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Childhood family disruption and adult height: is there a mediating role of puberty?

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ABSTRACT

Background and objectives: Childhood family background is known to be associated with child growth and development, including the onset of puberty, but less is known about the influence of childhood family disruption on outcomes in later life. Given the associations between early family disruption and childhood development, we predicted that there may be long-term health-relevant consequences of childhood disruption.

Methodology: Using data from a large US interview sample (*n* = 16 207), we test if death or divorce of parents, at different childhood periods, was associated with adult stature, and whether age at puberty mediated this relationship, for men and women.

Results: *Men*: parental death and divorce during early childhood was associated with shorter adult height, and later puberty. Later puberty was associated with shorter adult height. Path analyses demonstrated that the relationship between parental divorce and height was completely mediated by age at puberty; although parental death was only partially mediated by age at puberty. *Women:* the father's death during early childhood was associated with earlier puberty, which was in turn associated with shorter adult stature. The relationship between paternal death and height is entirely mediated by age at puberty; no evidence of a direct relationship between childhood family disruption and adult height.

Conclusions: Early childhood familial disruption is associated with shorter height for men, and is partially mediated by later puberty. For women, the relationship between father's death, and height was completely mediated by earlier puberty. These findings indicate that disruption during childhood can have long-reaching health repercussions, particularly for boys.

INTRODUCTION

In high-income settings, childhood familial disruption is known to be consistently associated with earlier puberty and earlier first births for girls [1]. Researchers working within the framework of life history theory have interpreted these observations as a response to adverse early environments: adversity is a cue to shortened life expectancy, making it prudent for the individual to expedite maturation and reproductive effort to more rapidly launch from the natal home and, in evolutionary terms, to avoid leaving no genetic legacy (i.e. to adopt a 'fast' life history strategy [2]. Less is known about how early family disruption influences outcomes in later life, however. Further, less is known about how family disruption influences boys' reproductive and health outcomes, and how about reproductive and health outcomes may be linked across the lifespan. Claims made in the literature about girls are sometimes implied to be true for boys too [3], but this has not been properly tested. Another issue that is implicit in research on this topic is that an individual on a 'fast' or 'slow' life history trajectory will express all associated traits as either fast or slow. This assumes that a life history strategy is an allometric suite of related traits which responds to environmental cues about life expectancy. This has also not been tested and the assumption that all life events in, say, a fast strategy have to be reached at younger ages is not necessarily correct. In societies where the nuclear family is the norm [4], one indicator of adverse early environment might be familial disruption such as the dissolution of the parental partnership. By way of a stress response to such events (which may incorporate both physiological and psychological stress), children may react by adopting a fast life-history strategy. Here, we aim to fill several gaps in the literature. We test how family disruption is linked to height in adulthood; we test whether any relationship between family disruption and height is mediated through the influence of family disruption on puberty; and we test these relationships separately for boys and girls.

Research has shown that for girls in high income populations, family reconfiguration is generally associated with earlier onset of puberty [5, 6], first sex, and first birth. For boys, the relationship is less clear, with research suggesting that in some cases puberty is delayed in response to childhood family disruption [7, 8], although not always [9]. This sex difference is possibly because stress releases cortisol which impedes the production of testosterone, and delays male puberty [10].

While the logic underlying an accelerated life history response to disruptive conditions should largely be the same for men and women, we might expect that the intermediate relationship between childhood stress and puberty to be different between the sexes, as there are differences between men and women's reproductive strategies (Fig. 1), and different hormonal mechanisms at work [11]. Previous research from Dominica found that male hormonal responses were more sensitive to father absence than were those of young women [11], for instance. In the current study, we test whether there is a relationship between childhood familial disruption and adult height, and if this relationship is mediated by age at puberty, developing the idea that there are life history trade-offs between growth and reproductive maturation. However, it should be noted that this model is likely to apply to high-income populations only, where the nuclear family is the norm and where sufficient calories are readily available for individuals to accelerate their life history even under relatively adverse conditions. In low-income settings, familial disruption may have different and more variable influences on children's life history outcomes [3]. This is partly because the extended family may buffer children from such disruption in some populations, and partly because family disruption may primarily lead to nutritional stress which may be expected to delay both male and female pubertal maturation, although this is still likely to lead to shorter stature and earlier death.

