Developmental influences on fertility decisions by women: an evolutionary perspective

D. A. Coall1,2, M. Tickner1, L. S. McAllister3 and P. Sheppard4

1School of Medical and Health Sciences, Edith Cowan University, Joondalup, Western Australia, Australia
2School of Psychiatry and Clinical Neurosciences, University of Western Australia, Crawley, Western Australia, Australia
3Department of Anthropology, University of California, Santa Barbara, CA 93106, USA
4Department of Population Health, London School of Hygiene and Tropical Medicine, London, UK

Developmental environments are crucial for shaping our life course. Elements of the early social and biological environments have been consistently associated with reproduction in humans. To date, a strong focus has been on the relationship between early stress, earlier menarche and first child birth in women. These associations, found predominately in high-income countries, have been usefully interpreted within life-history theory frameworks. Fertility, on the other hand—a missing link between an individual’s early environment, reproductive strategy and fitness—has received little attention. Here, we synthesize this literature by examining the associations between early adversity, age at menarche and fertility and fecundity in women. We examine the evidence that potential mechanisms such as birth weight, childhood body composition, risky health behaviours and developmental influences on attractiveness link the early environment and fecundity and fertility. The evidence that menarche is associated with fertility and fecundity is good. Currently, owing to the small number of correlational studies and mixed methodologies, the evidence that early adversity predicts fecundity and fertility is not conclusive. This area of research is in its infancy; studies examining early adversity and adult fertility decisions that can also examine likely biological, social and psychological pathways present opportunities for future fertility research.

1. Introduction

Human fertility is a tricky business at the best of times. There is compelling evidence that fertility decision-making and the likelihood of a pregnancy going to term are influenced by psychosocial, nutritional, disease, toxic and material poverty elements of the maternal environment surrounding pregnancy [1–5]. However, the influence that women’s developmental environments have on fecundity and fertility has received little attention. This is surprising as models from both the energetics and the psychosocial stress literature developed out of life-history theory frameworks predict associations between the early environment and fertility [6]. Perhaps this lack of focus is partly owing to the perceived control we have over reproduction: in high-income nations, reproduction is a matter of choice not development. To address this research gap, we will examine the impact that the developmental environment has on a woman’s subsequent fecundity (the ability to produce offspring) and fertility (the number of offspring produced).

The importance of the developmental environment for subsequent growth, development, reproduction and adult phenotypes is central to many disciplines, including evolutionary ecology, ethology, developmental health, developmental psychology and epidemiology [7–12]. Multi-cellular plants and animals that develop in heterogeneous environments show some level of developmental plasticity that provides the time necessary to assess, and adjust to, both the biological and social developmental environments [3,13–18]. The evolutionary
significance of this plasticity is that it enables organisms to develop functionally integrated reproductive strategies that maximize an individual's fitness in variable environments [14,19,20]. Indeed, it is the compromises created by the trade-offs between growth, maintenance and reproduction that fashion the developmental plasticity of life-history traits [21].

Theory and evidence suggest that at an ultimate level, local mortality rates are the environmental cues that organisms respond to when unconsciously adjusting the timing of their life-history traits that ultimately contribute to fertility (e.g. [4,21]). How this may be 'perceived' by an individual at a proximate level is discussed below. Comparative studies of mortality rates and variation in life-history traits across mammalian species suggest, after adjustment for body size, that mortality rates are the major mediating factor between ecology and life-history traits [22]. Both across and within species, higher mortality rates are associated with earlier maturation, larger litter sizes, smaller offspring and producing more offspring in a lifetime. Therefore, organisms have a probability of dying from external factors (predation, starvation) and allocate their resources to reproduction accordingly [22–25]. This is one life-history perspective that will be expanded below, but first we approach the issue of how organisms ‘perceive’ their early environment.

