INFLUENCE OF FAMILY DISRUPTION/FATHER ABSENCE ON DAUGHTERS' AGE AT MENARCHE: A GENETICALLY AND ENVIRONMENTALLY CONTROLLED SIBLING COMPARISON STUDY

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by

Jacqueline M. Tither

Department of Psychology

University of Canterbury

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Abstract

Previous research has demonstrated that exposure to family disruption/father absence (due to parental relationship dissolution) is a significant risk factor for early pubertal development in daughters. Moreover, the earlier in life that this exposure occurs, the greater the risk of these outcomes for girls. Two opposing classes of explanation have been proposed for this reliable finding. First, evolutionary-based developmental experience models have proposed that father absence may actually cause early pubertal development in daughters through mechanisms that remain to be elucidated. Second, this association may arise from either a genetic or a family-wide environmental confound. To discriminate between these two competing classes of explanation (i.e., *causal* vs. *noncausal*), a retrospective study employing a community sample of full biological sister pairs was conducted in New Zealand. This study examined menarchael age in (a) a primary group comprising age-discrepant biologically disrupted/father absent sister pairs (n = 68), and (b) a matched control group comprising age-discrepant biologically intact/father present sister pairs (n = 93). According to the causation model, if greater exposure to family disruption/father absence causes earlier pubertal development in girls, then in families in which (a) full biological sisters are discrepant in age, and (b) the younger sister has experienced more prolonged father absence than has her older sister, younger sisters should be at greater risk for earlier pubertal development. By contrast, if a genetic or family-wide environmental confound explains this association, full biological sisters should

not systematically differ in pubertal timing as a function of birth order, even if they have experienced different amounts of father absence.

The unique contribution of the current study to this area of inquiry is its employment of a differential sibling exposure design to test the explanatory value of the two opposing classes of explanation (i.e., causal versus noncausal). This genetically and environmentally controlled sibling design was utilised (a) to test the central hypothesis that the birth order/age discrepancy (older versus younger) between sisters would interact with family type (biologically disrupted vs. biologically intact) to predict the size of sibling differences in menarcheal age, and (b) to test for potential moderating effects of paternal dysfunction. Consistent with evolutionary causal models, the current sibling comparison study revealed that within biologically disrupted/father-absent families, younger sisters (who had more prolonged exposure to father absence) had earlier menarcheal ages than did their older sisters. The current study was therefore not only able to distinguish between the two competing classes of explanations, but its findings plausibly supported a *causal* rather than a *noncausal* explanation for the association between father absence and earlier pubertal timing in girls. Moreover, it revealed that this association is more nuanced than previously thought, because the accelerating effect of family disruption/father absence on daughters' menarcheal timing was moderated by fathers' functioning in the family.

The current study has eight important limitations that can be used to direct future research. These limitations are detailed along with proffered suggestions (where applicable) for addressing them in future studies. Possible mediating mechanisms for the earlier menarcheal timing found in daughters

from biologically disrupted/father absent families are also proposed. Finally, the implications of the current study's findings for both parents and daughters in biologically disrupted/father absent families are discussed.

Chapter One: Introduction

This thesis—which represents my contribution to the burgeoning body of research examining the effects of early rearing experiences on pubertal development—has its inception in a small footnote appended to an article written by Jerome Barkow (1984) entitled "The distance between genes and culture". In it, he states:

> Draper and Harpending argue for developmental pathways strongly affecting behavior, but do not discuss whether physique might be affected. A prediction that father-absent boys should be more mesomorphic than father-present ones would be consistent with their framework and would also permit it to be tested. For girls the prediction would involve not differences in physique but in age at menarche, which would presumably be earlier for father-absent girls. No data are available to test either of these hypotheses, but the present author is now planning an appropriate study. (Barkow, 1984, p. 378)

Although at the time of Barkow's (1984) writing he duly noted the lack of available data to test his central hypothesis—that is, that developmental pathways would not only affect human behaviour but also physique—this is certainly not the case today. Indeed, in the three decades following Barkow's *a priori* prediction that father-absent girls would exhibit earlier menarcheal timing than would other girls, an extensive body of research has been conducted to test this very idea. Moreover, while the proposal that father absence may actually causally influence pubertal timing is still somewhat contentious, a growing number of theorists would now concur with Barkow's percipient hunch, myself included.

When, as an honours student in Psychology, I was first introduced to the proposition that father absence actually causes earlier pubertal timing in girls, I was not only intrigued but also determined to discover more about the veracity of this proposal. Therefore, when I embarked upon this thesis, I started out by first studying the variation found in pubertal timing in humans and familiarising myself with the key factors that influence it. Consequently, starting this thesis with a discussion about the important features of human pubertal development—especially that of females'—both in terms of its stages *and* of the variation in pubertal timing that manifests within individuals, seems pertinent.

Adolescence versus puberty

In the human life course, the developmental period that bridges childhood and adulthood is undoubtedly one of the most dramatic. This time period is often interchangeably referred to as *adolescence* or *puberty*, but strictly speaking, these terms are not synonymous (Sisk & Zehr, 2005). Rather, there are important differences between these two terms that need to be considered (Dorn, Dahl, Woodward, & Biro, 2006). To elaborate, *adolescence,* (derived from the Latin *adolescere* which means "to grow up" [Orsman, 2001, p. 14]) is a broad term that encompasses all of the dramatic physical and psychosocial changes and experiences that characterise the teenage years (Steingraber, 2007). However, the timeframe during which adolescence occurs varies in terms of how it is defined: some authors argue that it encompasses the second decade of life, while others claim that it extends right through to the early twenties (Dorn et al., 2006). Although the term *adolescence* has a number of definitions, for the purpose of this discussion it can be usefully defined as "the interval between childhood and the assumption of adult roles and responsibilities, a broad interval of maturation that

encompasses physical, mental, and emotional development, as well as coincident cognitive changes and changes in social roles" (Dorn et al., 2006, p. 33). Hence, according to this definition, *adolescence* encompasses all facets of maturation: the physical, social, cognitive and emotional. *Puberty,* however, derived from the Latin *puber* meaning adulthood (Orsman, 2001, p. 922), is a more specific term that refers to the profound physical changes that serve to transform the juvenile body into its reproductively capable, fertile adult form (Pinyerd & Zipf, 2005). While the majority of these physical changes occur during the adolescent period, some do not. For example, some of puberty's biologic processes such as *adrenarche* actually begin during the first decade of life, often between the ages of 6 to 8 years.

During adolescence, the anatomy of the human brain undergoes dramatic and widespread restructuring (Casey, Getz, & Galvan, 2008; Grumbach & Styne, 2003; Konrad, Firk, & Uhlhaas, 2013; Lenroot & Giedd, 2006; Paus, 2005; Sisk & Zehr, 2005; Steingraber, 2007; Styne & Grumbach, 2011). Some of this change is elaborative (i.e., cell numbers grow, dendrites become more complex, and axons sprout), while some of it is retrograde (i.e., some cells die and some synapses are pruned) (Pinyerd & Zipf, 2005; Sisk & Zehr, 2005; Steingraber, 2007). There is a linear increase in white matter, along with an initial increase—followed by a commensurate decrease—in grey matter, suggestive of synaptic pruning (Sisk & Zehr, 2005; Steingraber, 2007). Measurable changes in brain wave patterns also occur (Dorn & Rotenstein, 2004; Grumbach & Styne, 2003; Sisk & Zehr, 2005; Styne & Grumbach, 2011), which are indicative of increasing brain function complexity, especially over the frontal associative cortex (Anokhin, Birbaumer, Lutzenberger, Nikolaev, & Vogel, 1996). This increase in brain complexity is not surprising, given that adolescence is a time when the ability to solve complex problems in an adult fashion first emerges (Grumbach & Styne, 2003; Styne &

Grumbach, 2011). As Sisk and Zehr (2005) note, "a biological hallmark of adolescence is the remarkable remodelling of cortical and limbic circuits, which leads to the acquisition of adult cognition, decision making strategies and social behaviors" (p. 163).

However, this increase in brain complexity does come at a price: it is accompanied by a reduction in brain plasticity and cognitive flexibility (Steingraber, 2007). This is exemplified by a dramatic loss in ability to assimilate new and complex skills after puberty, such as learning to ride a bicycle, play a musical instrument, or speak a second language without an accent (Grumbach & Styne, 2003; Steingraber, 2007; Yun, Bazar, & Lee, 2004). Moreover, adolescence also encompasses significant psychological changes, such as the need for high levels of stimulation, and the acceleration of strong emotions.

It has been noted, however, that puberty is not "a de novo event" (Grumbach, 2002, p. 3). Rather, it is part of a continuum of events involving the development of the hypothalamic-pituitary-gonadal (HPG) axis and the endocrine system that starts when the individual is in the womb and extends (somewhat disjointedly) through to adulthood (Blondell, Foster, & Dave, 1999; Grumbach, 2002). Puberty actually makes two temporally distinct appearances in the life course of a human being (Steingraber, 2007). Its hormonal circuitry is first activated in utero but a few months after birth this circuitry becomes nascent (Steingraber, 2007). The function of this "juvenile puberty" is unknown, but it has been suggested that it may serve to prepare the endocrine system for its reactivation during adolescence (Steingraber, 2007).

Puberty

The second appearance of puberty, during adolescence, arises from the awakening of a complex neuroendocrine system that is not yet well understood (i.e., its

primary mechanism has not yet been elucidated) (Terasawa, 2001). However, what is well-known is that both the tempo and timing of pubertal maturation are under the control of two separate signalling pathways: the hypothalamic-pituitary-adrenal axis and the hypothalamic-pituitary-gonadal (HPG) axis (Grumbach, 2002; Grumbach & Styne, 2003; Styne & Grumbach, 2011). From an endocrinal viewpoint, the physical changes associated with puberty stem from two temporally overlapping processes: *adrenarche* and *gonadarche* (Dorn & Rotenstein, 2004; Styne & Grumbach, 2011). While these pubertal processes may appear to occur simultaneously and to be caused by the same phenomenon, they are, in fact, distinct and largely independent pubertal components (Counts et al., 1997; B. J. Ellis, 2004; Pinyerd & Zipf, 2005; Styne & Grumbach, 2011). Evidence for this independence includes the fact that functional maturation of the HPA axis generally starts during the least active phase of the HPG axis, and the fact that children with atypical pubertal development may only experience one or other of these processes (Del Giudice et al., 2009). For example, many girls with Addison's disease will experience gonadal puberty but not adrenarche; conversely, girls with Turner syndrome may experience normal adrenarche but not undergo complete gonadal puberty (Del Giudice et al., 2009).

Recent research, however, suggests that adrenarche and gonadarche may not be entirely independent processes. For example, although environmental effects may uniquely affect the timing of adrenarche and gonadarche, recent genetic research suggests that the timing of these processes may be largely regulated by the same set of genes (Van den Berg et al., 2006). Moreover, early adrenarche may also predict early gonadarche, especially in girls. Hence, while adrenarche and gonadarche may be considered to be distinct components of pubertal maturation, they each play their part

in the increased secretion of the sex steroid precursors and the sex steroids that characterise puberty (Dorn & Rotenstein, 2004; B. J. Ellis, 2004; Grumbach, 2002).

Adrenarche

In normal pubertal development, the earliest phase is *adrenarche*, a process with enigmatic origins that precedes *gonadarche* by approximately 2 years (Grumbach, 2002; Grumbach & Styne, 2003; Styne & Grumbach, 2011). Adrenarche involves the amplified secretion of adrenal androgens as a result of the functional maturation of the HPA axis and generally occurs somewhere between the ages of 6 and 9 years in both sexes (Auchus & Rainey, 2004; Del Giudice et al., 2009; Dorn & Chrousos, 1997; Styne & Grumbach, 2011). This process has been described as the "awakening of the adrenal glands" (Dorn & Chrousos, 1997, p. 25), and is characterised by both structural and hormonal alterations (Del Giudice et al., 2009). During adrenarche, the adrenal cortex undergoes expansion in both size and mass, but one of its three regions—the *zonas reticularis*—which is the site of androgen synthesis, especially enlarges (Del Giudice et al., 2009; Styne & Grumbach, 2011). These changes result in an increasing concentration of adrenal androgens in the body, especially dehydroepiandrosterone (DHEA) and its sulphate (DHEAS), and also androstenedione. Bodily concentrations of these adrenal androgens gradually increase throughout the first two decades of life (with higher levels found in males than females) until they peak in the third decade and then start to decrease (a process known as *adrenopause)* (Del Giudice et al., 2009; Dorn & Chrousos, 1997; Dorn & Rotenstein, 2004).

In early adrenarche, these increased androgen concentrations generally have no external manifestations, but in later adrenarche, they are associated with a number of visible bodily changes. These include pubic and axillary hair growth, a skeletal growth

spurt, changes in body odour, and oilier hair and skin (sometimes accompanied by acne) (Dorn & Chrousos, 1997). Aside from these external manifestations, animal studies suggest that increased androgen concentrations may also have direct effects on the central nervous system, possibly influencing neural plasticity, memory, and behaviour (Del Giudice et al., 2009).

However, the exact role of adrenarche in pubertal development requires further explanation, given that it neither initiates puberty (in a physiological sense), nor is it related to sexual development. Moreover, its timing varies quite considerably, it can occur prematurely in either sex, and its triggering mechanism remains unknown (Del Giudice et al., 2009).

Gonadarche

Gonadarche (or *hypothalamic-pituitary-gonadal* maturation) involves the functional maturation of the testes and ovaries. It is activated by the action of macroneurons of the hypothalamus that secrete gonadotropin-releasing hormone (GnRH) (Blondell et al., 1999). Prior to the onset of puberty, the hypothalamus gonadostat is extremely sensitive to minute concentrations of androgens and estrogens (i.e., sex steroids) (Pinyerd & Zipf, 2005). Consequently, GnRH secretion is suppressed, which in turn blocks the pituitary gland from secreting luteinizing hormone (LH) and follicle-stimulating hormone (FSH) (Pinyerd & Zipf, 2005). However, at the end of childhood, the suppressive effects of androgens and estrogens on the hypothalamus are alleviated, which results in increased secretion of GnRH, LH, and FSH (Pinyerd & Zipf, 2005).

Normal pubertal development is therefore characterised by dramatic hormonal changes in the body originating from the hypothalamus (Pinyerd & Zipf, 2005). During

childhood, the hypothalamus secretes small amounts of GnRH (Pinyerd & Zipf, 2005). However, at the onset of puberty, secretion of this hormone increases dramatically (Pinyerd & Zipf, 2005). GnRH primarily functions to regulate the growth, development, and function of the gonads (i.e., the ovaries in girls; the testes in boys) (Pinyerd & Zipf, 2005). It also prompts the pituitary gland to start secreting follicle-stimulating hormone (FSH) and luteinizing hormone (LH) (Pinyerd & Zipf, 2005). These hormones (also known as gonadotropins) have sex-specific effects on maturational processes. In boys, LH stimulates production of testosterone and FSH prompts production of sperm (Pinyerd & Zipf, 2005). In girls, FSH stimulates the development and maturation of ovarian follicles, and both LH and FSH are essential for ovulation (Pinyerd & Zipf, 2005).

In sum, pubertal maturation starts with increased GnRH pulses from the hypothalamus, which in turn prompt the secretion of sex steroids. Ongoing exposure to these steroids then produces the secondary sexual characteristics that characterise the reproductively capable adult phenotype. This process produces the notable physical changes that occur during this *second* pubertal period, including the adolescent growth spurt, the development of primary sex organs (genitals and gonads), and the appearance of secondary sexual characteristics (e.g., pubic and axillary hair in both sexes; facial [and other body] hair and an enlarged larynx [that results in a deeper voice in boys]; and breasts in girls) (Grumbach & Styne, 2003; Styne & Grumbach, 2011).

Physical manifestations of puberty

In boys, the first physical change associated with adolescent puberty is increased testicular volume (above 3 ml, consistent with Tanner G2 Stage) (Parent et al., 2003; Terasawa & Fernandez, 2001), followed by pubic hair growth, and increased penis size (Parent et al., 2003; Terasawa & Fernandez, 2001). This testicular volume increase

tends to occur between the ages of 9.5 and 13.5 years (average 12 years) and results from reactivation of the HPG axis. This reactivation enhances the sensitivity of the tissues of the testes, which in turn causes T concentrations to rise above prepubertal levels (Dorn et al., 2006). This results in increased thickness of the seminiferous tubular lining, formation of a lumen, and differentiation and growth of Sertoli and Leydig cells, resulting in a proliferation of Leydig cells (Terasawa & Fernandez, 2001). However, because this increase in testicular volume can only be ascertained by conducting a thorough evaluation in the context of a physical examination, it has been argued that when taking medical histories, there is no reliable recallable event that can be used to time pubertal maturation in boys.

Puberty in girls involves a series of changes that usually starts towards the end of the first decade of life and unfolds over a period of approximately 4.5 years. In girls, puberty starts with a period of accelerated growth. This results from the stimulating effect on epiphyseal growth due to an increase in estrogen levels in early puberty (Terasawa & Fernandez, 2001). However, this initial pubertal marker in girls is difficult to determine as it necessitates the collection of accurate height measurements several times per year. A second signalling pathway stimulates the adrenal gland to start producing androgens, a process which causes the growth of pubic hair (*pubarche*). In the temporal process of pubertal development, *thelarche* and *pubarche* are relatively early events, while menarche is a relatively late-stage event (Steingraber, 2007).

In sum, the various stages of puberty are well understood. In term of its physical manifestations, normal pubertal development (as identified by Tanner, 1962; Marshall & Tanner, 1969, 1970) starts in boys with enlargement of the testicles, followed by the appearance of pubic and axillary hair (i.e., pubarche), enlargement of the penis, and, finally, *spermarche* (i.e., initial development of sperm in testicles). Skeletal and muscular

development are relatively late features of puberty in males. In girls, puberty's physical manifestations start with the development of breast buds (i.e., *thelarche*) and skeletal growth, followed by the appearance of both pubic and axillary hair (i.e., *pubarche*), and, finally, *menarche* (i.e., the first menstrual period) is attained. However, of all the pubertal stages in girls, menarche is often viewed as being the most dramatic. It has been extensively studied, especially in terms of variation in its timing among individuals, and a number of factors that appear to influence this variation are discussed in more detail later in this chapter.

Interestingly, however, the sequence of pubertal events, although not exactly the same for each individual, shows far less variability (in both sexes) than do the timing and tempo of such events (Tanner, 1962). Notably, while the different stages of puberty have not changed over time, over the last few centuries there have been dramatic alterations in both its timing and tempo. Therefore, it is the variation evident among individuals during this "adolescent puberty"—this profound developmental transition that for the majority culminates in the ability to sexually reproduce (Pinyerd & Zipf, 2005)—that will be discussed over the next few pages.

In girls, puberty normally starts between the ages of 8 and 14 years, and in boys, between the ages of 9 and 14 years (Blondell et al., 1999). However, both pubertal timing and pubertal tempo show considerable variability. For example, Michelle Surbey (1990), in her sample of Canadian girls, found that menarcheal age ranged from 9 through to 18.5 years. Therefore, a rather unusual feature of human sexual maturation is its variation (in both timing and tempo) across individuals. For example, it has been noted that chronological age at onset of puberty can vary by up to 4-5 years among normal individuals experiencing comparable life conditions (Parent et al., 2003; Tanner, 1962).

Secular trends in pubertal timing have also been noted. For example, from the mid-19th century until the mid-20th century, the average age at menarche in European girls dropped rapidly and steadily from 17 to 13 years. Likewise, a similar trend has been noted in the United States, although it was not until the beginning of the 20th century that useful data on this became available (Marshall & Tanner, 1968; Tanner & Eveleth, 1975; Wyshak & Frisch, 1982).

Pubertal status versus pubertal timing

Because two distinct aspects of pubertal maturation—*pubertal status* and *pubertal timing*—are often confounded, they require careful definition (Steinberg, 1987). *Pubertal status* refers to a stage or level of pubertal development at a given point in time (i.e., it is an absolute variable), whereas *pubertal timing* is relative to expected pubertal development at either a specific chronological age or within a defined reference group (i.e., it is a relative variable). *Pubertal timing*, therefore, is an individual differences variable and is often categorised as being early, on time, or late, relative to a specific reference group, commonly the individual's same-age peers (Alsaker & Flammer, 2006; Marceau, Ram, Houts, Grimm, & Susman, 2011; Shirtcliff, Dahl, & Pollak, 2009; Susman et al., 2010).

Sex differences

Although "off-time" pubertal timing seems to present more problems for females than it does males, its effects on both sexes have been investigated. Consequently, since the 1930s, the effects of both early and late pubertal timing have been well-documented. Specifically, classic early studies found that the psychological consequences of early pubertal timing varied by sex: generally early maturation tended to be relatively positive for boys (especially with regard to their social development)

whereas for girls, the reverse was true (e.g., Faust, 1969; Jones, Bayley, & Jones, 1948; Stolz & Stolz, 1944). However, results have been somewhat inconsistent across studies (see Susman, Dorn, & Schiefelbein, 2003).

The majority of recent pubertal timing research has focussed on variation in girls; male pubertal timing has received far less research attention (although one recent review of pubertal timing and internalising problems also included male participants [see Negriff, Susman, & Trickett, 2011]) There are both theoretical and empirical reasons for this inequity (see B. J. Ellis, 2004). At an empirical level, menarche provides a clear, easily quantified, and relatively memorable marker of female pubertal timing, whereas some authors would argue that no such analogous marker of male pubertal timing exists (e.g., see B. J. Ellis, 2004). However, this view has recently been challenged by researchers utilising timing of onset of ejaculations (i.e., *oigarche*) as an analogous marker of pubertal timing in boys (e.g., Kaltiala-Heino, Koivisto, Marttunen, & Frojd, 2011).

Variation in pubertal timing

There are at least four possible explanations for the wide range of variation in pubertal development found both within and across all human populations: (a) it is random (i.e., nonadaptive), (b) it arises from alternative reproductive strategies, (c) it arises from conditional reproductive strategies, or (d) it arises from a combination of alternative and conditional reproductive strategies (see Belsky, 2012; Surbey, 1998).

The first possibility is that the variation found in human pubertal timing is random; that is, it arises from either *nonadaptive phenotypic plasticity* (Stearns, 1992), or genetic variation that does not confer any selection advantage (Surbey, 1998). Specifically, because numerous traits in any given population will have values that are

normally distributed, observing random variation around the mean for a particular trait is not unexpected (Surbey, 1998). Therefore, even though such variation may well be predictable (at least to some extent) from environmental conditions, and it may even be heritable, it is not necessarily adaptive. To illustrate this point, Surbey (1998) describes how some reproductive phenomena show significant seasonal variation. Timing of the first menstrual cycle in humans, for example, is not randomly distributed across the calendar year; rather, it peaks in particular months (Surbey, 1998). However, this particular phenomena, albeit interesting, does not appear to confer any adaptive advantage to humans, and is in all likelihood a vestige of seasonal breeding patterns still evident in other mammals (Surbey, 1998). Moreover, while particular genes may predispose an individual to earlier or later pubertal timing (or, for that matter, curly or straight hair), and while these genetic predispositions may, in fact, be heritable, their effects on the resulting phenotype in terms of its reproductive fitness may be negligible. Therefore, although menarcheal timing in humans exhibits wide variation, appears to be heritable, and the correlation between mothers' and daughters' age at menarche typically approximates $r = .30$ (Damon, Damon, Reed, & Valadian, 1969; Johnston, 1964; Surbey, 1990, 1998), this does not necessarily mean that it is adaptive. However, whether this variation in pubertal timing is adaptive or not, a heritability score that is higher than zero will still produce behaviour–genetic effects involving transgenerational transmission of both developmental rate and correlated behavioural attributes (Surbey, 1998).

A second possibility is that the existence of *alternative life history strategies*—that is, adaptive genotypic differences that produce distinctive phenotypes—constitutes a second source of variation in pubertal timing (Surbey, 1998). Therefore, in this case, variation in pubertal timing reflects heritable predispositions (Surbey, 1998). For

example, selection operates on some populations to produce morphs that display alternative reproductive strategies, including differential pubertal timing and behavioural reproductive strategies (Surbey, 1998). Such alternative strategies are produced by *diversifying selection*, which divides previously continuous distributions of values on specific life history traits into bi- or even tri-modal curves (Surbey, 1998). Strictly speaking, however, because bi- or tri-modal distributions are not typically found within human populations for life history traits (e.g., distributions for markers of pubertal timing such as menarcheal age are continuous), the use of the term "alternative life history strategies" (in terms of how it has been traditionally defined) to describe the patterns of variation found in human pubertal timing is somewhat incongruent (Surbey, 1998). Moreover, when it is adopted to describe the between-person variation found in life history traits, it has been the subject of some debate (Surbey, 1998). For example, Stearns (1976, 1980) proposes that this term is not applicable to the within-species variation found in life history traits, and moreover, that life history tactics are not the properties of individuals, but rather, of populations (Surbey, 1998).

Third, it is possible that the observed phenotypic variation in pubertal timing is the product of *conditional strategies* that have evolved over time (Surbey, 1998). If this were the case, variation in pubertal timing would be arising from *adaptive developmental plasticity* in response to specific environmental cues, and therefore environmental effects—not genotypic variation—would account for the observed phenotypic variation found in pubertal timing. To illustrate how a conditional strategy would work in a given context, Surbey (1998) describes how, in order to improve their chances of obtaining a mate, males in a number of insect species will select one strategy—out of a ubiquitous range of possible mating strategies available to all males

of that species—to fit a particular environmental circumstance (e.g., Thornhill, 1980). Therefore, it is the ability to be able to correctly select the optimal strategy in a given environment, rather than the strategy adopted per se, that is both genetically coded and adaptive (Surbey, 1998). The employment of conditional strategies is to be expected in environments where individuals are forced to contend with changing sets of circumstances during development (Surbey, 1998, p. 74). Hence, under this view, the developmental trajectory of any given individual might be interpreted as the chronology of conditional strategies that he or she has selected over time (Surbey, 1998).