The premise for the model depicted in Fig. 1 is that variation in childhood rearing environment, including extrinsic mortality risk and type and quality of parental care, leads to the development of adaptive alternate life history strategies. Thus, children who are subjected to disrupted rearing environments are prompted to adopt a life history strategy that maximises their likelihood of reproducing before dying. Following the internal prediction hypothesis [12], this does not require predicting future mortality rates. Rather, the assaults associated with disruptive childhood environments (e.g. high levels of stress) directly affect the individual's health, in turn shortening that individual's life expectancy. When faced with adverse childhood conditions, an individual is prompted to pursue a life history strategy that is the best that can be done, given the environmental constraints they face. Our model does not, however, assume that all life history traits will be
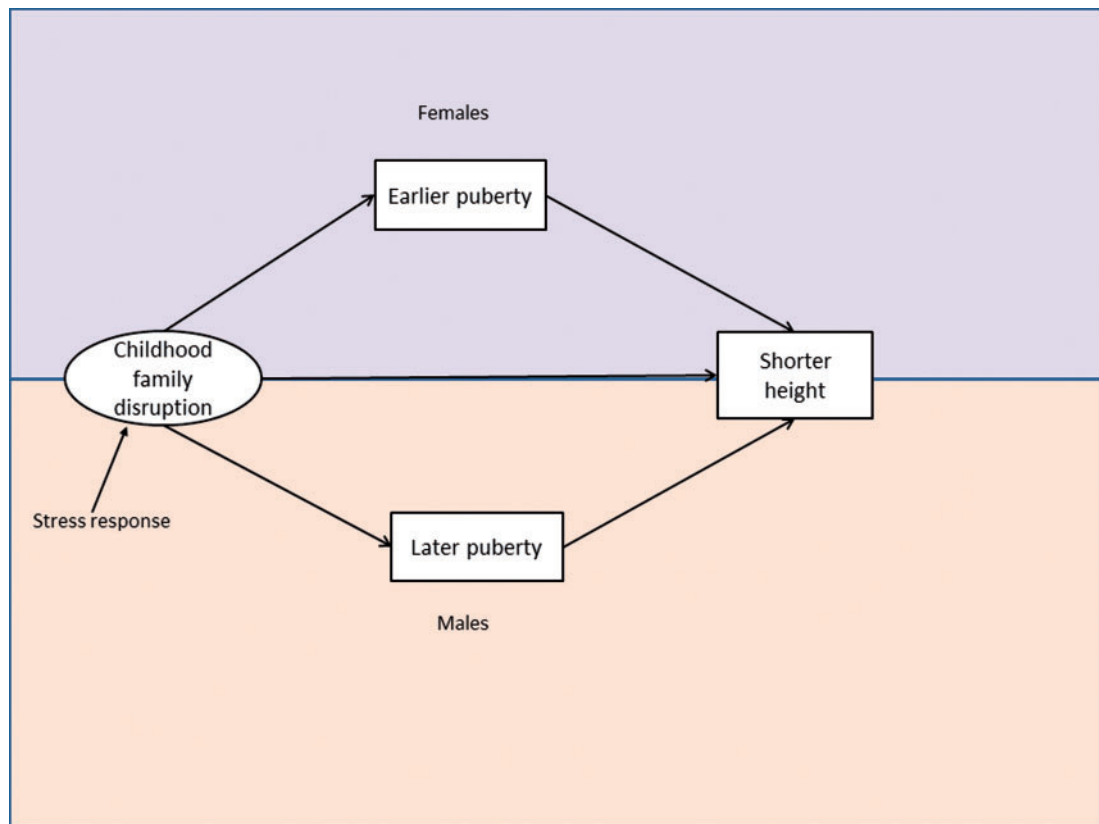
Figure 1. Schematic representation of the life history-driven theoretical relationship between family disruption, puberty and height

accelerated in the same way, because of the different hormonal mechanisms involved for boys and girls. For example, in the proposed model, although male puberty is delayed (i.e. indicating a 'slow' life history), adult men end up with shorter stature (i.e. fast). However, despite different pathways, both sexes are expected to attain shorter adult stature as a direct result of the physiological consequences of suffering childhood stress (Fig. 1, middle arrow). Indirectly, we expect to see different pathways via puberty, for males and females (Fig. 1, outer arrows) due to different hormonal and reproductive constraints faced by each sex.

For males, childhood disruption produces psychological stress which raises cortisol and inhibits testosterone production, delaying pubertal onset [13] (Fig. 1, first lower arrow). Later puberty results in shorter height for males during puberty (Fig. 1, second lower arrow), although some may catch up later, as boys undergo a late pubertal growth spurt [14]. In the model we propose, the direct effects of childhood disruption may constrain the male's ability to catch up in height (Fig. 1, middle arrow).

For females, childhood disruption is associated with accelerated age at menarche in non-nutritionally

stressed populations. This is attributed to the psychological stress response that reduces levels of anabolic hormones like androgen which promote skeletal growth and lean tissue, but are associated with less adipose fat [15] (Fig. 1, first upper arrow). Elevated cortisol is also a consequence of stress, which increases insulin production, and together they produce higher levels of fatty tissue [15]. Increased fatty weight gain during adolescence expedites female puberty. Earlier age at puberty results in taller stature at the time but ultimately these women tend to attain shorter adult height than their later-maturing peers because of their shorter period of growth [14] (Fig. 1, second upper arrow). Figure 2 shows the hormonal pathways that underlie the theoretical model shown in Fig. 1.