2. Influence of the developmental environment

A range of alternative (although not mutually exclusive) models examine how adverse developmental environments affect development, reproduction and adult health. One paradigm suggests that the physiological embodiment of the early environment provides a ‘weather forecast’ of future environments. If accurate and consistent with the adult environment, physiological adjustments to these forecasts are likely to provide a fitness advantage [26,27]. These forecasts are often referred to as predictive adaptive responses [27,28]. If the environments are not consistent, a mismatch will occur between the individual and their adult environment, reducing fitness [28,29]. However, within life-history theory, the first priority is survival. Various models portray physiological changes as ‘making the best of a bad start’ (e.g. [30–32]). This is a form of downside risk protection. Childhood provides the time necessary to make assays of both the biological [3,17] and social [14,16] developmental environments and adjust reproductive strategies accordingly. A woman born into an adverse environment must adjust her physiology and behaviour to ensure her own survival and reproduction, avoiding lineage extinction, rather than ‘predict’ a future environment that may never exist [15]. Still other models see early adversity as a detrimental disruption to development, no matter what the context is. These models are often referred to as ‘silver spoon’ (the converse as ‘leaden/wooden spoon’) models [33] and suggest that there is a lifelong fitness advantage (good environment ‘silver spoon’) or disadvantage (poor environment ‘wooden spoon’) associated with the early environments that individuals embody. These models are not necessarily adaptive: for example, the poor ‘wooden spoon’ environment is disruptive, creating a disadvantage across the lifespan. In this paper, we contend that the embodiment of the early experience of biological and social environments is a crucial component of facultative reproductive strategies, and thus fertility. However, in such a long-lived species as humans, these pathways are not set in stone as predictive adaptive responses suggest. Crucially, reproductive strategies can be modified throughout a lifetime as subsequent environments influence physiology, behaviour and psychology [4,34–38]. We will begin by discussing the adaptive and maladaptive explanations of childhood stress before examining associations between early stress and age at menarche and fecundity and fertility.

3. Stress: adaptive and maladaptive

The impact of the developmental environment is often discussed in terms of stress. The dominant bio-medical models of stress during development predominately focus on stress as a disruptive, maladaptive (or no longer adaptive) process that in chronic form has long-term consequences for morbidity and mortality. This perspective aligns most closely with the disadvantaged ‘wooden spoon’ model above. The Adverse Childhood Experiences Study conducted in the USA shows that stressful life events have been linked with an increase in risk factors for adult diseases and mortality [39–41]. The biological embedding of this early experience [42] and the negative influence stress has throughout the lifespan, which is often discussed in terms of allostatics [43,44], predicts that the impact of early stress on fertility should be disruptive, reducing fertility.

Two models developed out of life-history theory frameworks examine the potential adaptive association between the early environment and fertility. Both the energetics and the psychosocial stress literature predict associations between the early environment and higher fertility [6] by way of earlier first births. Whether these models are more appropriately regarded as ‘weather forecast’ or ‘making the best of a bad start’ models is currently debated [28,30,36,38]. The energetics literature predicts associations between the early energy and energy balance environments, but the direction of the expected effect depends on the context. Improved health and nutrition is often cited as the reason for the secular decrease in age at menarche, i.e. earlier maturation, in high compared with low socio-economic groups, and urban versus rural communities [45–47]. On the other hand, when conditions are very poor (e.g. malnutrition), it makes evolutionary sense to grow slowly, delaying maturation and reproduction [3,17]. In high-income settings, where poor nutrition is no longer a constraint on reproduction, high-quality childhood environments enable children to develop slowly and extend childhood to enhance other aspects of embodied capital, including somatic investment and social learning [26]. Given the length of gestation and birth spacing in humans, and reproductive lifespans being terminated well before old age thanks to menopause, catch-up pregnancies are not an option [48]. Consequently, delaying reproduction under good conditions ultimately reduces fertility.

Life-history models also propose that under some circumstances poor environments, such as higher levels of familial and social stress, which are associated with higher cortisol levels in children [18], can accelerate reproductive timing [16,49,50]. In environments of higher extrinsic mortality rates (e.g. death from violence, accidents), experienced subjectively as the health and survival of people in your social group (psychosocial stress) [51], it makes adaptive sense for individuals to grow faster and mature earlier, increasing their probability of reproducing at all [52]. In addition, producing more offspring throughout their reproductive lifespan, and necessarily
investing fewer resources in each, maximizes the chance of at least some offspring surviving (e.g. [25,53–55]). This in turn minimizes the probability that one’s own genes will vanish from future generations. In this context, organisms that reduced parental investment per offspring and focused resources on offspring production, increasing fertility, rather than on investment in each offspring, were more likely to survive and reproduce, providing protection against lineage extinction [24,56,57]. Currently, the mechanism by which early stress accelerates reproduction is debated. Although the mechanism may interact with nutrition and energy balance, that is unlikely to be the entire picture as early psychosocial stress is generally recognized as an independent predictor of earlier reproduction [35,55,58].

