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Daniel Nettle

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Research

Flexibility in reproductive timing in human females: integrating ultimate and proximate explanations

Daniel Nettle*

Centre for Behaviour and Evolution, Institute of Neuroscience, Newcastle University, UK

From an ultimate perspective, the age of onset of female reproduction should be sensitive to variation in mortality rates, and variation in the productivity of non-reproductive activities. In accordance with this prediction, most of the cross-national variation in women's age at first birth can be explained by differences in female life expectancies and incomes. The within-country variation in England shows a similar pattern: women have children younger in neighbourhoods where the expectation of healthy life is shorter and incomes are lower. I consider the proximate mechanisms likely to be involved in producing locally appropriate reproductive decisions. There is evidence suggesting that developmental induction, social learning and contextual evocation may all play a role.

Keywords: human behavioural ecology; life history; reproductive strategies; developmental plasticity; developmental programming

1. INTRODUCTION

Like many other animals, human beings exhibit considerable within-species variation in behaviour. One parameter which demonstrates this very clearly is the age of onset of childbearing in women. The average age at first birth (AFB) varies from under 18 to over 25 across a set of 17 small-scale societies [1]. When all of the world's contemporary nations are considered, the range of variation is even wider, with more than 10 years' difference between the youngest- and oldest-AFB countries [2].

Variation in AFB is an excellent case study for investigating human behavioural flexibility, for a number of reasons. First, there is well-developed theory, and comparative evidence from other species, concerning which factors are likely to affect reproductive timing, and these can be brought to bear on the human case. Second, evolutionary biologists [2] and social scientists working with no direct reference to evolutionary theory [3] have converged on rather similar ideas in this domain. Thus, this is an area of research that can be used to banish the twin misconceptions that evolutionary explanations are necessarily at odds with those of the social sciences, and that to take an evolutionary approach entails de-emphasizing social context (see [4] for discussion of these misconceptions). Third, variation in reproductive timing has received attention both from anthropologists concerned primarily with ultimate questions (e.g. what are the fitness consequences of delaying childbearing?), and psychologists concerned with proximate mechanisms (e.g. how do women decide when to begin childbearing?). It has long been acknowledged within behavioural biology that ultimate and proximate explanations need to be integrated, but in many areas this integration remains an aspiration rather than being a reality (see [5] for the human case, and [6] more generally). Reproductive timing may be a domain where such integration can begin. Finally, there is well-characterized variation in AFB both at the between-population level, and at the within-population level. Studying within-population patterns helps overcome the limitations of comparing populations that may differ in multiple ways, and also in adjudicating between competing hypotheses about the mechanisms involved [7].

In this paper, then, I examine both ultimate (§2) and proximate (§3) causes of variation in AFB in humans, drawing in particular on my own research on British women. My aim is to provide an overview of the current evidence on this particular topic, but, more broadly, to demonstrate the power of taking an integrative evolutionary approach, encompassing both functional and mechanistic concerns, in explaining human behavioural flexibility.

2. FLEXIBILITY IN REPRODUCTIVE TIMING: ULTIMATE EXPLANATIONS

In general terms, high-mortality regimes favour relatively early reproduction, whereas low-mortality regimes favour delaying the onset longer [8,9]. As usual with an evolutionary problem, the reasons for this can be expressed in terms of costs and benefits. On the benefit side, females delaying reproductive onset may be able to produce higher quality offspring in the end, because of the extended period of pre-reproductive somatic investment and resource accumulation they can make. On the cost side, every time unit of delay increases the probability that the

*daniel.nettle@ncl.ac.uk

One contribution of 14 to a Theme Issue 'Evolution and human behavioural diversity'.