Finally, despite the fact that these alternative and conditional reproductive strategies perspectives differ in terms of what they each emphasise (i.e., the former emphasises genotypic differences whereas the latter emphasises environmentally-triggered processes), they are not necessarily mutually exclusive (Belsky, 2012). Instead, it may be useful to adopt both of these perspectives in order to best account for the variation found in human pubertal development (i.e., a hybrid theoretical formulation) (Belsky, 2012). However, the key point of difference between these two types of strategies may well be their respective susceptibility to environmental influences. That is, alternative reproductive strategies may be far more vulnerable to contextual regulation than are conditional reproductive strategies (Belsky, 2012). Two recent studies that provide support for this differential susceptibility perspective indicate that both physiological reactivity (Ellis, Shirtcliff, Boyce, Deardorff, & Essex, 2011) and an estrogen-receptor gene (Manuck, Craig, Flory, Halder, & Ferrell, 2011) can be utilised to determine the extent to which rearing experiences regulate subsequent pubertal development in females (Belsky, 2012).

Measuring puberty

Pubertal development can be measured using various objective and/or subjective measures, which are often interchangeably referred to as measures of pubertal development, or measures of pubertal status (Dorn et al., 2006). While the utilisation of some of these measures is more appropriate during certain pubertal phases than others (and some are sex-specific) each of them can be used in specific ways to describe particular pubertal processes (comprehensively reviewed in Dorn et al., 2006).

Objective measures of pubertal development

1. Physical examination using Tanner criteria. A five-stage clinical system for measuring pubertal development in both boys and girls (adapted from Reynolds & Wines, 1951) was developed in the early 1960s by British paediatricians William Marshall and James Tanner (1969, 1970). Traditionally Tanner staging has been viewed as the "gold standard" against which all other methods have been judged (Dorn et al., 2006). The Tanner system involves categorising pubertal changes such as breast development (both size and contour) in girls, genital development (including testicular volume) in boys, and pubic hair distribution in both sexes, into stages (Blondell et al., 1999; Dorn et al., 2006; Marshall & Tanner, 1969, 1970). For example, at Stage 1 of the Tanner system, there is no evidence of any external manifestations of gonadal activation, whereas by Stage 5, full maturation has been attained (i.e., all requisite physical signs indicative of this level of maturation are present) (Dorn et al., 2006). More recently, a number of modifications to Tanner staging for boys have been made (see Biro, Lucky, Huster, & Morrison, 1995).

2. Pubertal maturation by areola development. For girls, measurement of change in areola diameter comprises another method of measuring pubertal maturation by physical examination. This is considered by some to be a more accurate measure of pubertal maturation in girls than other methods, because accuracy of measurement is not affected by adipose tissue (Dorn et al., 2006). However, although areola measurement may be a more objective measure, fewer studies have used this methodology (Dorn et al., 2006). Therefore, with the exception of one author who cites norms (Grumbach, 2002), little normative data of areola measurements is available. It is most useful as a measure of pubertal maturation for girls during adrenarche and gonadarche (similar to Tanner staging), and it may be especially relevant for use in longitudinal studies (Dorn et al., 2006).

3. Pubertal maturation by testicular volume. In several research studies and in clinical settings, pubertal development in boys has been categorised using testicular volume measurement (Dorn et al., 2006). This type of measurement has most commonly been obtained through the use of a Prader orchidometer. This apparatus consists of a string of 12 or 14 numbered wooden or plastic beads that increase in size from about 1 to 25 millilitres, or from 1 to 35 millilitres, respectively. This measurement procedure is simple: the string of beads is compared with the patient's testicles, and the volume is then read off the bead that most closely matches each testis in size. Testicular volume measurements of 1–3 ml are considered to be prepubertal, those of 4-11 ml are considered to be pubertal, and those of 12–25 ml are considered to be adult. In other studies, callipers, rather than the orchidometer, have been used to measure the length and width of the testes (Dorn et al., 2006).

Most paediatric endocrinologists would argue that testicular volume measurement is superior to visual judgements of developmental changes in the scrotum

or in the length and width of the penis (Dorn et al., 2006). Thus, it is a more precise measure of the onset of puberty in boys that may be especially useful for determining prepubertal versus peripubertal development in research studies. Because increased testicular volume is generally the first visible manifestation of puberty in boys, its careful measurement is the optimal way to determine the shift from being prepubertal (i.e., Stage 1: within the adrenarche phase) to Stage 2 and above (i.e., the beginning of the gonadarche phase). Because increased testicular volume is usually only visible when health care providers use palpation, self- or parent-report may underestimate pubertal development in this early phase. However, when it is conducted accurately, testicular volume measurement is a particularly useful method for determining the actual onset of pubertal processes (Dorn et al., 1996).

4. Hormone concentrations. In the 1980s, the first studies measuring serum hormone concentrations (i.e., adrenal androgens, gonadal steroids, and gonadotropins) and emphasising a biopsychosocial perspective were conducted (Brooks-Gunn & Graber, 1994; Brooks-Gunn & Warren, 1989; Halpern, Udry, Campbell, & Suchindran, 1993; Nottelmann, Susman, Dorn et al., 1987; Nottelmann, Susman, Inoff-Germain et al., 1987; Susman, Inoff-Germain, Nottelmann, & Loriaux, 1987; Susman et al., 1985; Susman, Nottelmann, Inoff-Germain, & Dorn, 1987; Udry, Billy, Morris, Groff, & Raj, 1985; Udry & Talbert, 1988). However, the earliest stage of adrenarche was excluded from these studies (the youngest participant was already 9 years old), and most of these studies also simultaneously used physical examination to measure pubertal stage. It is widely believed that it is not possible to categorise an individual into a particular pubertal stage based on a single hormone concentration, especially given that tables indicating ranges of hormones by stage show considerable variability and overlap, both within and between pubertal stages in boy and girls. New methodologies for measuring

hormonal concentration have recently been developed, including the testing of urine, blood spot and saliva, but each of these methods has its advantages and limitations (reviewed in Dorn et al., 2006).

5. Gonadal ultrasound. It is possible to use ultrasound to examine and measure ovarian volume. However, norms of these measures have not yet been determined, and the link between ovarian volume and other pubertal markers has not yet been elucidated. Although a recent study reported significant differences in ovarian volume between prepubertal and peripubertal girls (Herter, Golendziner, Flores, Becker, & Spritzer, 2002), and it is positively correlated with both pubertal stage and chronological age, ovarian volume has not yet been used to categorise individuals into pubertal stages. It can be more precisely measured using a transvaginal probe, but this more costly and invasive method is unlikely to be either feasible or necessary in adolescents (Dorn et al., 2006). Moreover, the lack of norms across puberty and the resultant difficulties in interpretation mean that in most studies, the use of measures of ovarian volume by ultrasound does not constitute a particularly useful marker of pubertal development (Dorn et al., 2006).

Testicular volume has also been determined using ultrasound, but rather than specifically focusing on adolescents, the studies reported in the literature have mainly focused on comparing methods of determining testicular volume in adults. One study that compared the measurement of testicular volume using callipers, the orchidometer, and ultrasound found the latter to be the superior method (Fuse, Takahare, Ishii, Sumiya, & Shimazaki, 1990), but measurement using callipers was found to be inaccurate. However, another study that compared the use of ultrasound with the use of an orchidometer found that the former method underestimated testicular size (Dorn et al., 2006).

6. Age at Spermarche. Research on adolescent males has examined the age at *spermarche*, also known as the onset of spermatozoa production, nocturnal emissions, or *oigarche* (reviewed in Dorn et al., 2006). Studies utilising different sample sizes and measurement methods (e.g., examining morning urine samples versus self-reports) have found that age at spermarche tends to occur between 13–14 years of age, and thus it is a relatively early pubertal event. However, this body of research has revealed considerable variability in the developmental markers that co-occur with spermarche (e.g., testicular volume and stage of pubic hair), thus making it difficult to determine whether it is age or testicular volume that determines sperm production. Another issue with age at spermarche is that it is a dichotomous variable and can only be used to determine if an individual is prepubertal (i.e., not producing sperm) or pubertal (i.e., is producing sperm). Therefore it is an objective measure that cannot be used to make any finer discrimination than this.

Subjective measures of pubertal development

1. Age at menarche. Age at *menarche* (i.e., age at first menstruation), which is usually obtained from either parental or self-reports, is often used to determine pubertal status and is usually obtained via a questionnaire or an interview. Usually, the girl herself is asked "How old were you when you got your first period?" and/or the parent (usually the mother) is asked "How old was your daughter when she got her first period?" The response is then recorded (either in years, or in both years and months). An alternative method is to determine gynaecological age by subtracting the month and year of menarche from the interview date. For example, if an adolescent girl had attained menarche 3 years and 6 months prior to the interview date, her gynaecological

age would then be assessed at 3.5 years. The variable of gynaecologic age is then used in the analyses, rather than the actual age at menarche (reviewed in Dorn et al., 2006).

2. Pubertal stage using parental or self-reports. Parental and self-reports are also used to define pubertal stage (often as a substitute for physical examinations) (Dorn et al., 2006). There are two main methods: (a) the adolescent and the parent are asked to examine images (either line drawings or photographs) of the Tanner stages in order to report on the adolescent's pubertal stage, or (b) the Pubertal Development Scale (Petersen, Crockett, Richards, & Boxer, 1991) is used to report on the adolescent's pubertal stage. This scale does not include images but instead asks questions about changes in the adolescent's height, body hair, and skin. It also includes questions about voice changes and facial hair for boys, and about breast development for girls (reviewed in Dorn et al., 2006).

Implications of "off-time" pubertal timing

Variation in timing of pubertal maturation in adolescent girls has received substantial research attention in Western countries. The most consistent finding to emerge from this extensive area of inquiry is that "off-time" pubertal maturation (i.e., pubertal development that is either earlier or later than that of same-age peers) is associated with a variety of disadvantageous outcomes for girls.

The negative health implications for late-maturing girls include bone density, menstrual, and fertility issues in adulthood. For example, late menarcheal age is a risk factor for osteoporosis, including forearm osteoporosis (Chevalley, Bonjour, Ferrari, & Rizzoli, 2008). Moreover, it is a risk factor for experiencing multiple miscarriages (Bracken, Bryce-Buchanan, Stilten, & Holford, 1985; Martin, Brinton, & Hoover, 1983; Wyshak, 1983;). Later age at menarche is also associated with irregular menstrual

cycles in women. One study, for example, found that in the 10 years following the onset of menstruation, women with later ages at menarche tended to have both longer and more variable menstrual cycles than did those with earlier menarcheal ages (Wallace, Sherman, Bean, Leeper, & Treloar, 1978).

Undoubtedly, however, the most consistent finding to emerge from the extensive body of research that has been conducted in Western societies is that *early* pubertal maturation in girls is associated with an extremely dismaying array of health and psychosocial outcomes, some of which are outlined below.

Early maturing girls have been found to be at heightened risk for long-term unhealthy weight gain (e.g., Ness, 1991; Wellens, Malina, Roche, Chumlea, Guo, & Siervogel, 1992). For example, longitudinal research has revealed a positive association between earlier pubertal maturation and Body Mass Index at age 50 (Hulanicka, Lipowicz, Koziel, & Kowalsko, 2007). It is also associated with heightened risk for high blood pressure in adulthood (see Hulanicka et al., 2007). Moreover, early maturing girls are at heightened risk of having multiple miscarriages, for teenage pregnancy, and for giving birth to low-weight or stillborn neonates (reviewed in B. J. Ellis, 2004).

Early menarche is also associated with both short- and long-term differences in hormonal profiles (Apter, Bolton, Hammond, & Vihko, 1984). That is, young women who experience early menarche tend to have earlier onset of ovulatory cycles than do their later maturing peers (Apter & Vihko, 1983; MacMahon et al., 1982; Henderson, Ross, Judd, Krailo, & Pike, 1985). Specifically, one longitudinal study found that for girls who attained menarche before the age of 12, it took approximately just 1 year for 50% of their menstrual cycles to be ovulatory, whereas for those who had experienced menarche between the ages of 12.0-12.9, and ≥13.0 years, this process took 3 and 4.5 years to occur, respectively (Apter & Vihko, 1983).

Early pubertal maturation is also associated with an increased risk in later life of developing a variety of cancers of the reproductive system, and it is also a significant risk factor for breast cancer (Drife, 1986; Kampert, Whittemore, & Paffenbarger, 1988; Negri et al., 1988; Pike et al., 1981; Ravnihar, MacMahon, & Lindtner, 1971; Valaoras, MacMahon, Trichopoulos, & Polychronopoulou, 1969; Vihko & Apter, 1986). Notably, compared to girls who have later menarcheal ages (≥13 years), those who attain menarche before the age of 12 have double the relative risk of developing breast cancer at a young age. Moreover, menarcheal age is inversely associated with the incidence of endometrial cancer; one study demonstrated that girls who attained menarche at the age of 15 or older were one-third less likely to develop endometrial cancer in later life than those who had attained menarche at the age of 10 or younger (McPherson, Sellers, Potter, Bostick, & Folsom, 1996).

There are also a number of negative psychological implications for girls who experience "off-time" pubertal timing. Specifically, girls who are the earliest in their peer group to attain *thelarche* report feeling greater anxiety and having a more negative self-image than do their later-maturing peers (Steingraber, 2007). Moreover, early-maturing girls are more likely to express dissatisfaction with their height and weight, and to experience disturbances in body image, eating disorders, adjustment disorders, anxiety, depression, and suicide attempts than are their later-maturing peers (comprehensively reviewed in Mendle, Turkheimer, & Emery, 2007; but also see Faust, 1969*;* Martin 1996; Stolz & Stolz, 1944; Susman, Nottleman, Inoff-Germain, Loriaux, & Chrousos, 1985*;* Zuckerman, 2001).

Early pubertal timing is also associated with a range of negative behaviours in girls. For example, early-maturing girls are more likely to engage in antisocial behaviours than their on-time or late-maturing peers (Flannery, Rowe, & Gulley, 1993).

That is, they are at greater risk for delinquency (Stattin & Magnusson, 1990), both independently (Haynie, 2003) and in conjunction with peer effects (Caspi, Lynam, Moffitt, & Silva, 1993). Early pubertal maturation also puts girls at greater risk of experiencing violent physical victimisation (Haynie & Piquero, 2006). Early maturing girls are also more likely to engage in early and frequent tobacco smoking, problematic alcohol consumption, and other forms of substance abuse than are their later-maturing peers (e.g., Dick, Rose, Viken, & Kaprio, 2000; Mezzich, Tarter, Giancola, Lu, Kirisci, & Parks, 1997; Patton et al., 2004; Stattin & Magnusson, 1990; Steingraber, 2007; Tschann et al., 1994; Westling, Andrews, Hampson, & Peterson, 2008; Wichstrom, 2001; Wiesner & Ittel, 2002; Wilson et al., 1994). Finally, early pubertal maturation is also associated with both sexual promiscuity (e.g. Caspi & Moffitt, 1991; Flannery et al., 1993; Stattin & Magnusson, 1990) and risky sexual behaviour in girls, possibly mediated through having older boyfriends (Mezzich et al., 1997).

Given that early pubertal timing is associated with such disadvantageous outcomes for girls (and considering that the above list is not exhaustive) it is essential that all predisposing risk factors (and their nuances) be elucidated through research.

Genetic and environmental influences on pubertal timing

Due to the negative health and psychosocial implications of "off-time" pubertal development in girls, a clear understanding of its aetiology is important. On rare occasions, such variation is the consequence of some underlying medical condition. For example, early pubertal development can be a secondary effect of a central nervous system lesion, while delayed puberty can be a consequence of pituitary pathology, a gonadotropin-releasing hormone (GnRH) deficiency, or undiagnosed chronic conditions such as inflammatory bowel disease (see Palmert & Hirschhorn, 2003). However, while

most patients referred to paediatric endocrinology clinics with variations in pubertal timing are given diagnoses of either idiopathic precocity or constitutionally delayed pubertal development, much is still unknown about the aetiology of these conditions (Palmert & Hirschhorn, 2003).

The factors that regulate pubertal timing within the general nonclinical population *also* require further clarification. Therefore, in order to delineate the biological, psychological, and environmental predictors of pubertal development, various investigators have examined a number of pubertal antecedents. While the relative contributions of many of these factors are not yet clearly understood, what is well established is that although environmental factors clearly play an influential role in the regulation of the neuroendocrine axes that affect pubertal development, their influence is superjacent to significant genetic control (Palmert & Hirschhorn, 2003).

Therefore, the first section of this discussion will describe some of what is known about the underlying genetic influences on variation in pubertal timing, while the latter part will discuss some of the important environmental influences that appear to regulate it.

Genetic influences on variation in pubertal timing

Behavioural genetic studies comparing twins and siblings have established that a substantial proportion of the variance found in pubertal timing is accounted for by genotypic differences (e.g., Farber, 1991; Kaprio, Rimpela, Winter, Viken, Rimpela, & Rose, 1995; Rowe, 2002; Treloar & Martin, 1990). Specifically, twin studies have found that monozygotic twins exhibit greater similarity in pubertal timing than do dizygotic twins (e.g., Kaprio et al., 1995; Pickles et al., 1998; Rowe, 2002; Treloar & Martin, 1990). Consequently, many researchers have concluded that more than half of the variation

found in pubertal timing is genetically controlled (e.g., Kaprio et al., 1995; Loesch, Huggins, Rogucka, Hoang, & Hopper, 1995; Meyer, Eaves, Heath, & Martin, 1991; Palmert & Hirschhorn, 2003; Treloar & Martin, 1990).

In terms of the timing of *menarche*, most research investigating its genetic basis has compared pubertal timing in female relatives (Mendle et al., 2006). For example, studies examining the resemblance in menarcheal timing between (a) sisters (Boas, 1932; Kaprio et al., 1995; Reymert & Jost, 1947); (b) twins (Kaprio et al., 1995); and (c) mothers and daughters (Campbell & Udry, 1995; Graber, Brooks-Gunn, & Warren, 1995) have demonstrated that timing of menarche is substantially heritable. Specifically, age at menarche is more strongly correlated in monozygotic twin pairs than in either dizygotic twin pairs or in sibling pairs (e.g., Doughty & Rodgers, 2000; Kaprio et al., 1995; Meyer et al., 1991; Rowe, 2002; Treloar & Martin, 1990). Consistent with these findings, large studies utilising twin designs in Australia, Europe, and the United States have concluded that approximately 50-80% of the variation found in menarcheal timing is explained by genetic effects (reviewed in B. J. Ellis, 2004). Furthermore, the positive correlation found between menarcheal timing in mothers and daughters (Campbell & Udry, 1995; Graber et al., 1995) indicates that mothers with earlier menarcheal ages tend to give birth to daughters who also experience early menarche (e.g., Campbell & Udry, 1995; Graber et al., 1995; Kim & Smith 1998; Surbey, 1990).

In addition to these behaviour genetic analyses, molecular genetic analyses are providing new insight into the genetic regulation of pubertal timing. For example, recent molecular genetic investigations have begun to identify allellic variants of genes that are associated with variation in (a) menarcheal timing (Comings, Muhleman, Johnson, & MacMurray, 2002; Stavrou, Zois, Ioannidis, & Tsatsoulis, 2002), and (b) the

timing of development of secondary sexual characteristics (Kadlubar et al., 2003). However, because this promising line of investigation is relatively new, specific genetic determinants of pubertal timing are, in the main, yet to be elucidated (Palmert & Hirschhorn, 2003).

In light of this genetic evidence, it has been proposed by some authors that rearing effects associated with early pubertal timing (e.g., experiencing family disruption/father absence, which is central to this thesis) are simply an artifact of genes that are common to both parents and daughters (Belsky, 2012). However, research by Rowe (2000) that used a genetically informative design to explicitly test this proposal did not find unequivocal support for it. Rather, (using a boxing metaphor to summarise his findings) Rowe (2000, p. 165) concluded that, "the behavioral genetic view gave no knock-out punches", and, moreover, "the evolutionary life history view…threw a few hard punches."

Environmental influences on variation in pubertal timing

While behaviour genetic analyses have clearly demonstrated that pubertal timing is under significant genetic control, these same analyses have also produced clear evidence of the importance of shared environmental influences on menarcheal age (Ellis et al., 2003). For example, Farber (1981) reported a positive relationship between the degree of consanguinity and pubertal timing. Specifically, in terms of age of menarche, monozygotic twins reared together were the most similar (average difference = 2.8 months), followed by monozygotic twins who were reared apart (average difference = 9.3 months), followed by dizygotic twins reared together (average difference = 12 months). However, the fact that monozygotic twins reared apart were most similar to dizygotic twins reared together (in terms of similarity in

menarcheal ages) suggests that the degree to which girls share common environments—as well as genes—influence individual differences in menarcheal timing (Ellis et al., 2003). Comparisons between mother–daughter dyads and sister–sister dyads provide further evidence of shared environmental effects on pubertal timing (reviewed in B. J. Ellis, 2004). Specifically, although the members of such dyads share the same degree of consanguinity, correlations for age at menarche are higher among sister–sister dyads than among mother–daughter dyads. This finding clearly indicates that being reared in the same family environment increases similarity in pubertal timing (reviewed in Malina, Ryan, & Bonci, 1994). However, it is important to note that some environmental influences on pubertal timing are also likely to have a *nonshared* environmental component because it is probable that some of them will affect siblings who are being reared in the same home differently (B. J. Ellis, 2004). For example, the specific environmental influences being examined in this thesis (i.e., differential sibling exposure to family disruption/father absence and associated factors) are likely to have nonshared effects on sisters' pubertal development.

While genetic factors clearly play a role in regulating pubertal timing, their influence occurs in concert with the influence of environmental factors—both physical and psychosocial—that also play a role in its regulation. Therefore, the second part of this discussion will examine a number of the environmental influences that appear to influence pubertal timing, including geographic factors, intrinsic factors that are unique to individuals (i.e., prenatal growth, body weight, physical activity, and dietary factors), extrinsic factors that are shared by family members (i.e., familial socioeconomic status, parental education, and levels of family conflict) and family composition variables (i.e., family size, presence of step-parents, step-brothers and half-brothers, and birth order),

and parental absence (*especially* father absence). However, while many of the factors being examined are interrelated, for pragmatic reasons I will discuss them individually.

Geographic factors

Aspects of the physical environment in which girls are reared have been implicated in their subsequent pubertal timing. For example, previous research has consistently demonstrated that girls who live in urban areas attain menarche at earlier ages than those who reside in rural areas. This effect has been found in a number of different countries (e.g., the United States [Matchock & Susman, 2006], Spain [Marrodan, Mesa, Arechiga, & Perez-Magdaleno, 2000], and Poland [Charzewska, Ziemlanski, & Lasecka, 1976; Hulanicka & Waliszko, 1991]). Numerous studies have also found an association between being reared at higher altitudes and delayed menarche (Eveleth & Tanner, 1976). Specifically, this effect has been found in Europe (Valsik, 1965) and in Sherpa populations in Nepal, where researchers found that girls living at higher altitudes had significantly later menarche than those residing at more moderate altitudes (Bangham & Sacherer, 1980). An earlier, widely held view that girls mature earlier in warmer climates, such as the tropics, than they do in temperate or arctic climates has been debunked by several authors (reviewed in Bojlen & Bentzon, 1968; Singh, 1972). However, recent research by Dossus et al. (2013) examining pubertal timing in women born in different locations across France has demonstrated an inverse dose-response relationship between latitude and ultraviolet radiation dose and age at menarche. Furthermore, these analyses suggest that pubertal maturation may be influenced by childhood light exposure (Dossus et al., 2013).

Intrinsic factors unique to individuals

A second group of influences on pubertal timing are more intrinsic to the individual (Fisher & Eugster, in press). Because the neuroendocrine system that controls pubertal onset comprises multiple signalling pathways, it is extremely vulnerable to disruption (Steingraber, 2007). For example, a variety of upstream factors (e.g., prematurity and low birth weight, overweight or obesity, or exposure to endocrine-disrupting chemicals in the environment) have the potential to accelerate pubertal onset by altering the regulation of the GnRH-secreting neurons (Steingraber, 2007). Therefore, it is reasonable to assume that many aspects of the individual's developmental milieu will affect both the timing and tempo of his or her subsequent pubertal maturation. Although it does not comprise an exhaustive list, some of the key environmental modifiers associated with pubertal development are discussed below.

Prenatal growth. Intrauterine growth, birth weight, and the tempo of early weight gain—especially in terms of its effects on foetal programming and pubertal timing—has been the subject of increasing research attention (e.g., Adair, 2001; Fisher & Eugster, in press). Both premature birth and retarded intrauterine growth are events that are associated with increased risk for early puberty in girls (reviewed in Steingraber, 2007). For example, being small for gestational age is a risk factor for idiopathic central precocious puberty (Deng et al., 2012)

Body weight. One of the most robust research findings is that excess weight predicts earlier pubertal timing in girls (Fisher & Eugster, in press). Obesity, which has become increasingly prevalent in children over the last few decades, is a known endocrine disrupter that dramatically alters leptin, aromatase, and insulin levels in the body (Steingraber, 2007). Moreover, it has been reliably established that heavier girls tend to experience *thelarche* earlier than their lighter peers (Steingraber, 2007).