4. Fertility: a missing link between early environment and fitness

In the childhood psychosocial stress literature from high-income countries, there has been a theoretical and empirical focus on examining the associations between a more stressful childhood social environment and earlier menarche [2,49,50,59]. Menarche has been conceptualized as a good indicator of the reproductive strategy being pursued. Menarche, of course, does not imply sexual activity, let alone early reproduction, but cross-cultural studies often find that age at menarche, age at first sexual intercourse and age at first birth are correlated (e.g. [60]), although not always [37,61]. However, a missing link between an individual’s early stress, reproductive strategy and fitness, is fertility. Here, we are focusing on fertility, which is necessary to achieve reproductive success. It must be recognized that fitness is also dependent on subsequent child survival, which we do not consider here. Although fertility has been central to some theoretical accounts [29,50], it has not been to others [49]. Therefore, it is somewhat surprising that fertility has received comparatively little attention in the empirical evolutionary psychosocial stress literature from high-income nations. In the following section, owing to the empirical focus on menarche as a reproductive strategy marker, we examine the associations between age at menarche and fecundity and fertility before tackling the key question: does childhood psychosocial stress predict adult fecundity and fertility? We finish the section with evidence from experimental psychology studies of the early environment and fertility.

5. Age at menarche and fecundity and fertility

Throughout much of the life-history literature, age at menarche has been promoted as a valid indicator of an individual’s reproductive strategy. Menarche is a measure of sexual maturity, is necessary for fecundity and indicates a woman is transitioning into a period of fertility. Fecundity is the ability of an individual to conceive and have a live birth. As such, fecundity can never be measured directly and is only estimated from the components of reproduction. Fertility is the direct measure of the number of live births an individual has. While this is a clearly defined measure, the literature examining direct associations between menarche and fertility is scant.

Females are generally not considered fecund at menarche [62]. The most common reason proposed for adolescent subfertility is the absence of ovulation during the early post-menarcheal menstrual cycles [48,62]. The time until onset of regular ovulatory cycles is highly variable [63,64]. The percentage of fertile cycles increases steadily with age since menarche [65,66]. When the analysis is conducted between menarcheal age groups, in comparison to late matures, ovarian function increases more rapidly in women with earlier menarche [67].

In addition, a woman’s age at menarche is consistently associated with other aspects of her reproductive strategy. An earlier menarche has been associated with a longer reproductive lifespan [68], a younger age at first sex [69–71] and first pregnancy [72,73], an increased risk of teenage pregnancy [74] and a shorter time to pregnancy [60,72,75]. As each of these studies adjusted for a range of potential confounders (e.g. socio-economic status), this evidence suggests earlier matures are more fecund and more likely to become pregnant at a younger age.

This is a useful avenue of investigation; however, the associations between menarche and reproductive hormones and adult reproductive outcomes are not conclusive [6]. Indeed, especially in high-income populations, it does not necessarily follow that women who reach sexual maturity at a certain age are locked into a cascade of adult reproductive events [2]. Life-history theory is usefully employed to understand this variance as it addresses how aspects of the developmental environment can impact the pathways by which an individual may reach similar outcomes, such as early menarche, but generate diverging adult phenotypes and intergenerational trade-offs [34,35]. Therefore, to gain a more complete understanding of developmental influences on reproductive strategies, it is necessary to also consider the impact the early psychosocial environment has on fecundity and fertility.

6. Does childhood psychosocial stress predict adult fecundity and fertility?

A more adverse childhood psychosocial environment has been associated with a range of reproductive traits including earlier menarche, earlier first sexual intercourse, increased desire to have children, increased readiness for children, earlier expected family formation, more sexual partners, instability of marital relations, increased risk of unplanned pregnancies, shorter time from menarche to first conception and earlier first pregnancy and first childbirth [76–79]. Moreover, experiencing more adverse childhood events has been associated with an increased risk of poorer pregnancy outcomes [80]. Scan attention, however, has been paid to fecundity and fertility. Therefore, both theoretical and empirical work suggest that investigating associations between the early psychosocial environment and fecundity and fertility in humans is likely to be fruitful.

