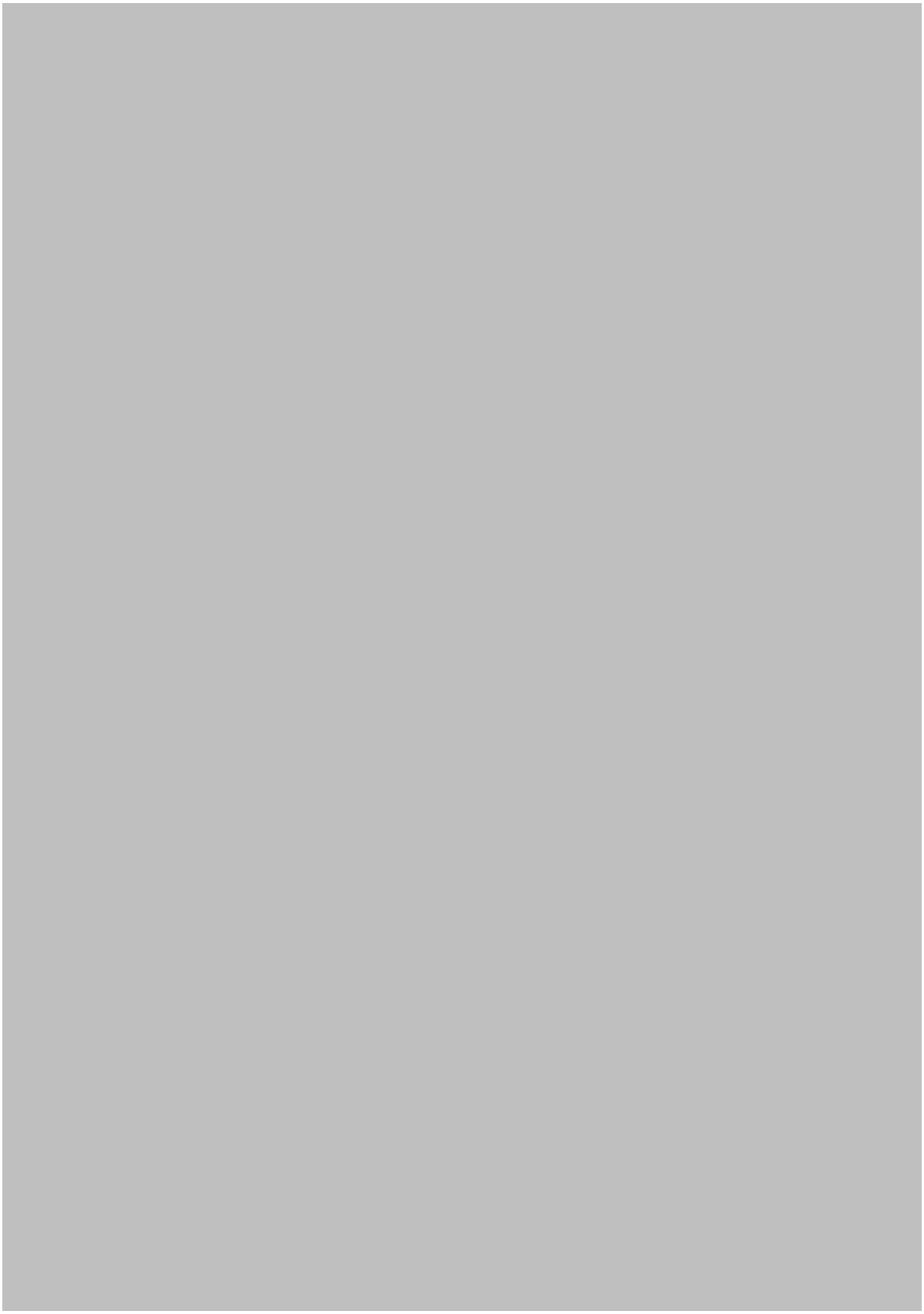
APPENDIX 20: BODY COMPOSITION PERCENTILE CHARTS

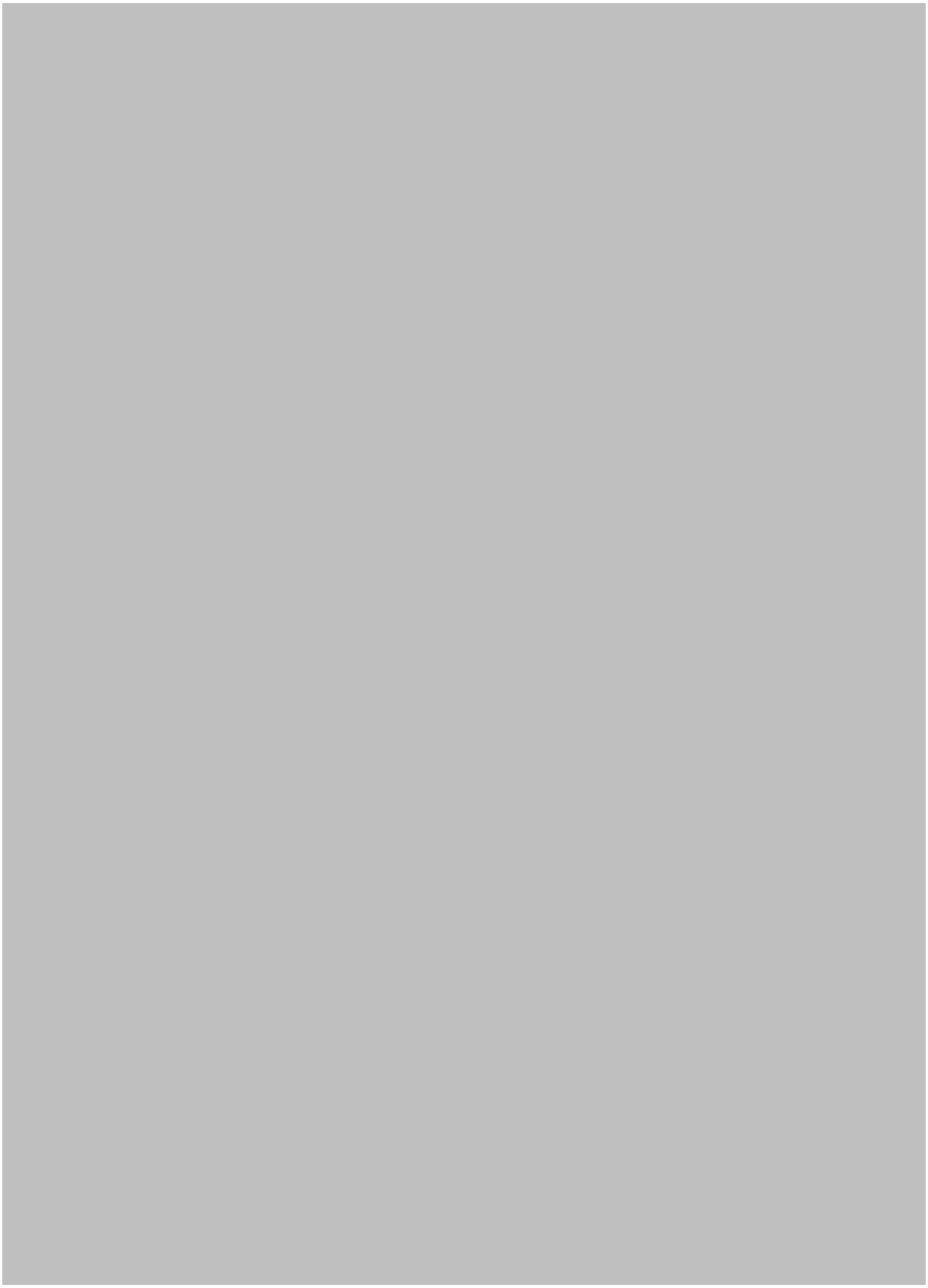


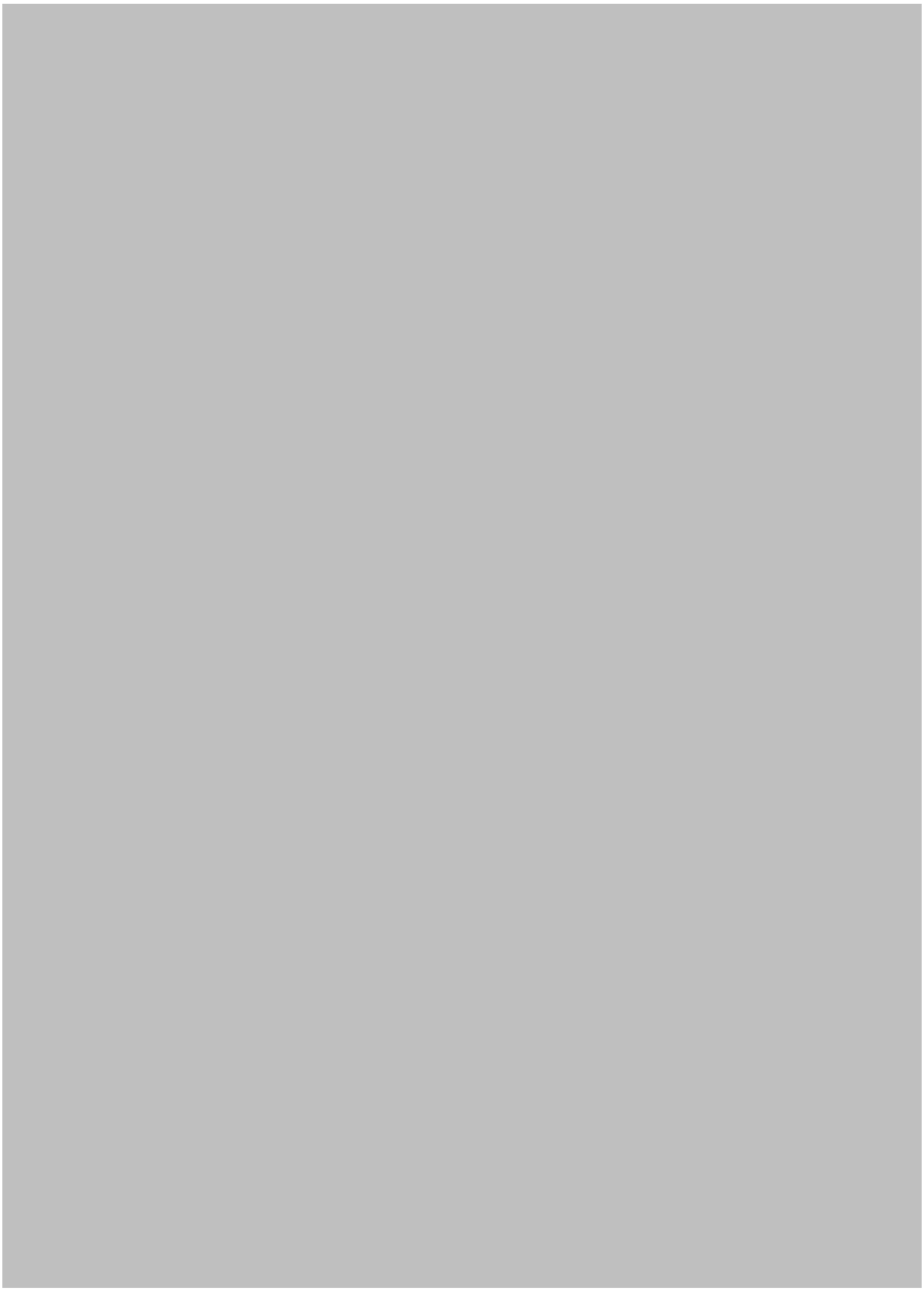


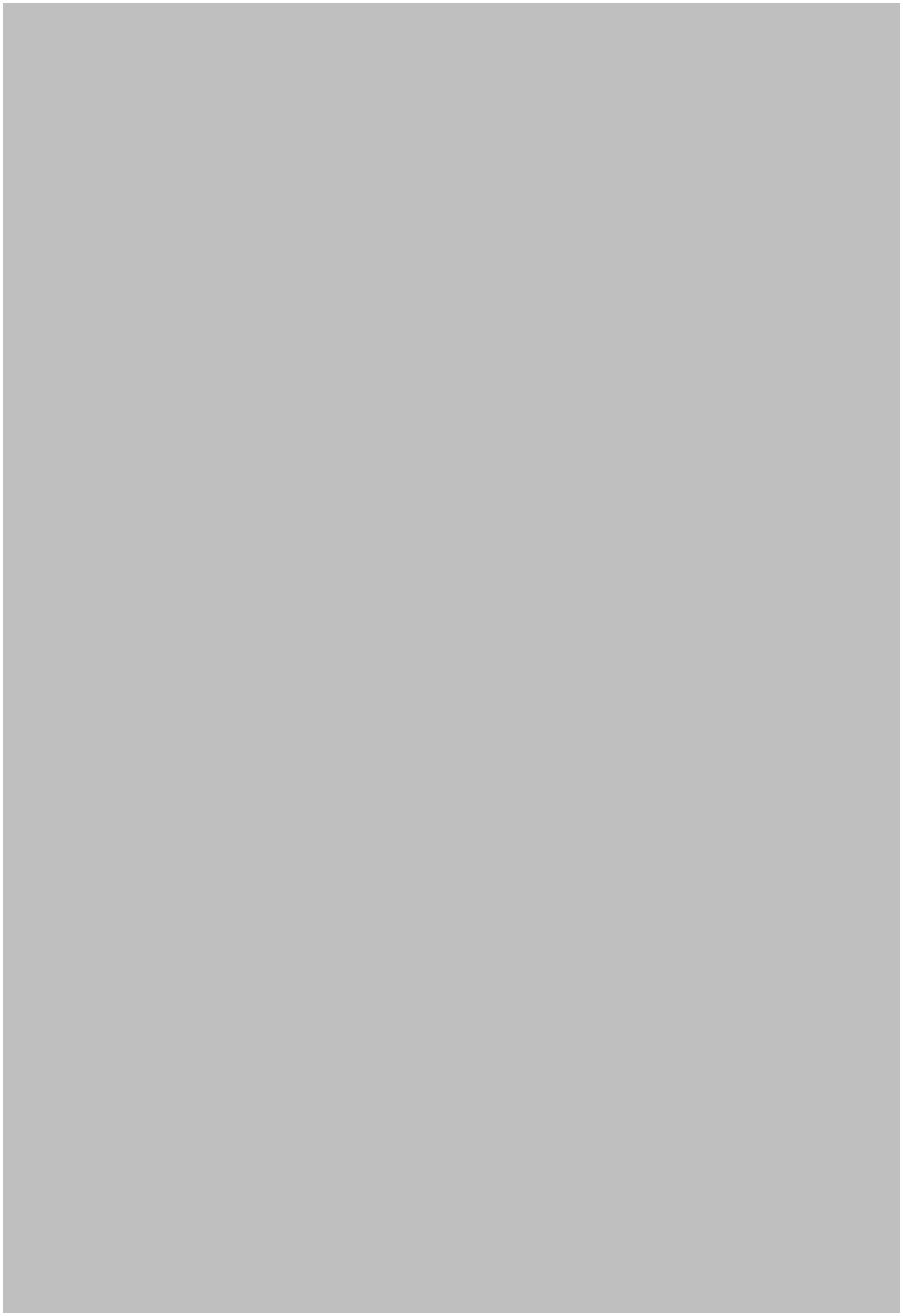












P3M:14:451-460	*I: You mentioned this would be quite hard, but I'm going to ask it anyway. Erm, what do you think life would be like if you didn't have the diagnosis?* P3M: I mean, my seven year old, she's been a handful from the minute she was born, she didn't sleep for the first two years, and at times, I mean, she's known in the family as, she's a lovely lovely little girl, a lot of life in her, you know, you have to sort of reign her in a little bit, and I don't know if P3 would be the same. P3, P3's