



HAL
open science

Childhood environmental harshness predicts coordinated health and reproductive strategies: A cross-sectional study of a nationally representative sample from France

Hugo Mell, Lou Safra, Yann Algan, Nicolas Baumard, Coralie Chevallier

► To cite this version:

Hugo Mell, Lou Safra, Yann Algan, Nicolas Baumard, Coralie Chevallier. Childhood environmental harshness predicts coordinated health and reproductive strategies: A cross-sectional study of a nationally representative sample from France. *Evolution and Human Behavior*, 2018, 39 (1), pp.1-8. 10.1016/j.evolhumbehav.2017.08.006 . ijn_03065806

HAL Id: ijn_03065806

https://hal.science/ijn_03065806

Submitted on 16 Dec 2022

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

Contents lists available at [ScienceDirect](http://ScienceDirect.com)

Evolution and Human Behavior

journal homepage: www.ehbonline.org

Childhood environmental harshness predicts coordinated health and reproductive strategies: A cross-sectional study of a nationally representative sample from France

Hugo Mell ^{a,c,*}, Lou Safra ^a, Yann Algan ^b, Nicolas Baumard ^c, Coralie Chevallier ^{a,c}^a Laboratoire de Neurosciences Cognitives (LNC), Département d'Etudes Cognitives, INSERM U960, Ecole Normale Supérieure, PSL Research University, F-75005 Paris, France^b Sciences Po, OFCE, 27 Rue Saint-Guillaume, 75007 Paris, France^c Institut Jean Nicod, Département d'Etudes Cognitives, CNRS UMR8129, Ecole Normale Supérieure, PSL Research University, F-75005 Paris, France.

ARTICLE INFO

Article history:

Initial receipt 25 January 2017

28 August 2017

Final revision received 28 August 2017

Available online xxxxx

Keywords:

Psychosocial acceleration theory

Childhood adversity

Life History Theory

Reproductive strategies

Health strategies

Structural equation modeling

ABSTRACT

There is considerable variation in health and reproductive behaviours within and across human populations. Drawing on principles from Life History Theory, psychosocial acceleration theory predicts that individuals developing in harsh environments decrease their level of somatic investment and accelerate their reproductive schedule. Although there is consistent empirical support for this general prediction, most studies have focused on a few isolated life history traits and few have investigated the way in which individuals apply life strategies across reproductive and somatic domains to produce coordinated behavioural responses to their environment. In our study, we thus investigate the impact of childhood environmental harshness on both reproductive strategies and somatic investment by applying structural equation modeling (SEM) to cross-sectional survey data obtained in a representative sample of the French population ($n = 1015$, age: 19–87 years old, both genders). This data allowed us to demonstrate that (i) inter-individual variation in somatic investment (e.g. effort in looking after health) and reproductive timing (e.g. age at first birth) can be captured by a latent fast-slow continuum, and (ii) faster strategies along this continuum are predicted by higher childhood harshness. Overall, our results support the existence of a fast-slow continuum and highlight the relevance of the life history approach for understanding variations in reproductive and health related behaviours.

© 2017 Elsevier Inc. All rights reserved.

1. Introduction

People engage in numerous behaviours that bear a high cost to the individual and to society: smoking, poor engagement in health-promoting behaviours, overconsumption of high calory foods, teen-pregnancy, etc. In OECD countries, for instance, “lifestyle” conditions linked to tobacco use, excessive diets and physical inactivity are now responsible for most years of lost life (Hurst & Sassi, 2008). Adolescent childbearing has also been a major concern for policy makers because of the potential health costs and loss of education and labor opportunities for teenage mothers (Hoffman, Foster, & Furstenberg, 1993; Miller, 2000). Therefore, identifying the determinants of health and reproductive decisions is of vital importance. Here, we argue that behavioural diversity for health and reproductive decisions should not come as a surprise and should be construed as the predictable outcome of humans' evolutionary make-up.

Specifically, we investigate the idea that health and reproductive decisions are adjusted during development to the way individuals perceive the harshness of their environment. Harshness here refers to extrinsic morbidity-mortality, which encompasses all external sources of death and disability that are largely beyond the individual's control (Ellis, Figueredo, Brumbach, & Schlomer, 2009). Put simply, the hypothesis is that focusing on one's health or delaying reproduction to invest in other areas of life might be less beneficial in environments where mortality is high than in environments where mortality is low. The degree of environmental harshness experienced during childhood may therefore place individuals on a reproductive and health path that is calibrated to their ecology. While previous studies have already highlighted such effects of harshness on reproduction and health behaviours independently, we go further by integrating variation in both domains to take into account the coordinated nature of people's allocation strategies.

Life History Theory (Roff, 2002; Stearns, 1992) provides a general framework to investigate variation in allocation decisions. It states that the life history strategy of any organism is the product of the interaction between tradeoffs among traits and environmental factors that affect mortality and fertility rates (Stearns, 2000). Drawing on these fundamental insights, evolutionary psychologists started to investigate

* The raw data and R script used in this study are available through the Open Science Framework at: <https://osf.io/8k5y4/>.

* Corresponding author at: CNRS UMR 8129 – INSERM U960, Département d'Etudes Cognitives, Ecole Normale Supérieure, 29 rue d'Ulm, 75005 Paris, France.

E-mail address: hugo.mell@ens.fr (H. Mell).

how specific adversity events occurring during ontogeny, could be used by individuals as cues to adjust their strategies (Belsky, Steinberg, & Draper, 1991; Chisholm et al., 1993). This led to a fruitful body of research often designated by the name “psychosocial acceleration theory” (Nettle, Frankenhuys, & Rickard, 2012). Specifically, it predicts that individuals living in harsh environments should exhibit overall “fast” strategies, whereas individuals living in favorable environments are expected to show overall “slow” strategies (Ellis et al., 2009). The “fast” end of this fast-slow continuum is generally characterized by a shortened period of growth associated with an early onset of reproduction (early sexual maturation and first reproductive event), a higher number of offspring with a lower investment per offspring, lower body maintenance and a reduced lifespan; whereas the slow end of the continuum has the opposite characteristics (Ellis et al., 2009). According to this theory, having a faster strategy in harsh environments increases an individual's chances to reproduce before dying, whereas a slow strategy in favorable environments would allow for a longer growth period, which in turn, would lead to larger future reproductive benefits. In many species, the level of environmental harshness in which mature individuals will reproduce is uncertain. Adaptive mechanisms of phenotypic plasticity might therefore have evolved to orient individuals' life history strategy based on the level of harshness experienced during the juvenile period. There are two main reasons why life history strategies should be conditioned on early life harshness. First, if early harshness is reliably correlated with post-juvenile harshness, cues of harshness gathered during development should be used as a ‘weather forecast’ to trigger a faster strategy (this is the so-called ‘external-PAR’ hypothesis; see Rickard, Frankenhuys, & Nettle, 2014). Second, if stressful events in early life cause irreversible damage to an organism's soma, the individual should also pursue a faster life history strategy since the probability of early death or disability is increased (this is the so-called internal-PAR hypothesis; see Rickard et al., 2014). Both pathways are not mutually exclusive and they predict that childhood adversity events that might serve as cues of later harshness or that directly impair the individual's somatic state should lead to faster life history strategies. Various features of an individual's socioecology have been argued to provide potential cues of harshness to guide life history decisions (see Ellis et al., 2009 for an extensive discussion). Socioeconomic status (SES) is one important dimension since virtually all forms of morbidity and mortality decrease linearly with SES in western countries (Chen, Matthews, & Boyce, 2002). Personal knowledge of deaths and exposure to violence should also directly affect individuals' estimates of environmental harshness. In addition, level of parental investment might also convey useful information about harshness in the parents' environment.