Correlates of adult stature

There is some limited existing evidence that childhood family disruption is associated with a number of social and health disadvantages for children later in life. For instance, parental divorce is associated with children's poorer mental health in adulthood [16], as are negative pre-divorce conditions—high
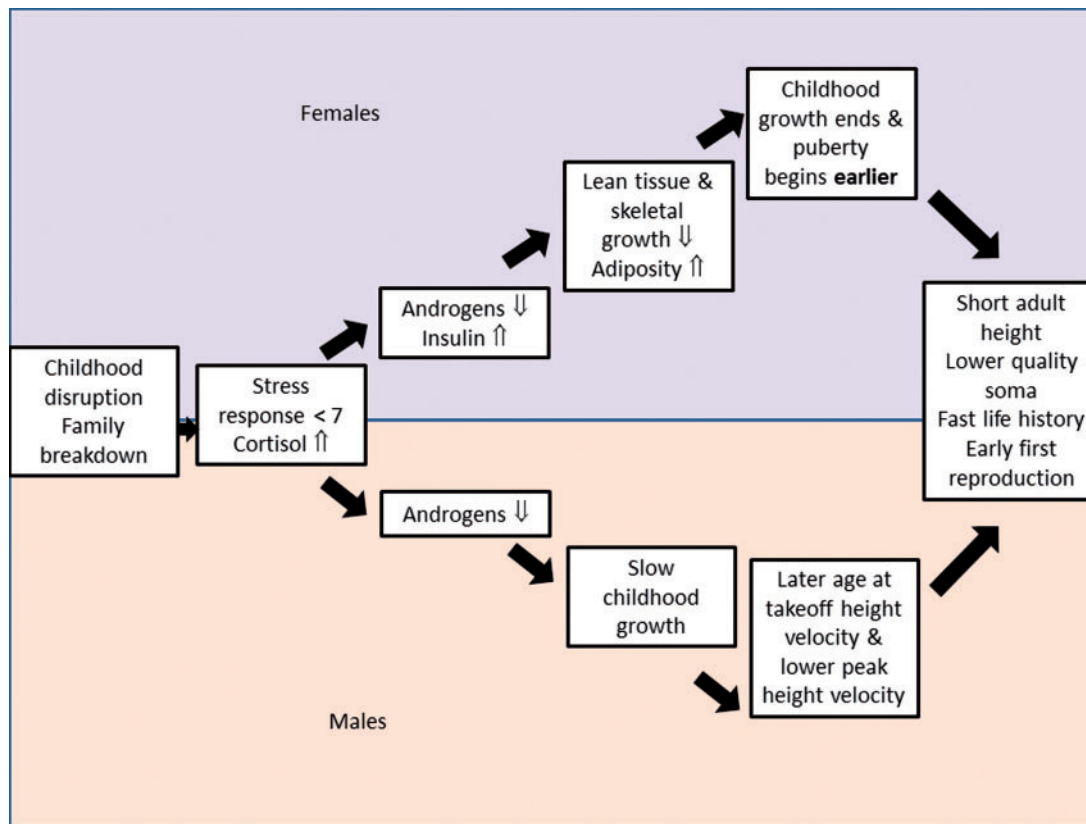
Figure 2. Schematic representation of the mechanisms underlying the theoretical model in Fig. 1. Diagram kindly contributed by Meredith Reiches

levels of marital conflict explain much of the effect of divorce on children's educational attainment, economic success and psychological wellbeing [17]. Few studies have addressed the influence of family disruption on physical health, but, one British study found that parental divorce before age six was associated with shorter adult stature, although there was no effect of parental death on stature [18]. Another study found that family conflict, including parental divorce, before age seven was associated with stunted growth during childhood [19].

Short adult stature is associated with a variety of negative health outcomes, including general poor health [20], heart disease [21], type II diabetes [22], poor psychosocial outcomes [20] and a higher risk of suicide [23]. Overall, taller people report better quality of life, including being generally more positive about their lives, and are less likely than shorter individuals to report negative experiences including sadness and physical pain [24]. The relationship is not completely straightforward, however, as some research has shown that taller stature is positively correlated with certain cancers [25]. The relationship between height and health is in some cases curvilinear, where for women, being of average height is associated with lower mortality risk [26]. Height is the product of both prenatal and postnatal development; the result of an interplay of genes, and childhood nutrition and health. Although lower socioeconomic status is associated with reduced height [27], overall less is known about social factors that influence childhood growth and ultimately adult height. The relationship between health and height goes in both directions: while height is clearly associated with numerous health outcomes, poor health also affects height. Exposure to pathogens and malnutrition during childhood, for example, can lead to stunted growth, affecting adult stature [28].

The relationship between height and age at pubertal maturity has been well researched for women [29]. Evidence shows that there is typically a positive association between age at menarche and height, because of the energetic trade-offs that mammals face between growth and reproduction. Skeletal maturation is imperative for human reproduction and explains much more of the variation in age at menarche than does adiposity [29]. Human female

reproduction incurs a high physiological cost meaning that when resources are aimed towards reproductive effort, females tend to stop investing in growth [30, 31]. In industrialised settings, there has been a secular trend over time in both age at menarche and height as economies prosper, with menarche occurring at younger ages and height gradually increasing [32]. In such economies, where nutrition is less of a limiting factor, girls grow faster. Young men also need to reach a height threshold in order to mature, but their growth spurt is later in the pubertal process than is girls' and continues after pubertal maturation. Taller boys tend to reach puberty earlier than their shorter peers, at least in high-income economies [33]. This negative correlation between puberty and height for boys might indicate that boys who are in particularly good condition can mature earlier but also grow tall. Such factors that influence adult height thus also appear to result in differential health outcomes in adulthood.