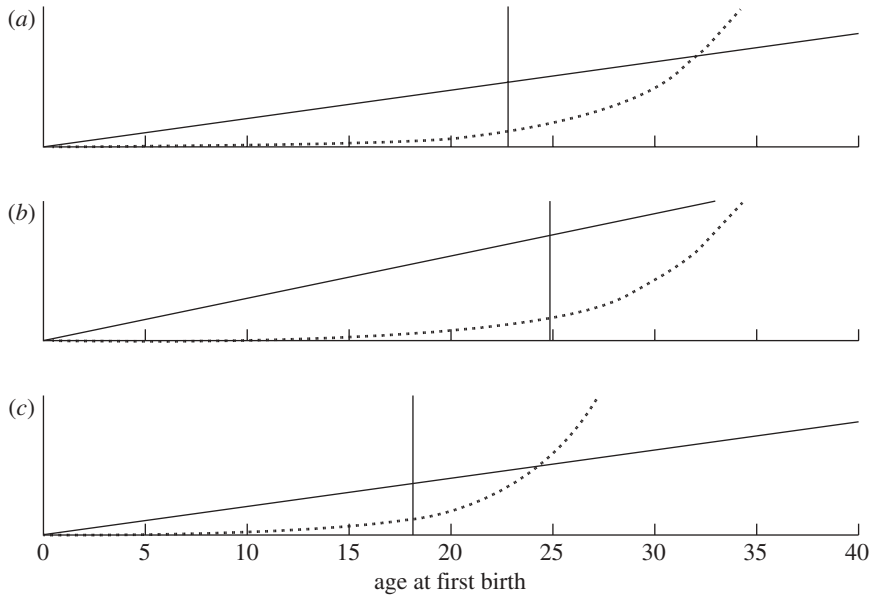

Figure 1. Illustrative model of the predicted response of AFB to changes in the costs and benefits of delaying childbearing. Assuming that benefits (solid line) accrue linearly with every year's delay, and that costs (dotted line) increase exponentially as years of possible reproduction run out, then selection favours an age of onset which balances costs and benefits (the vertical line). Increasing the benefit of each year's delay, for example through labour force participation becoming more productive, moves the optimum to a later age (*b* versus *a*). Increasing the costs of delay, for example through an increase in mortality rate, moves the optimal age earlier (*c* versus *a*).

individual will die or become incapacitated before she is able to complete her reproductive career. Selection favours a point where the trade-off between these costs and benefits is optimized (figure 1). Any increase in mortality rates will move the optimum point younger, while any increase in the benefits of delay (for example, a greater improvement in eventual offspring quality for every unit of pre-reproductive delay) will move it to an older age. The mortality-rate prediction is supported by evidence from experimental evolution [10,11], and also by comparative data: across mammal species, there is an extremely strong relationship between mortality rates and AFB [12,13].

Although within-species variation need not be conditioned by the same factors as between-species variation, in this instance this does seem to be the case, because a very similar pattern can be detected across human populations. Using data for over 100 countries, Low *et al.* [2] showed that female life expectancy (LE) is strongly but nonlinearly associated with mean AFB. This study only measures variation in the cost of delaying reproduction (i.e. the risk of failing to complete reproduction by waiting too long). Behaviour should also be sensitive to variation in the benefits of delaying. In humans, a large part of the benefit of delaying will be in the form of the extra-somatic resources (possessions, housing, land, money and so on) a woman can accumulate in the pre-reproductive period. However, the return on this kind of activity will vary according to local conditions; where women's economic activities have a high return, the benefits of delaying childbearing will be greater than where the return is low. We should thus predict that AFB will respond to women's economic opportunities, as well as to mortality schedules.

To test this, I extended Low *et al.*'s [2] analysis by collating mean AFB (source [14]), and female LE at birth [15] for all available countries (116 countries, UK excluded, data from 2001, dataset available on request). In addition, I included the mean female income (in 2001 purchasing-power equivalent US$, logged for skewness, from [15]), as a proxy for the return on women's economic activities. As figure 2 shows, there are strong associations between AFB and LE, and AFB and ln(income). The partial correlation of AFB and ln(income) controlling for LE is significant ($r = 0.52$, $p < 0.01$), as is the partial correlation of AFB and LE controlling for ln(income) ($r = 0.35$, $p < 0.01$). The best-fitting regression model contains both independent variables (AFB $= 0.08$ LE $+ 1.41$ ln(income) $+ 6.60$; adjusted $r^2 = 0.74$, p for both variables and overall <0.001). This suggests that both mortality rates and economic opportunities make independent contributions to explaining typical AFB. Together, they account for 74 per cent of the variation. Thus, a very simple model of the costs and benefits of delaying reproduction predicts accurate behaviour surprisingly accurately at the national level.