Although the theoretical link between harshness and fast strategies is not straightforward (Baldini, 2015), empirical studies have repeatedly found associations between proxies of harshness and fast reproductive strategies. For instance, fertility at the country level is predicted by disease diversity, which is a strong indicator of extrinsic morbidity and mortality, even after controlling for various economic, cultural and sociodemographic factors (Guégan, Thomas, Hochberg, de Meeûs, & Renaud, 2001). At the individual level, high socioeconomic deprivation and low parental care during childhood are associated with earlier reproduction (Nettle, 2011), with an earlier onset of puberty (Belsky, Steinberg, Houts, Friedman, DeHart, Cauffman, Roisman, Halpern-Felsher, Susman, and NICHD Early Child Care Research Network, 2007; Belsky, Steinberg, Houts, Halpern-Felsher, and NICHD Early Child Care Research Network, 2010; Ellis, 2004; Ellis & Essex, 2007; Moffitt, Caspi, Belsky, & Silva, 1992; Tither & Ellis, 2008) and more sexual partners (Simpson, Giskevicius, Kuo, Sung, & Collins, 2012). Internal features such as lower birthweights, also predict early reproduction in a longitudinal study of the British population, even after controlling for other socioecological variables (Nettle, Coall, & Dickins, 2009). Hence, people coming from harsher backgrounds

develop overall faster reproductive strategies that manifest in a coherent manner for various sexual traits.

In parallel to these effects on reproduction, exposure to harsh environments also influences resource allocation to body maintenance (Cabeza de Baca & Ellis, 2017; Del Giudice, 2014a). There is indeed a well-documented social gradient in preventive health behaviours (Stringhini, 2010) and part of the disinvestment in health observed in people with lower SES could be due to initial disparities in life expectancies (Nettle, 2010). Indeed, subjective socioeconomic standing is associated with reported effort in looking after one's health in a cross-sectional sample of the American population, and the effect of subjective socioeconomic position is fully mediated by perceived extrinsic mortality risks (Pepper & Nettle, 2014). Inter-individual differences in risky behaviours such as risky sexual behaviours, alcohol or drug use, which are in part predicted by early exposure to harsh environments in longitudinal studies of adolescent behaviours (Belsky, Schlomer, & Ellis, 2012; Belsky, Steinberg, Houts, Halpern-Felsher, and NICHD Early Child Care Research Network, 2010; Brumbach, Figueredo, & Ellis, 2009; Hartman, Li, Nettle, and Belsky, in press; Simpson et al., 2012), could also be interpreted as a disinvestment in body maintenance in response to higher extrinsic mortality risks.

Overall, there is therefore consistent empirical support for the application of Life History Theory principles to the study of allocation strategies in human populations. Nevertheless, most studies have focused on the impact of harshness on a few life history traits restricted to one of the two allocation domains previously highlighted, i.e. reproductive or somatic efforts. Yet, based on psychosocial acceleration theory, we actually expect clusters of correlated traits across these domains, reflecting functional suites of multiple traits that aim toward short-term returns in harsh conditions and long-term returns in favorable environments (Belsky et al., 1991; Figueredo et al., 2006, 2005; Giskevicius, Delton, Robertson, & Tybur, 2011; Reale et al., 2010). Indeed, if the fast-slow continuum is a broad axis of variation relevant to human life history strategies, all else being equal, individuals who adopt fast reproductive strategies should also exhibit lesser investments in their embodied capital (Kaplan, Lancaster, & Robson, 2003). To our knowledge, only one study (Brumbach et al., 2009) explicitly assessed life history strategies with traits related to both reproductive and somatic investments in a single sample and showed that exposure to harsh events during adolescence predicted faster strategies across domains in young adulthood. In the current paper, we further test the existence of coordinated fast-slow strategies by analyzing data from a cross-sectional survey of a nationally representative sample of the French population specifically designed to test the existence of such a fast-slow continuum. We used structural equation modeling to test the prediction that part of the variation in reproductive and somatic effort is predicted by individual differences in exposure to harsh events during childhood. Specifically, we predicted that: 1) It is possible to identify a latent construct reflecting individuals' Life History Strategies which influences decisions pertaining to both reproductive and health choices; 2) Individuals' Life History Strategies fall along a fast-slow continuum. 3) Childhood environmental harshness has an influence on Life History Strategies, such that increased harshness leads to faster behaviours on the fast-slow continuum. In addition, we also tested whether specific harsh events were better predictors of individuals' future life history strategy.

Lastly, it should be stated that despite our nationally representative sample, the cross-sectional design of the study does not allow us to make causal inferences about the reported relationships and it constrained us to use retrospective measures of environmental harshness, which are known to be error prone (Hardt & Rutter, 2004). The use of retrospective measures also prevents us from disentangling the effects of more refined dimensions of harshness that are thought to affect life history strategies independently, such as the mean level of extrinsic morbidity-mortality (harshness per se) vs. the variation across space and time around that mean (Belsky et al., 2012; Ellis et al., 2009; Frankenhuys, Panchanathan, & Nettle, 2016).

2. Methods

2.1. Sample and procedure

Our sample consisted of 1015 French males ($N = 447$) and females ($N = 568$), aged 19 to 87 years old (mean: 52.5 ± 14.3 sd) and recruited online by the French polling institute Ipsos. Initially, 11,000 people received an electronic invitation from the institute to take part in our online study. Subjects willing to participate first had to answer a demographic survey which collected information about their gender, age, location, household composition, marital status, socio-professional category, occupational status, annual income and educational status. The quota sampling method was applied to select a fraction of the individuals based on these demographic variables, in order to obtain a representative sample of the French population. The retained subjects were then asked to answer a second survey grouping all the items pertaining to life history traits and environmental harshness during childhood. Two of our key reproductive variables, namely *age at first birth* and *number of children*, were relevant only for people who had already reproduced. Therefore, among the 1691 participants who completed the questionnaire, we restricted our final study sample to individuals who already had children at the time of the study ($N = 1063$). We also calculated each participant's number of absurd answers (e.g. number of years spent smoking greater than the participant's age) and non-response. Participants with a total number at least three standard deviations above the sample mean were excluded ($N = 48$). Our final sample size was 1015 participants.

2.2. Variables of interest

Participants were asked to answer questions pertaining to their childhood environment and their adult reproductive and health strategy. We now present a summary of the various areas covered by our questionnaire (full questionnaire available in Appendix A).