a thinker. You know, she'll go off and sit and write, and stuff like that. Whereas P3S is running about, acting crazy, like I said, a normal seven year old to me. So I don't know, because obviously I don't know what P3 would have been like if she hadn't had the diagnosis.	Personality/ behaviour/ change	pos and neg	_P3S was always active/ P3 is more thoughtful _Would P3 be more like P3S if she didn't have the disorder?	_Unsure whether the disorder has changed P3's personality	_Uncertainty/ difference
P3M:14:460-468	P3M: Erm, but even just being able to put her in, I say ordinary clothes that kids her age wear because, her classmates, they wear jeans. I cannot get a pair of jeans to fit P3, and we've spent a lot of money buying clothes and having them altered so she can have a pair of jeans. Even just finding her a pair of boots recently was a nightmare because she was a very wide fit, and she's got a high instep, and just something as simple as that, it can take you at least two weeks looking in the shops just to find something that fits her property, just because of the size she is and everything. Erm, so sometimes, sometimes she gets upset.	Physical change/ being different/ impact	neg	_Difficult to buy clothes for P3 that fit her properly _This upsets P3	_P3's size is problematic for her	_Frustration/ expensive/ time-consuming/ a nightmare/ upsetting
P3M:14:469-470	P3M: She's not dressing the same way, as her other friends are, but I try to get her dressed the same way as much as possible.	Being different/ physical changes/ coping	neg	_P3 has to dress differerntly to her friends _P3M tries to get similar clothes	_P3's size is problematic for her	_Different/ frustration
P3M:14-15:470-475	P3M: But I don't, I don't honestly know, I don't know what it would have been like…. You could say maybe that life would have been a bit easier, but maybe it wouldn't because, I've said, I've had to look at different ways of disciplining my seven year old, because the way I was doing it just wasn't getting through to her at one point	Change/ impact/ coping	NA	_Life may have been easier if P3 didn't have the disorder, but it may not	_May have had different problems	_Uncertainty/ pros and cons