The associations between early adversity and fecundity and fertility have been investigated using prospective data on 6477 women from the British National Child Development Study (NCDS; [81]). Diverse measures of childhood adversity (e.g. financial hardship, a carer with low interest in education, family structure disruption) were consistently associated with an increased likelihood of ever becoming pregnant in women who had never married. Although all of these factors have been associated with, and therefore may be confounded by, lower socio-economic status in the literature, the above association was robust to adjustment for menarcheal age, body mass index (BMI), smoking, social class and education. Interestingly, none of these effects were found in women who had ever been married, for whom early adversity was associated with a
longer time to pregnancy; however, adjustment for social class and education in adulthood removed all associations [81]. This suggests that the impact of an adverse early environment on fertility may be moderated by marital status and mediated by the adult socio-economic environment in slow reproductive strategies. It makes sense that an individual’s reproductive physiology should respond to both early and adult environments. The potential association between additional parental and family resources with delaying pregnancy may be usefully interpreted within a life-history theory framework.

In this study, witnessing abuse, conflict in the home or maternal alcoholism (recalled at 41 years of age) were associated with an increased risk of a woman being told by a doctor she could not have children [81]. Consistent with this, retrospective US studies found adverse childhood experiences were associated with an increased risk of experiencing fertility difficulties, irregular menstruation and amenorrhea [82,83]. Conversely, in a racially heterogeneous, low socio-economic US sample, sexual abuse before 14 years of age was associated with reporting more pregnancies and children [79]. This evidence shows the association between early stress and fertility from retrospective studies is currently mixed.

Using the 1934–1944 Helsinki Birth Cohort, women who as children (4–11 years of age) were evacuated from Helsinki during WWII gave birth to more children, in comparison to women who were not evacuated [61]. As evacuees came from more adverse family backgrounds and had larger family sizes (in 1940), the study was extended to the entire evacuation population and same-sex sibling comparisons were made. In this analysis, the association between evacuation and fertility was not found [84]. Although the within-sibling design is powerful and controls for familial factors, the assumed similarity of experience and condition before and after separation for evacuated and un-evacuated siblings who were no doubt of different ages and health status seems unlikely. Indeed, the characteristics of the children chosen to be evacuated are likely to be influenced by their parents’ reproductive strategies [85]. Finally, in a prospective study of women born in Newcastle-upon-Tyne in 1947, low socio-economic status and poor housing conditions at birth were associated with earlier first birth, and poor housing was also associated with higher completed fertility. When both outcomes were tested simultaneously, age at first birth completely mediated the relationship between poor housing and total fertility [37].

These studies suggest that individuals’ early psychosocial environments are associated with their subsequent fertility. The finding of increased fertility associated with early stress being confined to the never-married women may reflect repeated environmental cues associated with an accelerated reproductive strategy. As fertility is increasing rather than decreasing in its association with early adversity, this pattern would not appear to fit adaptive suppression or maladaptive disruptive explanations. Conversely, that childhood adversity was associated with reduced fecundity in the ever married women is not consistent with the early stress hypothesis. It is possible, however, that this may reflect plasticity in response to the adult environment. The increased social, emotional and financial resources that can be available within a stable pair bond may have changed the allocation of resources to reproduction and maintenance such that temporarily delaying reproduction may be beneficial [3,6,18]. Further investigation is needed to establish whether there is any adaptive significance of these interactions between the childhood and adult environments.

It is worth noting that research conducted using the NCDS has also found that low paternal involvement and a lower birth weight were independently associated with an earlier age at first birth, an increased risk of teenage pregnancy and the intention to start a family before 20 years of age [77,86]. In addition, women who became teenage mothers showing distinct patterns of growth and psychological development compared to matched controls who did not become teenage mothers [74]. Therefore, using data collected in the NCDS, it has been shown that childhood adversity is associated with earlier reproduction and an increased probability of ever falling pregnant, potentially increasing fitness.