2.2.1. Environmental harshness

The level of environmental harshness experienced during childhood was assessed with a survey consisting of 24 items, reflecting various aspects of childhood environment that previous studies had found to be associated with one or several life history traits in adulthood (Griskevicius et al., 2011; McCullough, Pedersen, Schroder, Tabak, & Carver, 2012; Nettle & Cockerill, 2010; Simpson et al., 2012). The first seven items captured general features of the family unit during participants' childhood. Sample items include "Have you ever lived with a stepfather?" and "Were you ever placed in an institution or in a foster family?". A three-item "parental investment" scale was used to assess participants' perception of the parental care they received during childhood, with items such as "My parents always seemed to care about what I was doing.". A "parenting style" scale of three items captured the harshness of parental education, with items such as "Some of the punishments I received when I was a child now seem too harsh to me.". Participants were also asked if they had been the victim of psychological, sexual or physical abuse during childhood and whether these episodes were caused by people in or outside their families. A set of seven questions concerned the exposure to other particular familial difficulties (e.g. "Did you live with one or several people who had spent time in prison?") and were regrouped into a single index. Participants were also asked whether they had suffered a long illness requiring a hospitalization before the age of seven and a "neighborhood stability" scale collected information about the stability of their growing-up environment with two items ("How many times did you move?" and "How many times did you change school?"). Lastly, participants' childhood socioeconomic status (SES) was measured by a scale developed by Griskevicius and colleagues (Griskevicius et al., 2013) from the following three items: "My family usually had enough money for things when I was growing up.", "I grew up in a relatively wealthy

neighborhood." and "I felt relatively wealthy compared to the other kids in my school.". Cronbach's alphas were superior to 0.8 for all the scales used in the analysis, suggesting good internal consistencies.

2.2.2. Reproductive strategy

Participants' reproductive strategy was assessed using four items from the literature (Nettle et al., 2009; Simpson et al., 2012): number of children ("How many children have you had?"); age at first birth (calculated from the age of the participant and the reported birth date of their first child); age at first (consented) sexual intercourse; and number of short-term sexual partners. For the last two items, participants could choose not to answer the question by selecting an "I don't want to answer" response.

2.2.3. Somatic strategy

Participants' somatic strategy was also assessed using four items previously used in the literature (Pepper & Nettle, 2014): body mass index (BMI), calculated based on reported height and weight following the standard formula used in the biomedical field; general health status ("How is your health in general?"); health effort ("How much effort do you make to look after your health and ensure your safety these days?"); and level of cigarette's consumption ("In total, during how many years did you smoke daily or almost daily?"). The responses for this last item were divided by the participant's age to allow for more a meaningful comparison between young adults and older participants.

2.3. Analysis

All statistical analyses were carried out in R 3.0.3 (<https://www.r-project.org/>). Since this study aimed to investigate a specific theoretical model that involved a latent construct, namely "life history strategies", structural equation modeling (SEM) was used as our main multivariate analysis method. Although our variables of interest showed overall low percentages of missing responses (ranging from 0 to 6.5%), multiple imputation techniques were used to preserve sample size and avoid biased estimations of model parameters. Twenty complete datasets were generated by fully conditional specifications for categorical and continuous data using the *r* package *mice* (Buuren & Groothuis-Oudshoorn, 2011). This package allows the use of different imputation methods depending on the type of variable with missing entries. Predictive mean matching was used for numeric variables, logistic regression imputation for binary data and proportional odds model for ordered categorical variables with more than two levels.

In order to assess the effect of harshness during childhood on life history strategies later in life, a synthetic harshness measure had to be constructed based on the associated survey items. Drawing on the methodology used by Brumbach et al. (2009), environmental harshness was modelled as an emergent variable rather than a reflective latent variable. Indeed, environmental harshness is arguably better conceived as an emergent variable since harsh events during childhood can be thought of as risk factors (like particular genetic variants, smoking and poor diet for cardiovascular diseases) that are not necessarily correlated with one another, but that all contribute to the cumulative probability of developing a particular outcome; in our case a faster or slower strategy. For example, having been exposed to the death of a sibling, hospitalized for a long illness or lived with a stepfather are three events that we can theoretically expect to increase the probability of developing a faster strategy, but that might often occur independently. Furthermore, we do not expect that all harsh events will have effects of the same magnitude on the cumulative risk of developing a particular life history strategy. Instead, some events might be better accounted for in a general harshness score when they are attributed heavier weights. One simple method to model this type of emergent variables used by Brumbach et al. (2009) is to sum individual *z*-scores for each harshness item. The use of *z*-transformed scores confers more weight to the most highly dispersed items and therefore reflects the implicit assumption that rare

harsh events should be better predictors of fast strategies. Thus, the experience of rare events like losing one's mother or having been the victim of physical abuse will contribute more to an individual's harshness score than more frequent events, such as having changed school a couple of times.

Nonetheless, such an assumption might not hold in all cases and it would be valuable to compute weights of the harshness items based on their predictive power rather than implicitly through the degree of dispersion of their distributions. Such an approach can be implemented in SEMs using unknown weight composites, which capture the collective effects of a set of causes on a response variable (Grace & Bollen, 2008). In this case, the composite score is computed via a set of weights that maximize variance explanation in the dependent variable and hence allows to compare the relative contribution of the hypothesized causes to the overall predictive power of the composite. Thus, after

fitting a SEM following the methodology previously used by Brumbach and colleagues (Brumbach et al., 2009; Fig. 1A), harshness was also modelled as an unknown weights composite in a subsequent SEM to gain these inferential benefits (Fig. 1B).

Whether harshness was computed as a sum of z-scores or as a composite, it was used as a predictor of the latent variable capturing individuals' general life history strategies. This latent construct was modelled as a unique factor capturing the covariation between all life history indicators (i.e. reproductive and somatic items). Yet, one might expect that items within each domain will show some additional degree of correlation that will not be captured by the single general factor. For example, subjects suffering from hereditary diseases would probably tend to declare a poor health state and higher efforts in looking after their health even though it might not be linked to a faster or slower reproductive strategy. To deal with this issue we elaborated on the single factor