Current study

The current study aims to test whether there is a relationship between childhood familial disruption and adult height, for both sexes. Due to the life history trade-off between growth and reproduction, we expect there to be a relationship between age at puberty and adult height. With this in mind, we develop a path model to test if childhood family status is associated with reduced adult height for both men and women and, where it is, if this relationship is mediated by age at puberty.

METHODS

Data

We use an existing dataset, collected in the United States from 1938 to 1963 by Alfred Kinsey and colleagues (n = 16 207). Data was collected by in-depth face-to-face interviews. The original Kinsey survey data contain detailed information about respondents' social and sexual lives, and also contain valuable demographic information related to the current study. Each interviewee was asked about their childhood circumstances (retrospectively collected), ages for development of physical markers of sexual maturity and height was measured at the time of interview (i.e. as adults). The original Kinsey survey culminated in two 'Kinsey Reports' on human

sexuality providing great detail about both the sample and descriptions of their overall findings [34, 35].

Models

We developed a path model to test the association of childhood disruption on pubertal timing, and of both childhood disruption and pubertal timing, on adult height. We present two models, one for each sex, separately. We defined childhood familial disruption as the death of either natural parent, or parental divorce, at two stages in childhood: from birth to age seven, and from age 8 to 15 years. No parental death or divorce (i.e. two natural parents) was treated as the reference category. We structured our models this way because some authors suggest that the period before age seven is critical to development, and children's experiences during this early period have long-term impacts on later life events [3], although this time sensitivity is not always empirically apparent [36], especially for boys [7]. By analysing familial disruption at two stages during childhood, we are able to uncover whether any observed relationship between familial disruption and puberty or height is time-sensitive. There is evidence to suggest that conditions during early childhood also influence hormonal trajectories during adrenarche and puberty [37, 38], so we expect that familial disruption before age seven years will have more of an impact on age at puberty and adult height in this sample compared with those who experienced disruption during later childhood.

Some children suffered both parental divorce and death during their childhoods. To ease interpretation and to maintain statistical independence between the categories, we recoded those children to whichever divorce age-group category they fell into (divorce always occurs before the death of a parent). Similarly, for children whose parents both died, we categorised them into the age group that child was when the first parental death occurred. Children whose parents both died during the same childhood period were omitted from the analysis, but this did not change the results.

Height was measured in inches (women: *mean* 64.14, SD 2.52; men: mean 69.39, SD 2.83). Initially we also included a quadratic term for height but as this variable was non-significant in our models we removed it. Age at puberty is a measure we constructed based on the average age at three self-reported pubertal landmarks: for men these are, ages at voice breaking, pubic hair development and

Table 1. Mean age at puberty in years, and mean height in inches, for each category of childhood family breakdown

Family disruption	Men			Women		
	n (%)	Height Mean (SD)	Age at puberty Mean (SD)	n (%)	Height Mean (SD)	Age at puberty Mean (SD)
Intact family	6196 (70.20)	69.54 (2.78)	13.25 (1.28)	5053 (71.72)	64.24 (2.49)	12.18 (1.31)
Mum died 0–7	386 (4.37)	68.66 (3.05)	13.62 (1.49)	303 (4.30)	63.97 (2.65)	12.26 (1.33)
Dad died 0–7	465 (5.27)	68.91 (3.05)	13.69 (1.55)	342 (4.85)	63.93 (2.65)	12.07 (1.38)
Parents divorced 0–7	701 (7.94)	69.16 (2.86)	13.51 (1.49)	520 (7.38)	63.82 (2.50)	12.20 (1.28)
Mum died 8–15	234 (2.65)	69.21 (2.96)	13.33 (1.46)	218 (3.09)	63.70 (2.61)	12.38 (1.52)
Dad died 8–15	410 (4.65)	69.33 (2.72)	13.49 (1.54)	318 (4.51)	64.09 (2.45)	12.32 (1.34)
Parents divorced 8–15	434 (4.92)	69.15 (2.79)	13.48 (1.37)	291 (4.13)	64.25 (2.41)	12.28 (1.33)
Range		0.88 inches	0.44 years		0.55 inches	0.31 years

SD = standard deviation.

first ejaculation (mean 13.34 years, SD 1.35); for women, these are breast development, pubic hair development and first menstruation (mean 12.20 years, SD 1.32). Although these measures combine both primary and secondary sexual characteristics, and we are concerned with puberty as a measure of reproductive capability, these traits are correlated and thus provide a more holistic marker of pubertal development than do single measures usually used in other research. Table 1 provides sample sizes, mean ages at puberty and mean heights in inches (and standard deviations for both) stratified by each category of childhood familial disruption for both men and women. We controlled for total number of siblings (including co-resident half and step siblings) as large sibships have been shown to be associated with shorter stature [39]. We also controlled for socioeconomic status (SES), and year of birth, to account for secular trends in age at puberty and height. SES is a subjective measure, asked retrospectively at the time of the interview, of how poor or wealthy the respondent deemed their family to be while growing up. We used a generalised structural equation model (GSEM) to gauge the relationship between childhood family disruption and height, while allowing us to test if age at puberty mediates any relationships found. GSEM was used because the primary independent variable is categorical, which regular SEM cannot handle. Analyses were performed using Stata© version 13.