We can apply exactly the same reasoning to the explanation of within-population variation as between-population variation. There is considerable social divergence in reproductive timing within affluent Western populations, leading to the emergence of 'teenage pregnancy' as a recognized social issue in some countries [16,17]. Teenage pregnancy is concentrated in the poorest social strata [18], and is basically a by-product of the fact that in these groups the whole age distribution of childbearing is shifted younger, pushing the left tail into the teenage years. Geronimus *et al.* [3] showed that the risks of mortality and morbidity in the poorest urban US communities are

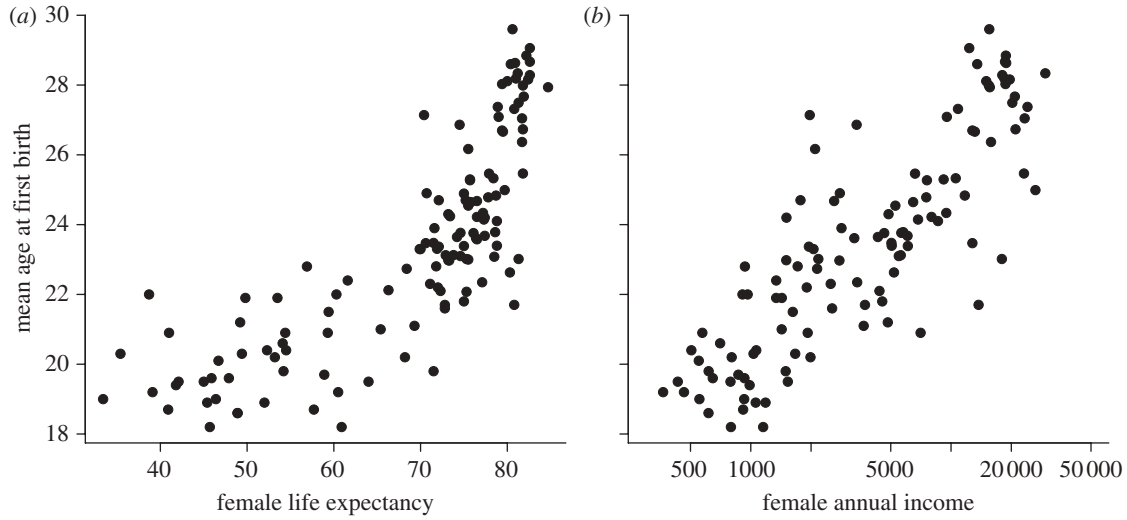

Figure 2. (*a*) Relationship between female LE and average AFB first across 116 countries; (*b*) Relationship between average female income (2001 US$ PPP) and average AFB across 116 countries. After Low *et al.* [2]. Sources as described in text.

sufficiently elevated that delaying childbearing to the US normative age would entail significant reductions in average reproductive success. Note that this is a convergent explanation to that given by Low *et al.* [2] for the cross-country pattern. Once again, Geronimus *et al.* [3] focus only on the costs of delay, whereas the benefits should also be relevant.

I investigated the within-country variation in England using the Office of National Statistics' division of English neighbourhoods into deciles of socioeconomic deprivation (1 = most deprived, 10 = most affluent). Socioeconomic deprivation is assessed using multiple indices deriving from the UK Census and other sources, based on income, housing, education, access to services and the material environment (see [19]). Female LE (for 1994–1999) for each of the deciles of neighbourhoods has been calculated from national statistics by Bajekal [20]. Mean AFB comes from the Millennium Cohort Study [21], a longitudinal survey of a large, representative sample of British families who had a child in 2000–2001. The Millennium Cohort Study data record which decile of deprivation the family's neighbourhood of residence falls in. (Note that the Millennium Cohort Study uses a smaller scale resolution of neighbourhoods, and a slightly different set of indices of deprivation, from those used in Bajekal's work. The effects of this discrepancy are likely to be slight; see [19] for further details.) I calculated mean AFB for each decile by taking the age of the mother at the child's birth in Millennium Cohort families living in England where there are no older siblings reported ($n = 4816$). Female income comes from a later Millennium Cohort Study survey (2006), and was calculated by taking the estimated marginal means of female gross weekly pay (for those women who are working), for each decile of neighbourhoods, controlling for the woman's age and the number of hours worked per week ($n = 4142$).

As table 1 and figure 3 show, across the deciles of increasing socioeconomic position, LEs become longer, women's incomes become higher, and AFB gets correspondingly later. The variables are so closely associated (all $r_s > 0.95$) that it is impossible to attempt regression, but the qualitative pattern is the same as that of the cross-national data. However, the within-country variation appears much larger than the between-country pattern predicts. I used the cross-country regression equation to predict AFB for each of the deciles of English ward (after roughly annualizing and dollarizing the income variable by multiplying by 50 and 1.5), and the predicted divergence in AFB between the most deprived and most affluent neighbourhoods is only of the order of 1 year, whereas the observed difference is almost 7 years (table 1).