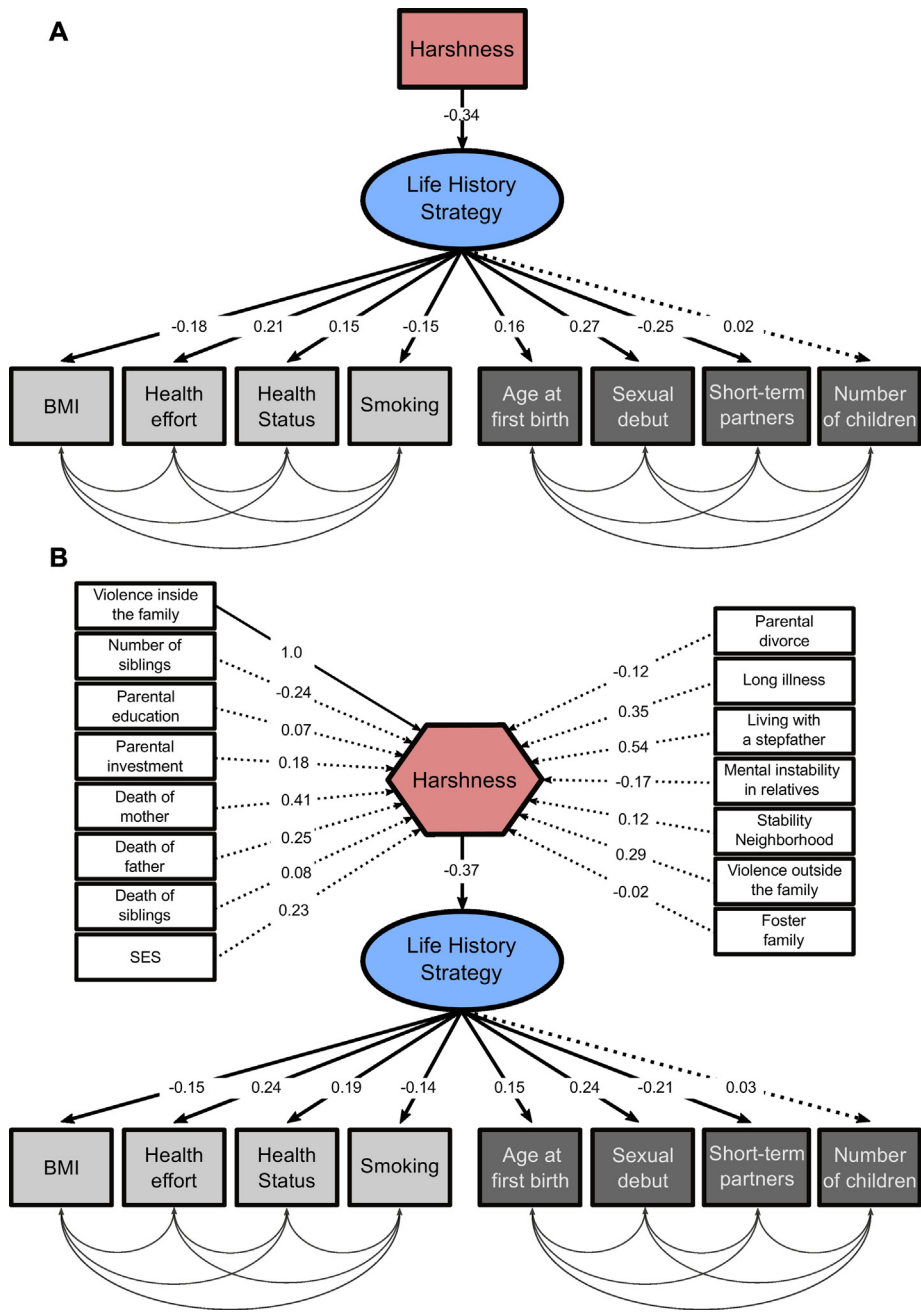


Fig. 1. Path diagrams of the structural models fitted showing for clarity only the structure of the residual covariances in the measurement model and standardized regression weights for harshness and reflective indicators. Significant paths at the 5% level are represented with a plain arrow A. Model 1, harshness is modelled as a weighted sum B. Model 2, harshness is modelled as a latent composite.

model by allowing for correlations between residual errors of items within the same domain (i.e. only between somatic and reproductive items respectively). This way the model's implied covariance matrix captures the correlations between items that are not explained by the latent life history factor but that can still be theoretically expected due to various unmeasured causes.

The latent variable reflecting individuals' life history strategy was scaled by fixing its variance to 1 in both SEMs. Composite variables also need to be scaled for identification purposes by fixing the coefficient of one of the causal indicator. Therefore, in the second SEM, harshness was scaled by setting the path from *violence in the family* to 1. The latter item measured whether participants had been victim as a child of physical, sexual or psychological abuse caused by people in their family. Its significance was assessed through the partially reduced form of the model, which directly estimates the pathways from the harshness items to the latent variable without the use of a composite (Grace, Anderson, Olf, & Scheiner, 2010). Finally, since our study sample covers a wide age range (19–87 years old), age was used as an auxiliary variable to control for its effects on life history indicators. SEM models were fitted using the R packages *lavaan* (Rosseel, 2012) and the function *runMI* of the R package *semTools* (semTools Contributors, 2016) was used to combine the results obtained for the 20 imputed datasets. Parameter estimates and standard errors were pooled using Rubin's rules (Rubin, 2004). The MLMV estimator was used for its robustness to departures from normality since this assumption did not hold for all reflective indicators. Hence the dependent variable *health status* with four ordered levels had to be treated as continuous to allow the use of this robust maximum likelihood estimator. The large size of our sample and the absence of floor or ceiling effects in this variable justified such a treatment (see Appendix B Fig. B.1). Finally, the chi-square statistics and the related fit indices were pooled using the method described in Li, Meng, Raghunathan, and Rubin (1991).

3. Results

3.1. Correlation matrix and descriptive statistics

Based on psychosocial acceleration theory, we expected correlations between all life history measures and with childhood environmental harshness in a pattern consistent with the relationships implied by the fast-slow continuum. Table 1 reports descriptive statistics and the correlation matrix for the variables included in the first SEM (Fig. 1A). An extended table including the individual harshness indicators used in the second SEM (Fig. 1B) is available in Appendix B (Table B.1).

The raw correlation matrix shows low but significant correlations among some of the life history indicators and with the global harshness score. Furthermore, the direction of the effect is consistent with the theory for every significant correlation. Hence, to further explore this

pattern and to assess the theoretical model presented in the introduction, we fitted the two structural equation models represented in Fig. 1 on the data. Fit indices and parameters estimates are reported separately for each model in the following sections.

3.2. SEM with harshness as a sum of z-scores

3.2.1. Model fit

Table 2 reports fit indices for the SEMs. The chi-square test yielded significant *p*-values for the first SEM. However, the large sample size of our study ($N = 1015$) prevents us from interpreting this statistic as evidence for a discrepancy between the sample and the model-implied covariance matrix. The chi-square statistic is indeed known to be particularly sensitive to sample size, which can lead models fitted on large samples to be systematically rejected (Schermelleh-Engel, Moosbrugger, & Mäüller, 2003). We therefore focus on several approximate fit indices, the Comparative Fit Index (CFI), Standardized Root Mean Square Residual (SRMR) and Root Mean Square Error of Approximation (RMSEA), which eliminate the issue of sample size dependency (Kline, 2016). The RMSEA value of 0.046, associated with a close-fit test giving a *p*-value of 0.63, suggests an approximately good fit of the model. CFI and SRMR values of respectively 0.93 and 0.030 are also consistent with a close fitting model. Therefore, the approximate fit indices reveal no strong misspecification for this model.

3.2.2. Measurement model: the life history strategy latent factor

All life history variables included in the model loaded significantly on the general life history latent factor except “number of children” (Fig. 1A; see Appendix B Table B.2 for an extensive list of model coefficients). Inspection of the estimated covariance however, shows that “number of children” is not independent of the other reproductive items but correlates with “age at first birth” ($r = -0.36, p < 0.001$). Yet, even though the moderate correlation between “number of children” and “age at first birth” is consistent with the theory, it is not part of the general pattern captured by the life history factor. For all other life history items, the pattern of covariation follows our predictions: higher scores on the life history factor are associated with lower BMI (standardized $c = -0.18, p = 0.01$), higher self-reported efforts in looking after one's health (standardized $c = 0.21, p = 0.005$), higher self-reported general health status (standardized $c = 0.15, p < 0.001$), a lesser proportion of life spent smoking daily (standardized $c = -0.15, p < 0.001$), later age at first birth (standardized $c = 0.16, p < 0.001$), later sexual debut (standardized $c = 0.27, p < 0.001$) and fewer short-term sexual partners (standardized $c = -0.25, p < 0.001$); and therefore correspond to the theoretical description of a slow strategy. Hence, the latent life history construct in the first SEM is consistent with the proposed fast-slow continuum.

