Life-history theory, chronic childhood illness and the timing of first reproduction in a British birth cohort

David Waynforth*

Department of Population Health and Primary Care, The Norwich Medical School, University of East Anglia, Norwich, Norfolk NR4 7TJ, UK

Life-history theoretical models show that a typical evolutionarily optimal response of a juvenile organism to high mortality risk is to reach reproductive maturity earlier. Experimental studies in a range of species suggest the existence of adaptive flexibility in reproductive scheduling to maximize fitness just as life-history theory predicts. In humans, supportive evidence has come from studies comparing neighbourhoods with different mortality rates, historical and cross-cultural data. Here, the prediction is tested in a novel way in a large ($n = 9099$), longitudinal sample using data comparing age at first reproduction in individuals with and without life-expectancy-reducing chronic disease diagnosed during childhood. Diseases selected for inclusion as chronic illnesses were those unlikely to be significantly affected by shifting allocation of effort away from reproduction towards survival; those which have comparatively large effects on mortality and life expectancy; and those which are not profoundly disabling. The results confirmed the prediction that chronic disease would associate with early age at first reproduction: individuals growing up with a serious chronic disease were 1.6 times more likely to have had a first child by age 30. Analysis of control variables also confirmed past research findings on links between being raised father-absent and early pubertal development and reproduction.

Keywords: mortality; risk; evolutionary medicine; father absence; maturity

1. INTRODUCTION

Natural selection acts on the timing of reproductive maturation and first birth, in part, because mortality rates differentially affect the fitness costs and benefits of early reproduction. Under high mortality or in risky environments, individuals who mature and reproduce early are likely to have higher reproductive success than those who reproduce later, because they are less likely to die before having an opportunity to reproduce. Early maturation and reproduction are not without costs, however, and early reproduction is usually expected to be associated with a smaller body size at maturity and, *ceteris paribus*, less total energy available for reproduction and lower lifetime reproductive success [1,2].

In life-history theory, a distinction is made between extrinsic and intrinsic causes of mortality: extrinsic mortality is from causes outside of the control of the individual, whereas intrinsic mortality causes are those in which death can be hastened or delayed as a function of an individual's energy allocation decisions [1]. For example, physiological ageing can theoretically be delayed by allocation of energy to cellular maintenance and away from reproductive effort. The prediction that first reproduction should occur earlier under high adult mortality more specifically is that first reproduction should occur earlier under high *extrinsic* mortality. It is not the case that maturity should occur early when an individual can reallocate energy to increase their likelihood of survival, unless the benefits of the energy reallocation are outweighed by fitness costs. In practice, many

causes of mortality appear to have both extrinsic and intrinsic components [1,3]. For example, someone living in a neighbourhood with a high homicide rate could alter their behaviour to maximize their survival, and thus reallocate time and energy to influence mortality risk.

There is evidence in a variety of species across different taxa for the evolution of adaptive flexibility in the timing of reproductive maturity and reproductive effort in early adulthood depending on extrinsic mortality [4,5]. For example, Stibor [6] exposed *Daphnia* (a freshwater invertebrate) to chemicals associated with the presence of predators. With these chemicals present without their associated predation risk, *Daphnia* matured and reproduced at an earlier age than an unexposed control group.

A number of researchers have applied the mortality risk life-history theoretical framework to age at first reproduction or menarche in humans. A difficulty in doing so is that it requires measuring mortality risk or finding a suitable proxy measure that isolates mortality risk from other factors that would cause shifts in allocation of effort to current versus future reproduction. A handful of researchers have analysed regional or national mortality statistics for correlations between age at first reproduction and mortality rates. Wilson & Daly [7] compared neighbourhoods in the Chicago area with different mortality rates, and a substantial proportion of the difference in mortality between neighbourhoods being due to differences in homicide rates. Teenage birth rates in the highest-mortality neighbourhoods were much higher than in low-mortality areas. This pattern has been replicated in other nations and in comparisons between nations and time periods [3,8–10], and at the level of the

individual implies that those who see or are aware of young adult deaths around them tend to discount the future, and have children at a young age as life-history theory predicts. One problem with some of these studies is that life-expectancy reduction owing to homicide in deprived areas is typically quite modest, at up to about a 5-year decrease for African American men in the most dangerous Los Angeles neighbourhoods [11].