Table 1. Mean female weekly gross income (UK£), LE at birth, healthy LE at birth and AFB, for English neighbourhoods divided into deciles on the basis of the index of multiple deprivation (1 = most deprived, 10 = most affluent). For sources, see text. Healthy LE is the number of years of good health a person could expect if rates of mortality and morbidity remain unchanged.

decile	income	LE	healthy LE	mean AFB
1	217	78.0	51.7	22.7
2	235	78.9	56.0	24.6
3	233	79.1	58.0	25.2
4	254	79.7	58.7	27.3
5	250	80.1	59.9	27.4
6	285	80.5	62.3	27.9
7	280	80.7	64.7	28.8
8	304	81.0	65.7	29.3
9	313	81.1	66.9	29.2
10	353	81.2	68.5	30.0

Why should the within-society socioeconomic variation be so much greater than the between-country pattern leads us to expect? One possibility is that there are effects of inequality above and beyond those of absolute conditions [22]. That is, it may have a greater effect on behaviour to have an income of $10 000 in a population where the mean income is $20 000 than in one where $10 000 is the mean [23]. Another possibility is that LE is a poor proxy

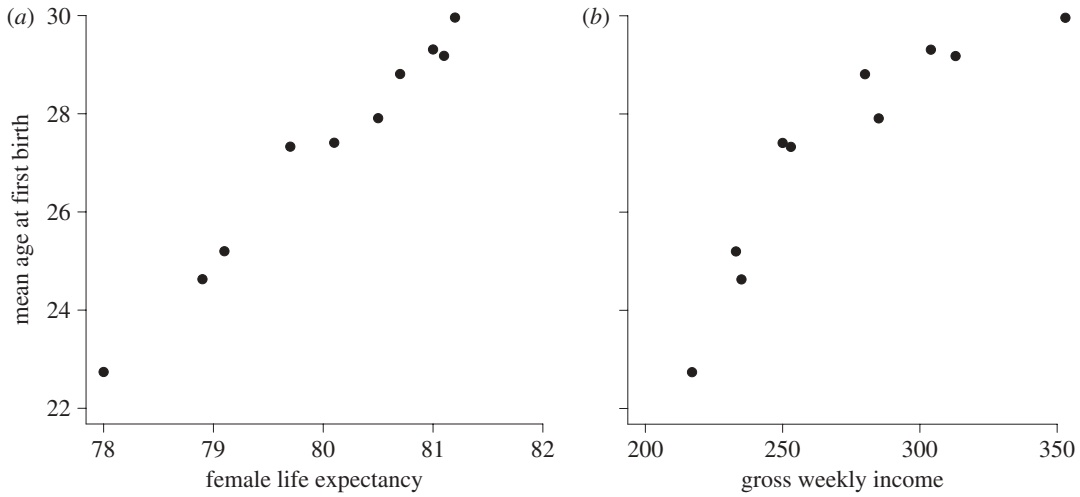

Figure 3. (*a*) Mean AFB against female LE and (*b*) mean gross weekly income (UK£) for contemporary England. Data points represent groups of neighbourhoods classified on the basis of socioeconomic deprivation. For sources see text.

for variation in health prospects within developed countries, where the biggest discrepancies between the rich and the poor are actually in the burden of extra morbidity rather than extra mortality across the life course [24]. The expectation of healthy life—which is the number of years of good health a person can expect—shows a much sharper socioeconomic gradient than does total LE. For example, the difference between the least and most deprived deciles of neighbourhood in female healthy LE is 16.8 years, when compared with 3.2 for total LE (table 1, penultimate column). Chronic ill health has a negative effect on a person's ability to conceive, bear infants to term, and care for offspring, and so it makes sense that increased morbidity would have a similar effect on reproductive decisions as increased mortality does [25]. Thus, it may be that incorporating morbidity as well as mortality would more accurately predict the socioeconomic differences in AFB in the UK data.

A simple analysis suggests that this may be the case. I used the expectation of healthy life for each decile of neighbourhood to calculate the age at first childbearing that a woman would need to adopt to satisfy the rule 'begin childbearing at such an age that you can on average expect to be in good health until your oldest grandchild is five, given where you live' (and assuming that your child will adopt the same AFB as you). The predicted AFB given by this rule is remarkably close to the actual behaviour (figure 4). There is no *a priori* justification for the choice of this particular rule as the maximand, but it does not seem an unreasonable one, and it serves to make the point that women's behaviour seems to be responding systematically to the local expectation of healthy life.