However, because obesity both contributes to, and is a consequence of, early puberty, its role in accelerating thelarche and pubarche has not yet been clarified (Steingraber, 2007). Proposed mediators of the association between obesity and pubertal timing include gut peptides, adipcytokines, and leptin (Fisher & Eugster, in press).

Physical activity. It has also been proposed that reduced levels of physical activity, especially inactivity associated with increased electronic media use (including television viewing) also accelerates pubertal timing (Steingraber, 2007). Increased calorie intake often accompanies children's use of electronic media. Therefore, reduced levels of physical activity and increased calorie consumption may be working in concert to produce obesity, which in turn may be provoking earlier pubertal timing (Steingraber, 2007).

Dietary factors. The impacts of specific dietary exposures on pubertal timing have also been extensively examined (Fisher & Eugster, in press). For example, vitamin D deficiency, and greater consumption of cow's milk and animal protein during childhood are associated with earlier pubertal timing (Fisher & Eugster, in press). There is some evidence that being breastfed in infancy may offer girls some protection from early puberty; however, this association requires further investigation (Steingraber, 2007). Other nutritional factors thought to influence pubertal timing have been comprehensively reviewed by Cheng et al. (2012).

Extrinsic factors shared by family members

A group of extrinsic factors that tend to be shared by family members are also associated with variation in pubertal timing. These include family and parental influences such as socioeconomic status, parental education, and family functioning variables such as family conflict.

Socioeconomic status and parental education. Recent research in the United States has suggested that lower socioeconomic status (SES) is related to earlier pubertal timing (Braithwaite et al., 2009; Davison, Susman, & Birch, 2003; Ellis & Essex, 2007; Lee et al., 2007). However, research findings for this association have been mixed. For example, a comprehensive review conducted by Parent et al. (2003) reported inconsistent results for this association, especially between developing and developed countries. Moreover, measurement of SES is often based on either parental education or household income, but these measures can produce evidential differences. For example, a study by Windham, Bottomley, Birner, and Fenster (2004) found that lower maternal education predicted earlier pubertal timing, whereas no such relationship was found for household income. However, more recent studies have reported that lower parental education (Davison, Susman, & Birch, 2003; Ellis & Essex, 2007; Lee et al., 2007) *and* lower household income (Davison, Marshall, & Birch, 2006; Ellis & Essex, 2007) independently predict earlier pubertal timing.

Family Conflict. Researchers have also proposed that poor family functioning predisposes children to early pubertal timing. In accordance with this prediction, variables associated with problematic family functioning have been found to be associated with early pubertal timing, such as family conflict at the age of 7 (Moffitt, Caspi, Belsky & Silva, 1992), and increased family tension and more distant relations with siblings (Leek, 1991). Moreover, more tense family relationships **(**Kim & Smith 1998), more conflictual or distant relationships in mother–daughter dyads (Kim & Smith 1998; Steinberg, 1988, 1989), and more conflictual relationships in daughter–parent triads (Graber et al., 1995; Kim & Smith, 1998), all predict earlier menarcheal timing in daughters.

Family variables associated with variation in pubertal timing

Previous research on rodents, nonhuman primates, and prairie dogs suggests that within these mammalian species, certain social cues act as influential regulators of timing of pubertal maturation (reviewed in Matchock & Susman, 2006). For example, first vaginal oestrus in juvenile female mice can be accelerated by the actual presence of male mice, but also by the mere presence of their urine. Puberty in rodents can be delayed by the presence of grouped females and also by the mere presence of their urine, which is thought to contain pheromonal cues that mediate changes in reproductive status within juvenile rodents.

These social effects may not be confined to nonhuman animals. Recent research suggests that social factors may also dynamically affect human pubertal timing. Although the socioendocrinological processes involved have not yet been elucidated, it has been proposed that the timing of human pubertal maturation is especially sensitive to social cues related to family composition (see Matchock & Susman, 2006). Therefore, this final group of factors associated with variation in pubertal timing includes family size, birth order and sibling configurations, presence of step-parents, step-siblings and half-siblings, and, finally, the absence versus presence of biological parents.

Family size. An important aspect of the early social environment for humans is the size of our immediate family of origin (a factor that is largely determined by our total number of siblings). Because theorists have suggested that family size may have a regulatory effect on pubertal timing, the association between family size and menarcheal timing has received considerable research attention. The research findings, however, have been somewhat mixed. Whereas some studies have found a positive relationship between age at menarche and total number of children in the

family (e.g., in India [Singh, 1972]; Canada [Jenicek & Demirjian, 1974]; Slovakia [Valsik, Stukovsky, & Bernatova, 1963]; the United Kingdom [Morris, Jones, Schoemaker, Ashworth, & Swerdlow, 2010; Roberts & Dann, 1967; Roberts, Danskin, & Chinn, 1975]; and the United States [Malina, Katzmarzyk, Bonci, Ryan, & Wellens, 1997]; other studies have not found this association (e.g., in Australia [Jones, Leeton, McLeod, & Wood, 1972]; Nigeria [Oduntan, Ayeni, & Kale, 1976]; and Canada [Surbey, 1990]).

Birth order. The effect of birth order on timing of menarche has also been extensively investigated and the findings are also mixed. For example, Jones et al. (1972) found a positive association between having a greater number of younger brothers (but not sisters) and a later menarcheal age. Moreover, while some authors have found that earlier-born sisters in sibships tend to have later menarcheal timing than do their later-born sisters (e.g., Roberts & Dann, 1967), other studies have not found this association (e.g., Jones et al., 1972; Matchock & Susman, 2006; Singh, 1972). However, it has been suggested that in order to find birth order effects, it is necessary to simultaneously classify the mean age at menarche by family size and birth order, a procedure which, if applied to the data, may resolve these apparent inconsistencies (see James, 1973).

Presence of step-parents, step-brothers and half-brothers. Along with the stress of experiencing parental separation or divorce, children are often exposed to other significant family-structure changes. For example, subsequent repartnering of the biological parents often culminates in the introduction of step-parents, step-siblings, and half-siblings into the child's home. Research has revealed that a number of these changes in family structure are associated with accelerated pubertal maturation in children, especially the presence of stepfathers (Mekos, 1991; Mekos, Hetherington, &

Clingimpeel, 1992) and stepmothers (Mekos et al., 1992). Matchock & Susman (2006) also found that the presence of half-brothers and stepbrothers was associated with earlier menarche in girls.

Family composition. It is an intriguing and well-replicated research finding that girls who are raised in stressful family environments are more likely to experience earlier pubertal timing (relative to their same age peers) than girls who are not. For example, experiencing a greater number of stressful life events in childhood is associated with earlier menarche (e.g., Kim & Smith, 1998; Surbey, 1990). Moreover, recent theory and empirical research (e.g., Belsky, Steinberg, & Draper, 1991; Ellis & Garber, 2000; Ellis, McFadyen-Ketchum, Dodge, Pettit, & Bates, 1999; Graber et al., 1995) suggest that *both* family processes *and* family composition (especially father absence) during childhood may causally influence pubertal development in girls.

Mother absence and absence of both parents. The suggestion that family composition, especially biological parent absence, may influence timing of pubertal maturation has a lengthy research history. Seemingly one of the earliest investigations of the effects of biological parent absence on pubertal timing was a study conducted by Whiting (1965), which utilised cross-cultural ethnographic data, and found an association between exposure to mother absence (i.e., a stressor) in the first two weeks of life and earlier menarcheal age. Later research by Bogaert (2005, 2008), however, found no such effect of maternal absence. Early studies also found that the loss of both biological parents is associated with earlier menarcheal timing (Hulanicka, 1989; Lucsak & Laska-Mierzejewska, 1990; Surbey, 1990).

Early research on father absence. A number of early studies examining the effects of exposure to biological father absence found that this too was associated with earlier pubertal timing (Hulanicka, 1989; Jones et al., 1972; Milicerowa, 1968). The earliest of

these studies, conducted by Jones et al. (1972), examined the association between early exposure to father absence and age at menarche in a sample of 400 Australian adult females of lower socioeconomic status. These authors found that daughters who were under 6 years of age when their fathers left the family home experienced significantly earlier menarche than their counterparts who were still living with their fathers when they attained menarche (Jones et al., 1972). Several early studies conducted in Poland also found that father absence (as a result of divorce) was associated with earlier menarcheal timing in girls (Hulanicka, 1989; Milicerowa, 1968).

Recent research on father absence. Since Barkow (1984), Belsky et al. (1991), and B. J. Ellis (2004) published their evolutionary-based theorising regarding the association between father absence and earlier pubertal timing in girls, researchers have recently started to consider their research findings in light of these authors' predictions. Consequently, the proposition that biological father absence actually accelerates daughters' pubertal timing has now been extensively investigated. In this area of inquiry, a father-absent family has typically been defined as a family in which the biological father does not reside with his offspring. Importantly, this definition of father absence has generally required the biological father to be absent as the result of relationship dissolution, and not for any other reason (i.e., not due to military deployment or any other work-related or pragmatic absences, nor due to his death). Typically, also, father absence has occurred prior to the children entering puberty.

Numerous correlational studies have found an association between family disruption/father absence and earlier pubertal development in daughters. In particular, the link between father absence and early menarcheal timing has proved to be a very robust finding (e.g., Campbell & Udry, 1995; Kim & Smith, 1998; Moffitt, Caspi, Silva, & Belsky, 1992; Quinlan, 2003; Romans, Martin, Gendall, & Herbison,

2003; Surbey, 1990; Wierson et al., 1993). Studies examining the association between family disruption/father absence and early pubertal development in daughters have been conducted in a number of countries (including Australia, the United Kingdom, Canada, Chile, France, Germany, New Zealand, Poland, and the United States). Moreover, these studies have employed a variety of research designs and methods, and have examined a wide range of population samples. For example, a number of studies have assessed the effects of family disruption/father absence on pubertal timing prospectively during adolescence (e.g., Campbell & Udry, 1995; Ellis & Garber, 2000; Ellis et al., 1999; Hetherington & Kelly, 2002; Moffitt et al., 1992; Rowe 2000; Wierson et al., 1993). Other studies examining this association have assessed age at menarche retrospectively in adult samples (Alvergne, Faurie, & Raymond, 2008; Doughty & Rodgers, 2000; Hoier, 2003; Jones et al., 1972; Jorm, Christensen, Rodgers, Jacomb, & Easteal, 2004; Kiernan & Hobcraft, 1997; Ossa, Munoz, Amigo, & Bangdiwala, 2010; Quinlan, 2003; Romans et al., 2003; Surbey, 1990). The effects of father absence on pubertal timing have been examined in convenience samples (Hoier, 2003; Surbey, 1990; Wierson et al., 1993); broadly representative community or national samples (Doughty & Rodgers, 2000; Ellis et al., 1999; Jorm et al., 2004; Moffitt et al., 1992; Quinlan, 2003); and community-based samples, including clinical and psychopathology samples which have been matched with carefully chosen controls (Ellis & Garber, 2000; Romans et al., 2003). While most of these studies had age at menarche as the dependent variable, several prospective studies focussed instead on the development of secondary sexual characteristics (Ellis & Garber, 2000; Ellis et al., 1999; Rowe 2000).

Despite this plethora of samples, methods, and designs, this body of research has converged upon one remarkably robust research finding: girls who do not reside with

their biological fathers (as a result of parental relationship dissolution) tend to experience earlier pubertal development than do their peers who are raised in father-present homes (e.g., Campbell & Udry, 1992; Hulanicka, 1986, 1989; Jones et al., 1972; Luczak & Laska-Mierzejewska, 1990; Mekos, Hetherington, & Clingempeel, 1992; Milicerowa, 1968; Moffitt et al., 1992; Surbey, 1990).

Because numerous studies have revealed that the earlier the father leaves the family home, the earlier his daughters tend to go through puberty, the effects of (a) the duration of exposure to father absence, and (b) the age at onset (i.e., the chronological age at which it is first experienced) of father absence on pubertal timing have both been examined. This research has revealed that both earlier onset and longer duration of father absence is associated with more marked acceleration of pubertal timing. Specifically, researchers have found that a child's experience of father absence before the age of variously, 6 (Jones et al., 1972), 9 (Moffitt, Caspi, & Belsky, 1990), or 11 (Surbey, 1990), or experiencing a greater number of years of father absence before the age of 10 (Moffitt et al., 1990), is associated with earlier timing of menarche and/or pubertal development. Therefore, because lengthier exposure to father absence appears to exacerbate its accelerating effect on daughters' pubertal timing (see B. J. Ellis, 2004), the association between early father absence and early pubertal timing has often been characterised as a dose-response effect (see Ellis et al., 2003).

There have, however, been some exceptions to this finding. For example, research by Campbell and Udry (1995) demonstrated an interaction between father absence and ethnicity. That is, these authors found a significant effect of father absence on daughters' age at menarche for all participants except the African-American girls in the sample. Kim and Smith (1998) also did not find an association between family structure and pubertal timing, but given that the effect sizes in these studies are

typically quite small, and the fact that these authors had assessed age at menarche in a very small sample (n = 18), this finding is not especially surprising.

In sum, numerous correlational studies have revealed that exposure to family disruption/father absence is indeed associated with accelerated pubertal timing in daughters. Specifically, the correlations found in these studies, although statistically significant, tend to be small, typically representing accelerations in menarcheal timing ranging from approximately 2 to 8 months (B. J. Ellis, 2004). However, although the effect sizes in these studies are small, this does not mean that they are inconsequential. For example, B. J. Ellis (2004) highlights the significance of previous findings regarding the early onset of ovulatory menstrual cycles in early maturing girls (Apter & Vihko, 1983) in the following extract: "the time from menarche until 50% of cycles are ovulatory is approximately 1 year if menarche occurs before age 12 and 4.5 years if menarcheal age is 13 or older" (p. 936). This means that even seemingly minor accelerations in menarcheal timing can have extremely important reproductive implications for adolescent girls (B. J. Ellis, 2004).

Finally, it is important to note that these previous empirical tests of the effects of family disruption/father absence on daughters' pubertal timing have contained a basic confound. That is, they have confounded genetic effects and environmental effects, with the result that they have been unable to determine causation–which is an important focus of this thesis. Therefore, although this body of research has demonstrated a replicable phenemonon, the correlational designs utilised by investigators preclude selection effects from being ruled out. That is, it is possible that the reliable association found between family disruption/father absence and earlier menarcheal timing in daughter derives from pre-existing differences between biologically intact/father present and biologically disrupted/ father absent families

(Tither & Ellis, 2008), with genetic or socioeconomic differences being possible candidates.

Competing explanations for the association between family disruption/father absence and daughters' pubertal timing

Three competing classes of explanation have been proposed to account for the well-replicated association between biological family disruption/father absence and earlier pubertal development in girls (Tither & Ellis, 2008).

Explanation 1. Family disruption/father absence and related factors may actually cause earlier pubertal development in daughters.

This first possibility has been proposed by evolutionary-based models of developmental experience such as *paternal investment theory* (Draper & Harpending, 1982, 1988; B. J. Ellis, 2004) and *psychosocial acceleration theory* (Belsky et al., 1991; Chisholm, 1999). Both of these models, which posit that biological family disruption/father absence and related factors place daughters at heightened risk for earlier pubertal and sexual development, have at their centre the concept of *conditional adaptation* (Tither & Ellis, 2008).

Theory and empirical research in the field of evolutionary biology have recently recognised that the idea that single optimal survival and reproduction strategies evolve within most species is improbable (Boyce & Ellis, 2005; Gangestad & Simpson, 2000; Gross, 1996). This is because the optimal strategy in any given context varies as a function of that environment's particular characteristics, which means that a strategy that promotes survival and reproductive success in one context may well produce failure in another (Boyce & Ellis, 2005). Therefore, it has been noted that rather than favouring the evolution of a single optimal reproduction and survival strategy within a

particular species, selection pressures tend instead to favour *adaptive phenotypic plasticity*, or "the capacity of a single genotype to produce a range of phenotypes (manifested in morphology, physiology, and/or behaviour) in response to particular ecological conditions that recurrently influenced fitness during a species' evolutionary history" (Boyce & Ellis, 2005, p. 289). However, these alternative phenotypes do not develop randomly; rather, they are the products of a process that (a) involves systematic dealings between genes and environment, and (b) has been shaped by natural selection to increase both the ability and proclivity of individuals to monitor their developmental environments, and (based on these readings) adjust their phenotypes to suit the local conditions (Boyce & Ellis, 2005).

According to Boyce and Ellis (2005), in order for organisms to flourish in a particular niche, they must have the capacity to respond to immediate contingencies. The importance of this capacity means that selection should favour a hierarchy of mechanisms that enable organisms to both monitor, and react adaptively to incoming environmental data. Boyce and Ellis (2005) propose that the psychological mechanisms that underpin general and social intelligence will be at the top of this hierarchy, and that this higher group of mechanisms serve to enable the organism to respond quickly and flexibly to environmental changes (i.e., to both opportunities and threats) in their immediate environment. However, located further down this hierarchy are other mechanisms that monitor the slower and more common changes occurring in the environment (Boyce & Ellis, 2005). This group includes physiological, anatomical, endocrinal, and developmental mechanisms, and these often take the form of *conditional adaptations* (Boyce & Ellis, 2005). In the following extract, Boyce and Ellis (2005) describe these conditional adaptations as being

> evolved mechanisms that detect and respond to specific features of childhood environments, features that have proven reliable over evolutionary time in predicting the nature of the social and physical world into which children will mature, and entrain developmental pathways that reliably matched those features during a species' natural selective history. Conditional adaptations…underpin development of contingent survival and reproductive strategies and thus enable individuals to function competently in a variety of different environments. (p. 290)

While both psychosocial acceleration theory and paternal investment theory have adopted the concept of *conditional adaptation*, these theories have also evolved over time from shared origins, so, at this point, a chronology of their development seems useful. Specifically, these causal models of the association between father absence and early pubertal development in daughters have their inception in a seminal paper published in 1982 by two anthropologists, Patricia Draper and Henry Harpending. Notably, however, rather than adhering to the commonly held view that father-absent homes are somehow defective in comparison with their father-present counterparts and that being reared in such a family inevitably produces deleterious results for children, these authors took the opposite view, proposing instead that father-absent and father-present households were "differing but equally sound structural types within which mating and provisioning of the young can occur" (Draper & Harpending, 1982, p. 256). Specifically, these authors proposed that when framed within a larger and more inclusive evolutionary context, father absence and father presence can each be viewed as having adaptive value. To this end, the existing body of

research literature on father absence was reinterpreted by these authors in light of theory drawn from evolutionary biology (Belsky, 2000; Draper & Harpending, 1982).

Arguably the most novel aspect of this paper was its adoption of the construct of *reproductive strategies,* drawn from the field of behavioural ecology (see especially Trivers, 1974) and from life history theory (Belsky, 2000). Trivers' (1974) parental investment theory proposes that in order to reproduce, an organism must attend to three fundamental tasks: (a) physical growth and development; (b) mating; and (c) parenting. However, while it is obvious that species vary dramatically in terms of how much effort their members expend on each of these tasks, substantial *within-species* variation is also found in terms of how such effort is allocated. This variation is evident among humans: different people apportion significantly different amounts of effort to mating and parenting respectively. However, it is important to note that such allocations are typically nonrandom. Specifically, life history theory assumes that selection will tend to favour phenotypic mechanisms that allocate resources (which are limited) to maintenance, growth, and reproduction over a lifetime in a manner that will maximise fitness (Belsky, 2000; Surbey, 1998). However, tradeoffs among these fitness components are inevitable, because once resources are allocated to one component they are no longer available to be allocated to another. Therefore, the concept of reproductive strategies—that is, behaviours that evolve to maximise an individual's reproductive success—was not only fundamental to Draper and Harpending's (1982) initial analysis, but also to their organisation of a range of findings related to parenting, interpersonal, and sexual behaviours in the context of heterosexual relationships (Belsky, 2000).

Central also to Draper and Harpending's (1982, 1988) theorising is the idea that humans have evolved to detect and encode information about specific aspects of their

early rearing contexts, and that the nature of the information collected then biases individuals towards adopting particular reproductive strategies over others in adulthood (Draper & Harpending, 1982, 1988). Specifically, these authors propose that during early childhood, children display an evolved, sensitive period for learning about (a) the status of their mother's pair bond, and (b) her disposition towards males. Therefore, they propose that a key function of early life experience (during approximately the first 5 years of life) is to produce within children an understanding of two important aspects of their rearing environments: (a) the level of paternal investment in the family, and (b) the quality of the relationships between males and females. Furthermore, Draper and Harpending's theory posits that once acquired, these understandings then have the effect of canalising particular developmental tracks in children, which, in turn, have predictable effects on their subsequent reproductive behaviour.

While Draper and Harpending (1982, 1988) focused on the differential effects of father absence and father presence on the subsequent reproductive strategies of *both* boys and girls, for the purposes of this thesis I will restrict my discussion to these authors' theorising about girls' development. Specifically, these authors propose that girls who experience relatively low levels of paternal investment and adversarial male–female relationships in their early family environments discern that parental investment by males is *not* essential for reproduction. The developmental track "chosen" by these girls is characterised by early onset of sexual activity and reproduction, and a willingness to enter into new sexual relationships, which tend to constitute relatively unstable pair bonds. Conversely, girls who experience relatively more amicable male–female relationships in their early family environments and relatively high levels of paternal investment discern that parental investment by males *is* important for

reproduction. The developmental track "chosen" by these girls is characterised by delayed onset of sexual activity and reproduction and reduced willingness to enter into sexual relationships, thereby encouraging the formation of relatively stable pair bonds with caring and reliable male partners. Either way, this theory proposes that each girl "selects" a developmental pathway based on the type of adult social environment into which she is born. Moreover, the pathway selected is likely to have promoted reproductive success in similar social environments during human evolutionary history.

Essentially, then, Draper and Harpending (1982) propose that girls who are reared in homes where the fathers are absent (as a result of relationship dissolution) will develop behavioural patterns that reflect a belief that pair bonds and paternal investment are neither reliable nor enduring. They will engage in sexual activity and reproduction earlier, and they will expend a greater amount of their energy on mating than on parenting. Conversely, girls reared in father-present homes will have exactly the opposite expectations. When this latter group of individuals reaches biological maturity, they will defer sexual activity, choosing instead to channel their energy into developing and maintaining close pair bonds (Draper & Harpending, 1982, 1988).

Draper and Harpending's (1982) provocative paper ultimately spearheaded substantial theoretical developments and empirical studies on the role of early experience in shaping reproductive strategies. Research investigating the effects of father absence on the development of female reproductive strategies largely supports Draper and Harpending's (1982, 1988) theorising. It is common, for example, for adolescent girls from father-absent homes to exhibit precocious sexual interest in males, while at the same time expressing negative sentiments towards both masculinity and males, and being disinterested in establishing exclusive long-term emotional and sexual

relationships with them (Belsky et al., 1991; Bereczkei & Csanaky, 1996; Draper & Harpending, 1982; Hetherington, 1972).

At the time of its publication, Draper and Harpending's (1982) argument was novel, because it had framed the effects of early rearing experiences in the family within evolutionary terms, rather than viewing them in terms of mental health (which had been the usual approach up until then) (Belsky, 2012). Therefore, these authors' emphasis on pair bonds, parental investment, reproductive strategies, and reproductive fitness in order to understand the effects of these experiences was a new approach (Belsky, 2012). However, it was not without its critics, who argued that these analyses had two important deficiencies: (a) they did not elucidate the specific developmental mechanisms by which early experiences would shape reproductive behaviour; and (b) they did not advance any novel predictions (Belsky, 2000, 2012).

Because this paper prompted so much theoretical and empirical interest, however, it did not take long for these lacunae to be addressed. While Draper and Harpending (1982) had restricted their scope to the effects of early rearing experiences in the family on the subsequent psychology and behaviour of offspring, predictions about the differential effects of particular family types on children's subsequent physical development soon materialised. For example, within 2 years of its publication, Draper and Harpending's paper had prompted Jerome Barkow (1984) to propose that experiencing father absence versus father presence during childhood would not only systematically affect children's psychology and behaviour, but it would also provoke sex-specific changes in their physiques. Specifically, Barkow (1984) predicted that father-absent boys would develop more mesomorphic physiques than would father-present boys, and that father-absent girls would attain menarche earlier than would father-present girls. Moreover, Barkow posited that not only were his predictions in line with

Draper and Harpending's (1982) initial theoretical framework, but that they also offered the opportunity to test it.

Moreover, less than a decade after Barkow (1984) made his novel predictions, an alternative evolutionary theory, which has since come to be known as *psychosocial acceleration theory,* was advanced by Belsky et al. (1991) (although, to be precise, this theory actually focuses on the *regulation,* not just the acceleration, of development [Belsky, 2012]). This theory has derived a number of more specific models that have focused on the role of familial stressors in early pubertal timing and reproduction (Belsky et al., 1991; Chisholm, 1993, 1996; Wilson & Daly, 1997). However, while Draper and Harpending's (1982, 1988) theory had restricted its focus to father absence versus father presence, Belsky et al.'s (1991) theory increased the range of predictor variables, thereby focusing on a much broader range of family and environmental stressors. These authors, however, retained Draper and Harpending's (1982) framework of "evolved, sensitive-period learning of reproductive strategies" (p. 264), proposing that

> a principal evolutionary function of early experience—the first 5 to 7 years of life—is to induce in the child an understanding of the availability and predictability of resources (broadly defined) in the environment, of the trustworthiness of others, and of the enduringness of close interpersonal relationships, all of which will affect how the developing person apportions reproductive effort. (Belsky et al., 1991, p. 650)

This revised theory now focused on the various environmental circumstances within families that are stressful for children, including being raised by a sole parent; having low socioeconomic status; receiving inconsistent, coercive, or inadequate parenting (from either parent); and experiencing discordant parental relationships.