Other studies have included variables less closely linked to mortality but which are indicators of unstable or poor environments, such as father-absent child-rearing and measures of childhood stress [12–15]. This introduces a greater potential influence of other costs and benefits of investing in current versus future reproduction, of which a number are possible that are not directly related to mortality risk. For example, there may be different payoffs to fathers of investing heavily in offspring quality in stable versus unstable social and economic conditions that affect the benefits of delaying reproduction in order to accrue resources to invest in offspring [16].

Data that may be closer to a natural experiment in which only extrinsic mortality risk varies between study participants are chronic health conditions such as diabetes and severe asthma, which are unlikely to be significantly affected by shifting allocation of effort away from reproduction towards survival (e.g. through immunity), have comparatively large effects on adult mortality and life expectancy, and are less likely to vary systematically with the income or social class of the family that the affected individual was born into.

It may seem improbable without a life-history theoretical perspective that chronic health conditions would associate with early reproduction, but a number of studies have shown that adolescents with chronic conditions are more likely to engage in sexual activity and risky behaviours that often covary with sexual activity [17–20]. Here, data from the 1970 British birth cohort study (BCS70) of all recorded births in a one week period in 1970 (initial study $n = 17\,198$ births) were analysed to compare age at first reproduction in individuals with and without a serious chronic disease diagnosed by age 10.

2. METHODS

(a) *Sample*

The BCS70 sample consisted of 17 198 births in the UK between 5 and 11 April 1970. Follow-up data were collected for the entire sample or part of it at ages 5, 10, 16, 26, 30, 34 and 38. Here, data collected at age 10 in a health interview and at 30 years old by telephone interview were analysed [21,22]. Data were complete for 9099 individuals at both ages 10 and 30, with the remainder missing owing to a number of causes, but in most cases for not providing information on some variables. Ninety-six per cent of the BCS70 sample classified themselves as ethnically White, with the highest proportion of other ethnicities being Indian (0.9%, 99 individuals) and Pakistani (0.6%, 66 individuals).

(b) *Variables and statistical procedures*

Health data including the presence of chronic illness were collected at age 10 by participants' school nurse and the community medical officer. The data included lists of past and present health problems and use of health services,

and these were used to determine whether the participant had a chronic condition diagnosed between birth and age 10.

Inclusion criteria in studies of behavioural correlates of chronic conditions have varied from analysis of a single condition to include almost all self-reported cases of chronic illness. Here, the focus is on chronic conditions diagnosed in childhood that reduce life expectancy while not causing severe physical or mental disablement or disfigurement. Examples of included conditions are cancers, severe asthma, type I diabetes and epilepsy. Examples of excluded conditions are those that are usually profoundly disabling, including Down's syndrome, neural tube defects and cystic fibrosis. Conditions that are primarily acute, such as life-threatening neonatal conditions were also excluded (for example, Ramstedt's pyloric stenosis), as were chronic conditions that are less likely to reduce life expectancy, including mild asthma, eczema and benign heart murmurs diagnosed in infancy.

Because social class and educational attainment are often associated with reproduction [23], parental socioeconomic status (SES) when the participant was aged 10 was included as a covariate in the statistical model. SES data were coded using the 1980 Registrar General's classification of social class based on occupation [24]. The SES of the head of household in which the child lived was used. If the father was absent or not employed and the mother was working, then the mother's SES classification was used.

Educational attainment is usually found to be an important predictor of the timing of first birth, with higher levels and leaving ages of education associated with delayed first birth [25]. In addition, it is possible that those with a chronic disease will have lower educational attainment due to their ill health, and will as a consequence have ages at first reproduction similar to healthy individuals with the same educational attainment. To control for education, the individual's age when they left full-time education was coded on a three-point scale by grouping all those who left education before age 17; from 17 to 18; and post-18. These ages represent the British legal school-leaving age of 16, and being in full-time education beyond the two national examination milestones of 'O' levels and 'A' levels.

It is plausible that raising a child with a serious chronic condition will place a stress on marriage that results in an increased probability of divorce or separation. Because being raised father-absent is itself a risk factor for early reproduction, father absence is another route via which chronic illness may associate with early reproduction. Being raised father-absent owing to parental divorce or separation was statistically controlled for using a binary variable coded as one if the father was present up to the time of the 1980 (age 10) interview, or two if he was absent.