This section has shown that consideration of the costs and benefits of delaying childbearing can predict the pattern of observed variation in women's AFB quite well, both between and within societies. Thus, women are clearly responding to ecological context. However, this observation alone cannot tell us *how* they internalize information from the environment and use it to alter their life histories. To address that question, we must turn from issues of ultimate causation to those of proximate mechanism.

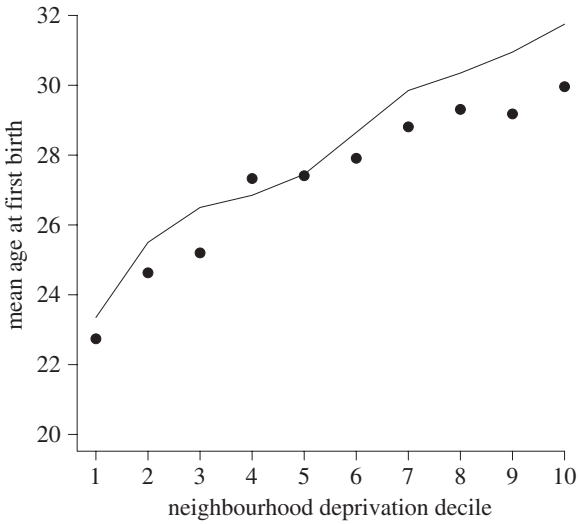

Figure 4. AFB observed (points) and predicted by the simple rule 'begin childbearing at such an age that you can on average expect to be in good health until your oldest grandchild is five' (line), for English neighbourhoods divided into deciles according to the index of multiple deprivation. For sources, see text.

3. FLEXIBILITY IN REPRODUCTIVE TIMING: PROXIMATE EXPLANATIONS

We have seen that women are highly responsive to the affordances and hazards of their local environment. We can also be sure that they don't generally have access to complete actuarial information in order to make their decisions. What, then, are the processes which link ecology to behaviour? There are several classes of mechanism which could in principle be involved (see [7]). The most obvious of these are genetic polymorphisms, developmental induction, social learning, contextual evocation and what I shall call higher order cognitive processes. I now briefly examine each of these in turn.

(a) *Genetic polymorphisms*

There are well-established genetic effects on timing of puberty [26], and thus it is plausible that there might

be heritable influences on AFB. There may have been some population-specific genetic evolution favouring early AFB in humans, for example in the case of pygmy populations, which are convergently genetically adapted to high-mortality ecological regimes that favour short growth and early maturation [27,28]. However, it seems unlikely that genetic factors could explain differences in AFB more generally. There is abundant gene flow within and between societies, which constantly works against local adaptation. AFB differences within the UK track neighbourhood characteristics extremely closely (see §2), and the population is not genetically structured by neighbourhood to anything like the degree that would be required for this pattern to be explained by genetic differences. Moreover, AFB responds far too quickly to shifts in the ecology as countries develop for it to be driven mainly by genetic change. However, gene × environment interactions, whereby people with a certain genotype respond more strongly to environmental inputs than others, may well be important and account for some of the variation within social groups experiencing the same broad environment [29].

(b) *Developmental induction*

Developmental induction (often known as developmental programming in the biomedical literature) describes mechanisms where specific early-life environmental inputs cause the organism to develop an alternate adult phenotype. The relevant inputs can operate post-birth, as in the triggering of the gregarious form of the desert locust by early-life cues of crowding [30], or pre-birth, as in the metabolic and hormonal changes in rat offspring whose mothers are calorically restricted during pregnancy [31]. Belsky *et al*. [32] suggested a special role for early-life conditions in calibrating female life-history strategy in humans, by hypothesizing the existence of a developmental induction mechanism of the form 'if you receive low investment in the first few years of life, your prospects are poor, so mature fast and reproduce young'. There have been a large number of empirical tests of this hypothesis and related variants. The measures of low early-life investment have included low birthweight, lack of paternal involvement and lack of closeness to parents. The most usual measure of maturational tempo has been age at menarche, though some studies have focused on other variables such as age at first intercourse, interest in infants during adolescence or teenage pregnancy. Regardless of which early-life measures and which outcomes are investigated, studies have tended to find effects consistent with the predictions of the hypothesis (e.g. [33–45]).