Furthermore, such stressors were ordered in a causal chain. That is, Belsky et al. (1991) predicted that exposure to family stress (from a wide range of possible sources) would produce more discordant and less supportive family relationships which, in turn, would promote a reproductive strategy that was both precocious and promiscuous, and would involve low levels of parental investment. Conversely, these authors predicted that a more supportive family context would produce family relationships that were more congenial and supportive which, in turn, would promote a reproductive strategy characterised by later development and more monogamous attitudes and behaviour.

Moreover, Belsky et al. (1991) (consistent with Barkow's [1984] prediction) made a second important addition to Draper and Harpending's (1982, 1988) theory: the addition of puberty as a new outcome variable. Thus, while Draper and Harpending had only tested predictions related to behavioural and psychological development, Belsky et al. (1991) (see also Surbey, 1990) extended the theory to include predictions about physical development in different ecological contexts. Specifically, these authors predicted that contexts in which (a) paternal investment in children, and (b) supportive long-term relationships between males and females were unlikely would promote a type of reproductive strategy characterised by earlier pubertal timing, precocious sexuality, and unstable pair-bonds. Such a strategy would function to promote reproduction at a relatively early age. Hence, the revised theory now included a novel prediction: girls whose early rearing experiences and contexts are relatively more stressful (or less supportive) will experience accelerated pubertal maturation, within their own individual range of plasticity (Belsky et al., 1991).

Belsky et al. (1991) thus predicted that rearing experiences before puberty would influence pubertal timing, and that, collectively, these processes and events would influence both sexual behaviour in adolescence and pair bonding in adults.

Essentially, these authors argue that individuals engage in either a 'quantity or quality' pattern of mating and child rearing. The pattern that is actually evinced by individuals is determined by their early experience, which in turn affects their behavioural and psychological development in predictable ways, which in turn influences their somatic development.

Throughout the course of human natural selective history, such a strategy may well have reliably increased the reproductive success of ancestral females reared in stressful family environments. As Chisholm (1996, p. 21) proposes "when young mammals encounter conditions that are not favourable for survival—that is, the conditions of environmental risk and uncertainty indexed by emotional stress during development—it will generally be adaptive for them to reproduce early."

This psychosocial acceleration model, however, begs the question "Does stress actually accelerate pubertal development in girls?" Research suggests that contrary to Belsky et al.'s (1991) theory, physical stress, which includes factors such as poverty, lack of nutrition, and disease, does not accelerate pubertal development. Rather, recent studies suggest that physical stress may actually cause pubertal maturation in girls to be delayed, thus channelling energy away from growth and reproduction towards maintenance (i.e., survival) (reviewed in B. J. Ellis, 2004; see also Ellison, 1990; Surbey, 1998). Conversely, however, numerous studies have found that socioemotional stress, which includes factors such as experiencing conflictual family relationships, divorce, parental psychopathology, and lack of parent/child connectedness, *is* associated with accelerated pubertal development in girls (e.g., reviewed in B. J. Ellis, 2004; see also Ellis et al., 1999; Graber et al., 1995**;** Steinberg, 1988). Finally, researchers have found that girls reared in father-absent homes tend to enter puberty a few months earlier than do girls raised in father-present homes (Moffitt et al., 1992; Surbey, 1990; Wierson et al.,

1993). Moreover, this appears to be a dose-response relationship, given that several studies have found that the longer the duration of father absence, the earlier the onset of menarche (Moffitt et al., 1992; Surbey, 1990).

More recently, *paternal investment theory*, as formulated by B. J. Ellis (2004), has been proposed to explain the association between family disruption/father absence and earlier pubertal and sexual development in girls (see also Ellis et al., 1999, 2003; Ellis & Garber, 2000). It should be noted that while paternal investment theory is also fundamentally based on Draper and Harpending's (1982, 1988) original theorising, it is also a variant of psychosocial acceleration theory. However, whereas Belsky et al. (1991) in their attempt to identify the environmental factors that regulate the development of reproductive strategies, chose to extend the range of the predictor variables far beyond father absence to include the quality of both parent-child and parental relationships (and their determinants), Ellis and colleagues' formulation of paternal investment theory retains Draper and Harpending's (1982) emphasis on the importance of paternal influence on reproductive strategies. As B. J. Ellis (2004) proposes:

> Girls detect and internally encode information specifically about the quality of paternal investment during approximately the first 5 years of life as a basis for calibrating the development of (a) neurophysiologic systems involved in timing of pubertal maturation and (b) related motivational systems, which make certain types of sexual behavior more or less likely in adolescence. (p. 938)

Therefore, although paternal investment theory is also a variant of psychosocial acceleration theory, it is more a particularised theory due to its very specific focus on (a)

the role of the father in the family, (b) maternal behaviour towards males, and (c) maternal sexual attitudes. Moreover, paternal investment theory proposes that the quality of paternal investment plays a unique and central role in the regulation of daughters' sexual development, with its effects occurring over and above all other sources of psychosocial stress and support (B. J. Ellis, 2004). However, it too retains Draper and Harpending's (1982) framework of "evolved, sensitive-period learning of reproductive strategies" (p. 264) by proposing that early experiences provide girls with vital understandings of paternal investment and the quality of relationships between males and females, which then provide input into the regulatory mechanisms that determine sexual development (B. J. Ellis, 2004). Specifically, this theory proposes that girls who experience relatively low levels of paternal investment and adversarial male–female relationships in their early family environments perceive that parental investment by males is not essential for reproduction. Moreover, it hypothesises that these girls will (a) develop in a manner that accelerates pubertal timing (and the onset of sexual activity and reproduction), and (b) be oriented towards forming relatively unstable pair bonds (B. J. Ellis, 2004). Conversely, girls who experience relatively more amicable male–female relationships in their early family environments and relatively high levels of paternal investment perceive that parental investment by males *is* important for reproduction, and are hypothesized to develop in the opposite manner (B. J. Ellis, 2004). Either way, this theory proposes that the pathway selected by the individual girl is likely to have promoted reproductive success in similar social environments during human evolutionary history (see especially Belsky et al., 1991).

Explanation 2. The association between family disruption/father absence and earlier pubertal development in daughters may arise from a family-wide environmental confound.

A second possibility is that the association between family disruption/father absence and earlier pubertal development in daughters is noncausal because it derives from some family-wide environmental confound (Tither & Ellis, 2008). Specifically, family-wide environmental effects are causal factors that are shared within, but that vary among, families. Therefore, when attempting to account for the association between family disruption/father absence and earlier pubertal development in daughters, such effects are problematic because it is quite possible that a family-wide environmental confound is causing both family disruption/father absence and earlier pubertal development (Tither & Ellis, 2008). Poverty, for example, is a possible candidate, especially given that recent studies conducted in the United States have revealed that it is associated with *both* earlier pubertal development in girls *and* higher rates of family disruption/father absence (Braithwaite et al., 2009; Davison et al., 2003, Ellis & Essex, 2007). Therefore, if poverty—or some other family-wide environmental factor—is causing both earlier pubertal timing and family disruption/father absence, then the association between these two factors is noncausal (i.e., the "family disruption/father-absence effect" on pubertal timing derives from a third, unmeasured environmental variable) (Tither & Ellis, 2008).

Explanation 3. The association between family disruption/father absence and earlier pubertal development in daughters may arise from a shared genetic confound.

A third possibility is that that the association between family disruption/father absence and earlier pubertal development in daughters is noncausal because it derives from some shared genetic confound (also known as a gene-environment correlation) (Tither

& Ellis, 2008). Specifically, earlier-maturing girls also tend to (a) have earlier sexual debuts, (b) marry earlier, and (c) give birth for the first time at earlier ages, than do their later-maturing counterparts (reviewed in B. J. Ellis, 2004). This covariation may occur for two reasons: (a) early pubertal timing may actually provoke earlier sexual behaviour and reproduction in girls, or (b) pubertal, sexual, and reproductive timing in girls may be genetically correlated traits (Rowe, 2002). Earlier reproduction is itself associated with both heightened risk of relationship dissolution and paternal investment of a poorer quality (e.g., Amato, 1996; Bennett, Bloom, & Miller, 1995). Because mothers who mature earlier tend to have daughters who also exhibit earlier pubertal timing (e.g., Malina et al., 1994; Salces, Rebato, Susanne, San Martin, & Rosique, 2001*)*, the relation between timing of puberty and family disruption/father absence may, therefore, be noncausal; that is, it may derive from genetic transmission of pubertal timing and associated behavioural characteristics (Belsky et al., 1991; Kim & Smith, 1998; Moffitt et al., 1992; Rowe, 2000; Surbey, 1990).

In sum, mothers who themselves experience earlier onset of puberty and sexual activity may be genetically transmitting this tendency to their daughters (see Dunne et al., 1997). However, because young mothers also tend to form less stable relationships with the fathers of their children, their daughters may also be disproportionately exposed to early father absence (e.g., Amato, 1996). Thus, the association between family disruption/father absence and early pubertal maturation in daughters may simply be the result of genetic transmission of the timing of pubertal maturation and associated behavioural characteristics (Belsky et al., 1991; Kim & Smith, 1998; Moffitt et al., 1992; Rowe, 2000; Surbey, 1990). Findings from molecular genetic research that demonstrate the influence of allelic variations on pubertal timing are consistent with this particular noncausal explanation (e.g., Kadlubar et al., 2003; Stavrou et al., 2002).

It is also possible that absent fathers are genetically transmitting a tendency towards early pubertal and sexual development to their daughters. For example, Comings et al. (2002) have proposed a specific version of the genetic transmission hypothesis based on a variant of the X-linked androgen receptor (AR) gene. Specifically, some fathers carry X-linked genes that are associated with aggression, impulsivity, promiscuity, and associated patterns of relationship discord and dissolution (thus increasing their probability of being absent fathers), which they then transmit to their daughters (Comings et al., 2002). In daughters, these genes are associated with earlier menarcheal age, precocious sexual behaviour, and father absence. Thus, these authors have offered an alternative genetic explanation of the association between family disruption/father absence and earlier pubertal development, in which fathers who carry these *AR* alleles are more likely to both be absent fathers *and* to pass these alleles on to their daughters, in whom they produce a tendency towards earlier menarcheal age (and behavioural problems associated with sexual risk). This theory has found some support in molecular genetic research conducted by Comings et al. (2002), who examined two clinical samples: (a) female outpatients who had volunteered for a weight control programme, and (b) males receiving inpatient treatment for substance abuse. However, it did not find support in later molecular genetic research conducted by Jorm et al. (2004), who examined general population samples. Therefore, further research is required in order to clarify these conflicting findings (Tither & Ellis, 2008).

Previous attempts to address potential genetic and family-wide environmental confounds

Whereas evolutionary-based causal explanations (i.e., psychosocial acceleration theory and paternal investment theory) have explicitly proposed that family disruption/father absence causes earlier pubertal timing in daughters, the two

opposing noncausal explanations outlined above have instead implicated potential genetic and family-wide environmental confounds. Therefore, determining whether or not family disruption/father absence is causally influencing daughters' pubertal timing, especially in light of these potential genetic and family-wide environmental confounds, has presented researchers with a major methodological challenge.

Most prior attempts to distinguish between these opposing classes of explanation (i.e., causal vs. noncausal) have involved researchers testing for the association between family disruption/father absence and daughters' pubertal timing while controlling for a range of potential confounds (Tither & Ellis, 2008). Such control variables have included (a) physical characteristics of the children themselves (including initial levels of pubertal development [N. B. Ellis, 1991; Graber et al., 1995; Steinberg, 1988], weight, body fat percentage, and biliac diameter [e.g., Campbell & Udry, 1995; Graber et al., 1995; Moffitt et al., 1992]); (b) parental characteristics (including both maternal age at menarche [Campbell & Udry, 1995; Graber et al., 1995; Kim & Smith, 1998; Surbey, 1990] and maternal sexual and reproductive history [Ellis & Garber, 2000; Kim & Smith, 1998a; Quinlan, 2003]); and (c) family-wide factors, (notably race [ethnicity] and socioeconomic status [reviewed in B. J. Ellis, 2004]). While in most cases, controlling for these variables made no meaningful differences to the observed associations between family environment and timing of puberty (reviewed in B. J. Ellis, 2004), a covariate adjustment method such as this does have a major limitation: while the researcher selects and measures a censored and somewhat arbitrary set of control variables, the remaining genetic and environmental factors remain unmeasured (Tither & Ellis, 2008).

Effectively testing the association between family disruption/father absence and earlier pubertal timing in daughters while at the same time controlling for potential

genetic and family-wide environmental confounds therefore necessitates the utilisation of genetically and environmentally controlled research designs that include environmental measures (Tither & Ellis, 2008). It is, however, extremely difficult to ascertain the independent contributions of uncontrolled environmental factors such as father absence/presence on pubertal timing, because practical and ethical constraints prevent the random assignment of children into these different fathering conditions (Mendle et al., 2006). Consequently, attempts to identify such unique contributions, independent of other genetic and environmental factors that also affect the likelihood that parents will have offspring who are genetically and/or environmentally at heightened risk of early puberty, have been rare (Tither & Ellis, 2008).

Children-of-Twins design

One notable attempt to address this conundrum, however, was the utilisation of a genetically controlled children-of-twins (CoT) research design by Mendle et al. (2006) to test the effects of family composition (i.e., family disruption/father absence/stepfather presence) on menarcheal age. In this study, the CoT methodology, which measures specific family variables and utilises data collected from other family members (see D'Onofrio et al., 2003; Turkheimer, D'Onofrio, Maes, & Eaves, 2005), was used to test for environmentally mediated effects on menarcheal timing (Mendle et al., 2006). While twin models have previously been employed to partition sources of variation in pubertal timing into genetic and environmental components, such models do not allow for the testing of specific effects of family disruption/father absence on pubertal timing because discordance among twins for parental relationship dissolution is very uncommon (i.e., childhood family composition is generally identical for twins). However, Mendle et al.'s (2006) study overcame this problem by using a specific

version of the CoT design which, rather than comparing age at menarche *within* pairs of female monozygotic twins (who are usually concordant for family dissolution), instead compared menarcheal timing among their offspring (i.e., first cousins who were *discordant* for family dissolution).

Specifically, this study utilised a sample of female identical twins who had each married and given birth to children. However, in each set of twins in this study, one twin was still in an intact relationship with her children's biological father, whereas her twin was not. Comparisons were then made between the twins' respective offspring, who (naturally) had genetically identical mothers (thus being in receipt of exactly the same maternal genetic risk for family disruption/father absence *and* for pubertal timing) but were differentially exposed to family disruption/father absence. Such a design allows for the testing of specific effects of family disruption/father absence on age at menarche. Take, for example, a set of (hypothetical) identical twins, Jane and Kate, who are discordant for relationship dissolution. If Jane's daughters (who have experienced family disruption/father absence) exhibit earlier pubertal timing than do Kate's daughters (whose father is still present), then a causal (i.e., environmentally mediated) influence of family disruption/father absence on pubertal timing is then supported (albeit provisionally) (Mendle et al., 2006). However, in this study, Mendle et al.'s (2006) findings did not support a causal interpretation of the effect of family composition on age at menarche (Tither & Ellis, 2008).

Limitations of the Children-of-Twins design

Mendle et al's. (2006) utilisation of the CoT design undoubtedly constitutes a major advance in this area of inquiry. Especially noteworthy is the design's ability to successfully address some of the most significant limitations of the correlational

research designs previously employed to examine the effects of changes in family structure on pubertal timing (Tither & Ellis, 2008). Nonetheless, it is important to note that the CoT design itself has a number of important limitations (Tither & Ellis, 2008). First, because the fathers of monozygotic-twins' offspring are typically *not also* monozygotic twins, this design is actually only 50% genetically controlled (Tither & Ellis, 2008). This is problematic because it means that this design does not account for fathers' unique genetic contributions to their offspring, including those that may influence their children's pubertal timing (Eaves, Silberg, & Maes, 2005). Second, it does not account for fathers' unique environmental contributions to their immediate family contexts that may also influence their children's pubertal timing (Eaves, Silberg, & Maes, 2005). Third, it does not control for environmental risk factors for earlier pubertal timing that are unique to only one of the adult twins in each pair and her offspring (D'Onofrio et al., 2003, 2005). A final important limitation of the CoT design is that it is underpowered to test for moderators of the effects of family disruption on daughters' pubertal timing, such as parents' functioning in the family (Tither & Ellis, 2008).

The current sibling comparison study

In order to be able to effectively test the potential *causal* influence of amount of exposure to family disruption/father absence on pubertal timing, it is important to employ genetically *and* environmentally controlled research designs that incorporate environmental measures (Tither & Ellis, 2008). One promising candidate is a differential sibling exposure design. Use of such a design in this area of inquiry would involve making comparisons, in terms of their respective pubertal timing, between full biological siblings who have been differentially exposed to family disruption/father absence (Tither & Ellis, 2008). Because full biological siblings share

exactly the same biological parents *and* usually reside together, such an approach would successfully address a number of the most problematic limitations of previous research designs that have been employed to examine the effects of family disruption on pubertal development, including the CoT approach (Tither & Ellis, 2008).

As reviewed above, it has already been reliably established that lengthier exposure to family disruption/father absence is associated with earlier pubertal timing in daughters. However, in order for a causal explanation for this association to be supported, the following conditions should be met. First, in families in which (a) full biological sisters are discrepant in age, and (b) younger sisters have lengthier exposure to father absence than do their older sisters (due to an earlier onset, which occurs in early childhood), these younger sisters should tend to exhibit earlier pubertal development than do their older sisters (Tither & Ellis, 2008). Second, age-discrepant sisters from biologically intact/father present families should not exhibit this within-family systematic difference in pubertal timing, thereby ruling out the possibility that it is a birth-order/birth-spacing effect (Tither & Ellis, 2008). Conversely, however, in order for the alternative *noncausal* explanation for this association to be supported (i.e., that it arises from either a genetic or family-wide environmental confound), a different condition should be met. That is, in both biologically disrupted and biologically intact families, age-discrepant full biological sisters should *not* exhibit systematically different pubertal timing as a function of birth order/birth spacing, despite the fact that within biologically disrupted families, such sisters have histories of differential exposure to family disruption/father absence (Tither & Ellis, 2008).

Therefore, utilising a differential sibling exposure design involves comparing markers of pubertal timing in full biological sisters: (a) who are discrepant in age, (b)

who have experienced the termination of their biological parents' union as a result of relationship dissolution during their childhoods (and prior to the younger sister entering puberty), and (c) who (at least in the case of the younger sister) have lived primarily with their biological mother from the time that their biological parents stopped residing together (Tither & Ellis, 2008). A key feature of this differential sibling exposure design, then, is the age discrepancy between the sisters in each family because, in the biologically disrupted families at least, it is this age gap that determines the sisters' differential exposure to family disruption/father absence (Tither & Ellis, 2008). For example, take a hypothetical (initially) biologically intact family in which there is an age gap of exactly 5 years between Anne, the older sister, and Robyn, the younger sister. If, due to relationship dissolution, their biological father stops residing with the family from the time that Anne is 10 years old and Robyn is 5 years old, then Anne will experience exactly 5 more years of life in a biologically intact family during her childhood than will Robyn. Conversely, compared to Anne, Robyn will experience (at least) 5 more years of biological father absence during her childhood.

Specific a priori predictions of the current study

Utilising such a differential sibling exposure design would allow me to effectively test the potential *causal* influence of amount of exposure to family disruption/father absence on age at menarche, in particular. Specifically, if I find that systematic differences in menarcheal timing between younger and older sisters are only evident in biologically disrupted families (i.e., these differences are not found within biologically intact families), I can then rule out birth order/birth spacing effects, and I can instead infer a causal relationship between length of exposure to biological family

disruption/father absence and earlier menarcheal timing. Conversely, however, if the association between lengthier father absence and earlier menarcheal timing in girls arises from either a family-wide environmental or a genetic confound, my findings should meet an entirely different condition. Specifically, if this association is noncausal, I should only find systematic differences in menarcheal timing *between* the two family types; no such differences should be evident within individual families. That is, if this association arises from some shared environmental confound (such as poverty) or a shared genetic confound, then I should find that age-discrepant sisters pairs from biologically disrupted/father-absent families do not systematically differ in terms of their menarcheal timing as a function of age.

Advantages of the current study's design

Therefore, in light of the urgent need for the utilisation of genetically and environmentally controlled research designs to examine the reliable association between family disruption and father absence, in the current study I employed a differential sibling exposure design to examine the effects of sisters' differential exposure to family disruption/father absence (i.e., a nonshared factor) on age at menarche. This constitutes an important methodological advance in this area of inquiry because, unlike the aforementioned correlational and CoT designs, this design incorporates vital genetic *and* environmental controls (Tither & Ellis, 2008).

First, unlike traditional quantitative genetics methods that determine heritability estimates by comparing levels of similarity between individuals who differ in terms of their genetic relatedness, this design controls for genetic effects through randomisation (Tither & Ellis, 2008). This approach assumes that genetic differences between sisters (e.g., in terms of pubertal timing) are randomly

distributed across the birth order (Tither & Ellis, 2008). Specifically, there is no reason to expect that either younger sisters, who in this case happen to have had more prolonged exposure to father absence, or older sisters, who in this case happen to have had less prolonged exposure to father absence, are at elevated genetic risk for any kind of pubertal outcome (i.e., either earlier or later) as a function of their particular birth order (Tither & Ellis, 2008).

Second, this differential sibling exposure design controls for family-wide environmental effects (i.e., factors that are shared within but that vary between individual families) through the use of within-family analyses (Tither & Ellis, 2008). Specifically, making direct comparisons between full biological siblings who are concordant for family-wide factors purported to influence age at menarche (e.g., family size, socioeconomic status, and ethnicity) avoids the methodological confounds associated with comparing genetically unrelated individuals from different families who may well be discordant for influential family-wide factors (Tither & Ellis, 2008).

Third, this differential sibling exposure design allows for a comparison group to be included in the sample, thus allowing other variables that may affect the outcome variable to also be accounted for (Tither & Ellis, 2008). Specifically, by comparing the magnitude of sibling differences in menarcheal age across both a primary sample of sister pairs from biologically disrupted families *and* a matched control sample of sister pairs entirely raised in biologically intact families, I can effectively account for other variables which may affect age at menarche, such as birth order/birth spacing effects and number of older/younger siblings (e.g., see Matchock & Susman, 2006).

The major advantage of utilising this differential sibling exposure design is that it enables the measurement of the effects of non-shared environmental factors (in this case sisters' differential exposure to biological family disruption/father absence) on a specific developmental outcome (in this case age at menarche), while simultaneously controlling for both genetic and family-wide environmental effects (Tither & Ellis, 2008). This design, therefore, offers the unique opportunity to discriminate between the opposing classes of explanation outlined above (i.e., causal versus noncausal) for the well-replicated association between lengthier exposure to family disruption/father absence (and associated factors) and earlier pubertal timing.

The current study has therefore utilised a differential sibling exposure design to distinguish between these two competing classes of explanation. The central hypothesis tested was that the birth order/age discrepancy (older versus younger) between sisters would interact with family type (biologically disrupted vs. biologically intact) to predict the magnitude of sibling differences in menarcheal age (Tither & Ellis, 2008). Specifically, because birth order/age discrepancy is a proxy for differences in exposure to family disruption/father absence *only* in biologically disrupted families, (i.e., this is not the case in biologically intact families), it should only produce systematically larger sibling differences in menarcheal age in this first group of families (Tither & Ellis, 2008).

Siblings' differential exposure to family disruption/father absence comprised the measured nonshared environmental influence in this study. However, numerous factors are known to covary with family disruption/father absence, including (but not restricted to) child neglect and abuse; exposure to stepfathers, stepsiblings, and half-siblings; financial security; family mobility; and maternal mental health (see Amato, 2000; Daly & Wilson, 1998; McLoyd, 1990). The current study was unable to determine which of these related nonshared environmental factors (either measured or

unmeasured) had the *most* causal influence (Tither & Ellis, 2008). Rather, it was restricted to testing a less specific hypothesis: that differential exposure to father absence (and associated factors) *causes* significant differences in menarcheal timing between sisters. Moreover, while the current study specifically tested for unique effects of family disruption/father absence and associated factors on menarcheal age, its design controlled for, but did not negate, potential genetic and family-wide environmental influences (Tither & Ellis, 2008).