RESULTS

The results from the GSEMs are shown in Table 2, for both men and women. Overall we found no significant

associations with family disruption during *later childhood* and either puberty or height, for either sex, although we did find associations between early family disruption and height. Results for men and women are reported separately, as the results are different for each sex. For visual ease, Figs 1 and 2 display only the significant pathways derived from the models, but all pathways were modelled, as shown in Table 2.

Men

The death of either parent, or their divorce, during early childhood, was associated with delayed puberty and reduced stature, in men, but no relationship if these events occurred during later childhood. As illustrated in Fig. 2, the association between divorce and height is fully mediated by age at puberty. Age at puberty was directly associated with height, with later maturing boys being of shorter stature as adults. Both SES and year of birth were significantly associated with age at puberty, and height, but in opposite directions: later-born, high SES boys had earlier puberty and were taller. Number of siblings was positively associated with puberty but not directly associated with height. In sum, the death of either parent is directly and indirectly, by way of delayed puberty, associated with shorter adult stature. Parental divorce is only indirectly linked to shorter height, via delayed puberty. Figure 2 is a diagrammatic representation of the relationships between these variables.

Women

For women, the father dying during early childhood was associated with earlier puberty, although no

Table 2. Results from a generalised structural equation model showing beta coefficients and associated 95% confidence intervals for family breakdown on age at puberty and on adult height

	Women n = 4602 coefficient	95% CI		Men n = 4753 coefficient	95% CI	
Age at puberty (years)						
Ref. Intact family						
Mum died 0–7	−0.01	−0.20	0.19	0.30**	0.11	0.49
Dad died 0–7	−0.19*	−0.37	−0.01	0.30**	0.13	0.48
Parents divorced 0–7	0.03	−0.11	0.17	0.22**	0.08	0.37
Mum died 8–15	0.09	−0.13	0.31	0.01	−0.25	0.25
Dad died 8–15	−0.01	−0.19	0.17	0.13	−0.05	0.32
Parents divorced 8–15	0.07	−0.12	0.25	0.15	−0.03	0.32
Siblings	0.08***	0.07	0.10	0.05***	0.04	0.07
SES	−0.03*	−0.06	−0.01	−0.09***	−0.12	−0.07
Year of birth	−0.02***	−0.02	−0.01	−0.02***	−0.02	−0.01
Intercept	46.49	38.91	54.06	47.92	40.54	55.30
Adult height (inches)						
Age at puberty	0.12***	0.06	0.17	−0.07*	−0.13	−0.01
Mum died 0–7	−0.03	−0.40	0.34	−0.42*	−0.80	−0.03
Dad died 0–7	−0.14	−0.49	0.21	−0.36*	−0.71	−0.01
Parents divorced 0–7	−0.26	−0.53	0.02	−0.13	−0.42	0.17
Mum died 8–15	−0.36	−0.78	0.06	0.19	−0.32	0.71
Dad died 8–15	−0.08	−0.43	0.26	0.29	−0.10	0.67
Parents divorced 8–15	0.09	−0.25	0.44	−0.15	−0.51	0.21
Siblings	−0.04**	−0.08	−0.01	−0.02	−0.05	0.02
SES	0.20***	0.15	0.25	0.26***	0.21	0.32
Year of birth	0.01	−0.01	0.01	0.04***	0.03	0.04
Intercept	54.04	39.21	68.88	1.16	−14.28	16.61

CI = confidence interval.
*$P < 0.05$, **$P < 0.01$, ***$P < 0.001$.

other type of childhood family disruption showed a significant association, and none directly impacted height. Later age at puberty was significantly associated with taller adult height, providing evidence that the effect of the father's death on height appears to be completely mediated by age at puberty, in this model, as illustrated in Fig. 3. Number of siblings and SES were associated with both outcomes, but again in opposite directions. Women with more siblings had later puberty and were ultimately shorter as adults. High SES women had earlier puberty but were taller adults. We controlled for year of birth to account for the secular trend to earlier puberty [40]. Our results support this and show that time influences height only by way of puberty. In addition, the death of the father during early childhood is associated with earlier age at

puberty, which is in turn associated with shorter stature. Parental divorce and maternal death are not significantly associated with either outcome.