An obvious limitation of these findings is that they are based on correlational data. Thus, it is difficult to show conclusively that the developmental events cause the maturational acceleration, rather than both being the result of some third factor. This third factor could be shared environment (certain social conditions, for example, causing father to invest less *and* daughters to mature faster). It could also be genotype, if, for example, the same genetic variants expressed in fathers caused them not to invest and expressed in daughters caused them to mature early [46,47]. The best studies address these problems in one of two ways. Some employ genetically and environmentally controlled designs, such as comparing siblings growing up together but who differ in the relevant exposure [45], and still find positive results. Others exploit experiments of nature. Pesonen *et al*. [42], for example, compared the reproductive behaviour of people from Helsinki who had been evacuated away from their birth families during the second world war, with those who had remained. As evacuation was decided largely randomly with respect to family characteristics, this is a quasi-experimental design. Former evacuee women had early menarche and more children in total than the controls (although their AFB was not significantly earlier).

There are also a number of other reasons why the developmental induction mechanism for calibrating maturational tempo is plausible. First, receiving low early-life investment demonstrably affects LE (for example, for the case of birthweight, see [48]), and therefore, it *ought* to affect the individual's optimal AFB. Second, developmental induction is most likely to be favoured by selection where the organism needs to specialize early in life in order to develop the contextually appropriate phenotype. This is true for reproductive development. To bear children early, a woman needs to cease stature growth and reach menarche early [49]. To do this, a whole suite of hormonal and growth changes are required several years earlier [41,50,51]. Thus, phenotypic specialization needs to be underway by middle childhood if very early childbearing is going to be possible. Thus, the idea that early-life factors (within the first 5 years) might have evolved as calibrational cues is cogent. Finally, there is evidence for similar mechanisms in other female mammals. Female rat pups that receive low amounts of maternal licking and grooming reach puberty earlier, and are more likely to conceive with the first male they encounter, than those receiving high levels of maternal licking and grooming [52,53]. In these animal models, genuinely experimental manipulations can be employed to show unequivocally that the effects are causal.

However, although the case that early-life conditions accelerate adolescent maturational timing seems compelling, it is not necessarily true that adolescent maturational timing will map onto timing of first birth in a population where childbearing generally begins a considerable time after maturation. We recently examined this issue in two ways, using data from the National Child Development Study. First, we found that intended age for reproduction, stated at age 16, was in fact a good predictor of realized AFB, even though childbearing was often a decade or more later [54]. Thus, reproductive behaviour does seem to get relatively set by late adolescence. Second, we took the more direct approach of examining whether early-life conditions predict age at first pregnancy directly. We found that not being breastfed, separation from mother in childhood, residential disruption and lack of paternal involvement all had independent, and additive, accelerating effects on age

at first conception two decades or so later, even controlling for family socioeconomic position and for the cohort member's mother's age at her birth [49].

Developmental induction mechanisms could be important in linking behaviour to ecological conditions, since harsh environments induce lower parental investment per offspring [55]. This may then predispose the offspring to earlier AFB via developmental induction. For the within-country case, this means that we should predict a significant mediation of the relationship between low socioeconomic position and early childbearing by early-life parental investment received. Although we did find such a mediation effect in the National Child Development Study data [49], it was small, with most of the effect of socioeconomic conditions operating in ways not captured by the mediator. Thus, though developmental induction by parental investment received may be important, it is certainly not the only mechanism at work.

(c) *Social learning*

Two kinds of social learning have been discussed which are relevant to variation in AFB. The first is the observation of what is happening to others in the environment as they go about their lives. Qualitative research by Geronimus [56] found that US teen mothers were aware of how the health of the women around them had weathered over time, and could relate this to their own reproductive goals. Wilson & Daly [57] showed that LE in Chicago neighbourhoods strongly predicted onset of reproduction, and suggested that seeing others in the social environment die activates domain-specific psychological mechanisms producing reproductive motivation. Since this is a form of contextual evocation, I return to it below.