The information surrounding the childhood psychosocial environment and its potential impact on fertility and fecundity is growing. With time, analyses are more likely to integrate theoretical predictions and hypothesis-testing into their analyses. For example, to examine the influence of early stress and any associated developmental trajectory to changes in fertility, it would be useful to examine the potential moderation by age at menarche, first sex, pregnancy and childbirth. Not everyone who experiences early stress goes on to accelerated reproductive development and timing; those that do are likely to be of the highest risk [35,37]. Perhaps this is reflected in the different findings for never- and ever-married women in the NCDS [81]. Evidence and theory support the idea of moderating factors throughout the lifespan that may be fruitfully explored by demographers and evolutionary anthropologists as they move into this area [87].

7. Does the association between early stress and fertility interact with current stress?

The interaction between the hypothalamic–pituitary–adrenal axis and hypothalamic–pituitary–gonadal axis means current stress is likely to disrupt or adaptively delay reproduction. When circumstances are temporarily unfavourable for reproduction, it may be pertinent for the woman to focus scarce resources on her own health and maintenance and support of existing offspring, suppressing this specific reproductive event [3,18,32]. Evidence from different disciplines supports this. The most direct evidence comes from Mayan women in the highlands of Guatemala. Women who experienced higher cortisol levels in the first three weeks of pregnancy were almost three times more likely to lose their pregnancy during that period [88]. However, the developmental environment is also important. Across mammalian species, although juvenile and adult mortality rates were highly correlated, juvenile mortality is a stronger predictor of fecundity than adult mortality [22]. A nation-level analysis of the impact that early and current mortality rates have on adolescent pregnancy rates also shows that infant mortality has a strong association with reproductive strategies, which is partly mediated by the adult mortality environment [4]. At the individual level, this increased mortality may be experienced as stress. Women who experience higher levels of childhood adversity are at an increased risk of re-victimization and experiencing new stressors in adulthood and pregnancy [89,90]. Thus, adult stress may mediate, moderate or confound the impact of childhood stress on fertility.

Two studies examining the association between childhood stress and fertility have been able to take into account adult or current stress. In a retrospective study of pregnant
and non-pregnant women, where childhood stress reduced fertility and fecundity, adjusting for trauma experienced since 18 years of age slightly attenuated the early stress associations [82]. Similarly, in a study of incarcerated women, experiencing physical or sexual abuse before 16 years of age, having a parent with a history of drug abuse, and any lifetime sexual abuse were associated with reduced fecundity independent of the impact of current stressors [83]. Therefore, even in this disadvantaged group of women with chronically high stress, childhood stress remained an independent predictor of adult fecundity.

8. Experimental evidence: early environment and adult fertility choices

Recent experimental evidence from USA studies supports the notion that reproductive decisions, made in response to the current environment, are moderated by individuals’ childhood environments [91,92]. The common experimental method used is priming, whereby individuals’ perceptions of their environment are temporality shifted through exposure to a composed stimulus, the prime, e.g. reading a newspaper article suggesting that extrinsic mortality is increasing. In general, these studies have shown that fertility preferences are susceptible to priming. In both European and Chinese samples, priming higher mortality risk increases desire for children and interest in babies in both sexes [93,94]. However, few studies have focused on the moderating effect of childhood environment [91,92]. Theoretical arguments suggest it is likely that childhood environments have long-term effects on reproductive preferences and behaviour. However, facultative life-history strategies may be dormant under relatively positive environmental conditions, with individuals instead adhering to local cultural norms and expectations [91,92]. Evidence suggests that individuals from harsh or adverse childhood environments, when primed for current environmental harshness, may express the fast life history they were sensitized to during childhood. When primed with high mortality risk, individuals from harsh childhood environments prefer an earlier age at first birth [91,92], but unexpectedly, smaller preferred completed fertility [92]. Conversely, when exposed to primes of resource scarcity, individuals from harsh childhood environments preferred later ages at first birth and equivalent completed fertility to controls [92]. In response to primes of high mortality risk, individuals from non-harsh childhood environments preferred later ages at first birth [91], or equivalent preferred ages at first birth and completed fertility compared to controls [92]. As discussed previously, the low response from non-harsh childhood environment individuals to cues of current environmental harshness may be because this period of harshness is temporary and it is adaptive to delay reproduction, while individuals with faster life-history strategies may assume more environmental fluctuation and benefit from quicker adaptation to environmental changes. Thus, experiencing a harsh environment in adulthood may encourage expression of latent life-history strategies cued during childhood.