DISCUSSION

These findings add to the literature on the aetiology of adult stature and extend existing research on the effects of early childhood on adult health. Our results suggest that stress during early childhood brought on by familial disruption can influence reproductive and health outcomes later in life. This is in line with some previous literature which suggests that stress during a critical developmental period in early childhood is important in determining life history outcomes such as puberty [41]. This may be unsurprising as the precursors to
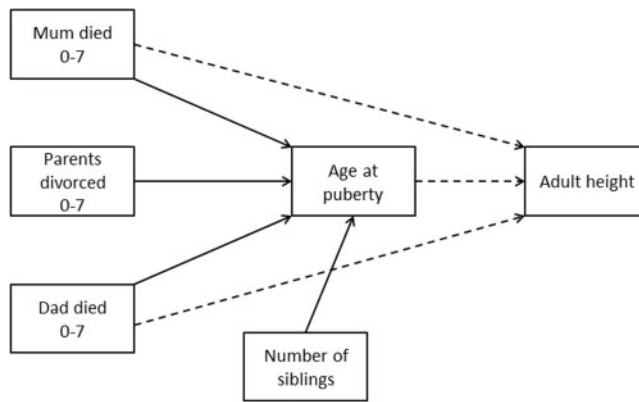
Figure 3. Schematic representation of the relationship between early childhood family stress, puberty, and adult height, for men. Negative correlations are denoted by dashed lines. This model is adjusted for childhood SES and year of birth
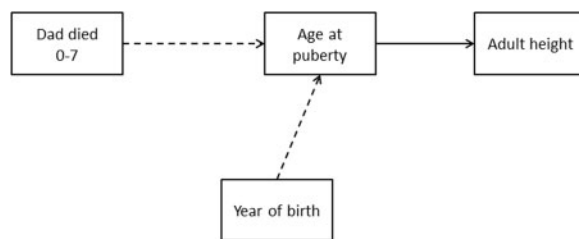

Figure 4. Schematic representation of the relationship between early childhood family stress, puberty and adult height, for women. Negative correlations are denoted by dashed lines. This model is adjusted for SES and number of siblings

puberty (e.g. adrenarche) start at around age 6–8 years for both sexes [42]. On the other hand, some research has shown that age at puberty is associated with change of family structure, or other family stress, during later childhood [7, 43], suggesting that pubertal trajectories are plastic even after they have begun.

Using data from a large US sample, we tested a path model of adult stature based on life history theory. Consistent with this model, puberty mediated the relationships between childhood family background and adult stature, but differentially for men and women. If adult height is a marker of lifetime health status, this indicates that family breakdown during early childhood has long-reaching health repercussions, particularly for boys, manifested throughout the lifespan.

For men, childhood parental divorce affected height only through age at puberty: the response to divorce is delayed puberty, which culminates in shorter stature. The death of either parent was associated with delayed puberty as well as showing a direct effect of shorter height. Familial disruption during childhood has been found to delay puberty in men previously in this population, although this was

only significant for families with absent mothers—boys who lived with a single father, or a father and stepmother, had later puberty compared with those from intact families and those from father-absent households [8]. Similarly, in a UK 1958 birth cohort, father absence during *later* childhood was associated with delayed voice-breaking in boys [7], while in contemporary US, father absence was associated with earlier puberty in boys (and girls) while mother absence was not [9]. Clearly the relationship between family structure and male puberty is complex: both the type and the timing of family disruption may matter for boys' maturation, but different types and timings of disruption may have different effects on boys. These differences may be the product of context-dependent settings. Additionally, the loss of a parent during childhood may constitute loss of household resources; this is somewhat supported by the finding that low SES and large sibships are associated with delayed puberty and reduced adult height in this study. However, this does not explain why the death of a parent has a long-term, direct impact on adult stature, but not divorce; it is possible that absent parents still contribute to the household wealth after divorce. Furthermore, this

contradicts what other studies have found: in two British cohort studies divorce, the associated family stress (but not the death of a parent) was associated with stunted growth and shorter adult stature [18, 19]. Height is a heritable trait [44] so it is possible that short parental stature is passed on to children, and is also associated with a higher likelihood of premature death. This potential confounding is not possible to test with these data as we have no information about parental height. Similarly, we would have liked to control for birth weight and length, if the data had been available, as these are associated with adult height [45], and would precede parental death or divorce in our models. Another avenue for future research, with appropriate data, would be to examine the relationship between height and individual morbidity and/or mortality as a function of age at puberty and childhood familial disruptions.

For women, father absence during early childhood has previously been associated with earlier age at menarche in numerous studies from high-income contexts [5, 6, 46]. As expected, therefore, the death of a father by age seven was associated with early puberty for women. We might expect that parental divorce would show a similar association (assuming that most girls live with their mothers after divorce). However, using the same data we previously found that family living situation between ages 6–8 years was not correlated with puberty [8]. In this previous study, there was no significant difference in age at puberty for girls living with single parents or parents and stepparents, compared with girls living in intact families. This suggests the influence of family disruption on girls' age at puberty may also be complex, and perhaps specific to particular types of family disruption. Most of the relevant literature on this issue comes from studies in high-income contexts, where divorce is more common than death. The current study uses older data, which may explain why death is more important for women than divorce is. Again, this does not explain why divorce appears to impact boy's puberty [11]. We found no evidence of a direct effect of father's death on adult height, suggesting that early childhood stressors impact pubertal development, and that there is then a knock-on effect on other life history outcomes, as suggested by Ellis [3]. This is also what we would expect given the life history trade-offs between growth and reproduction: girls who experience earlier puberty will cease growing earlier and be shorter adults.