Dysfunctional paternal behaviour as a potential moderator

Another important methodological advantage of the current sibling design is that it can be used to test for moderators of the effects of biological family disruption (unlike the CoT design) (Tither & Ellis, 2008). Clearly, all fathers are not created equal. Consequently, it is unlikely that the mere presence of the biological father in the home will automatically produce positive outcomes for children; however, it is probably not guaranteed that father absence will always produce negative child outcomes either (Tither & Ellis, 2008). Consistent with this reasoning, research by Jaffee, Caspi, Moffitt, Taylor, and Dickson (2001) demonstrated that simply residing in an intact biological family does not automatically (or, in fact, uniformly) benefit children. Rather, the putative benefits of residing in a biologically intact family can be negated if the biological father has a history of antisocial behaviour. That is, the group of children in Jaffee et al.'s (2001) study whose fathers exhibited low levels of antisocial behaviour appeared to benefit from paternal presence in the home, because these children exhibited *lower* rates of conduct problems. The reverse was true, however, for the group of children whose fathers exhibited high levels of antisocial behaviour; for this group, more prolonged coresidence with their fathers was associated with *higher* rates

of conduct problems. In light of Jaffee et al.'s (2001) findings, it is likely that characteristics of the biological parents, the wider family, and the ecological context will moderate the effects of family disruption/father absence on daughters' menarcheal timing (Tither & Ellis, 2008). This is consistent with past research that suggests that variation in paternal behaviour across families predicts daughters' menarcheal age (Ellis & Essex, 2007; Ellis et al., 1999; Steinberg, 1988). Because paternal behaviour and functioning in the family are likely to have important moderating effects on children's wellbeing, possible moderating variables that were tested in this study included perceived paternal warmth and paternal behavioural adjustment (Tither & Ellis, 2008). Consequently, among the biologically disrupted/father-absent sister pairs in this sample, I examined the interaction between birth order/birth spacing of siblings and fathers' dysfunctional behaviour in prediction of age at menarche. Therefore, a second broad but non-directional hypothesis—that differential exposure to paternal dysfunction would affect menarcheal age—was also tested (Tither & Ellis, 2008).

Life history theory as a metatheoretical framework

This thesis therefore aims to evaluate the explanatory worth of two opposing classes of explanation (i.e., causal vs. noncausal) for the reliable association between exposure to family disruption/father absence (due to parental relationship dissolution) and early pubertal timing in daughters. Importantly, the first class of explanation, which proposes that father absence due to parental separation/divorce may actually cause early pubertal timing in daughters (e.g., B. J. Ellis, 2004), is underpinned by an evolutionary-developmental psychology perspective. Evolutionary developmental psychology has been defined as

> the application of the basic principles of Darwinian evolution, particularly natural selection, to explain contemporary human development. It involves the study of the genetic and environmental mechanisms that underlie the universal development of social and cognitive competencies and the evolved epigenetic (i.e., gene-environment interactions) processes that adapt these processes to local conditions; it assumes that not only are behaviours and cognitions that characterize adults the product of selection pressures operating over the course of evolution, but so also are children's behaviours and minds. (Bjorklund & Pellegrini, 2002, p. 4)

As noted by B. J. Ellis (2005), life history theory (Charnov, 1993; Roff, 1992; Stearns, 1992) can be usefully employed as a metatheoretical framework for the study of pubertal timing from an evolutionary-developmental psychology perspective. That is, life history theory attempts to explain the timing of pubertal development (and other reproductive milestones) in terms of human beings having evolved strategies that determine their final distribution of limited metabolic resources across three competing domains: growth, maintenance and reproduction (B. J. Ellis, 2005).

Within the metatheoretical framework provided by life history theory, both *psychosocial acceleration theory* and *paternal investment theory* (detailed earlier in the Introduction) comprise middle-level theories of pubertal timing (B. J. Ellis, 2005). That is, they each apply the assumptions of life history theory to the question of how early rearing environments influence girls' subsequent pubertal timing. However, each of these theories has produced its own hypotheses and predictions that have subsequently been tested by researchers (B. J. Ellis, 2005). For example, Belsky et al. (1991) contend that depending on a girl's particular early rearing environment, one of two divergent

developmental pathways, thought to enhance reproductive success in that type of environment, will be canalised. One pathway is characterised by a stressful family context (including parental discord, high levels of stress, and/or inadequate financial resources) accompanied by negative child rearing practices (i.e., "harsh, rejecting, insensitive, [and/or] inconsistent" parenting) during infancy and early childhood (Belsky et al., 1991, p. 651). This type of early rearing environment produces (a) insecure attachments to parents and associated behavioral problems during childhood; (b) faster somatic development (i.e., earlier pubertal timing) during adolescence; and (c) faster reproductive strategies (i.e., "earlier sexual activity, short-term unstable pair bonds", [and] limited parental investment") in adulthood (Belsky et al., 1991, p. 651). The other pathway is characterised by a harmonious family context (including parental closeness and adequate financial resources) accompanied by positive child rearing practices (i.e., "sensitive, supportive, responsive, [and] positively affectionate" parenting) during infancy and early childhood (Belsky et al., 1991, p. 651). This type of early rearing environment produces (a) secure attachments to parents and fewer behavioral problems during childhood; (b) slower somatic development (i.e., later timing of puberty); and (c) a slower reproductive strategy (i.e., "later sexual activity, long-term enduring pair bonds, [and] greater parental investment") in adulthood (Belsky et al., 1991, p. 651). Thus, in Belsky et al.'s (1991) view, attachment style (i.e., insecure vs. secure) is determined by the girl's early rearing context and the child-rearing practices that she experiences. Whichever attachment style that she acquires from her early rearing context then influences her somatic development in adolescence (i.e., earlier vs. later pubertal timing), which in turn influences her reproductive strategy in adulthood (i.e., earlier vs. later onset of sexual activity; short-term vs. long-term pair bonds; and limited vs. greater parental investment) (Belsky et al., 1991).

Overview of the current study's aims

In sum, utilising a differential sibling exposure design affords the opportunity to conduct a genetically and environmentally controlled empirical study to test the potential causal influence of family disruption/father absence on daughters' menarcheal timing. Due to the genetic and environmental controls inherent in this design, it provides a unique method for testing whether more prolonged exposure to father absence and associated factors actually cause earlier menarcheal timing, and, if so, whether levels of paternal dysfunction moderate this (Tither & Ellis, 2008).

In conclusion, the main aim of this thesis is to empirically test an important central hypothesis—that family disruption/father absence causes earlier menarcheal timing in daughters—by conducting an empirical study that for the first time incorporates a genetically and environmentally controlled differential sibling exposure design. If this central hypothesis is supported, a broader hypothesis—that differential exposure to paternal dysfunction influences menarcheal age—can then be tested.

Chapter 2: Method

To ensure that all participants had already attained menarche, the current study required the use of a retrospective design (i.e., a design that is based on examining existing data) (Tither & Ellis, 2008). It also had a number of other important requirements. First, it required that the sisters in each pair be (at least) several years apart in age in order to ensure that sizeable enough differences existed between sisters from biologically disrupted families in terms of the nonshared environmental factor of interest (i.e., exposure to family disruption/father absence) (Tither & Ellis, 2008). Second, it required the younger sister in each biologically disrupted pair to have experienced biological family disruption that resulted in her (at least) no longer coresiding with her father prior to her attaining menarche (Tither & Ellis, 2008). Third, it required that information about exactly the same developmental time span (i.e., childhood through to early adolescence) be collected from both sisters in each pair in order to ensure that equivalent, complete data about age at menarche and family environment was obtained (Tither & Ellis, 2008). Finally, it required that all sisters from biologically intact families had experienced uninterrupted father presence from birth through to age 16 (Tither & Ellis, 2008). Therefore, in order to meet these requirements, all participants in the current study had to be at least 16 years of age (Tither & Ellis, 2008).

All sister pairs in the current study also had to be full biological siblings. However, obtaining a sample of full biological sister pairs whose family history involved the biological parents' relationship terminating prior to the younger sister attaining menarche and (at least) the younger sister not residing with her biological father after the parental relationship breakup was challenging (Tither & Ellis, 2008). Given the

rarity of such sister pairs and the difficulty of obtaining them using normal sampling methods, recruiting this particular group of participants for the current study necessitated the use of targeted advertising (Tither & Ellis, 2008). However, because the present research involved within-family comparisons that effectively controlled for both genetic and family-wide environmental effects, the disadvantages of using a self-selected sample such as this were mitigated (Tither & Ellis, 2008). Furthermore, as elaborated below, the demographics of the sample were representative of the general Christchurch population (Tither & Ellis, 2008).

Participants

Recruitment. Sixty-eight pairs of sisters from biologically disrupted families and 93 pairs of sisters from biologically intact families were recruited from urban areas in New Zealand (Tither & Ellis, 2008). This was achieved through the use of poster advertisements placed on campus notice boards and inside buses, and through advertising circulars distributed to approximately 6,500 residential letterboxes. Following an initial screening interview for family structure, respondents who met the selection criteria were invited to complete the main questionnaire (Tither & Ellis, 2008).

For the purposes of the current study, biologically disrupted families comprised families in which the biological parents' relationship had terminated through divorce or separation and the parents had ceased residing together prior to the younger sister attaining menarche (Tither & Ellis, 2008). Biologically intact families comprised families in which the biological parents had resided together (either in married or de facto relationships) for the duration of both of the sisters' childhoods (i.e., from birth to the age of 16) (Tither & Ellis, 2008). The younger sisters in the sample ranged from 16 to 44

years of age, with a mean age of 27.27 (*SD* = 6.6); the older sisters ranged in age from 19 to 52 years of age, with a mean age of 33.92, (*SD* = 6.86) (Tither & Ellis, 2008). In the biologically disrupted families, the average age of younger sisters when the parents' relationship terminated was 5.41 years (*SD* = 3.35); the average age of older sisters when this occurred was 11.79 years (*SD* = 3.61) (Tither & Ellis, 2008). The average age difference between sisters in biologically disrupted families was 6.48 years (*SD* = 2.06), and, in biologically intact families, it was 6.83 years (*SD* = 2.19) (Tither & Ellis, 2008). All participants were fluent speakers of English; information about other language(s) spoken in the home was not collected (Tither & Ellis, 2008).

Demographics. Due to the fact that the current sample was not randomly selected but self-selected (i.e., recruited using advertising), it was necessary to ensure that it was a demographically normal sample (Tither & Ellis, 2008). To check this, the current sample was compared to a general population sample, which comprised a birth cohort of women born in Christchurch, New Zealand, in 1977 (the Christchurch Health and Development Study [CHDS]) (Tither & Ellis, 2008). The CHDS data on biologically intact versus biologically disrupted families were provided by Ellis et al. (2003).

These demographic comparisons found marked similarities between the two samples (Tither & Ellis, 2008). The average age of participants in each sample was approximately the same. The occupational status of each father in each sample was classified using Elley and Irving's (1976) index of socioeconomic status for New Zealand. However, while the Elley–Irving coding classifies families into six socioeconomic groups based on the reported occupation of the father, the CHDS comparison sample had collapsed this classification down to a three-level classification as follows: 1 = Levels 1, 2 (professional, managerial); 2 = Levels 3, 4 (clerical, technical, skilled); and 3 = Levels 5, 6 (semiskilled, unskilled, unemployed). Therefore, in order to

allow meaningful comparisons to be made between the current sample and the CHDS sample, I also used the three-level classification system utilised by the CHDS researchers described above (see Fergusson & Woodward, 2000). Each participant was asked to describe in detail her father's primary occupation during her teenage years. Each participant was also asked to indicate the highest educational qualification attained by her mother, which was then coded using a three-level classification as follows: 1 = no formal educational qualifications; 2 = high school qualifications; and 3 = postsecondary certificate or degree. Mother's age at first birth was also reported by each sister. As shown in Table 1, the current sample and the CHDS sample closely resemble each other across all variables and across both biologically disrupted and biologically intact family types. In both samples, biologically intact families were more commonly found within the European New Zealander ethnic group than among other ethnic minorities (Tither & Ellis, 2008).

Furthermore, compared to their biologically disrupted counterparts, biologically intact families were characterised by later maternal age at first birth, higher paternal occupational status, and higher maternal educational status in both samples (see Table 1) (Tither & Ellis, 2008). Therefore, these comparisons revealed that the demographic profiles of sister pairs from both biologically disrupted and biologically intact families in the current sample were demographically normal, in that they closely resembled their respective family types in the general population, both in terms of their respective similarities and of their respective differences (Tither & Ellis, 2008).

Procedure

Poster advertisements were placed on the inside of Christchurch Metropolitan buses and on notice boards at Victoria University of Wellington and at the University of

Canterbury campuses in New Zealand (see Appendix A). The advertising circulars were distributed to residential letterboxes in selected mixed socioeconomic areas, primarily in Christchurch (see Appendix B) but also to households in Timaru, Blenheim, and Wellington (see Appendix C). These poster advertisements and circulars clearly described the type of family composition that was required for the study, what participation in the study would involve, and the researcher's contact details.

Potential participants who subsequently contacted the researcher were initially screened to determine whether or not their family composition fitted the research criteria. This initial screening interview was conducted via telephone using a questionnaire that requested the potential participant's contact details and asked questions about the structure of their family of origin (see Appendix D). This screening questionnaire was then used to identify appropriate sister pairs. If a sister pair met the research criteria, they were subsequently invited to participate in the study. Contact with both sisters was achieved by asking the sister who had first approached the researcher to contact her sister to see if she would also agree to participate in the study. If the potential participant who made the initial approach had multiple sisters, she was asked to first contact the sister who best met the research criteria and invite her to participate, and, if she declined, to contact the next most appropriate sister and so on. If both sisters agreed to participate, they were then invited to fill out the main questionnaire using one of three options: (a) completing a secure online version of the questionnaire by logging onto a specific website; (b) completing a hard copy of the questionnaire in the laboratory on the University of Canterbury campus; or (c) completing a hard copy that was posted out to their home address.

Participants who selected the online option were individually emailed a set of instructions and their login details (i.e., a unique sister pair code, a unique participant

number, and a unique password) (see Appendix E). All sister pairs who completed the online version were asked to complete it on the same day and at the same time as each other. They were asked to refrain from discussing the questionnaire with each other until such a time as they had both completed it. The information and consent forms (see Appendix F), and the debriefing form (Appendix G) were incorporated into the online version of the questionnaire. Once participants had completed the online questionnaire, they were emailed and asked to indicate which form of acknowledgement they wanted to receive (i.e., initially either a $10 petrol voucher or a $10 grocery voucher, but later in the study, participants were offered a $15 grocery voucher). This voucher was then posted out to them along with a receipt stating what form their acknowledgement had taken (i.e., the voucher type and its dollar value), and a postage paid return envelope. They were asked to sign and date the receipt and return it in the envelope provided.

Participating sister pairs who chose to complete a hard copy of the questionnaire in the laboratory on campus were asked to book an appointment at a time that suited both parties. The two sisters then completed the main questionnaire at the same time, but seated in separate rooms. Participants were first asked to read an information sheet that gave general information about the study, informed them that they could withdraw from the study at any time, and assured them of the complete confidentiality of all data gathered in the study and of their anonymity (see Appendix H). Once they had read the information sheet, participants who still wanted to participate in the study were then given a copy of the consent form to read, sign, and date (see Appendix I). Each participant was then given a copy of the main questionnaire (prenumbered with a unique sister pair code and a unique participant number) and an envelope.

Table 1

Demographic Comparisons in Biologically Disrupted and Biologically Intact Families: The Current Sibling Study versus the Christchurch Health and Development Study

	Current Sibling Study		CHDS	
Demographic variables	Biologically disrupted families	Biologically intact families	Biologically disrupted families	Biologically intact families
Race/ethnicity (%)				
European New Zealander	79	87	76	92
Maori/Polynesian	16	11	24	8
Other	5	2		
Fathers' occupation (%)				
Professional, managerial	13	30	13	26
Clerical, technical, skilled	52	54	46	57
Semiskilled, unskilled, unemployed	35	16	41	17
Mothers' education (%)				
Postsecondary certificate or degree	16	25	10	26
High school qualifications	32	31	20	31
No formal educational qualifications	52	44	70	43
Mean age (in years) of mothers at first birth (*SD*)	21.3 (3.4)	23.6 (3.8)	21.8 (4.3)	24.3 (3.9)

Note. CHDS = Christchurch Health and Development Study. From "Impact of fathers on daughters' age at menarche: A genetically and environmentally controlled sibling study," by J. M. Tither and B. J. Ellis, 2008, *Developmental Psychology, 44*, p. 1414. Copyright 2008 by the American Psychological Association. Adapted with permission of the author.

The questionnaire took each participant no more than 45 minutes to complete. After completing the main questionnaire, each participant was asked to place it in the envelope provided. They were then asked to read a debriefing sheet (see Appendix J) and asked if they had any questions about the study. Finally, participants were asked to indicate which form of acknowledgement they wanted to receive (i.e., initially either a $10 petrol voucher or a $10 grocery voucher, but later in the study, participants were offered a $15 grocery voucher). Once they had received their acknowledgement voucher, they were asked to sign and date the receipt provided.

Participants who chose to participate via post were each sent (in separate envelopes) a copy of the relevant information sheet (which varied depending on the incentive value being offered; see Appendix K and Appendix L), a copy of the consent form (see Appendix I), a copy of the main questionnaire (prenumbered with a unique sister pair code and a unique participant number) and a postage paid return envelope. Participants were asked to return the consent form (signed and dated) and the completed questionnaire in the postage paid return envelope. Sister pairs who completed the postal version of the main questionnaire were asked to fill it out at the same time on the same day. They were asked to refrain from discussing the questionnaire with each other until such a time as they had both completed and returned it. They were also asked to indicate which form of acknowledgement they wanted to receive (i.e., initially either a $10 petrol voucher or a $10 grocery voucher, but later in the study, participants were offered a $15 grocery voucher). Upon receipt of the completed consent form and questionnaire, participants were then posted out a copy of the debriefing form (see Appendix J), their acknowledgement voucher, a receipt stating what form their acknowledgement had taken (i.e., the voucher type and its dollar value) and a postage paid return envelope. They were asked to sign and date the

receipt and return it in the envelope provided.

Measures

Each participant, whatever her chosen method of participation, was asked to complete the main questionnaire without reviewing her answers with her participating sister. The following questions and scales were among the measures completed by each participant.[1]

Family structure. To determine whether sister pairs came from biologically disrupted or biologically intact families, participants completed an initial set of screening questions. Participants were initially asked, "Were you born into a two-parent household or a single-parent household?" If participants indicated that they were born into a single parent household, they were then asked whether they lived with their father or their mother. Of the sister pairs from single parent families, only those in which the younger sister had lived with the mother (and not the father) were included in the sample. This ensured that the younger sister in each pair had experienced an earlier onset/longer duration of father absence than had her older sister. This first group of sister pairs was classified as having come from biologically disrupted families.

Of the remaining participants (i.e., those not born into single-parent homes), the question was then asked, "If you were born into a two-parent household, are your birth parents still living together?" Participants who indicated that their birth parents were no longer living together were then asked, "Were you under the age of 16 when your birth parents stopped living together?" Those who indicated that they had been born into two-parent family homes but, prior to the age of 16 years, had experienced the loss of their birth father from the family home, were then asked to give their exact age (in

[1] Scales that are not germane to the current study were included in the main questionnaire, and are therefore not described here.

years and months) when their birth father first stopped residing with them. A number of other selection criteria were then applied in order to select appropriate sister pairs who met the research criteria for coming from biologically disrupted families. For example, participants who had experienced father absence as a result of the death of their birth father were excluded from the sample. Classification of girls into biologically intact or biologically disrupted families was based solely on birth father presence/absence status; the presence of stepfathers (or any other type of father figure) was ignored (Tither & Ellis, 2008). Therefore, in order to examine the effects of birth father absence on timing of menarche due to biological parental separation and/or divorce, the final sample was restricted to sister pairs from families in which: (a) the younger sister had experienced the loss of the birth father from the family home prior to menarche (but only due to parental separation and/or divorce, not to paternal mortality), and (b) the younger sister had primarily resided with her mother after the termination of her birth parents' relationship (Tither & Ellis, 2008). This ensured that in all cases, the younger sister had experienced an earlier onset/longer duration of father absence than had her older sister (with the *minimum* difference in duration equal to the age gap between the two sisters) (Tither & Ellis, 2008). This second group of sister pairs was also classified as having come from biologically disrupted families.

Finally, the remaining sister pairs, who had given affirmative answers to the question "If you were born into a two-parent household, are your birth parents still living together?", were classified as having come from biologically intact families (i.e., both sisters had resided continuously with both of their birth parents from birth through to (at least) the younger sister's 16th birthday).

Age at menarche. To obtain a 'retrospective' report of age at menarche from each participant, the questionnaire asked, "How old were you when you first menstruated

(got your period)?" Participants were asked to indicate their age (in both years and months) when they first menstruated. However, because previous research suggests that approximately 90% of women can accurately recall age at menarche to within 1 year (Bean, Leeper, Wallace, Sherman, & Jagger, 1979) but not to the actual month (Knaul, 2000), responses to this question were scored in years (Tither & Ellis, 2008). Earlier studies comparing self-reports of age at menarche collected during adolescence with recalled age at menarche after some time has elapsed have found retrospective reports such as these to be very reliable (Tither & Ellis, 2008). For example, participants' self-reports of their actual age at menarche collected in a prospective cohort study (Koprowski, Ross, Mack, Henderson, & Bernstein, 1999) were strongly positively correlated (r = .83) with their recalled age at menarche data collected in a retrospective study conducted several years later (Koprowski, Coates, & Bernstein, 2001). Moreover, in a number of other retrospective studies, reported age at menarche was strongly positively correlated even when measured at two extremely disparate time points (i.e., self-reports collected during adolescence and again 17–37 years later) with correlations in these studies ranging from .66–.79 (Casey, Dwyer, Coleman, Krall, Gardner, & Valadian, 1991; Cooper et al., 2006; Damon et al., 1969; Livson & McNeil, 1962; Must et al., 2002).

The current study found a weaker correlation between sisters' age at menarche in biologically disrupted families (n = 68, r = .24, p < .05) than in biologically intact families (n = 93, r = .36, p < .001) (Tither & Ellis, 2008).

Father warmth. To assess each participant's perception of parental warmth during childhood, the Parental Bonding Inventory ([PBI]; Parker, Tupling, & Brown, 1979) was completed by each sister. The PBI is a 'retrospective' scale administered to adults (over 16 years). It consists of two subscales, namely the 'care' scale, and the

'overprotection' or 'control' scale. Several studies have confirmed that the PBI has satisfactory validity and reliability (Parker, 1989). For example, respondents' sex, social class, education levels, and age do not appear to have any significant effect on their scoring of the PBI (Parker, 1990). Moreover, the particularly high concordance between sibling ratings found in Parker's (1990) study indicates that this instrument is validly assessing actual (as opposed to just perceived) parenting. PBI scores have also demonstrated remarkable stability. For example, in Parker et al.'s (1979) original study, the PBI demonstrated good test-retest reliability and internal consistency. A decade later, Wilhelm & Parker's (1990) study of 10-year test–retest reliability demonstrated that both paternal and maternal 'care' and 'over-protection' scores showed consistency over time (e.g., paternal 'care' scores were 21.9 vs. 21.4, $r = 0.72$; paternal 'overprotection' scores were 13.0 vs. 11.9, $r = 0.56$). Finally, a recent study has demonstrated that perceptions of parental 'care' and 'overprotection', as measured by the PBI, remained relatively stable over two decades, further attesting to the validity of this instrument (Wilhelm, Niven, Parker, & Hadzi-Pavlovic, 2005).

The PBI consists of 25 questions: 12 'care' items and 13 'overprotection' or 'control' items. It is used to measure participants' perceptions of their birth (or biological) parents' fundamental parenting styles, and is completed separately for biological mothers and fathers. When completing the PBI, each sister rated both their biological mother and father as they remembered them in the first 16 years of life (sample items: "My father/mother spoke to me with a warm and friendly voice" ('care' subscale); My father/mother seemed emotionally cold to me [reversed] ('care' subscale); "My father/mother did not want me to grow up" ('overprotection' or 'control' subscale'); "My father/mother invaded my privacy" ('overprotection' or 'control'

subscale'). These statements were responded to on a 4-point scale (1 = very unlike, 4 = very like).

Specifically, the 12-item 'care' subscale of the PBI completed for their biological fathers was used to measure participants' perceptions of paternal warmth during childhood. Items were keyed so that higher scores indicated higher levels of perceived paternal warmth (Cronbach's alpha = .93). Within sister pairs, the participants' ratings of perceived paternal warmth were strongly correlated (r [158] = .59, $p < .001$) (Tither & Ellis, 2008). Numerous studies have revealed that the 12-item 'care' subscale of the PBI is a reliable instrument over time (Wilhelm & Parker, 1990) and it is correlated with numerous aspects of mental health and behavioural adjustment in adolescents, including illicit drug and alcohol abuse (e.g., Chambers, Power, Loucks, & Swanson, 2001; Gerra et al., 2004; Martin, Bergen, Roeger, & Allison, 2004).

Father psychopathology. To assess whether participants had, during their childhoods, perceived paternal behavioural symptoms consistent with psychopathology, an 8-item checklist was completed by each sister (see Appendix M). This scale asked each participant to retrospectively indicate whether her father had suffered from a range of problems, including emotional problems, substance abuse problems, a history of psychiatric illness, imprisonment, and/or suicide attempts. The instructions read: "The next questions concern your father's mental health. Please think back to your childhood, up to the age of 16 years." Participants' responses to the checklist questions were made on a 3-point scale (yes, no, don't know). The first two items were included in order to assess relatively mild (and relatively common) behavioural issues ("Did your birth father suffer from nervous or emotional problems (such as anxiety or depression)?" "Did your birth father have trouble with drinking or other drug use?"). The final six items were included in order to assess far more serious

(and more uncommon) behavioural issues (e.g., "Did your birth father have any history of suicide/attempted suicide?" "Did your birth father have any history of offending involving violence?" "Did your birth father have any history of imprisonment?"). Sister pairs' perceptions of the presence of behaviours consistent with father psychopathology showed high rates of agreement, evidenced by high correlations between the number of items marked "yes" by both sisters (r [161] = .75, $p < .001$) (Tither & Ellis, 2008).

Computation of paternal dysfunction.