Table 1

Descriptive statistics and correlations from self-report data ($n = 1015$).

	BMI	Health status	Health effort	Smoking	Number of children	Age at first birth	Sexual debut	Short-term partners	Harshness	Age
BMI	–									
Health status	–0.20*	–								
Health effort	–0.12*	0.18*	–							
Smoking	0.04	–0.09*	–0.12*	–						
Number of children	0.13*	–0.00	0.01	–0.05	–					
Age at first birth	–0.09*	0.13*	–0.00	–0.05	–0.37*	–				
Sexual debut	–0.03	0.04	0.06*	–0.20*	0.04	0.18*	–			
Short-term partners	0.04	–0.00	–0.10*	0.19*	–0.07*	0.10*	–0.22*	–		
Harshness ^a	0.05	–0.14*	–0.07*	0.12*	0.04	–0.10*	–0.09*	0.04	–	
Age	0.11*	–0.14*	0.21*	0.04	0.17*	–0.15*	0.17*	0.07*	–0.06*	–
Mean	26.04	2.75	68.98	0.21	2.15	25.66	18.57	6.21	–0.06	52.52
SD	5.15	0.70	19.01	0.25	0.96	4.86	3.13	12.42	12.36	14.33
Range	13.6–58.8	1–4	0–100	0–0.91	1–7	14–56	10–61	0–160	–17.2–66.6	19–87

* $p < 0.05$.

^a Computed as a sum of z-scores, see Section 2.3.

Table 2
Statistical and practical fit indices for the structural equation models.

Model	χ^2	df	p	RMSEA	SRMR	CFI
Model 1	58.6	15	<0.001	0.046	0.030	0.93
Model 2	191	113	<0.001	0.026	0.028	0.83

3.2.3. Structural model: effect of harshness on the life history strategy factor

Concerning the structural part of the first SEM, the global score of harshness during childhood is negatively associated with the latent variable (standardized $c = -0.34$, $p < 0.001$). This relationship confirms the predictions of the theory since higher scores on the harshness index are negatively associated with a slower life history as reflected in a higher life history score.

3.3. SEM with harshness as a latent composite

3.3.1. Model fit

For the second model, as expected, the chi-square test yields a significant p -value. SRMR and RMSEA values were closer to zero (respectively 0.028 and 0.026) compared to the first SEM, which indicates a closer fit. On the other hand, the CFI index with a value of 0.83, which is inferior to the soft criterion of 0.90 (Bentler & Bonett, 1980), no longer indicates a close fit. The latter discrepancy might be due to the numerous degrees of freedom introduced by including the various harshness items/scales. Overall, the approximate fit indices still reveal no strong misspecification for this model.

3.3.2. Measurement model: the life history strategy latent factor

Coefficients related to the latent life history factor are very similar to those obtained for the first SEM. Although the particular values of the coefficients slightly vary, the overall pattern of covariation is identical: the variable “number of children” does not significantly correlate with the general factor but the remaining life history indicators covary as predicted by the fast-slow continuum (Fig. 1B). Indeed, higher scores on the life history strategy factor still reflect slower strategies characterized by lower BMI (standardized $c = -0.15$, $p = 0.03$), higher health efforts (standardized $c = 0.24$, $p < 0.001$), better health status (standardized $c = 0.19$, $p < 0.001$), a lesser proportion of life spent smoking daily (standardized $c = -0.14$, $p < 0.001$), later age at first birth (standardized $c = 0.15$, $p < 0.001$), later sexual debut (standardized $c = 0.24$, $p < 0.001$) and fewer short-term sexual partners (standardized $c = -0.21$, $p = 0.003$).

3.3.3. Structural model: effect of harshness on the life history strategy factor

Childhood harshness measured as a latent composite in the second SEM also predicted faster life history strategies. The use of a composite led to a slightly stronger association between these variables (standardized $c = -0.37$, $p < 0.001$; Fig. 1B, see Appendix B Table B.3 for an extensive list of model coefficients). However, the examination of the composite weights also reveals that this effect of childhood harshness is mostly driven by the item *violence in the family*. Indeed, only this item, which measured whether participants suffered from physical, sexual or psychological abuse caused by members of their family, contributed significantly to the effect of the composite (partially reduced model: standardized $c = -0.24$, $p < 0.001$). Marginal contributions to the composite's effects on the latent life history factor of *death of the mother* (standardized $c = 0.41$, $p = 0.055$) and *having lived with a stepfather* (standardized $c = 0.54$, $p = 0.061$) also emerged from this model. Lastly, we conducted a cross-validation analysis by computing harshness weights on one half of the sample and using these as a priori weights to calculate the harshness score and to predict life history strategies for the second half of the sample. This procedure was repeated 1000 times to reduce variability. The overall pattern confirmed our previous analyses: higher harshness scores significantly predicted faster

life history strategies (see Appendix B Table 4 for more details). The variance explained in life history strategies however decreased with an average R-square of 4%.

4. Discussion

Research in human behavioural ecology suggests that exposure to high levels of environmental harshness during ontogeny increases the probability of individuals adopting fast strategies. Previous studies have provided empirical support for this proposal by examining patterns of inter-individual differences often for various measures of either somatic or reproductive investments (Belsky, 2012; Pepper & Nettle, 2014). The present research further supports these findings by showing that, in a representative sample of the French population, distinct life history variables covary across both allocation domains in a theoretically coherent manner. The latent variable indeed contrasts individuals exhibiting i) traits suggestive of a lesser investment in their soma (smoking, lower self-reported health status, efforts in looking after one's health and higher BMI) and ii) a faster reproductive strategy (earlier sexual debut, age at first birth and higher number of sexual partners), with individuals showing the opposite characteristics. Furthermore, childhood harshness predicted scores reflecting faster strategies, which is consistent with our interpretation of this latent variable as the fast-slow continuum.

Thus, the emerging covariation pattern fits well with the idea of a broad fast-slow axis of life history variation. Nonetheless, it should be noted that the fast-slow continuum captures only a fraction of the variance in individual life history traits and that it will not necessarily be relevant for all of them. Such a result is not unexpected since life history traits are undoubtedly under the influence of multiple unmeasured causes, which can lead individuals' allocation strategies to depart from typical fast or slow combinations of traits. For example, BMI is sensitive to genetic factors (Locke et al., 2015) and its relationship with the intensity of physical activity is not completely linear, e.g., athletes tend to have high BMI but low percentage of body fat (Etchison et al., 2011). In such cases, the associated variance in BMI will not be captured by the fast-slow continuum and might even correlate in opposite directions with other life history indicators.

Similarly, many determinants of fertility might isolate it from the fast-slow continuum in developed countries and explain why *number of children* did not correlate with the latent variable. In our representative sample of a country where the demographic transition has long been completed, mean fertility is indeed close to two children per parent with little variation around this value. Cultural factors such as easy access to contraceptives, universal health care for both the child and the parents, widespread access to wage labor via economic markets for women and highly shared norms about family size might for example explain why fertility is disconnected from the fast-slow continuum (Colleran, 2016; Lawson & Borgerhoff Mulder, 2016). Eventually, several meaningful axes of variation are likely to emerge once one tries to capture finer inter-individual differences in life strategies across human populations and to identify particular socioecological factors that call for more diverse clusters of allocation strategies (Del Giudice, 2014b).