A second kind of social learning would be copying the reproductive behaviour of others. If such emulation were biased (for example, copy those who achieve the highest reproductive success), then it could lead to locally adaptive behaviour much of the time [58]. A particular form of this social learning has been discussed in relation to AFB, namely daughters copying from their mothers (vertical social transmission). The idea is potentially cogent, as theory shows that vertical social transmission can be favoured for behaviours which affect fertility, and for which the family environment is rather stable across generations [59]. There is a correlation between mother and daughter AFB in most Western samples [60], and in the National Child Development Study data we find an association between AFB and mother's age at cohort member's birth which is not reducible to continuities in socioeconomic position [49]. Thus, women may be copying the reproductive timing of their mothers to some extent.

(d) *Contextual evocation*

Contextual evocation (also sometimes called evoked culture; [61]) refers to situations where evolved, domain-specific psychological mechanisms respond to a particular class of environmental input by producing an appropriate motivational response. As mentioned above, one possibility for setting AFB is that observation of mortality in the surrounding environment cues evolved psychological mechanisms that activate reproductive motivation [57]. Chronic activation of these mechanisms would lead to the different reproductive schedules of populations in different environments. In support of this hypothesis, a number of psychological studies have found that merely making people think about death for a few minutes increases their stated desire to have children [62,63], or makes them more interested in infants [64]. Anthropologists may be sceptical about the link between these fleeting, hypothetical preferences and the actual behaviour of populations, but there is evidence of localized spikes in birth rates following unusual localized spikes in death rates [65,66]. Thus, death-related evoked motivation is a plausible mechanism to explain the general demographic finding that declines in death rates are followed by declines in birth rates.

It may not be just exposure to death which evokes early-fertility preferences. Davis & Werre [67] show in a large US sample that experience of agonistic interactions (being a victim of crime, being threatened, having fights, being offered drugs) at age 14 or 15 predicted subsequent early fertility and having a child out of wedlock, even controlling for a large number of contextual and individual factors. Thus, it could be that any environmental cue that suggests menace has a similar effect.

(e) *Higher order cognitive processes*

Psychologists often distinguish between relatively simple, automatic evolved heuristics on the one hand, and more cognitively elaborated, effortful, open-ended problem-solving processes on the other hand [68]. Whether this represents a true dichotomy is arguable; one could conceive of a graded scale of different cognitive processes each with more degrees of elaboration and complexity than the last. However, the distinction may be a useful idealization. The contextual evocation and social learning effects described above need only involve simple heuristics. Thus, it is an open question how elaborated the cognition which goes into the setting of reproductive goals is, and to what extent women can articulate the reasons for their preferences.

Many discussions of social variation in AFB within developed countries attribute little role to higher order cognitive processes like plans and intentions. Most of the biomedical literature on teenage pregnancy in the UK, for example, asserts that it is basically a mistake that arises from lack of skills in contraception [69]. However, it is not clear that this assertion is justified [70]. Qualitative researchers are generally struck with the sophistication with which young women can reason about their life situations and the impact of these on their reproductive decisions [17,56,71]. Young women appreciate that earlier fertility will reduce their chances to invest in their own pre-reproductive development and resources, but can also articulate that the cost of delay will be that they are not in a good position to complete their parental

and grandparental investment while still young and healthy [16,56]. British women who choose early motherhood tend to cite unhappiness in their childhoods (which could reflect threatening or harsh environments), and poor prospects for the future, as factors conditioning their decisions [72]. These relate rather neatly to the costs and benefits of delay in the simple theoretical model shown in figure 1. Thus, we should not underestimate the amount of insight women have into why certain behaviours might be adaptive in certain situations.

4. CONCLUSION: TOWARDS AN INTEGRATION OF FUNCTION AND MECHANISM

Section 2 showed that we can explain variation in AFB in terms of a response to the costs and benefits of delaying reproduction given local conditions. Section 3 showed that we can identify some of the psychological and developmental mechanisms which may be involved in mediating this response. The typology of mechanisms I have presented is somewhat artificial. For example, I have categorized the effects of parental behaviour before age 7 as developmental induction, but those of agonistic peer behaviour at age 14 as contextual evocation. In truth, there is much still unknown about the ontogenetic time-course, reversibility, domain-specificity and mutual interaction of the many types of inputs people receive from their local environments over the course of their prereproductive lives. Nonetheless, I hope to have shown that, in the case of flexibility in human reproductive timing, we can ask both ecological questions about ultimate causes, and psychological questions about mechanisms, and begin to unify the answers. If we can achieve this kind of integration in other domains too, we will begin to realize the potential of the broad evolutionary approach outlined by Tinbergen [73] for addressing problems in the human behavioural sciences.

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