Due to the likelihood that some daughters would have limited knowledge of the existence (or lack thereof) of paternal behavioural symptoms consistent with psychopathology, each birth father was categorised into one of three broad groups. Each group reflected a different overall degree of paternal dysfunction (i.e., low, moderate, high), and each father's categorisation was based primarily on the paternal psychopathology ratings provided by his two daughters (i.e., the scores they had given him on the 8-item father psychopathology checklist), and secondarily on the paternal warmth ratings provided by them both (i.e., the scores they had given him on the 'care' subscale of the PBI) (Tither & Ellis, 2008).

Because paternal dysfunction is by definition a between-families variable (i.e., levels will differ between but not within families), its calculation was by necessity based on information about the father provided by both sisters in each pair (Tither & Ellis, 2008). In order to compute the values of this variable, each sister pair's data was individually analysed, and the two sisters' responses to each item were compared. For example, in cases where the sisters' responses for an item were in agreement, a *yes/yes* response was coded as a *yes*; a *no/no* response was coded as a *no*; a *don't know/don't know* response was excluded from the analysis (Tither & Ellis, 2008). However, in cases

where the two sisters' responses for an item were discrepant, the following rubric was used: (a) if one sister checked *yes* and the other checked *don't know*, the item was coded as a *yes;* (b) if one sister checked *no* and the other checked *don't know*, the item was coded as a *no*; and (c) if the two sisters' responses contradicted each other (i.e., one sister checked *yes* and the other *no*), the item was excluded from the analysis (Tither & Ellis, 2008). This consensual information was then used to make provisional paternal psychopathology ratings on a 3-point scale (i.e., low, moderate, and serious). Fathers who received no affirmative answers for any of the eight items (signifying no behavioural problems) received a provisional paternal psychopathology rating of 0 (low); fathers who received at least one affirmative answer for the first 2 checklist items (signifying moderate behavioural problems) but no affirmative ratings for the final six items were given a rating of 1 (moderate); and fathers who received affirmative answers for any one or more of the final six items (signifying severe behavioural problems) were given a rating of 2 (serious) (Tither & Ellis, 2008). These provisional psychopathology ratings and composited ratings of father warmth (averaged across both sisters in each pair) were negatively correlated, r (161) = -.46, p<.001) (Tither & Ellis, 2008).

In line with Jaffee, Moffitt, Caspi, and Taylor's (2003) research, the paternal dysfunction measure that was subsequently used as a moderating variable in the analyses was primarily based on these provisional paternal psychopathology ratings. However, because it is possible that some loving fathers will exhibit symptoms consistent with psychopathology, and some negligent fathers will not, father warmth ratings were used to adjust the provisional paternal psychopathology ratings in situations where clear, consensual evidence of either low or high paternal warmth were found (Tither & Ellis, 2008). Such adjustments were confined to cases where ratings of

paternal warmth were at odds with the provisional paternal psychopathology ratings. Because paternal warmth is by definition another between-families variable (i.e., levels will differ between but not within families), its calculation was by necessity also based on information about the father provided by both sisters in each pair. To elaborate, because each daughter had provided her own warmth rating for her father, these ratings were then averaged across each sister pair, and in cases where the subsequent composited rating was sufficiently low or high, it was used to adjust the father's provisional paternal psychopathology ratings using the following rubric: (a) if his two daughters consensually rated their father as low on warmth (i.e., if he received a composited father warmth score of 2 or below) but his provisional psychopathology rating was in the low to moderate range, his provisional score was increased by one point (i.e., from low up to moderate [i.e., up from 0-to-1] or from moderate up to serious [i.e., up from 1-to-2]); and (b) if his two daughters consensually rated their father as high on warmth (i.e., if he received a composited father warmth score of 3 or higher) but his provisional psychopathology rating was in the moderate to serious range then his provisional score was reduced by one point (i.e., from serious down to moderate [i.e., down from 2-to-1] or from moderate down to low [i.e., down from 1-to-0]) (Tither & Ellis, 2008).

The resulting *paternal dysfunction* scores were unevenly distributed across family types, with fathers from biologically disrupted families exhibiting higher levels of paternal dysfunction both moderate and serious) than did those from biologically intact families. To elaborate, of the fathers from biologically disrupted families (*n* = 68), 46% received a paternal dysfunction score of 2, 21% received a score of 1, and 34% received a score of 0. By contrast, of the fathers from biologically intact families (n = 93), 13% received a paternal dysfunction score of 2, 17% received a score of 1, and 69% received

a score of 0 (Tither & Ellis, 2008). This finding corresponds with previous New Zealand research which revealed that biological fathers who reside only some—or none—of the time with their children are more likely to have lower socioeconomic status; be unemployed; experience anxiety and mental ill-health; abuse alcohol and drugs; engage in more abusive and illegal behaviours; and accrue more criminal convictions than are their counterparts who permanently reside with their children (Jaffee et al., 2001).

Chapter 3: Results

Tests of major hypotheses

The central hypothesis in the current study—that sister pairs from biologically disrupted families would be more discrepant in terms of their menarcheal timing than would sister pairs from biologically intact families—was tested using a 2 X 2 mixed ANOVA. Because ANOVA is always a nondirectional test, but theory in this case had yielded a clearly directional prediction, it was necessary to first confirm that the direction of the observed effect was consistent with *a priori* expectation and, if so, a *half-tailed* test of significance could then be employed (i.e., the usual *p* value derived from the upper tail of the statistical distribution would then be halved) (Overall & Rhoades, 1986). This first analysis had age at menarche as the dependent variable, and included one within-subjects factor (Sister; older vs. younger) and one between-subjects factor (Family type; biologically disrupted vs. biologically intact). The results of this initial analysis are shown in Figure 1, and were consistent with the directional *a priori* prediction. No main effects for Sister or Family type were found, but the Sister X Family type interaction effect was statistically significant (F (1, 159) = 2.81, $p < .05$ [half-tailed]). Comparisons between sister pairs from biologically intact families and those from biologically disrupted families revealed differently oriented slopes (see Figure 1) (Tither & Ellis, 2008). Closer scrutiny of the means for age at menarche revealed that older sisters from biologically intact families had a slight tendency to attain menarche at an earlier age (M = 12.52 years; SD = 1.29) than their younger sisters (M = 12.66 years; SD = 1.49). However, the reverse was true for sisters in biologically disrupted families. In these families, it was younger sisters (M = 12.34 years; SD = 1.52) who tended to attain menarche at earlier ages than their older sisters (M = 12.65 years;

SD = 1.41). Therefore, in line with the causal hypothesis, lengthier exposure to father absence/family disruption *was* associated with earlier menarcheal timing in this sample (Tither & Ellis, 2008). However, this significant interaction accounted for only a small amount of the variance (partial $\eta^2 = .02$).

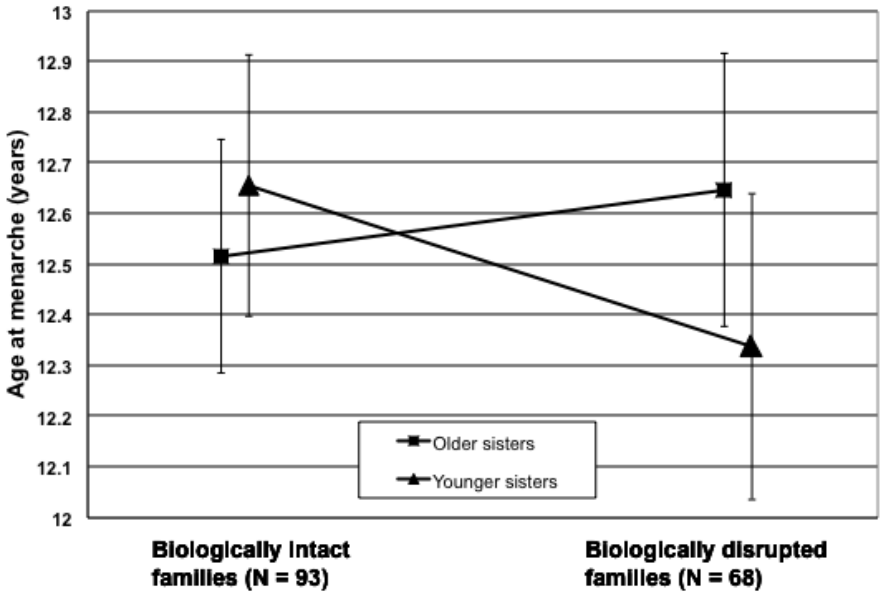

Figure 1. Differences between sisters in age at menarche in biologically intact versus biologically disrupted families. Error bars indicate 90% confidence intervals.

Note. From "Impact of fathers on daughters' age at menarche: A genetically and environmentally controlled sibling study," by J. M. Tither and B. J. Ellis, 2008, *Developmental Psychology, 44*, p. 1414. Copyright 2008 by the American Psychological Association. Adapted with permission of the author.

The next part of the analysis focused exclusively on the 68 biologically disrupted families in the sample. The possibility that fathers' functioning in these families (as measured by paternal dysfunction scores) would somehow moderate the effects of differential sibling exposure to family disruption/father absence on menarcheal timing was examined (Tither & Ellis, 2008). However, because in this case there were no clearly directional *a priori* predictions, a one-tailed test of significance was employed. A 2 X 3 mixed ANOVA was conducted, and this analysis had age at menarche as the

dependent variable, and included one within-subjects factor (Sister; older vs. younger) and one between-subjects factor (Paternal dysfunction; none, moderate, serious). A family-type factor (intact vs. disrupted) was excluded because in the biologically intact families, the small cell sizes for both moderate and serious paternal dysfunction had insufficient statistical power to detect interactions in a 3-way ANOVA (Tither & Ellis, 2008). The results of this analysis are shown in Figure 2. No main effects for Sister or Paternal dysfunction were found. However, a statistically significant interaction between Sister and Paternal dysfunction was found (F (2, 65) = 3.75, $p < .05$ [one-tailed]). This interaction indicated that the degree of discrepancy between sisters in age at menarche varied across the three different levels of paternal dysfunction, and accounted for 10% of the variance in age at menarche (partial $\eta^2 = .10$). Moreover, as Figure 2 suggests, it was earlier menarche in younger sisters from families with serious paternal dysfunction that was driving this significant interaction (Tither & Ellis, 2008).

To examine this interaction more closely, two contrasts were performed. The overall Type 1 error rate across comparisons was kept at $\alpha = .05$ by utilising the Bonferroni correction, where α was divided by two and set at .025. In the first contrast, age at menarche in the group of younger sisters from families with either no paternal dysfunction (M = 12.70 years; SD = 1.25) or moderate paternal dysfunction (M = 12.86 years; SD = 1.46) was compared with that of the group of younger sisters from families with serious paternal dysfunction (M = 11.84 years; SD = 1.61), using an independent-samples t-test (t (65) = 2.58, $p < .025$ [two-tailed], partial $\eta^2 = .09$). In the second contrast, age at menarche in older sisters from families with serious paternal dysfunction (M = 12.74; years; SD = 1.41) was compared with that of their younger sisters (M = 11.84 years; SD = 1.61), using a paired-samples t-test (t (30) = 2.69, $p <$ .025 [two-tailed], partial $\eta^2 = .20$). Thus, these contrasts revealed that younger sisters

from biologically disrupted families who were exposed to serious paternal dysfunction had significantly earlier menarcheal timing than either (a) younger sisters from biologically disrupted families who were not exposed to serious paternal dysfunction, or (b) their own older sisters (who had also been exposed to serious paternal dysfunction, but for a lengthier duration) (Tither & Ellis, 2008). Moreover, the magnitude of the within-family effects size was twice that of the between-family effect size (Tither & Ellis, 2008).

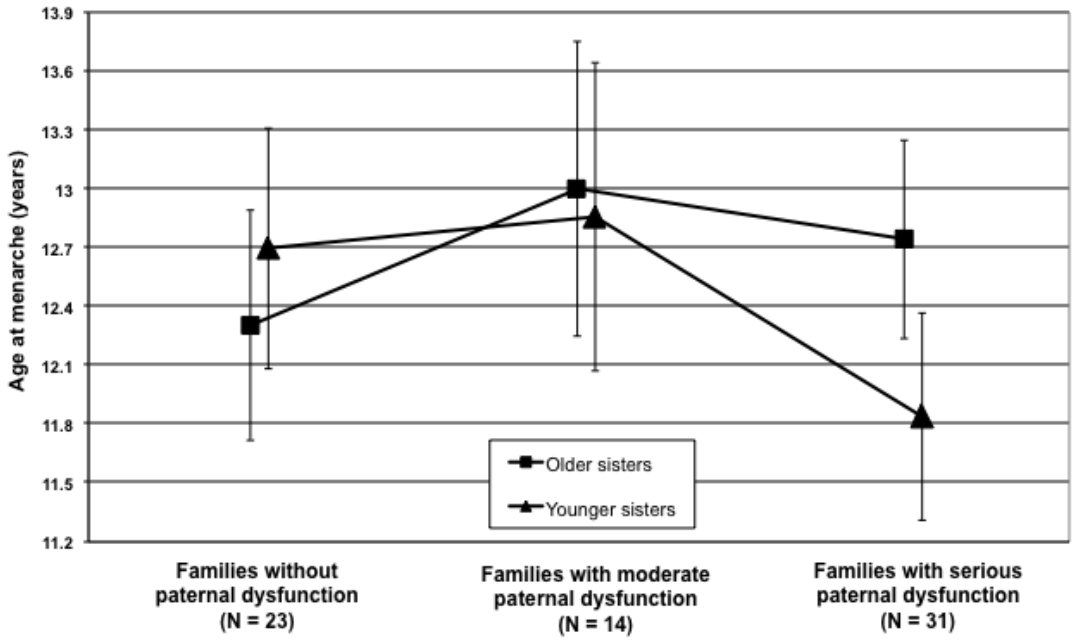

Figure 2. Differences between sisters in age at menarche as a function of paternal dysfunction in biologically disrupted families. Error bars indicate 95% confidence intervals.

Note. From "Impact of fathers on daughters' age at menarche: A genetically and environmentally controlled sibling study," by J. M. Tither and B. J. Ellis, 2008, *Developmental Psychology, 44*, p. 1415. Copyright 2008 by the American Psychological Association. Adapted with permission of the author.

Potential confounding effects of between-family differences

While the utilisation of a differential sibling exposure design provided the opportunity to examine the unique effect of differential sibling exposure to family

disruption/father absence (i.e., a specific measurable nonshared environmental factor) on age at menarche (i.e., a specific measurable developmental outcome), it is still possible that systematic differences between the two types of families in this sample could have affected this study's findings (Tither & Ellis, 2008). Specifically, although the current study (a) utilised a sibling comparison design which incorporated within-family analyses that simultaneously controlled for potential genetic and family-wide environmental confounds, *and* (b) included a biologically intact comparison group to control for main effects of birth order/birth spacing, it did not control for potential effects on pubertal timing of compositional differences that may exist between the biologically intact and disrupted families in the sample (Tither & Ellis, 2008). Specifically, despite somewhat inconsistent findings, past research examining the effects of family size and birth order on pubertal timing has suggested that number of siblings (especially older sisters) and birth order are two factors that may influence pubertal timing (reviewed in Malina et al., 1997; Matchock & Susman, 2006). Therefore, in the next part of the analysis, comparisons were made to ensure that the two types of families (i.e., biologically disrupted versus biologically intact) in the sample did not systematically differ in terms of (a) overall family size, (b) number of older sister, and (c) the relative birth order positions of the older and younger participating sisters (Tither & Ellis, 2008).

First, when the overall family-size calculations were restricted to full biological siblings only, the average number of children in disrupted families was 3.56 (SD = 1.18), whereas in intact families it was 3.96 (SD = 1.60). This difference was not statistically significant (t [159] = -1.81, p = .07; equal variances not assumed). Moreover, this slight imbalance in family size between the two types of families was not surprising, given that heterosexual couples who remain together have the opportunity to produce more

full biological offspring than do those who separate and/or divorce (Tither & Ellis, 2008). However, when half siblings were also included, this difference was eliminated. Specifically, when the calculations included both full and half siblings, the average number of children in disrupted families increased to 3.96 (*SD* = 1.42), which meant that they were now equivalent, in terms of their size, to intact families (Tither & Ellis, 2008).

Second, because past research had revealed that the number of older sisters is the most important aspect of family size for girls in terms of pubertal timing (Matchock & Susman, 2006), I also examined this variable's distribution across the two different family types. For the older sisters who participated in this study, the average number of older full biological sisters in intact families was .28 (*SD* = .63), whereas for their counterparts in disrupted families it was .16 (*SD* = .44), *t* (159) = -1.39, *p* = .17 (equal variances not assumed). By contrast, for the younger sisters who participated in this study, the average number of older full biological sisters in intact families was 1.76 (*SD* = .96), whereas for their counterparts in disrupted families it was 1.62 (*SD* = .71), *t* (159) = -1.10, *p* = .27 (equal variances not assumed). However, after counting both full and half siblings in disrupted families, the average number of older sisters increased to .22 (*SD* = .48) for the older participants from disrupted families and to 1.68 (*SD* = .76) for the younger participants from this family type, which meant that the two types of families were very similar in this regard (Tither & Ellis, 2008).

Finally, when birth order calculations were restricted to full biological siblings only, the average birth order position of older sisters from intact families was 1.69 (*SD* = 1.26), compared with 1.43 (*SD* = .74) for their counterparts in disrupted families, (*t* [159] = -1.64, *p* = .10; equal variances not assumed). By contrast, the average birth order position of younger sisters from intact families was 3.66 (*SD* = 1.46), compared

with 3.32 (*SD* = .98) for their counterparts in disrupted families, (*t* [159] = -1.72, *p* = .09; equal variances not assumed). Thus, in this sample, while older and younger sisters in both the biologically intact and the disrupted families exhibited similar relative birth order positions, sisters from biologically disrupted families tended to occupy slightly higher birth order positions than did their biologically intact counterparts (Tither & Ellis, 2008). However, when half-siblings were included in the calculations, this trend was no longer apparent (in the main, only sisters from disrupted families reported having half siblings) (Tither & Ellis, 2008). The inclusion of both full and half siblings in the calculations only served to increase the similarity between disrupted and intact families, because it moved the average birth order position of the older sisters within this family type up to 1.62 (*SD* = .86), and the younger sisters' up to 3.51 (*SD* = 1.07), respectively.

In sum, no meaningful differences in terms of three family composition variables thought to influence pubertal timing—family size, number of older sisters, and relative birth order positions—were found between the biologically intact and disrupted families in this sample, regardless of whether the calculation of these variables was restricted to full biological siblings, or included half siblings as well (Tither & Ellis, 2008). Therefore, in light of these analyses, potential confounding effects of between-family differences—in terms of these three family composition variables at least—on pubertal timing could be discounted (Tither & Ellis, 2008).

Chapter 4: Discussion

In line with previous research, the current study found a positive association between lengthier exposure to family disruption/father absence and earlier menarcheal age. As predicted by evolutionary developmental theories that posit that family disruption/father absence play a causal role in early pubertal timing, comparisons across the primary sample of sister pairs from biologically disrupted families and the matched control sample of sister pairs from biologically intact families revealed the existence of systematic differences in the magnitude of sibling differences in menarcheal age across family type (Tither & Ellis, 2008). Specifically, the current study found that within sister pairs from biologically disrupted families, younger sisters (who, prior to age 16, had experienced comparatively lengthier exposure to father absence than had their older sisters) tended to attain menarche earlier (on average 3-4 months earlier) than did their older sisters (who, prior to age 16, had experienced comparatively shorter duration of exposure to father absence than had their younger sisters) (Tither & Ellis, 2008). However, the reverse was true within sister pairs from biologically intact families; in these families, older sisters tended to attain menarche slightly earlier (on average approximately 1-2 months earlier) than did their younger sisters (Tither & Ellis, 2008). Although statistically significant differences were found between these two regression slopes, the effect size was relatively small, accounting for approximately 2% of the variance in age at menarche (Tither & Ellis, 2008).

The current study's findings regarding the effects of lengthier exposure to father absence on pubertal timing converge with previous research. However, its use of a genetically and environmentally controlled sibling comparison design—as opposed to the previously utilised correlational designs that confounded genetic and

environmental effects—constituted a major methodological advance in this area of inquiry (Tither & Ellis, 2008). The utilisation of this design extends previous research by incorporating within-family analyses, the findings from which lend plausible support to the proposal that family disruption/father absence and associated factors play a causal role in menarcheal timing (Tither & Ellis, 2008). Specifically, these within-family analyses suggest that the larger magnitude of sibling differences in menarcheal age found within sister pairs from biologically disrupted families is occurring independently of any genetic or shared family-wide environmental factors, and appears to be caused by the sisters' nonshared experiences associated with their differential exposure to family disruption/father absence and associated factors (Tither & Ellis, 2008). Furthermore, the between-family analyses that were conducted in the current study have established that this effect is not attributable to systematic differences between sister pairs from the biologically intact and the biologically disrupted families in terms of (a) family size, (b) number of older sisters, and (c) birth order (relative to other siblings) (Tither & Ellis, 2008).

However, the current study's relatively small effect size, although consistent with effect sizes reported in previous research examining the relation between lengthier exposure to family disruption/father absence and early menarcheal timing (Ellis & Garber, 2000; Moffitt et al., 1992; Quinlan, 2003; Surbey, 1990), warranted further investigation and explication (Tither & Ellis, 2008). Therefore, one possible explanation proposed for this finding—that paternal characteristics may moderate the main effect of family disruption/father absence on menarcheal age—was examined and is supported by the current study. Specifically, analyses conducted to investigate this possibility revealed that a sizeable moderating effect of paternal dysfunction superseded the small main effect of family disruption/father absence on menarcheal

age (Tither & Ellis, 2008). Interestingly, however, these analyses also revealed that family disruption/father absence seemed to noticeably accelerate menarcheal age in just one context: in biologically disrupted families in this sample characterised by serious paternal dysfunction (Tither & Ellis, 2008). Specifically, in this subset of biologically disrupted families comprising sister pairs who were differentially exposed to *both* family disruption/father absence *and* serious paternal dysfunction (e.g., substance abuse, violence, and other criminal behaviours), younger sisters (who, prior to age 16, had experienced comparatively lengthier exposure to family disruption/father absence but had resided with their seriously dysfunctional fathers for less time) attained menarche significantly earlier (i.e., 11 months earlier) than did their older sisters (who, conversely, prior to age 16, had experienced comparatively later/shorter duration of family disruption/father absence but had resided with their seriously dysfunctional fathers for longer) (Tither & Ellis, 2008). Moreover, this particular group of younger sisters from biologically disrupted families *also* attained menarche significantly earlier (i.e., 11 months earlier) than did their counterparts from biologically disrupted families who had experienced similarly high dosages of family disruption/father absence but whose families were not characterised by severe paternal dysfunction (Tither & Ellis, 2008).

When interpreting these findings, it is important to note that the relatively small effect of family disruption/father absence on age at menarche found in the current study may not be an accurate estimate of its actual effect in some children and adolescents (Tither & Ellis, 2008). This is because the current study did not examine the possibility that particular characteristics of the children themselves may also moderate this effect (Tither & Ellis, 2008). Specifically, while the current study tested whether this effect operated independently of genetic factors, the possibility that it might also occur

through interactions with genetic factors was not explored. As discussed in the Introduction, variations in both genes and development may make some daughters more or less vulnerable to family influences on menarcheal timing than others (see Belsky, 2012; Belsky et al., 2007). If this were the case, the relatively small effect of family disruption/father absence on menarcheal age documented in the current study would constitute an overestimate of its actual effect in less vulnerable populations of children and adolescents, but an underestimate of its actual effect in more vulnerable populations (Tither & Ellis, 2008).

A note on causal inference

Because the current study utilises a quasi-experimental design, it is able to support causal inferences. However, an important consideration when interpreting these results is that this sibling comparison methodology cannot conclusively demonstrate causation (Tither & Ellis, 2008). For example, consider the possibility that some girls possess a relatively rare trait, perhaps due to some *risky allele*, that causes them to experience early menarche *and* to exhibit behavioural problems severe enough to cause their biological parents to separate (Tither & Ellis, 2008). Girls who possess this trait are therefore at greater risk of experiencing both biological family disruption/father absence and early menarche than are girls who do not possess it. However, while such a trait ought to be randomly distributed across female siblings independent of birth order, it is also possible that it is not (Tither & Ellis, 2008). For example, the presence of such a trait in an older sister may significantly reduce the likelihood that any younger sisters will be born into the family (because the older sister's behavioural problems may cause the biological parents' relationship to terminate before any subsequent conceptions occur). Obviously, however, its presence

in a younger sister cannot reduce the likelihood of older sisters being born into the family in the same manner. Therefore, such an imbalance may result in a preponderance of younger sisters who possess this trait. If such a trait does exist in a population, researchers utilising a sibling comparison design who find that younger sisters are at heightened risk of attaining early menarche (relative to their older sisters) cannot definitively conclude that this difference is entirely caused by the sisters' differential exposure to family disruption/father absence (Tither & Ellis, 2008). Rather, some of this difference between sisters' age at menarche may be attributable to the differential distribution of the trait itself. Therefore, if such a trait does happen to exist, and, moreover, be unevenly distributed across younger and older sisters in the current study, it will have produced biased estimates of the effects of exposure to family disruption/father absence on age at menarche (Tither & Ellis, 2008). Hence, it is important to note that this differential sibling exposure research design can support credible—but not unequivocal—causal inferences.