Father absence may affect age at first reproduction through accelerated sexual maturity: early menarche and being raised father-absent are often found to be associated [12]. The BCS70 health survey (carried out by community health officers and nurses) included a count of the number of different signs of pubertal development present at age 10, and this count was included to determine whether those with chronic disease reached sexual maturity earlier, and whether pubertal development is a likely mechanism through which chronic disease could lead to early reproduction.

Cox regression was used to analyse age at first reproduction using the following model selection approach: all control variables were entered as covariates and retained in regression

Table 1. Pearson correlations between independent variables.

	child's sex	parental SES	education	father-absent by 10	signs of puberty (n)	serious chronic illness
child's sex (1, male; 2, female)	1	0.001	0.070**	0.024*	0.267**	0.000
parental SES (1, high; 6, low)	0.001	1	−0.334**	0.065**	0.009	0.003
education (1, low; 3, high)	0.070**	−0.334**	1	−0.081**	0.042**	−0.004
father-absent by age 10 (1, no; 2, yes)	0.024*	0.065**	−0.081**	1	0.023*	0.015
number of signs of puberty at 10	0.267**	0.009	0.042**	0.023*	1	−0.003
presence of serious chronic illness (1, no; 2, yes)	0	0.003	−0.004	0.015	−0.003	1

*Correlation is significant at the 0.05 level (two-tailed).
**Correlation is significant at the 0.01 level (two-tailed).

Table 2. Summary of alternative Cox regression models predicting age at first reproduction using the study variables. Results for step 1 represent change from a model without covariates. No combination of interaction effects significantly improved the −2 log-likelihood over the model shown in step 2 below.

	variables included in model	χ^2 for change from previous step in −2 log-likelihood	p-value and degrees of freedom for change from previous step
step 1: control variables only	sex SES education father absence number of signs of puberty at 10	774.4	>0.0001 (d.f. = 5)
step 2: control variables and chronic illness	sex SES education father absence number of signs of puberty at 10 Chronic illness	10.2	>0.002 (d.f. = 1)
step 3: all variables and interactions with chronic illness	sex SES education father absence number of signs of puberty at 10 chronic illness sex × chronic illness SES × chronic illness education × chronic illness father absence × chronic illness signs of puberty × chronic illness	4.9	>0.5 (d.f. = 5)

models, followed by entering the presence of chronic disease, then all main and interaction effects between chronic disease and the control variables entered together. Interaction effects were removed by backwards selection if they did not significantly change the model −2 log-likelihood value.

3. RESULTS

The most frequently occurring chronic conditions were epilepsy, type I diabetes, severe asthma, renal disease (the most common being nephrotic syndrome) and congenital heart defects with long-term implications (the most common of these was tetralogy of Fallot). These accounted for 58 of 84 cases of chronic disease, with the remaining cases consisting of fewer than 10 cases each of cancers, stroke and various congenital conditions such as sickle cell disease and Gaucher's disease. Being diagnosed with a serious chronic illness was not significantly correlated with any of the study control variables

(table 1). A total of 8.6 per cent of the sample was father-absent by age 10, and signs of puberty were not common: the mean number of pubertal signs was 0.17, with girls showing more signs of pubertal development than boys (table 1).

In Cox regression models of age at first birth, all main effects with the exception of number of signs of pubertal development significantly improved the model fit, and no interactions including chronic illness significantly influenced model −2 log-likelihood (table 2). Further analysis was carried out only for main effects.

Table 3 displays a summary of a Cox regression model predicting age at first birth, including all main effects. Consistent with prior studies using the BCS70 cohort [26], women had earlier ages at first birth than men. The results for education and parents' social class showed that both were significantly associated with age at first birth: remaining in full-time education for longer was associated with later first birth, with 32 per cent of

Table 3. Summary of Cox regression results for the effects of the explanatory variables included in step 2 of table 2 on age at first reproduction.