Priming experiments have also examined the effects of paternal absence and disengagement [95]. Women who were asked to think about an important time in their life when their father was unavailable had more sexualized thoughts, increased self-reported sexual permissiveness and negativity towards condom use compared to women asked to think about an important time in their lives where their father was present. These effects were specific to women’s sexual decision-making and paternal absence was key, with the absence of a close friend having no effect. If this priming of father absence can increase sexual risk-taking, experiencing prolonged paternal absence may increase risk-taking throughout adulthood, possibly resulting in earlier reproduction, higher fertility and perhaps lower investment in their children—hallmarks of a faster life-history strategy. Currently, these experimental studies are largely restricted to high-income countries and further research is required before the generalizability of these results can be established [96].

9. Mechanisms linking the developmental environment and fertility

In this section, we examine potential biological, social and psychological mechanisms that are consistent with life-history theory frameworks, and may provide pathways through which the early environment can influence fecundity and fertility. Integrating ultimate and proximate explanations of reproduction (e.g. reproductive timing) usefully synthesizes areas of research and generates potential future research directions [97,98]. Although not an exhaustive list, we examine the evidence that birth weight, childhood body composition, risky health behaviours and the developmental origins of attractiveness influence fertility.

(a) Birth weight and fertility

Fetal growth, often measured as birth weight, has lifelong consequences for reproductive health. Owing to the association between reduced fetal growth and increased morbidity and mortality throughout infancy, childhood and adulthood [7,99], it is usefully examined within a life-history context. Birth weight is often used as a measure of parental investment during pregnancy. This level of parental investment can be conceptualized as an early-life factor that influences a woman’s subsequent reproductive scheduling [77] or a consequence of her reproductive strategy that has implications for survival and reproduction in subsequent generations [35]. For example, a woman’s birth weight predicts her age at menarche [100,101], and in turn, a woman’s menarcheal age is associated with her offspring’s birth weight [102,103]. Moreover, as all the primordial follicles a woman will ever have are present in her fetal ovaries, it is likely the fetal environment will directly influence subsequent fertility, as it does in other animals [104].

Women who are born underweight are more likely to have a smaller uterus, smaller ovarian size, a reduced ovulation rate, and are less likely to reproduce [105,106]. A shorter gestational age, which is also associated with increased childhood mortality, has been associated with a lower probability of finding a partner and having children [105,107,108]. Strikingly, in this apparent disruptive physiology, women who are born small for gestational age are more likely to give birth at a younger age [105,109]. Similarly, women exposed to the Dutch Hunger Winter of 1944–1945 famine during pregnancy had more children, an earlier age at first birth, were more likely to have twins and less likely to remain childless [110]. However, other analyses using the same sample with different control groups have not found this association [111,112].
Currently, the developmental effects of reduced fetal growth on fertility in humans are complex, incompletely understood and may have both promoting and disruptive aspects.

(b) Body composition

Body composition directly influences both age at puberty and the ability to produce children. Female reproduction is constrained by skeletal growth and pelvic maturation [3], traits that are also correlated with age at menarche. Skeletal growth, which is strongly influenced by the quality of the early environment [113], matures around the late teenage years and early twenties when the female becomes capable of successful reproduction, in line with the life-history model of the trade-off between growth and reproduction. Although there are meagre data on body composition and completed fertility in humans, what evidence there is suggests that growth and body composition are independently related to fertility, and not simply as a function of age at menarche or first pregnancy.

Guatemalan women who had higher BMI scores as adolescents had earlier first births, and more children overall, although neither of these outcomes was associated with adolescent height [114]. Higher height-for-age at age two was a good indicator of adolescent BMI in these women. Also in Guatemala, well-nourished girls had improved childhood growth and achieved earlier menarche, first sex and first birth. This relationship held independently for each event [115]. In Bangladesh, heavier women are able to marry younger (independent of age at menarche), promoting earlier reproduction and higher lifetime fertility [116]. In the Gambia, taller women had later first births but also increased reproductive success because these children had improved chances of survival [117,118].