Overall, in the current study, it appears that men are more sensitive to family disruption than are women. Research on the effect of familial disruption and puberty has been scarce for males, mainly due to data constraints, and less reliable discrete markers of male puberty [3, 47]. Nevertheless, we might speculate that our results are in line with existing theoretical models as boys are generally more affected by environmental insults than are girls [11, 48, 49]. Women are perhaps able to buffer against the effects of early family disruption more easily in this somewhat historic population. However, the general lack of significant associations in the female model may instead be due to the opposing directions of expected effects of family disruption on puberty and height. From a simple nutritional perspective, family disruption is expected to slow growth and delay puberty, and delayed puberty in women is correlated with taller height. The existing empirical evidence suggests that family disruption tends to be associated with *earlier* puberty for girls, perhaps due to psychological stress accelerating development, and earlier puberty predicts shorter height in women [50]. While speculative, it is possible that in this historical US environment, the nutritional stress of family disruption and the psychosocial stress of family disruption may be cancelling one another out. Alternatively, the original Kinsey survey respondents range widely in age but the median year of birth is 1920, when nutritional factors may have been more important than in modern-day US. During this period, and the following decade when the respondents were growing up, the USA experienced substantial economic and social change. Major historical and cultural events such as World War II and the Great Depression would have impacted the economic, health and family lives of most US residents. It is likely therefore that the death of a parent, or parental divorce, would have had economic and social repercussions that changed over time. We may see weaker effects of family disruption for girls because its changing repercussions for girls washed out any clear effects on puberty or height; however, we do see clearer effects for boys, which may argue against this interpretation.

CONCLUSION

Familial disruption during early childhood has far-reaching repercussions for the health of both men and women. This study assesses adult height as one such health-relevant outcome. For men, family disruption was associated with adult height and this

relationship was partly mediated by age at puberty. Among women, we found evidence for a long-term impact of childhood disruption on height, however this is entirely mediated by age at puberty. Life history theory can contribute to our understanding of how events during early childhood play out throughout the lifespan. It is important to better understand the social influences on height, as adult stature is both a product, and a source, of human health. More empirical evidence may shed light on why this relationship differs between men and women.

REFERENCES

1. Quinlan RJ. Father absence, parental care, and female reproductive development. *Evol Hum Behav* 2003;**24**: 376–90.
2. Nettle D. Dying young and living fast: variation in life history across English neighborhoods. *Behav Ecol* 2010;**21**: 387–95.
3. Ellis B. Timing of pubertal maturation in girls: an integrated life history approach. *Psychol Bull* 2004;**130**: 920–58.
4. Sear R. Beyond the nuclear family: an evolutionary perspective on parenting. *Curr Opin Psychol* 2016;**7**:98–103.
5. Alvergne A, Faurie C, Raymond M. Developmental plasticity of human reproductive development: effects of early family environment in modern day France. *Physiol Behav* 2008;**95**:625–32.
6. Bogaert A. Menarche and father absence in a national probability sample. *J Biosoc Sci* 2008;**40**:623–36.
7. Sheppard P, Sear R. Father absence predicts age at sexual maturity and reproductive timing in British men. *Biol Lett* 2012;**8**:237–40.
8. Sheppard P, Sear R, Garcia JR. A not-so-grim tale: How childhood family structure influences reproductive and risk-taking outcomes in a historical U.S. population. *PLoS One* 2014;**9**:e89539
9. Bogaert A. Age at puberty and father absence in a national probability sample. *J Adolesc* 2005;**28**:541–6.
10. Campbell B, Leslie PW. Reproductive ecology of human males. *Am J Phys Anthropol* 1995;**38**:1–26.
11. Flinn MV, Quinlan RJ, Decker S *et al*. Male-female differences in effects of parental absence on glucocorticoid stress response. *Hum Nat* 1996;**7**:125–62.
12. Rickard I, Frankenhuis WE, Nettle D. Why are childhood family factors associated with timing of maturation? A role for internal prediction. *Perspect Psychol Sci* 2014;**9**:3–15.
13. Bribiescas RG. Reproductive ecology and life history of the human male. *Am J Phys Anthropol* 2001;**Suppl 33**:148–76.
14. Christie D, Viner R. Adolescent development. *BMJ* 2005; **330**:301–4.
15. Epel E. Psychological and metabolic stress: A recipe for accelerated cellular aging? *Hormones* 2009;**8**:7–22
16. Chase-Lansdale PL, Cherlin AJ, Kiernan KE. The long-term effects of parental divorce on the mental health of young adults: A developmental perspective. *Child Dev* 1995;**66**: 1614–34.
17. Furstenberg F, Teitler J. Reconsidering the effects of marital disruption. What happens to children of divorce in early adulthood? *J Fam Issues* 1994;**15**:173–90
18. Wadsworth M, Hardy RJ, Paul AA *et al*. Leg and trunk length at 43 years in relation to childhood health, diet and family circumstances; evidence from the 1946 national birth cohort. *Int J Epidemiol* 2002;**31**:383–90.
19. Montgomery SM, Bartley MJ, Wilkinson RG. Family conflict and slow growth. *Arch Dis Child* 1997;**77**:326–30.
20. Christensen TL, Djurhuus CB, Clayton P *et al*. An evaluation of the relationship between adult height and health-related quality of life in the general UK population. *Clin Endocrinol (Oxf)* . 2007 Sep; **67**:407–12.
21. Lawlor D, Taylor M, Davey Smith G *et al*. Associations of components of adult height with coronary heart disease in postmenopausal women: the British women's heart and health study. *Heart* 2004;**90**:745–9.
22. Lawlor D, Ebrahim S, Davey Smith G. The association between components of adult height and Type II diabetes and insulin resistance: British Women's Heart and Health Study. *Diabetologia* . 2002;**45**:1097–106.
23. Magnusson PKE, Gunnell D, Tynelius P *et al*. Strong inverse association between height and suicide in a large cohort of Swedish men: evidence of early life origins of suicidal behavior? *Am J Psychiatry* 2005;**162**:1373–5
24. Deaton A, Arora R. Life at the top: The benefits of height. *Econ Hum Biol* 2009;**7**:133–36.
25. Green J, Cairns BJ, Casabonne D *et al*. Height and cancer incidence in the Million Women Study: prospective cohort, and meta-analysis of prospective studies of height and total cancer risk. *Lancet* 2011;**12**:785–94.
26. Sear R. Height and reproductive success: is bigger always better? In: Frey U, Störmer C, Willführ K (ed.). *Homo Nova Hum without Illusion*. Heidelberg: Springer, 2010.
27. Power C, Manor O, Li L. Are inequalities in height underestimated by adult social position? Effects of changing social structure and height selection in a cohort study. *Br Med J* 2002;**325**:131–34.
28. Bogin B. 1999. *Patterns of Human Growth*. 2nd ed. Cambridge: Cambridge University Press.