An international adoption comparison

In order to explain the current findings, reference to the body of research examining pubertal timing in children who are internationally adopted early in life may be illuminating (Tither & Ellis, 2008). This research has found that children who are born into third-world countries but subsequently adopted into affluent Western countries experience significantly earlier pubertal timing than do either members of their birth cohort who remain in their countries of origin, or their same-age peers who are born and raised in their host countries (Domine, Parent, Rasier, Lebrethon, & Bourguignon, 2006; Mul, Oostdijk, & Drop, 2002; Teilmann, Pederson, Skakkebæk, & Jensen, 2006).

This body of research is intriguing for two reasons. First, it reveals that despite the chronic psychosocial and physical stress that surely would have characterised these foreign adoptees' early lives (including malnourishment, infection, neglect, and/or abuse) they are nonetheless at heightened risk of precocious pubertal timing (Tither & Ellis, 2008). Second, it indicates that the age at which impoverished children are internationally adopted moderates their subsequent risk for precocious pubertal timing. Specifically, this research has revealed that poor girls who are internationally adopted at *later* ages experience significantly earlier pubertal timing than do their peers who are adopted at *earlier* ages (Tither & Ellis, 2008). For example, a study examining menarcheal age in Indian and Bangladeshi girls who experienced significant early hardship but who were subsequently adopted into wealthy Swedish families found that the group of girls who were adopted at or over the age of 3 had a significantly earlier average age at menarche (mean = 11.1 years) than did the group who were adopted prior to the age of 3 (mean = 11.9 years) (Proos, Hofvander, & Tuvemo, 1991). Similarly, a large cohort study that compared pubertal timing in native Danes and girls adopted into Danish families from developing countries found higher rates of precocious puberty among the foreign adoptees than among the locally born Danes (Teilmann et al., 2006). However, this was especially true for later adoptees: while the rate of precocious puberty among foreign girls adopted before the age of 2 was 5 times higher than that of locally born Danes, it was 35 times higher among the group of foreign girls who were adopted after the age of 2 (Teilmann et al., 2006).

Interestingly, these adoption studies also indicate that alterations in factors other than nutrition are implicated in the subsequent pubertal precocity experienced by these international adoptees (Tither & Ellis, 2008). For example, although many of these internationally adopted children arrive in their new host countries in a malnourished

state, their height is generally more compromised than their weight (Parent et al., 2003). Moreover, the fact that many of these at-risk adoptees actually have normal body mass index at the time of their arrival (Parent et al., 2003) suggests that non-nutritional variables (such as stress) are influencing their subsequent early pubertal development (see especially Johnson, 2000). Consequently, it has been proposed that it is not only the improved nutrition associated with their adoption into these enriched environments, but also the concomitant reduction or removal of psychosocial stress, that is triggering the pubertal precocity evident in many of these international adoptees (Domine et al., 2006).

Collectively, therefore, these studies indicate that early exposure to significant physical and psychosocial stress followed by a marked improvement in living conditions is associated with earlier pubertal timing. Moreover, these findings indicate that lengthier exposure to stressors prior to adoption (i.e., later adoption into an enriched environment) is associated with greater risk for pubertal precocity. Specifically, out of all of these children, it is the later adoptees (i.e., the group who have experienced the most persistent exposure to impoverished environments in their early childhoods) who are responding to their arrival in greatly enriched new environments with the most markedly accelerated pubertal development. While this somewhat counter-intuitive finding requires further explication, Worthman (1999) argues that it concurs with the life history model, which, she states, "would predict that that girls experiencing persistent deprivation would react to a dramatic improvement in environmental quality by hastening reproduction in order to exploit a narrow window of resource availability" (p. 141). Thus, in line with Worthman's reasoning, the markedly accelerated sexual development found among these older adoptees may constitute an adaptive response that enables them to take advantage of a potentially

narrow window of reproductive opportunity offered by their new environments (Tither & Ellis, 2008).

When these international adoption findings and the current study's findings are examined together, two particular groups—which are comparable in terms of their experience of onset and potential alleviation of psychosocial stress—emerge as being the most susceptible to experiencing early menarche: older international adoptees and younger daughters exposed to seriously dysfunctional absent fathers. In terms of the current study, it is reasonable to assume that living with dysfunctional parents is one of the most significant sources of psychosocial stress for Western children (Tither & Ellis, 2008). As previously outlined in the Introduction, recent research focussing on the effects of coresidence with fathers with symptoms consistent with serious psychopathology has concluded that such living arrangements are in fact associated with a variety of deleterious effects for children (e.g., see Jaffee et al., 2001, 2003). In the biologically disrupted families in the current study, younger sisters stopped residing with their biological fathers at an average age of 5.4 years (Tither & Ellis, 2008). Therefore, the age at which biological family disruption/father absence was experienced by the subgroup of girls in the current study who were most susceptible to early menarche (i.e., younger sisters in the 31 sister pairs whose fathers exhibited symptoms consistent with serious psychopathology) is similar to the age at which the group of international adoptees who were most susceptible to early pubertal timing were adopted into greatly enriched environments (i.e., those adopted after the age of 3).

Consequently, in terms of the timing of alleviation of (or reduction in) exposure to a significant source of psychosocial stress, the experience of this group of younger sisters in the current study is comparable to that of the later international adoptees. That is, for these younger sisters, the timing of the probable stress reduction associated

with the departure of their seriously dysfunctional fathers from their family homes is analogous to the timing of a marked improvement in living circumstances experienced by later international adoptees. Furthermore, it is possible that this alleviation of a major source of psychosocial stress occurred during a sensitive age window for girls in each of these two groups—especially in terms of programming of maturation of the reproductive axis—with the result that pubertal timing was accelerated in these particular individuals (Tither & Ellis, 2008). And, if this were the case, then these improvements in circumstances presumably occurred too early in the developmental trajectories of the youngest children in the international adoption studies, and too late for the older sisters in the current study, to have markedly accelerated their pubertal maturation[2].

Therefore, when considered together, the findings of the current study and the international adoption studies outlined above strongly suggest that timing of exposure to severe psychosocial stress–in terms of both its onset and its reduction or alleviation–plays a prominent role in subsequent pubertal timing. This possibility raises an important interpretative issue for the present findings (Tither & Ellis, 2008). As outlined in the Introduction, when conceptualising and conducting this study, I adopted a *dose-response* metaphor. This decision was based on the assumption that lengthier exposure to family disruption/father absence would be associated with cumulatively

[2] When planning the present study, the reliable association between family disruption/father absence and early pubertal timing in daughters was construed as a dose-response relationship. That is, the younger the daughter was when the family disruption/father absence occurred, the lengthier/greater her exposure and the higher her risk of early pubertal timing. However, on a methodological note, had the older sisters in the present study been younger at the time of the parental relationship dissolution (i.e., had their *time-at-risk* been equivalent to that of their younger sister's), they too would have had lengthier/greater exposure to family disruption/father absence and, presumably, they too would have experienced the recalibration of pubertal timing exhibited by their younger sisters.

greater negative outcomes for daughters in terms of their subsequent pubertal timing (analogous to the effects of ongoing exposure to environmental toxins that become more toxic over time as they accumulate in the body) (Tither & Ellis, 2008). Consequently, for the purposes of this study, I treated timing of onset of father absence as a proxy for duration of exposure to family disruption/father absence. However, when attempting to interpret the current study's findings, it is important to note that a differential sibling exposure design such as this conflates the timing of onset and the duration of exposure to family disruption/father absence (Tither & Ellis, 2008). That is, because younger sisters from biologically disrupted families have experienced both earlier onset *and* longer duration of family disruption/father absence than their older sisters, it is not possible to unequivocally interpret the current study's findings regarding the effect of timing of onset versus the effect of duration of exposure to such psychosocial stressors. Therefore, a more parsimonious explanation of the accelerated pubertal timing found in younger sisters with seriously dysfunctional fathers may be that it constitutes a *sensitive period* effect rather than a dose-response effect (Tither & Ellis, 2008).

Adopting a sensitive period approach to explain the current study's findings is theoretically pertinent for two reasons. First, it concurs with Draper and Harpending's (1982) initial iteration of paternal investment theory that argued for the existence of a putative early sensitive period (during approximately the first 5 years of life) for the effects of father absence on daughters' subsequent sexual development (see the Introduction for a detailed account) (Tither & Ellis, 2008). Second, it concurs with recent developmental theories, especially Glen Elder's (1998) life course theory (Tither & Ellis, 2008). Essentially, Elder's theory has at its core a central premise: that life transitions affect developmental trajectories. The basic elements of the 'life course' are

the multiple trajectories of individuals and their developmental implications. Because life transitions (e.g., starting school, age at menarche, or age at first birth) always occur within a social context but these social contexts and time are in a constant state of flux, Elder argues that it is vital to take a holistic view of human development. He emphasises four principles, the first being historical time and place. This principle states that an individual's life course is embedded within, and influenced by, a particular time and place in history. Historical forces are important because they shape the timing of social transitions, and determine the opportunities and constraints that exist for any given cohort. Social transitions, in turn, influence behaviour and thus can have the effect of canalising particular developmental pathways. The second principle is timing in lives: the developmental implications of life transitions or experiences are determined in part by the timing of events in an individual's life. For example, differential effects on daughters' pubertal timing arising from the actual timing of exposure to family disruption/father absence are discussed below. The third principle is linked lives: individuals live their lives interdependently and, therefore, family members' fates are intertwined. For example, stressful social contexts affect individual families differently due to individual differences in family resources and strategies. Finally, the fourth principle is human agency: individuals construct their own life course via their decisions and behaviours. For example, individuals are often able to choose from a variety of possible pathways, but the opportunities and constraints encountered by individuals are also determined by their own particular social and historical milieu.

Therefore, when attempting to explain the systematically different developmental outcomes found in the current study within sister pairs from biologically disrupted families with serious paternal dysfunction, Elder's (1998) principles of timing in lives and linked lives seem most relevant. The crucial variable may not in fact be the

differential doses of both family disruption/father absence and serious paternal dysfunction experienced by the sisters in each pair; rather, it may be the actual timing of the family breakdown and of the concomitant reduction in exposure to paternal dysfunction in each individual sister's life course. Hence, experiencing a major reduction in exposure to serious paternal dysfunction (arising from disruption of the biological family) during early to middle childhood (i.e., within a putative sensitive developmental window)—but not during later childhood or adolescence—may constitute a key life transition that is capable of substantially altering a daughter's developmental trajectory in terms of her subsequent pubertal timing (Tither & Ellis, 2008).

Consideration of competing explanations

The reliable association between family disruption/father absence and earlier pubertal timing in girls has often been characterised in the literature as being a *noncausal* relationship. To recap, noncausal explanations of this association have posited that it arises from either genetic transmission of pubertal timing and associated behavioural characteristics, or from family-wide environmental confounds. However, alternative evolutionary-based developmental experience models that have also been proposed to explain this association contend that it may actually be *causal* (i.e., greater exposure to family disruption/father absence may actually cause earlier pubertal timing in girls) (e.g., Barkow, 1984; Blain & Barkow, 1988; Belsky et al. 1991; Draper & Harpending, 1982, 1988; B. J. Ellis, 2004). However, while extant research in this area of inquiry has reliably established that the association exists, such research has been unable to discriminate between these competing classes of explanation because, in the main, it has utilised correlational research designs that confound genetic and environmental effects (Tither & Ellis, 2008).

The current study's major contribution to this area of inquiry is its utilisation of a differential sibling exposure design. This design differs markedly from both the previously utilised correlational designs and the more recently utilised CoT design (Mendle et al., 2006). This is because it enables the measurement of the unique effects of a nonshared environmental factor—sister's differential exposure to family disruption/father absence—on a specific developmental outcome—age at menarche—while simultaneously controlling for both genetic and family-wide environmental effects (Tither & Ellis, 2008). Consequently, this differential sibling exposure design can be effectively employed to discriminate between these two competing classes of explanation (i.e., noncausal versus causal) for the association between family disruption/father absence and earlier pubertal timing, thereby effectively addressing the major methodological limitations of the extant research in this area of inquiry (Tither & Ellis, 2008).

This thesis is unusual because it has employed an empirical study to explicitly appraise three possible explanations for the reliable association found between family disruption/father absence and earlier pubertal timing in daughters. That is, rather than simply pitting the null hypothesis against a single theory, I have employed statistical hypothesis testing to appraise three different explanations for this association (i.e., two opposing classes of explanation: *causal* vs. *noncausal*) in terms of their explanatory value. Comparative theory appraisal such as this is unfortunately not a common enough practice in current psychological research (Haig, 2009), but this study can count among its main advantages the fact that it was designed in such a way as to be informative to theory (whatever its findings) while mitigating the effects of the *confirmation bias*.

As previously outlined in the Introduction, the two opposing classes of explanation currently under discussion (i.e., noncausal versus causal) for the

association between family disruption/father absence and earlier pubertal timing give rise to markedly different *a priori* patterns of predictions respectively. Specifically, if the association between lengthier family disruption/father absence and earlier menarche is noncausal (i.e., it arises from genetic or family-wide environmental confounds) I expected to find that sister pairs (irrespective of family type) would *not* report systematic within-pair differences in age at menarche (Tither & Ellis, 2008). That is, for a noncausal explanation to be supported, any systematic differences in reported menarcheal timing would *only* be found between—not within—the two different family types. By contrast, however, if the relationship between lengthier exposure to family disruption/father absence and earlier menarcheal timing is causal, I expected to find that younger sisters would report systematically earlier menarche than their older sisters in families in which: (a) full biological sisters were discrepant in age, and (b) younger sisters had lengthier exposure to family disruption/father absence than did their older sisters (Tither & Ellis, 2008).

In light of these *a priori* expectations, the current study's findings suggest that neither the genetic nor the family-wide environmental confounds explanations fully account for the association found in this study between family disruption/father absence and earlier pubertal timing (Tither & Ellis, 2008). That is, because systematic sibling differences in menarcheal age were not found within biologically intact sister pairs, these data do not support a noncausal explanation. Rather, and in line with the alternative causal explanation, systematic differences in reported age at menarche were *only* found within the biologically disrupted sister pairs. In sum, therefore, because sister pairs who experienced family disruption/father absence exhibited systematically greater differences in reported age at menarche than did sister pairs who resided with their biological fathers throughout their childhoods (i.e., up until the age of 16), the

current study's pattern of findings support the causal rather than the noncausal explanation (Tither & Ellis, 2008).

The present data, therefore, lend support to my central hypothesis that the birth order/age discrepancy (older versus younger) between sisters would interact with family type (biologically disrupted vs. biologically intact) to predict the magnitude of sibling differences in menarcheal age. However, the current findings also suggest that the small, but nonetheless well-replicated effect of family disruption/father absence on pubertal timing is not equally driven by all younger sisters who are exposed to family disruption/father absence. Rather, the effect seems to be being driven by the relatively small subset of girls who are exposed to serious paternal dysfunction in early childhood, and who are then, prior to attaining menarche, exposed to family disruption and father absence (Tither & Ellis, 2008).

In sum, these data lend plausible support to the argument advanced by evolutionary-based developmental experience models that family disruption/father absence and associated factors actually cause earlier pubertal development in girls (Tither & Ellis, 2008). However, because paternal characteristics appear to be moderating this association, these data more specifically support an interaction—in the biologically disrupted families at least—between fathers' functioning in the family and daughters' sexual development (Tither & Ellis, 2008). That is, in the current study it was not lengthier exposure to family disruption/father absence *per se* that had the most causal influence on pubertal timing; rather, it was differential exposure (in terms of timing and/or amount) to fathers who exhibited symptoms consistent with psychopathology that seemed most influential (Tither & Ellis, 2008). By contrast, and consistent with Scarr's (1992) concept of "good enough" parenting, in families in which biological fathers did not exhibit symptoms consistent with serious psychopathology

(i.e., fathers who were functioning within a normal range), such differential exposure to family disruption/father absence was not associated with increased risk of earlier age at menarche in daughters (Tither & Ellis, 2008).

These findings, therefore, call into question the assumption that pubertal timing in daughters is particularly sensitive to father absence *per se* as a proxy of paternal investment (see Ellis et al., 1999). Rather, it is the interaction between family disruption/father absence and fathers' functioning in the family that seems to be more influential in terms of daughters' subsequent pubertal timing. Moreover, these findings highlight the need to revise evolutionary–developmental models to reflect the importance of sensitive periods to changes in family conditions during daughters' development, especially in terms of their potential to exacerbate daughters' susceptibility to earlier pubertal timing (Tither & Ellis, 2008).

Metatheoretical considerations

On a final metatheoretical note, it seems useful to relate my thesis' findings to Nikolaas Tinbergen's (1983) explanatory framework and, in particular, to his four questions concerning any developmental phenomenon (i.e., "What is it for?"; "How did it evolve?"; "How did it develop?"; and "How does it work?"). In terms of understanding any type of developmental phenomenon, it is vital to distinguish among its ultimate, distal, and proximate causes (Tinbergen, 1963).

An ultimate cause is concerned with "why" a phenomenon occurs (i.e., the "What is it for?" question) (Belsky et al., 1991). In the case of the present study's findings, I would contend that the ultimate function of earlier menarcheal timing in younger sisters from biologically disrupted/father absent homes (with fathers who exhibited symptoms consistent with serious psychopathology) would be to enhance their

reproductive fitness. This phenomenon may have evolved in the environment of evolutionary adaptedness because it conferred a fitness benefit to those girls who responded to sudden and significant alleviations of psychosocial stress with accelerated somatic development (i.e., the "How did it evolve?" question).

Proximate and distal causes are concerned with "how" a process or phenomenon actually occurs (i.e., the "How did it develop?" and "How does it work?" questions) (Belsky et al., 1991). A proximate cause is temporally close to a given process or phenomenon, whereas a distal cause is further away from the proximate cause but is nonetheless inextricably linked to it (Belsky et al., 1991). Therefore, in the case of the present study's findings, I would contend that when extreme contextual stress caused by the presence of a father with symptoms consistent with serious psychopathology (i.e., a distal cause) is alleviated by that father's departure from the home (i.e., a proximate cause) this environmental improvement then has the effect of accelerating somatic development in girls. Thus, the resulting earlier menarcheal timing (with its associated earlier ovulatory cycles) then allows those daughters who are young enough at the time of their father's departure to take advantage of the window of opportunity afforded by this sudden and significant environmental improvement by rendering them physically capable of reproducing earlier.

Limitations of the current study

Detailed acknowledgement of limitations is often lacking in empirical research papers, which is unfortunate given that their inclusion serves several valuable purposes (Ioannidis, 2007). Specifically, limitations can assist the reader to contextualise the research findings, interpret their validity, and decide how credible the conclusions that have been drawn from them are (Ioannidis, 2007). Moreover, they provide some

measure of a given study's heuristic worth. Therefore, at this point, acknowledging that the current study has (at least) eight important limitations that can be used to direct future research seems useful. The first four limitations pertain to sample characteristics: the size of the sample of sister pairs from biologically disrupted families; the methods used to recruit the sister pairs from biologically intact and biologically disrupted families; the comparability of the sister pairs from biologically intact and biologically disrupted families in term of levels of paternal dysfunction; and the age range of participants across the total sample. The fifth and sixth limitations relate to the reliability and/or accuracy of the retrospective reports used to determine (a) ratings of paternal dysfunction and (b) age at menarche. The seventh limitation pertains to methodological issues regarding the control of genetic confounds, while the final limitation pertains to the absence of testing for mediating mechanisms. Each of these limitations is detailed in turn below, along with proffered suggestions (where applicable) for addressing it in future studies.

First, and most importantly, the relatively small sample size on which the present research is based may have produced inaccurate parameter estimations (Tither & Ellis, 2008). Future research could address this issue by studying larger numbers of sister pairs from biologically disrupted families in which sisters have experienced differential exposure to both family disruption/father absence and serious paternal dysfunction (Tither & Ellis, 2008).

Second, in order to be able to make the relevant sibling comparisons required to test the central hypothesis, extensive screening procedures were employed to identify a sufficient number of biologically intact and biologically disrupted families who met the research criteria. Consequently, this scrupulous screening process, while a vital element of the design, meant that a significant number of families were excluded from the study.

This situation raises the question of how much these research findings, based only on families that met the research criteria, would generalise to excluded families (Tither & Ellis, 2008). Although the answer to this question is not currently known, this is unlikely to be a major issue for the present research due to the fact that the demographics across the current study's total sample closely resemble those of a same-aged birth cohort from Christchurch, New Zealand (Tither & Ellis, 2008).

Third, while the present research compared the magnitude of sibling differences in age at menarche across a primary sample of sister pairs from biologically disrupted families and a matched control sample of sister pairs from biologically intact families, closer analyses revealed that levels of paternal dysfunction were not equivalent across these two family types. Unsurprisingly, seriously dysfunctional fathers were more prevalent in the biologically disrupted families, with the result that the primary and comparison samples were not symmetrical in this important regard. Therefore, it will be important in future research for comparisons to be made across primary samples of sister pairs from biologically disrupted families and matched control samples of sister pairs from biologically intact families that more closely resemble the biologically disrupted families in terms of levels of paternal dysfunction. That is, future samples should be better matched, not just in terms of the influential aspects of family composition affecting age at menarche that were considered in this study (i.e., birth order (relative to other siblings), birth spacing (age discrepancies between sisters), and number of older sisters), but also in terms of their relative distributions of paternal dysfunction. Although it would prove to be quite challenging to locate large numbers of sister pairs from families who have remained biologically intact despite the presence of serious paternal dysfunction, it would nonetheless be a very worthwhile endeavour in order to address this particular limitation.

Fourth, due to considerable variation in participants' ages in the sample, it is possible that the retrospective reports used to determine levels of paternal dysfunction in the current study were unreliable (Tither & Ellis, 2008). First, because participants in this research ranged in age from 18 to 53 years, it is possible that their recollections of their biological fathers were systematically affected by the (sometimes sizeable) differences between them in both the quantity of time that had elapsed since their childhoods, and their intervening life experiences (Tither & Ellis, 2008). That is, when younger respondents (e.g., those under 20 years of age) were asked to report on their father's behaviours and his disposition during their childhoods (e.g., his levels of drug and alcohol consumption, antisocial behaviours, and interpersonal warmth) they were basing their perceptions on relatively recent experiences. Hence, they may have provided more reliable reports than did older respondents (e.g., those over 30 years of age), whose perceptions were based on more temporally distant experiences. Second, individual responses given to these items might well vary over time due to the effects of both the passage of time and the participants' own life experiences (Tither & Ellis, 2008). For example, a daughter's perception of her father's substance abuse or interpersonal warmth during her childhood may differ depending on whether her recollections of him are sought at 18 years of age, or at 40 years of age. This limitation could be addressed in future research in (at least) two different ways: (a) by employing more rigorous screening procedures in order to obtain samples of sister pairs from biologically intact and biologically disrupted families that do not exhibit such wide age ranges, or (b) by employing a prospective design, which would obviate the need to employ retrospective reports from siblings in order to assess paternal dysfunction.

Fifth, both limited knowledge and variations in sibling knowledge of paternal behaviours and dispositions may have affected the accuracy of retrospective reports

used to determine levels of paternal dysfunction (Tither & Ellis, 2008). Specifically, in cases where the daughter's knowledge of her father was very limited, it is possible that rather than being accurate assessments of his actual behaviours and disposition during her childhood, her retrospective reports instead comprised subjective ratings reflecting her feelings and beliefs about him (Tither & Ellis, 2008). Although such variations in sibling knowledge are potentially problematic, the fact that the current study found high levels of agreement between siblings from biologically disrupted families in terms of their ratings of paternal symptoms consistent with psychopathology supports the validity of this assessment method in this case (Tither & Ellis, 2008). However, future research could employ a prospective design to obviate the need to use retrospective reports from siblings to assess paternal dysfunction. This would allow assessments of a father's functioning in the family to be based on objective data (such as the biological father's police records); self-report data (such as interviews with the father himself); and interviews with his daughters themselves and other close family members (especially the biological mother).

Sixth, the retrospective reports that were used in this study to assess age at menarche (to the nearest year) were potentially inaccurate (Tither & Ellis, 2008). Although repeated measurements of respondents' reported age at menarche over time have revealed high correlations (e.g., see Must et al., 2002), such retrospective reporting is potentially inaccurate for two reasons. First, increases in the amount of time elapsed between actually attaining menarche and recalling it tend to be accompanied by commensurate decreases in respondents' accuracy of recall. Second, such repeated measurements of reported age at menarche over time have revealed significant within-person variability (Koo & Rohan, 1997). However, despite these potential limitations, research by Casey et al. (1991) found that 84% of women (mean age = 50 years)

managed to accurately recall their age of menarche to within 1 year. Therefore, given the age of this sample, it is not unreasonable to expect that participants' reports of their menarcheal age (to the nearest year) in the current study were relatively accurate (Tither & Ellis, 2008). However, future research could address this limitation by employing a prospective design that combines objective measures (such as physical examinations) with parental and self-reports, in order to ensure that participants' menarcheal ages are recorded accurately (Tither & Ellis, 2008).