Attained height has its own impact on fertility as, although relatively shorter women are likely to have started reproduction earlier [74,118–120], smaller body size is also associated with adverse health outcomes [121]. The relationship between height and fertility therefore tends to be inverse U-shaped, with average or above average height women having the highest reproductive output, although this is not always the case and is heavily dependent on resource availability [119,122]. Indeed, reproduction while still growing, potentially through resource competition [123], may create trade-offs that reduce adult height [124]. Similar results have been found for BMI. In contemporary Finland, both men and women of average BMI as adolescents had more children overall. Both under-weight and overweight individuals had fewer children, which was attributed to the likelihood of extreme BMIs affecting partnership status [125]. This study was replicated in the US and found largely similar results [126].

These examples indicate that the relationship between body composition and fertility is not straightforward, although it appears that being average or slightly above average size is most beneficial for reproductive success. It is clear that there is an independent relationship between body size and fertility, rather than simply being a function of reproductive maturity.

In some cases, this may be owing to social practices (like a height preference in mates) but this is not the case across all cultures [118]. Alternatively, height and weight may indicate general health, which is also likely to influence fecundity and fertility. Fertility is thus the indirect product of events from early-life growth, including size at birth, age at puberty, adolescent height, age at first birth and attained adult height.

(c) Attractiveness and early stress

Risky health behaviours have been associated with both increased and decreased fertility and may be usefully interpreted within a life-history theory framework. Behaviours such as smoking, alcohol use, drug use, excessive weight gain and risky sexual behaviours, which are more likely in individuals who were brought up in families characterized by neglect, conflict and abuse [12,39,127–129], have also been associated with a more current-oriented reproductive strategy [130,131] and are likely to be associated with fertility [81,132].

Behaviourally, higher levels of risk-taking may reflect a shorter time preference. Risk-taking has been associated with an adverse childhood environment and was significantly higher in those who expected to have a short lifespan and believed the future was unpredictable [133]. A truncated life expectancy has been positively correlated with both age at menarche and early psychosocial stress in an American university sample [51] and a sample of 100 primiparous Australian mothers, half of whom were teenage mothers [134]. Thus, short time preference provides a potential mechanism by which early stress influences fertility.

Adverse childhood experiences may also influence fertility through risky sexual behaviours, however, the relationships are likely to be complex. On the one hand, adverse childhood experiences have been associated with an increased risk of sexual risk-taking behaviours [129] and contraction of sexually transmitted diseases [135], which in turn are associated with an increased risk of infertility [136]. At the same time however, childhood adversity has also been associated with earlier menarche, an increased risk of pregnancy at a younger age [86,134,137] and unplanned pregnancies [76], all of which have been associated with increased fertility.

Similarly, childhood weight gain and subsequent obesity may be a double-edged sword for fertility [32,138]. Both under- and over-nutrition are associated with reduced fertility [139]. Menstrual cycle functioning is closely linked to nutritional status, with under-nutrition reducing ovulation frequency [3]. Rapid childhood growth in response to an adverse childhood environment may accelerate sexual maturity and increase early fertility [35]. On the other hand, childhood maltreatment is associated with an increased risk of adult obesity [140,141], which is associated with a lower probability of finding a partner and conception, a longer time to pregnancy and reduced fertility, malnutrition during pregnancy and reduced placental function [82,125,142–144].

Whether the proximate mechanism involves chronic stress, altered time preference or increased weight gain, it is likely that these childhood experiences are embodied [42–44,58,145]. Generally, this is thought to be via dysregulation of the hypothalamic–pituitary–adrenal axis, resulting in altered stress reactivity [146]. At the molecular level, these developmental environments may influence fertility through epigenetic mechanisms that regulate stress hormones (e.g. glucocorticoids) via their receptors [1,13,147,148].

(d) Risky health behaviours and time preference

The evolutionary psychology literature suggests characteristics we find attractive reflect an individual’s potential fecundity or fertility [149]. Although it does make intuitive sense that attractive people have more offspring than less attractive people, they are also more likely to be successful in other aspects of their lives, which may create trade-offs between reproductive...
investment and embodied capital (e.g. career [26]). In humans, attractiveness is often but not always associated with increased fecundity and fertility [150,151]. Increased weight compared to normal weight, which may be associated with increased fecundity in low income populations [152], is associated with increased facial attractiveness and increased fertility in the Ache [153].