29. Ellison PT. Skeletal growth, fatness, and menarcheal age: a comparison of two hypotheses. *Hum Biol* 1982;**54**:269–81.

30. McIntyre MH, Kacerosky PM. Age and size at maturity in women: a norm of reaction? *Am J Hum Biol* 2011;**23**:305–12

31. Allal N, Sear R, Prentice A *et al*. An evolutionary model of stature, age at first birth and reproductive success in Gambian women. *Proc R Soc London B Biol Sci* 2004;**271**:465–70.

32. Malina RM. Secular trends in growth, maturation and physical performance: A review. *Anthropol Rev* 2004;**67**:3–31.

33. Kuzawa CW, McDade T, Adair L, Lee N. Rapid weight gain after birth predicts life history and reproductive strategy in Filipino males. *Proc Natl Acad Sci* 2010;**107**:16800–805.

34. Kinsey A, Pomeroy W, Martin C. 1948. *Sexual Behavior in the Human Male*. Bloomington: Indiana University Press.

35. Kinsey A, Pomeroy W, Martin C *et al*. 1953. *Sexual Behavior in the Human Female*. Bloomington: Indiana University Press.

36. Sheppard P, Snopkowski K, Sear R. Father absence and reproduction-related outcomes in Malaysia, a transitional fertility population. *Hum Nat* 2014;**25**:213–34.

37. Gettler LT, Mcdade TW, Kuzawa CW. Cortisol and testosterone in Filipino young adult men: Evidence for co-regulation of both hormones by fatherhood and relationship status. *Am J Hum Biol* 2011;**23**:609–20.

38. Núñez-De La Mora A, Chatterton RT, *et al*. Childhood conditions influence adult progesterone levels. *PLoS Med* 2007;**4**:0813–21.

39. Lawson D, Mace R. Sibling configuration and childhood growth in contemporary British families. *Int J Epidemiol* 2008;**37**:1408–21.

40. Onland-Moret NC, Peeters PHM, Gils CH. van *et al*. Age at menarche in relation to adult height: the EPIC study. *Am J Epidemiol* 2005;**162**:623–32.

41. Belsky J. The development of human reproductive strategies: progress and prospects. *Curr Dir Psychol Sci* 2012;**21**:310–316.

42. Patton GC, Viner R. Pubertal transitions in health. *Lancet* 2007;**369**:1130–1139.

43. Kim K, Smith PK. Childhood stress, behavioural symptoms and mother-daughter pubertal development. *J Adolesc* 1998;**21**:231–240.

44. Stulp G, Barrett L. Evolutionary perspectives on human height variation. *Biol Rev* 2015, in press.

45. Sorensen H, Sabroe S, Rothman K *et al*. Birth Weight and Length as Predictors for Adult Height. *Am J Epidemiol* 1999;**149**:726–9.

46. Ellis B, McFadyen-Ketchum S, Dodge KA *et al*. Quality of early family relationships and individual differences in the timing of pubertal maturation in girls: A longitudinal test of an evolutionary model. *J Pers Soc Psychol* 1999;**77**:387–401. American Psychological Association.

47. Gluckman P, Hanson M. Evolution, development and timing of puberty. *Trends Endocrinol Metab* 2006;**17**:7–12.

48. Helle S, Lummaa V, Jokela J. Sons reduced maternal longevity in preindustrial humans. *Science* 2002;**296**:1085

49. Rickard I, Russell AF, Lummaa V. Producing Sons Reduces Lifetime Reproductive Success of Subsequent Offspring in Pre-Industrial Finns. *Proc R Soc B Biol Sci* 2007;**274**:2981–88.

50. Hulanicka B, Gronkiewicz L, Koniarek J. Effect of familial distress on growth and maturation of girls: A longitudinal study. *Am J Hum Biol* 2001;**13**:771–76.