Seventh, the method used in the current study to control for genetic confounds is potentially problematic because it assumes (but does not ascertain) that the sister pairs in the total sample comprised full biological siblings (Tither & Ellis, 2008). Specifically, using randomisation of genetic effects across the birth order—without incorporating paternity testing of the sisters—in order to control for genetic confounds may produce biased estimates because this method does not take into account an estimated extrapair paternity rate of approximately 2% (Simmons, Firman, Rhodes, & Peters, 2004). Moreover, as a consequence of pre-existing relationship instability prior to actual relationship dissolution, the rate of extrapair paternity is likely to be higher in biologically disrupted families than in biologically intact families (Tither & Ellis, 2008). Therefore, the possibility that the current study's estimates of the causal effect of differential exposure to family disruption/father absence were biased by the presence of extrapair paternity cannot be ruled out (Tither & Ellis, 2008). However, in order for extrapair paternity to generate biased estimates, the following three conditions would need to be met: (a) the magnitude of the extrapair paternity rate within the sample would need to be substantively meaningful, (b) the distribution of extrapair paternity across sisters' birth order would need to be non-random, and (c) cases of extrapair versus inpair paternity would need to result in systematically (directionally) different

genetic effects on age at menarche (Tither & Ellis, 2008). Although it is unlikely that the current study met all three conditions, future research could address this limitation by incorporating DNA testing in order to ensure that all sister pairs in the research comprised full biological siblings (Tither & Ellis, 2008).

Finally, the current study did not test for mediating mechanisms (Tither & Ellis, 2008). In order to address this limitation, future research needs to be conducted in order to identify the intervening factors that explain the diverging patterns of pubertal development found between age-discrepant sisters from biologically disrupted families with seriously dysfunctional fathers in this study (Tither & Ellis, 2008). One promising candidate is siblings' nonshared childhood experiences. Therefore, possible intervening experiences that could be examined may include (but are not restricted to) siblings' differential exposure to the following: early childhood abuse and/or neglect; parental discord; the biological father after the parents' separation; and coresidence with stepfathers and other males (Tither & Ellis, 2008). Moreover, these intervening factors may be associated with particular neuroendocrine processes, which also need to be identified. These may include (but are not restricted to) differences between siblings in production of growth hormones and patterns of fat deposition prior to puberty (Tither & Ellis, 2008).

Implications of the current study's findings

In sum, it is reasonable to conclude from the present research that under certain circumstances exposure to family disruption/father absence markedly accelerates daughters' pubertal timing. Importantly, however, because daughters' pubertal timing was only accelerated under certain circumstances, the current data indicate that this particular type of alteration in family structure does not uniformly affect the pubertal

timing of all girls who experience it. Specifically, in the current study, a particular combination of factors had to be present before daughters' pubertal timing was accelerated: early exposure to serious paternal dysfunction (ascertained in this case from daughters' ratings of their father's behaviour and interpersonal warmth during their childhoods) followed by the early departure of the father from the family home (Tither & Ellis, 2008). Therefore, the current study has illuminated modifiable determinants of early menarcheal age that could be targeted for intervention (Tither & Ellis, 2008).

Conclusion

Because it is vital that all risk factors for accelerated pubertal timing, and their nuances, be clearly understood, this study is both timely and important. As clearly outlined in the Introduction, early pubertal development is associated with a wide range of negative physiological and psychosocial outcomes for girls. Moreover, for girls in New Zealand, the likelihood of experiencing serious paternal dysfunction followed by family disruption/father absence is relatively high. While the current study's findings may be particularly informative for biological parents who are contemplating terminating their relationship and thereby altering their domestic arrangements, they may also be useful for biological parents whose offspring are already living apart from their biological fathers. Enhanced knowledge of the potential risks associated with such common alterations to living arrangements may allow biological parents to make decisions that minimise potential harm to their children. Moreover, the current study's findings, which indicate that accelerated pubertal timing is most likely to be found in the group of daughters with seriously dysfunctional fathers who stopped residing with

them at an early age, may well be reassuring for parents of daughters in biologically disrupted families who do not fit these particular criteria.

Finally, and most importantly, prior to the present research, it was not known whether the reliable association between family disruption/father absence and early pubertal timing constituted a causal or a noncausal relationship. A first, important step, therefore, towards illuminating the link between father absence and early onset of puberty in girls was to determine causality. The unique contribution of the current study to this important area of inquiry is its employment of a differential sibling exposure design. This was utilised to first test the central hypothesis that the birth order/age discrepancy (older versus younger) between sisters would interact with family type (biologically disrupted vs. biologically intact) to predict the size of sibling differences in menarcheal age, and to subsequently test for potential moderating effects of paternal dysfunction. By employing this unique genetically and environmentally controlled sibling comparison design, the present research was able to distinguish between these competing explanations. That is, its findings plausibly support a causal rather than a noncausal explanation for the association between father absence and earlier pubertal timing in girls. However, this study has also revealed that this well-replicated association is more nuanced than previously thought, because it is clearly moderated by fathers' functioning in the family.

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Appendix A

College of Science
Department of Psychology
Tel: +64 3 364 2902
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SISTERS WANTED!

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This study has been reviewed and approved by the University of Canterbury Human Ethics Committee.

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Sisters Wanted: Earn $15
PH: 0800 862 788
(0800 UOC STU)

Appendix B

College of Science
Department of Psychology
Tel: +64 3 364 2902
Fax: +64 3 364 2181

UNIVERSITY OF CANTERBURY
Te Whare Wānanga o Waitaha
CHRISTCHURCH NEW ZEALAND

SISTERS WANTED!

ARE YOU A FEMALE AGED BETWEEN 18 AND 40 YEARS?

DO YOU HAVE A BIOLOGICAL SISTER
WHO IS AT LEAST 5 YEARS APART FROM YOU IN AGE?
(either older or younger)

EARN $10 EACH

By completing a simple questionnaire that takes about 45 minutes.

We are looking for women to participate in a study investigating the influence of family relationships on behaviour.

- You will complete a questionnaire that asks about your teenage years, your life now, and your family relationships.
- If your sister is willing to participate, she will also complete the questionnaire.

The questionnaire can be completed either in person or by post. Once the questionnaire is completed, you will be paid $10.00. Your sister will also be paid $10.00, if she participates.

Your responses in this research are strictly confidential and anonymous. This study has been reviewed and approved by the University of Canterbury Human Ethics Committee.

If you are interested, please contact **Jacqueline Tither** (Phone **364 2987; extn. 7845 or email: jmt68@student.canterbury.ac.nz)** for more information.

University of Canterbury Private Bag 4800, Christchurch 8020, New Zealand.
Tel: +64 3 366 7001, Fax: +64 3 364 2174 **www.canterbury.ac.nz**

Appendix C

College of Science
Department of Psychology
Tel: +64 3 364 2902
Fax: +64 3 364 2181

UNIVERSITY OF CANTERBURY
Te Whare Wānanga o Waitaha
CHRISTCHURCH NEW ZEALAND

SISTERS WANTED!

ARE YOU A FEMALE AGED BETWEEN 18 AND 40 YEARS?

DO YOU HAVE A FULL BIOLOGICAL SISTER WHO IS AT LEAST 5 YEARS APART FROM YOU IN AGE?
(either older or younger)

DID YOUR BIRTH PARENTS SEPARATE or DIVORCE BEFORE YOUR 18th BIRTHDAY?

EARN $15 EACH

by completing a simple questionnaire that takes about 45 minutes.

We are looking for women whose parents are divorced or separated to participate in a study investigating the influence of family relationships on behaviour.

- You will complete a questionnaire that asks about your teenage years, your life now, and your family relationships.
- If your sister is willing to participate, she will also complete the questionnaire.

The questionnaire can be completed either by post or via the internet (by logging on to a secure website). Once the questionnaire is completed, you will be paid **$15.00**. Your sister will also be paid **$15.00**, if she participates.

Your responses in this research are strictly confidential and anonymous. This study has been reviewed and approved by the University of Canterbury Human Ethics Committee.

If you are interested, please contact **Jacqueline Tither** on

Freephone: 0800 862 788 (0800 UOCSTU)

University of Canterbury Private Bag 4800, Christchurch 8020, New Zealand.

Appendix D

Background Information

Full Name: _____

Home Address: _____

_____

_____

Home Phone Number: _____

Cellphone Number: _____

Email: _____

Mother's Home Address: _____

(if different) _____

_____

Home Phone Number: _____

Cellphone Number: _____

Email: _____

Father's Home Address: _____

(if different) _____

_____

Home Phone Number: _____

Cellphone Number: _____

Email: _____

Sister's Home Address: _____

(if different) _____

_____

Home Phone Number: _____

Cellphone Number: _____

Email: _____

1) Were you born into a:

Two-parent household? ☐ Please go to **Question 3**

Single-parent household? ☐

2) If you were born into a single-parent household, who did you live with?

Your mother? ☐

Your father? ☐

Please turn to **Page 3**

3) If you were born into a two-parent household, are your birth parents still living together?

Yes ☐ Please turn to **Page 3**

No ☐

If no,

4) Were you under the age of 18 when your birth parents stopped living together?

No ☐ Please turn to **Page 3**

Yes ☐

If yes,

5) After your birth parents stopped living together, whom did you live with?

☐ Primarily my mother

☐ Primarily my father

☐ Parents had equal custody

☐ Other (Please describe) _____

6) How old were you when your birth parents first stopped living together?

_____Years & _____Months

Please turn to **Page 3**

Please record the following information about your brothers and sisters in the table below:

1) Age and first initial. The age and first initial of each of your brothers and sisters (**including yourself**) from oldest to youngest in the spaces provided.

2) Gender. The gender of each of your brothers and sisters (**including yourself**) by placing either an **F** (for female) or **M** (for male) in the box directly underneath their corresponding age.

3) Relation. The biological relationship of each one of your brothers and sisters to you:
   Me = For yourself, write "me"
   Full = If you share the same two biological parents, write "full"
   Half = If you share only one biological parent, write "half"
   Step = If you have different biological parents altogether (including adopted brothers and sisters), write "step"

Sample Table Only.

Order ==>	1st Born	2nd Born	3rd Born	4th Born	5th Born	6th Born	7th Born	8th Born	9th Born	10th Born
Age and first initial	44-J	43-M	41-S	39-B	23-R	18-N				
Gender	F	M	M	M	F	F				
Relation	step	step	full	me	half	half				

Please record your siblings details in the following table.

Order ==>	1st Born	2nd Born	3rd Born	4th Born	5th Born	6th Born	7th Born	8th Born	9th Born	10th Born
Age and first initial										
Gender										
Relation										

Appendix E

Subject: University of Canterbury 'Sisters Wanted' Study

Dear Participant

Thank you for volunteering to participate in this study.

Please print off a copy of the instructions given below, and follow them carefully.

1) Using Internet Explorer as your Browser, please paste this website into your browser window.

   http://db.psyc.canterbury.ac.nz/ellistest/info.cfm

2) Your Login Name is: (e.g., abc123)
   Your Password is: (e.g., none)

3) If something should happen to interrupt you while you are in the process of completing the questionnaire (e.g. your computer crashes, you have to log-off etc.,) please log on again using the new Login Name and Password below and start completing the questionnaire again from the beginning:

   New Login Name: (e.g., abc123a)
   Your Password is: (e.g., nonea)

4) While you are completing the questionnaire, if you are asked to give a number, please type it in numerals, not words (e.g. type '42' not 'forty-two' in the space provided).

Please note that although the information sheet says that you will be paid $10.00 for participating in this study, this has changed and should now read $15.00.

If you have any problems or questions, please don't hesitate to email me.

Thanks again
Jacqueline

Jacqueline Tither
Department of Psychology
University of Canterbury
Private Bag 4800
Christchurch
NEW ZEALAND
Phone: +64 3 364 2987; extn.7845
Fax: +64 3 364 2181

Appendix F

University of Canterbury Department of Psychology

Family Relationships and Behaviour Study

INFORMATION SHEET

You are invited to participate as a subject in the above-named study. The aim of this project is to examine the influence of family relationships on behaviour. Prior to participating in this research, please review this information sheet and consider the attached consent form. If, after reading the information sheet and consent form, you are interested in participating in this research, please click the "I consent" option. If you have any questions you would like to ask before consenting to participate in this study, please contact Jacqueline Tither at 364-2987, ext. 7845 or via email: jmt68@student.canterbury.ac.nz.

Your involvement in this project will involve you completing an online questionnaire. This questionnaire will ask you questions about your family of origin, your schooling, your relationships with your friends and parents, and rule-breaking behaviours during adolescence. Some of the questions will address aspects of your sexual behaviour. This study should take no longer than 45 minutes to complete and you will be paid $10.00 for your participation. You have the right to withdraw from the project at any time, including withdrawal of any information provided.

The results of the project may be published, but you may be assured of the complete confidentiality of data gathered in this investigation: the identity of

participants will not be made public under any circumstances. To ensure anonymity and confidentiality, all data will be identified by a code number only, and all identifying information will be stored separately from the data in a locked filing cabinet in a secure room.

This project is being carried out as a requirement for a Ph.D degree in Psychology by Jacqueline Tither, under the supervision of Dr. Bruce Ellis, who can be contacted at the Department of Psychology, University of Canterbury (telephone 364-2987; ext. 7845 or 8090; or via email jmt68@student.canterbury.ac.nz or bruce.ellis@canterbury.ac.nz). They will be pleased to discuss any concerns you may have about participation in the project.

This project has been reviewed and approved by the University of Canterbury Human Ethics Committee.

IMPORTANT CONSENT INFORMATION

I have read and understood the description of the above-named project. On this basis I agree to participate in this project, and I consent to publication of the results of the project with the understanding that anonymity will be preserved at all times. I understand that I may at any time withdraw from the project, including withdrawal of any information that I have provided.

NO, I do not want to complete the questionnaire at this time.

Yes, I understand and would like to continue.

Appendix G

ONLINE DEBRIEFING SHEET

Thank you for participating in the Family Relationships and Behaviour Study. The aim of this study is to investigate differences between members of the same family in terms of their behaviour during adolescence (e.g., school performance, age of first sexual activity, teenage pregnancy, aggressive behaviour, use of cigarettes and alcohol). Our method is to study pairs of sisters that differ in age and (in some cases) amount of time that they lived without their fathers in the home. The first goal of this research is to determine whether, within families, sisters who had more prolonged exposure to father absence during childhood have more adjustment problems during adolescence. If it turns out that they do, then the second goal will be to explain why. Accordingly, we asked you questions about such things as how closely you were monitored by your parents while you were growing up, how close you were to your father, what your friends were like, etc. We hope that this kind of information will provide clues to understanding differences between sisters in the same family. This research follows on from a previously published study on this topic:

> Ellis, B. J., Bates, J. E., Dodge, K. A., Fergusson, D. M., Horwood, J. L., Pettit, G. S., & Woodward, L. (2003). Does father absence place daughters at special risk for early sexual activity and teenage pregnancy? *Child Development, 74*, 801-821.

An electronic copy of this previous paper can be obtained by emailing Bruce Ellis at: bruce.ellis@canterbury.ac.nz

Because the current research focuses on family environments, sibling relationships, and adolescent adjustment, it was necessary to request sensitive information about your behaviour and life history. Although the results of this project may be published, you can be assured of the complete confidentiality of data gathered in this investigation: the identity of participants will not be made public without their consent. To ensure anonymity and confidentiality, all the

data collected will be identified by a code number only, and all identifying information will be stored separately from the data in a locked filing cabinet in a secure room. Please let us know if you would like a summary of the research results and we will get this information to you when it becomes available.

This project has been reviewed and approved by the University of Canterbury Human Ethics Committee, and is being carried out as a requirement for a Ph.D degree in Psychology by Jacqueline Tither, under the supervision of Dr. Bruce Ellis, who can be contacted at the Department of Psychology, University of Canterbury (364-2987; ext. 7845 or 8090). If this research has raised issues that were not apparent to you when you first consented to participate, please feel free to contact us to discuss any concerns you may have about your participation in the project. Moreover, you have the right to withdraw from the project at any time without penalty, including withdrawal of any information provided.

Given the personal nature of some of the questions in the questionnaire, there is the potential that some distress may have been produced. In the event that participation in this project has caused you any distress, contact details for Counselling and Advice Services available in the local area are provided below:

Citizens Advice Bureau	**0800 FOR CAB (0800 367 222)**
Campbell Centre - Presbyterian Support Service	**(03) 366 5472**
Relationship Services	**0800 RELATE (0800 735 283)**

Appendix H

University of Canterbury Department of Psychology

Family Relationships and Behaviour Study

INFORMATION SHEET

You are invited to participate as a subject in the above-named study. The aim of this project is to examine the influence of family relationships on behaviour. Prior to participating in this research, please review this information sheet and consider the attached consent form. If, after reading the information sheet and consent form, you are interested in participating in this research, please sign and date the consent form. If you have any questions you would like to ask before consenting to participate in this study, please do not hesitate to direct them to the researcher who is running the study today.

Your involvement in this project today will involve you completing a questionnaire. This questionnaire will ask you questions about your family of origin, your schooling, your relationships with your friends and parents, and rule-breaking behaviours during adolescence and beyond. Some of the questions will address aspects of your sexual behaviour. This questionnaire should take no longer than 45 minutes to complete and you will be paid $10.00 for your participation. After completing the questionnaire, please place it in the envelope provided and alert the researcher, who will then pay you.

Please note that you have the right to withdraw from the project at any time, including withdrawal of any information provided. The results of the project may be published, but you may be assured of the complete confidentiality of data gathered in this investigation: the identity of participants will not be made public without their consent. To ensure anonymity and confidentiality, all data will be identified by a code number only, and all identifying information will be stored separately from the data in a locked filing cabinet in a secure room.

This project is being carried out as a requirement for a Ph.D degree in Psychology by Jacqueline Tither, under the supervision of Dr. Bruce Ellis, who can be contacted at the Department of Psychology, University of Canterbury (364-2987; ext. 7845 or 8090). They will be pleased to discuss any concerns you may have about participation in the project.

This project has been reviewed and approved by the University of Canterbury Human Ethics Committee.

Appendix I

University of Canterbury Department of Psychology

Family Relationships and Behaviour Study

CONSENT FORM

I have read and understood the description of the above-named project. On this basis I agree to participate in this project, and I consent to publication of the results of the project with the understanding that anonymity will be preserved at all times. I understand that I may at any time withdraw from the project, including withdrawal of any information that I have provided.

NAME (please print): ……..

Signature:

Date:

Principal Investigator: Jacqueline Tither

Address: Department of Psychology
University of Canterbury
Private Bag 4800
Christchurch
Ph: 364-2987; ext. 7845
Email: jmt68@student.canterbury.ac.nz

Appendix J

University of Canterbury Department of Psychology

Family Relationships and Behaviour Study

DEBRIEFING SHEET

Thank you for participating in the Family Relationships and Behaviour Study. The aim of this study is to investigate differences between members of the same family in terms of their behaviour during adolescence (e.g., school performance, age of first sexual activity, teenage pregnancy, aggressive behaviour, use of cigarettes and alcohol). Our method is to study pairs of sisters that differ in age and (in some cases) amount of time that they lived without their fathers in the home. The first goal of this research is to determine whether, within families, sisters who had more prolonged exposure to father absence during childhood have more adjustment problems during adolescence. If it turns out that they do, then the second goal will be to explain why. Accordingly, we asked you questions about such things as how closely you were monitored by your parents while you were growing up, how close you were to your father, what your friends were like, etc. We hope that this kind of information will provide clues to understanding differences between sisters in the same family. This research follows on from a previously published study on this topic:

> Ellis, B.J., Bates, J.E., Dodge, K.A., Fergusson, D.M., Horwood, J.L., Pettit, G.S., & Woodward, L. (2003). Does father absence place daughters at special risk for early sexual activity and teenage pregnancy? *Child Development, 74*, 801-821.

An electronic copy of this previous paper can be obtained by emailing Bruce Ellis at: bruce.ellis@canterbury.ac.nz

Because the current research focuses on family environments, sibling relationships, and adolescent adjustment, it was necessary to request sensitive information about your behaviour and life history. Although the results of this project may be published, you can be assured of the complete confidentiality of data gathered in this investigation: the identity of participants will not be made public without their consent. To ensure anonymity and confidentiality, all the data collected will be identified by a code number only, and all identifying information will be stored separately from the data in a locked filing cabinet in a secure room. Please let us know if you would like a summary of the research results and we will get this information to you when it becomes available.

This project has been reviewed and approved by the University of Canterbury Human Ethics Committee, and is being carried out as a requirement for a Ph.D degree in Psychology by Jacqueline Tither, under the supervision of Dr. Bruce Ellis, who can be contacted at the Department of Psychology, University of Canterbury (364-2987; ext. 7845 or 8090). If this research has raised issues that were not apparent to you when you first consented to participate, please feel free to contact us to discuss any concerns you may have about your participation in the project. Moreover, you have the right to withdraw from the project at any time without penalty, including withdrawal of any information provided.

Given the personal nature of some of the questions in the questionnaire, there is the potential that some distress may have been produced. In the event that participation in this project has caused you any distress, contact details for Counselling and Advice Services available in the local area are provided below:

Citizens Advice Bureau	**0800 FOR CAB (0800 367 222)**
Campbell Centre - Presbyterian Support Service	**(03) 366 5472**
Relationship Services	**0800 RELATE (0800 735 283)**

Appendix K

University of Canterbury Department of Psychology

Family Relationships and Behaviour Study

INFORMATION SHEET

You are invited to participate as a subject in the above-named study. The aim of this project is to examine the influence of family relationships on behaviour. Prior to participating in this research, please review this information sheet and consider the attached consent form. If, after reading the information sheet and consent form, you are interested in participating in this research, please sign and date the consent form. If you have any questions you would like to ask before consenting to participate in this study, please contact Jacqueline Tither at 364-2987, ext. 7845 or via email: jmt68@student.canterbury.ac.nz.

Your participation in this project will involve you completing a questionnaire that asks questions about your family of origin, your schooling, your relationships with your friends and parents, and rule-breaking behaviours during adolescence. Some of the questions will address aspects of your sexual behaviour. This questionnaire should take no longer than 45 minutes to complete and you will be paid $10.00 for your participation. After completing the questionnaire, please place your signed consent form and the questionnaire in the return envelope provided and post them back to us. Once we have received your completed consent form and questionnaire, we will mail your payment out

to you (this will be either a petrol or grocery voucher (your choice) to the value of $10.00).

Please note that you have the right to withdraw from the project at any time, including withdrawal of any information provided. The results of the project may be published, but you may be assured of the complete confidentiality of data gathered in this investigation: the identity of participants will not be made public under any circumstances. To ensure anonymity and confidentiality, all data will be identified by a code number only, and all identifying information will be stored separately from the data in a locked filing cabinet in a secure room.

This project is being carried out as a requirement for a Ph.D degree in Psychology by Jacqueline Tither, under the supervision of Dr. Bruce Ellis, who can be contacted at the Department of Psychology, University of Canterbury (telephone 364-2987; ext. 7845 or 8090; or via email jmt68@student.canterbury.ac.nz or bruce.ellis@canterbury.ac.nz). They will be pleased to discuss any concerns you may have about participation in the project.

This project has been reviewed and approved by the University of Canterbury Human Ethics Committee.

Appendix L

University of Canterbury Department of Psychology

Family Relationships and Behaviour Study

INFORMATION SHEET

You are invited to participate as a subject in the above-named study. The aim of this project is to examine the influence of family relationships on behaviour. Prior to participating in this research, please review this information sheet and consider the attached consent form. If, after reading the information sheet and consent form, you are interested in participating in this research, please sign and date the consent form. If you have any questions you would like to ask before consenting to participate in this study, please contact Jacqueline Tither at 364-2987, ext. 7845 or via email: jmt68@student.canterbury.ac.nz.

Your participation in this project will involve you completing a questionnaire that asks questions about your family of origin, your schooling, your relationships with your friends and parents, and rule-breaking behaviours during adolescence. Some of the questions will address aspects of your sexual behaviour. This questionnaire should take no longer than 45 minutes to complete and you will be paid $15.00 for your participation. After completing the questionnaire, please place your signed consent form and the questionnaire in the return envelope provided and post them back to us. Once we have received your completed consent form and questionnaire, we will mail your payment out

to you (this will take the form of petrol, *The Warehouse* or grocery vouchers (your choice) to the value of $15.00).

Please note that you have the right to withdraw from the project at any time, including withdrawal of any information provided. The results of the project may be published, but you may be assured of the complete confidentiality of data gathered in this investigation: the identity of participants will not be made public under any circumstances. To ensure anonymity and confidentiality, all data will be identified by a code number only, and all identifying information will be stored separately from the data in a locked filing cabinet in a secure room.

This project is being carried out as a requirement for a Ph.D degree in Psychology by Jacqueline Tither, under the supervision of Dr. Bruce Ellis, who can be contacted at the Department of Psychology, University of Canterbury (telephone 364-2987; ext. 7845 or 8090; or via email jmt68@student.canterbury.ac.nz or bruce.ellis@canterbury.ac.nz). They will be pleased to discuss any concerns you may have about participation in the project.

This project has been reviewed and approved by the University of Canterbury Human Ethics Committee.

Appendix M

FP Questionnaire

The next questions concern your father's mental health. Please think back to your childhood, up to the age of 16 years.

Please circle **ONE** number in each scale.

1. Did your birth father suffer from nervous or emotional problems (such as anxiety or depression)?

1	**2**	**3**
Yes	No	Don't know

2. Did your birth father have trouble with drinking or other drug use?

1	**2**	**3**
Yes	No	Don't know

3. Did your birth father have any history of suicide/attempted suicide?

1	**2**	**3**
Yes	No	Don't know

4. Did your birth father suffer from any psychiatric illness?

1	**2**	**3**
Yes	No	Don't know

5. Did your birth father have any history of offending involving property?

1	**2**	**3**
Yes	No	Don't know

6. Did your birth father have any history of offending involving violence?

1	**2**	**3**
Yes	No	Don't know

7. Did your birth father have any history of being convicted of a criminal offence?

1	**2**	**3**
Yes	No	Don't know

8. Did your birth father have any history of imprisonment?

1	**2**	**3**
Yes	No	Don’t know