Investigations of the associations between early stress, reproductive strategies and attractiveness have found males who experience early stress dislike masculine female faces [154]. This preference for opposite-sex partners with more sex-specific facial traits also holds for individuals with accelerated reproductive strategies. Women who experienced early menarche and first sex showed a preference for more masculine male faces, while in males, early first sex was associated with a preference for more feminine female faces [155].

Consistent with the animal literature, evidence of a relationship between deviations from bilateral symmetry and developmental instability are found in the human literature [156]. In a study of early stress and facial symmetry, lower socio-economic status during childhood, but not in midlife, was significantly associated with reduced facial symmetry at age 83 [157]. Recent attempts to examine developmental influences on attractiveness, focusing specifically on childhood health, have not been supported [158,159]. Consistent with this interpretation, in a study of Latvian women, facial attractiveness was not associated with immune responsiveness; however, it was associated with common correlates of stress. Women who experienced higher cortisol levels were rated as less attractive, as were women who were too thin or overweight [160]. Fecundity has received less attention; however, associations with hormone levels have also been reported. In a Polish study, more symmetrical women had a 1.3% higher average salivary oestradiol over the menstrual cycle, peaking at 28% for mid-cycle levels of oestradiol [161]. Therefore, although current weight and obesity are likely to have direct consequences for attractiveness and fertility, the current evidence is inconclusive regarding the association between the early environment and adult attractiveness.

10. Conclusion

A broad life-history theory framework is useful for examining the adaptive, maladaptive and disruptive influences of the early environment on subsequent fecundity and fertility. There is an extensive literature examining the associations between the early psychosocial environment and myriad life course factors reflecting reproductive development, partnership formation, reproductive timing and reproductive outcomes. Therefore, the lack of attention that fertility has received in the literature is striking. Within this literature, fertility is a missing link between the early environment and fitness. We find that earlier menarche is associated with physiological and behavioural characteristics that increase fecundity at a younger age, increasing the probability of becoming pregnant and most likely fertility. The early adversity and fertility literature, which predominately comes out of the public health and epidemiology fields, is small, correlational and not yet conclusive. In three longitudinal analyses [37,61,81] and one retrospective study [79], early adversity increased fecundity and fertility. This is consistent with an adaptive allocation of resources to current reproduction in a high risk environment. However, the remaining retrospective analyses found a negative, most readily interpreted as disruptive, association [81–83]. Crucial to understanding the complexities of these associations are the mediating and moderating factors that contribute to fertility decisions throughout a woman’s life. Socio-economic status, owing to its relationship with resource availability, is crucial to this and must be explored carefully across the lifespan, not merely discarded as a confounder [97,98]. Therefore, a life-history strategy may respond to sensitive periods of development (e.g. pregnancy and infancy), yet they are not programmed and are best served by responding to the changing demands of the environment. This review also showed that mechanisms likely to be on the proximate causal pathway are (i) associated with both early adversity and fertility and (ii) usefully interpreted within a life-history theory framework including the woman’s fetal environment, early body composition, risky health behaviours and developmental influences on attractiveness. The interactions between adverse developmental environments and these pathways highlight future opportunities for fertility research.

Authors’ contributions. D.A.C., M.T., L.M. and P.S. drafted and reviewed the manuscript. All authors gave final approval for publication.

Competing interests. We have no competing interests.

Funding. We received no funding for this study.

Acknowledgements. This review came out of an interdisciplinary workshop ‘Integrating Evolutionary Models of Human Fertility Change’ at the National Evolutionary Synthesis Center (NESCent), North Carolina, USA. The authors thank the organizers Mary Shenk, Daniel Hruschka and Rebecca Sear, and attendees, for helpful comments. We also thank Toni Wain and Jim Chisholm for editorial comments.

References


76. Dietz PM, Spitz AM, Anda RF, Williamson DF, McAlister PM, Santelli JS, Nordenberg DF, Felitti VI, Kendler KS. 1999 Unintended pregnancy among adult women exposed to abuse or household dysfunction during their childhood. JAMA 282, 1359e64. (doi:10.1001/jama.282.14.13599)


85. Hidy SB. 2009 Mothers and others: the evolutionary origins of mutual understanding. Cambridge, MA: Harvard University Press.