In addition, our analysis also suggests that the calibration of life history strategies might be particularly sensitive to specific events. Indeed, the composite model revealed that when all harshness predictors were considered independently, only *violence in the family* contributed significantly to its effect on the latent variable, with marginal effects of *having lived with a stepfather* and *death of the mother*. These differential effects of harshness items could be interpreted in the light of theoretical models of adaptive developmental plasticity (Frankenhuis & Panchanathan, 2011a, 2011b). These models predict that the reliability (i.e. the strength of the association between a cue and a particular state of the environment) of the cues used by an organism to adjust its developmental trajectory should influence the timing and the

rigidity of the organism's commitment to a particular life strategy. Therefore, a higher cue reliability compared to other harsh events could be one property of the item *violence in the family*, beyond the fact that it is a particularly strong measure of low parental care. This would be the case if such violent behaviours from the caregivers have been more reliably associated with future harsh conditions over human evolutionary history compared to other types of harsh events. This interpretation is in line with the external-PAR hypothesis described in the introduction. Yet, an account based on the internal-PAR hypothesis might also be given here since violence inside the family can sometimes involve severe costs to the child's soma. Hence further studies are needed to arbitrate between the two hypotheses. Alternatively, the absence of significant coefficients for the other harshness predictors might be due to the retrospective and non-genetically informed design of the current study. Indeed, participants were asked to recall and judge adverse events that took place several decades ago in certain cases and the accuracy of their report could have been severely limited (Hardt & Rutter, 2004). However, this null finding could also arise from the fact that depending on their genes, individuals might not equally weight their developmental experience during the calibration of their life history strategies (Belsky et al., 2012; Belsky & Pluess, 2009). For instance, there is empirical evidence that the effects of harshness on female pubertal development are genetically moderated (Hartman, Widaman, & Belsky, 2015). In addition, the small number of positive realizations in our sample for rare events such as *death of mother*, *death of father* or *long illness* might have prevented the detection of meaningful effects.

Regarding the influence of childhood harshness on life history strategies, it should also be stated that the correlational nature and the cross-sectional design of the current study hinders inferences about the causal role of early adversity on future life history strategies. Indeed, the influence of the environment experienced later in development on life strategies could not be controlled for. However, several longitudinal studies in adolescents have already found that both early and later environments predict individuals' life strategies (Belsky, 2012; Belsky et al., 2010; Brumbach et al., 2009; Hartman et al., in press; Simpson et al., 2012). Moreover, recent works, studying the effect of external shocks (famine, epidemics, war, etc.) during fetal life and early childhood, have demonstrated that lack of resources has detrimental and durable effects on a range of outcomes later in life: health problems (Lin & Liu, 2014), attention deficits (de Rooij, Wouters, Yonker, Painter, & Roseboom, 2010), anti-social behaviours (Neugebauer, 1999), lower educational level (Lavy, Schlosser, & Shany, 2016), or lower probability of being married and getting a job (Almond, Edlund, Li, & Zhang, 2007). Hence, while life-history strategies remain flexible in the face of new information, at least part of the effect of childhood harshness measured here might reflect conditional adaptations to early life conditions.

Thus, despite the caveats mentioned above, the overall pattern measured in this study is consistent with the idea that people form coherent life history strategies that can be partly captured by a fast-slow continuum and shaped by early experience of harsh events. Such a general pattern in a developed country is not easily explained without adopting an evolutionary developmental perspective (Frankenhuis et al., 2016) and it will therefore be interesting to extend this work. For instance, future research should identify which fast and slow strategies hold or vary across the broader range of situations encountered by humans. One promising direction could be to implement statistical techniques such as SEMs with composite variables in longitudinal designs or capitalizing on relevant natural experiments. This way one could assess the respective contributions to the development of fast strategies of different harsh events measured at various time points during ontogeny.

To conclude, our results support the relevance of adopting an evolutionary framework to explore patterns of individual differences within and across human populations. Our study also highlights the relevance of approaches that consider whole suites of behaviours rather than single outcomes in order to test functional hypotheses related to Life History Theory. More importantly perhaps, this framework puts forward a

different way of construing important behavioural obstacles to health improvement in developed countries. Indeed, while vaccination, antibiotics and improved sanitation have greatly increased life expectancy, this process based on technological advances may have reached its limits. Recent works indeed suggests that the maximum lifespan of humans is subject to natural constraints (Dong, Milholland, & Vijg, 2016). By contrast, many years of life are still lost due to lifestyle factors, in particular in middle and lower social classes. Moreover, while the most important health issues in the 20th were due to infectious pathogens, the most important health issues of the 21st century are primarily due to "lifestyle" decisions (dietary risks, high body-mass index, and tobacco smoking). Despite these evidence, behavior-related causes of health are still ill-understood. For most people, dietary risks, high body-mass index, and tobacco smoking are seen as the result of lifestyle choices over which individuals have control (Hallsworth et al., 2016). Instead, the framework we put forward in this paper suggests that part of the variance observed in these at-risk behaviours can be traced back to evolved mechanisms geared to maximize short-term rewards over long-term investments in an environment that is perceived as dangerous.

Acknowledgments

This study was supported by the Institut d'Études Cognitives (ANR-10-LABX-0087 IEC and ANR-10-IDEX-0001-02 PSL*), the Institut national de la santé et de la recherche médicale (INSERM), an ERC consolidator grant (no. 8657) and an "Action Incitative" funding from the École Normale Supérieure. We are grateful to Anne L'Hôte for programming the survey and to Céline Dusautois for her help in preliminary analyses.

Conflict of interests

None.

Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.evolhumbehav.2017.08.006>.

References

- Almond, D., Edlund, L., Li, H., & Zhang, J. (2007). *Long-term effects Of the 1959–1961 China famine: Mainland China and Hong Kong (No. w13384)*. Cambridge, MA: National Bureau of Economic Research.
- Baldini, R. (2015). *Harsh environments and "fast" human life histories: What does the theory say?* *bioRxiv* 14647. <https://doi.org/10.1101/014647>.
- Belsky, J. (2012). The development of human reproductive strategies: Progress and prospects. *Current Directions in Psychological Science*, 21, 310–316. <https://doi.org/10.1177/0963721412453588>.
- Belsky, J., & Pluess, M. (2009). Beyond diathesis stress: Differential susceptibility to environmental influences. *Psychological Bulletin*, 135, 885–908. <https://doi.org/10.1037/a0017376>.
- Belsky, J., Schlomer, G. L., & Ellis, B. J. (2012). Beyond cumulative risk: distinguishing harshness and unpredictability as determinants of parenting and early life history strategy. *Developmental Psychology*, 48, 662–673. <https://doi.org/10.1037/a0024454>.
- Belsky, J., Steinberg, L., & Draper, P. (1991). Childhood experience, interpersonal development, and reproductive strategy: An evolutionary theory of socialization. *Child Development*, 62, 647. <https://doi.org/10.2307/1131166>.
- Belsky, J., Steinberg, L., Houts, R. M., Halpern-Felsher, B. L., & NICHD Early Child Care Research Network (2010). The development of reproductive strategy in females: Early maternal harshness → Earlier menarche → Increased sexual risk taking. *Developmental Psychology*, 46, 120–128. <https://doi.org/10.1037/a0015549>.
- Belsky, J., Steinberg, L. D., Houts, R. M., Friedman, S. L., DeHart, G., Cauffman, E., ... NICHD Early Child Care Research Network (2007). Family rearing antecedents of pubertal timing. *Child Development*, 78, 1302–1321. <https://doi.org/10.1111/j.1467-8624.2007.01067.x>.
- Bentler, P. M., & Bonett, D. G. (1980). Significance tests and goodness of fit in the analysis of covariance structures. *Psychological Bulletin*, 88, 588–606. <https://doi.org/10.1037/0033-2909.88.3.588>.
- Brumbach, B. H., Figueroa, A. J., & Ellis, B. J. (2009). Effects of harsh and unpredictable environments in adolescence on development of life history strategies: A longitudinal