	B	s.e.	Wald	d.f.	p-value <	exp(B)
sex (1, male; 2, female)	0.530	0.031	285.139	1	0.000	1.698
father-absent by age 10 (1, no; 2, yes)	0.209	0.047	19.667	1	0.000	1.232
number of signs of puberty at 10	0.001	0.028	0.001	1	0.974	1.001
parental SES (1, high; 6, low)	0.083	0.013	42.404	1	0.000	1.086
education (1, low; 3, high)	−0.382	0.022	295.583	1	0.000	0.683
presence of serious chronic illness (1, no; 2, yes)	0.515	0.147	12.351	1	0.000	1.674
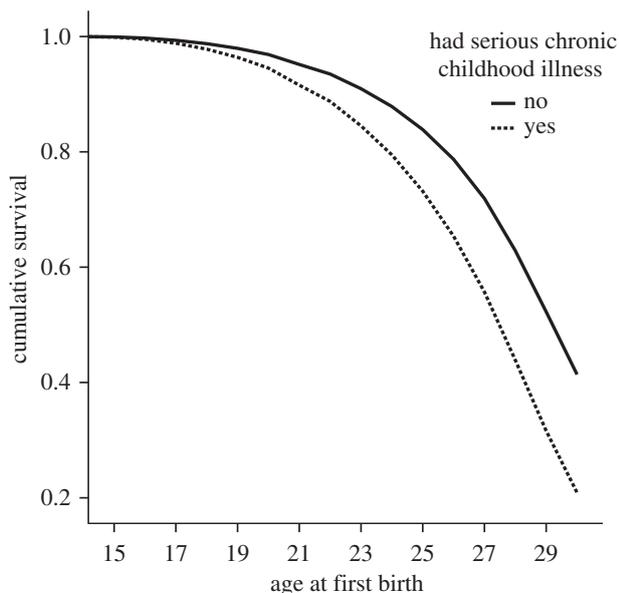
Figure 1. Cumulative survival curves for age at first birth (the proportion of individuals who have not had their first child by the age shown on the x-axis) for those with and without a chronic disease diagnosed by age 10.

those who continued education past age 18 having had a child by age 30 versus 57 per cent of those who left school at or below the legal school-leaving age of 16. Higher parental SES was associated with a reduced hazard of birth before age 30 (table 3), with SES coded as 1 for the highest social class and 6 as the lowest.

Being raised father-absent was associated with earlier first reproduction. The hazard of birth before age 30 for individuals raised father-absent was 1.23 times that of those with both parents present in the home (table 3). The presence of chronic disease also had a significant effect on the hazard of birth by age 30: those with a serious chronic disease had 1.67 times the risk of birth compared with those without a chronic illness. Figure 1 displays cumulative survival curves for age at first birth in individuals with and without chronic disease.

4. DISCUSSION

The results were consistent with the prediction that a shorter expected lifespan from causes outside of the individual's control should lead to earlier first reproduction, and were consistent with research on reproductive correlates of other measures of mortality risk, such as local homicide rates [7].

The present study did not successfully identify intermediary factors linking chronic illness to early reproduction. It was hypothesized that children with a serious chronic illness would have lower educational attainment and would be at greater risk of being raised father-absent, and via these intermediary variables would have earlier ages at first birth. However, chronic illness was uncorrelated with educational attainment, parental SES and father absence, and each of these variables was independently significantly associated with the timing of first reproduction.

Father absence may influence age at first reproduction, in part, via accelerated reproductive maturity in children raised father-absent. The results here did not suggest a similar pathway to early reproduction for individuals with chronic disease. Presence of chronic disease was not significantly correlated with number of signs of pubertal development at age 10, nor were signs of pubertal development at 10 associated with age at first birth. This is perhaps not surprising in a population in which reproduction usually begins many years after reproductive maturity: less than 1 per cent of the sample had a child before age 18.

The results were consistent with prior findings on associations between father-absent rearing and the timing of reproduction: father absence was associated both with earlier reproduction in offspring and with more signs of pubertal development at age 10 (table 1). Father absence was also significantly associated with lower parental SES and lower educational attainment, and was more common for girls. Although these correlations were small, the sex difference may be indicative of effects of evolved parental investment strategies, as boys require higher levels of parental investment than girls [27,28]. Thus, fathers may be less likely to desert a relationship with a son's mother because the costs are higher in terms of increased likelihood that a son will fail to survive and successfully reproduce after having lost his paternal investment.

Life expectancy for individuals with most of the chronic diseases included in the study has increased significantly in the past 50 years and continues to increase. Despite life expectancy remaining lower for those with serious chronic disease, differences in reproductive timing between those with and without chronic disease should be less evident in more recent cohorts, although it is not certain that reproductive responses will quickly follow rapidly changing mortality risk [10].

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