- test of an evolutionary model. *Human Nature*, 20, 25–51. <https://doi.org/10.1007/s12110-009-9059-3>.
- Buuren, S. v., & Groothuis-Oudshoorn, K. (2011). *Mice: Multivariate imputation by chained equations in R*. *J. Stat. Softw.*, 45. <https://doi.org/10.18637/jss.v045.i03>.
- Cabeza de Baca, T., & Ellis, B. J. (2017). Early stress, parental motivation, and reproductive decision-making: applications of life history theory to parental behavior. *Current Opinion in Psychiatry*, 15, 1–6. <https://doi.org/10.1016/j.copsyc.2017.02.005>.
- Chen, E., Matthews, K. A., & Boyce, W. T. (2002). Socioeconomic differences in children's health: how and why do these relationships change with age? *Psychological Bulletin*, 128, 295–329.
- Chisholm, J. S., Ellison, P. T., Evans, J., Lee, P. C., Lieberman, L. S., Pavlik, Z., ... Worthman, C. M. (1993). Death, hope, and sex: Life-history theory and the development of reproductive strategies [and comments and reply]. *Current Anthropology*, 34, 1–24. <https://doi.org/10.1086/204131>.
- Colleran, H. (2016). The cultural evolution of fertility decline. *Philos. Trans. R. Soc. B Biol. Sci.*, 371, 20150152. <https://doi.org/10.1098/rstb.2015.0152>.
- de Rooij, S. R., Wouters, H., Yonker, J. E., Painter, R. C., & Roseboom, T. J. (2010). Prenatal undernutrition and cognitive function in late adulthood. *Proceedings of the National Academy of Sciences*, 107, 16881–16886. <https://doi.org/10.1073/pnas.1009459107>.
- Del Giudice, M. (2014a). An evolutionary life history framework for psychopathology. *Psychological Inquiry*, 25, 261–300. <https://doi.org/10.1080/1047840X.2014.884918>.
- Del Giudice, M. (2014b). A tower unto heaven: Toward an expanded framework for psychopathology. *Psychological Inquiry*, 25, 394–413. <https://doi.org/10.1080/1047840X.2014.925339>.
- Dong, X., Milholland, B., & Vijg, J. (2016). Evidence for a limit to human lifespan. *Nature*, 538, 257–259. <https://doi.org/10.1038/nature19793>.
- Ellis, B. J. (2004). Timing of pubertal maturation in girls: An integrated life history approach. *Psychological Bulletin*, 130, 920–958. <https://doi.org/10.1037/0033-2909.130.6.920>.
- Ellis, B. J., & Essex, M. J. (2007). Family environments, adrenarche, and sexual maturation: A longitudinal test of a life history model. *Child Development*, 78, 1799–1817. <https://doi.org/10.1111/j.1467-8624.2007.01092.x>.
- Ellis, B. J., Figueredo, A. J., Brumbach, B. H., & Schlomer, G. L. (2009). Fundamental dimensions of environmental risk: The impact of harsh versus unpredictable environments on the evolution and development of life history strategies. *Human Nature*, 20, 204–268. <https://doi.org/10.1007/s12110-009-9063-7>.
- Etchison, W. C., Bloodgood, E. A., Minton, C. P., Thompson, N. J., Collins, M. A., Hunter, S. C., & Dai, H. (2011). Body mass index and percentage of body fat as indicators for obesity in an adolescent athletic population. *Sports Health*, 3, 249–252. <https://doi.org/10.1177/1941738111404655>.
- Figueredo, A. J., Vásquez, G., Brumbach, B. H., Schneider, S. M., Sefcek, J. A., Tal, I. R., ... Jacobs, W. J. (2006). *Consilience and life history theory: From genes to brain to reproductive strategy*. *Developmental Review*, 26, 243–275.
- Figueredo, A. J., Vásquez, G., Brumbach, B. H., Sefcek, J. A., Kirsner, B. R., & Jacobs, W. J. (2005). The K-factor: Individual differences in life history strategy. *Personality and Individual Differences*, 39, 1349–1360. <https://doi.org/10.1016/j.paid.2005.06.009>.
- Frankenhuis, W. E., & Panchanathan, K. (2011a). Balancing sampling and specialization: An adaptationist model of incremental development. *Proceedings of the Royal Society B: Biological Sciences*, 278, 3558–3565. <https://doi.org/10.1098/rspb.2011.0055>.
- Frankenhuis, W. E., & Panchanathan, K. (2011b). Individual differences in developmental plasticity may result from stochastic sampling. *Perspectives on Psychological Science*, 6, 336–347. <https://doi.org/10.1177/1745691611412602>.
- Frankenhuis, W. E., Panchanathan, K., & Nettle, D. (2016). Cognition in harsh and unpredictable environments. *Current Opinion in Psychiatry*, 7, 76–80. <https://doi.org/10.1016/j.copsyc.2015.08.011>.
- Grace, J. B., Anderson, T. M., Olff, H., & Scheiner, S. M. (2010). On the specification of structural equation models for ecological systems. *Ecological Monographs*, 80, 67–87.
- Grace, J. B., & Bollen, K. A. (2008). Representing general theoretical concepts in structural equation models: The role of composite variables. *Environmental and Ecological Statistics*, 15, 191–213. <https://doi.org/10.1007/s10651-007-0047-7>.
- Griskevicius, V., Ackerman, J. M., Cantu, S. M., Delton, A. W., Robertson, T. E., Simpson, J. A., ... Tybur, J. M. (2013). When the economy falters, do people spend or save? Responses to resource scarcity depend on childhood environments. *Psychological Science*, 24, 197–205. <https://doi.org/10.1177/0956797612451471>.
- Griskevicius, V., Delton, A. W., Robertson, T. E., & Tybur, J. M. (2011). Environmental contingency in life history strategies: The influence of mortality and socioeconomic status on reproductive timing. *Journal of Personality and Social Psychology*, 100, 241–254. <https://doi.org/10.1037/a0021082>.
- Guégan, J. F., Thomas, F., Hochberg, M. E., de Meeüs, T., & Renaud, F. (2001). Disease diversity and human fertility. *Evol. Int. J. Org. Evol.*, 55, 1308–1314.
- Hallsworth, M., Snijders, V., Burd, H., Prest, J., Judah, G., Huf, S., & Halpern, D. (2016). *Applying behavioral insights: Simple ways to improve health outcomes*. World Innovation Summit for Health: Doha, Qatar.
- Hardt, J., & Rutter, M. (2004). Validity of adult retrospective reports of adverse childhood experiences: Review of the evidence. *Journal of Child Psychology and Psychiatry*, 45, 260–273. <https://doi.org/10.1111/j.1469-7610.2004.00218.x>.
- Hartman, S., Li, Z., Nettle, D., & Belsky, J. (2017). External-environmental and internal-health early-life predictors of adolescent development. *Development and Psychopathology* (in press).
- Hartman, S., Widaman, K. F., & Belsky, J. (2015). Genetic moderation of effects of maternal sensitivity on girl's age of menarche: Replication of the Manuck et al. study. *Development and Psychopathology*, 27, 747–756. <https://doi.org/10.1017/S0954579414000856>.
- Hoffman, S. D., Foster, E. M., & Furstenberg, F. F. (1993). Reevaluating the costs of teenage childbearing. *Demography*, 30, 1–13. <https://doi.org/10.2307/2061859>.
- Hurst, J., & Sassi, F. (2008). *The prevention of lifestyle-related chronic diseases*. (OECD Health Working Papers No. 32).
- Kaplan, H., Lancaster, J., & Robson, A. (2003). Embodied capital and the evolutionary economics of the human life span. *Population and Development Review*, 29, 152–182.
- Kline, R. B. (2016). Principles and practice of structural equation modeling. *Methodology in the social sciences* (Fourth edition). New York: The Guilford Press.
- Lavy, V., Schlosser, A., & Shany, A. (2016). *Out of Africa: Human capital consequences of in utero conditions* (No. w21894). Cambridge, MA: National Bureau of Economic Research.
- Lawson, D. W., & Bergerhoff Mulder, M. (2016). The offspring quantity–Quality trade-off and human fertility variation. *Philos. Trans. R. Soc. B Biol. Sci.*, 371, 20150145. <https://doi.org/10.1098/rstb.2015.0145>.
- Li, K. -H., Meng, X. -L., Raghunathan, T. E., & Rubin, D. B. (1991). Significance levels from repeated p-values with multiply-imputed data. *Statistica Sinica*, 1, 65–92.
- Lin, M. -J., & Liu, E. (2014). *Does in utero exposure to illness matter? The 1918 influenza epidemic in Taiwan as a natural experiment* (No. w20166). Cambridge, MA: National Bureau of Economic Research.
- Locke, A. E., et al. (2015). Genetic studies of body mass index yield new insights for obesity biology. *Nature*, 518, 197–206. <https://doi.org/10.1038/nature14177>.
- McCullough, M. E., Pedersen, E. J., Schroder, J. M., Tabak, B. A., & Carver, C. S. (2012). Harsh childhood environmental characteristics predict exploitation and retaliation in humans. *Proceedings of the Royal Society B: Biological Sciences*, 280, 20122104. <https://doi.org/10.1098/rspb.2012.2104>.
- Miller, F. C. (2000). Impact of adolescent pregnancy as we approach the new millennium. *Journal of Pediatric and Adolescent Gynecology*, 13, 5–8.
- Moffitt, T. E., Caspi, A., Belsky, J., & Silva, P. A. (1992). Childhood experience and the onset of menarche: A test of a sociobiological model. *Child Development*, 63, 47–58.
- Nettle, D. (2010). Why are there social gradients in preventative health behavior? A perspective from behavioral ecology. *PLoS One*, 5, e13371. <https://doi.org/10.1371/journal.pone.0013371>.
- Nettle, D. (2011). Flexibility in reproductive timing in human females: Integrating ultimate and proximate explanations. *Philos. Trans. R. Soc. B Biol. Sci.*, 366, 357–365. <https://doi.org/10.1098/rstb.2010.0073>.
- Nettle, D., Coall, D. A., & Dickins, T. E. (2009). Birthweight and paternal involvement predict early reproduction in British women: Evidence from the National Child Development Study. *American Journal of Human Biology NA-NA*, 22(2), 172–179. <https://doi.org/10.1002/ajhb.20970>.
- Nettle, D., & Cockerill, M. (2010). Development of social variation in reproductive schedules: A study from an English urban area. *PLoS One*, 5, e12690. <https://doi.org/10.1371/journal.pone.0012690>.
- Nettle, D., Frankenhuis, W. E., & Rickard, I. J. (2012). The adaptive basis of psychosocial acceleration: Comment on beyond mental health, life history strategies articles. *Developmental Psychology*, 48, 718–721. <https://doi.org/10.1037/a0027507>.
- Neugebauer, R. (1999). Prenatal exposure to wartime famine and development of antisocial personality disorder in early adulthood. *JAMA*, 282, 455. <https://doi.org/10.1001/jama.282.5.455>.
- Pepper, G. V., & Nettle, D. (2014). Perceived extrinsic mortality risk and reported effort in looking after health: Testing a behavioral ecological prediction. *Human Nature*, 25, 378–392. <https://doi.org/10.1007/s12110-014-9204-5>.
- Reale, D., Garant, D., Humphries, M. M., Bergeron, P., Careau, V., & Montiglio, P. -O. (2010). Personality and the emergence of the pace-of-life syndrome concept at the population level. *Philos. Trans. R. Soc. B Biol. Sci.*, 365, 4051–4063. <https://doi.org/10.1098/rstb.2010.0208>.
- Rickard, I. J., Frankenhuis, W. E., & Nettle, D. (2014). Why are childhood family factors associated with timing of maturation? A role for internal prediction. *Perspectives on Psychological Science*, 9, 3–15. <https://doi.org/10.1177/1745691613513467>.
- Roff, D. A. (2002). *Life history evolution*. Sunderland, Mass: Sinauer Associates.
- Rossee, Y. (2012). Lavaan: An R package for structural equation modeling. *Journal of Statistical Software*, 48. <https://doi.org/10.18637/jss.v048.i02>.
- Rubin, D. B. (2004). *Multiple imputation for nonresponse in surveys*. Hoboken, NJ: Wiley classics library. Wiley-Interscience.
- Schermelleh-Engel, K., Moosbrugger, H., & Müller, H. (2003). Evaluating the fit of structural equation models: Tests of significance and descriptive goodness-of-fit measures. *Methods Psychol. Res. Online*, 8, 23–74.
- Simpson, J. A., Griskevicius, V., Kuo, S. I. -C., Sung, S., & Collins, W. A. (2012). Evolution, stress, and sensitive periods: The influence of unpredictability in early versus late childhood on sex and risky behavior. *Developmental Psychology*, 48, 674–686. <https://doi.org/10.1037/a0027293>.
- Stearns, S. C. (1992). *The evolution of life histories*. Oxford; New York: Oxford University Press.
- Stearns, S. C. (2000). Life history evolution: successes, limitations, and prospects. *Naturwissenschaften*, 87, 476–486. <https://doi.org/10.1007/s001140050763>.
- Stringhini, S. (2010). Association of socioeconomic position with health behaviors and mortality. *JAMA*, 303, 1159. <https://doi.org/10.1001/jama.2010.297>.
- Tither, J. M., & Ellis, B. J. (2008). Impact of fathers on daughters' age at menarche: A genetically and environmentally controlled sibling study. *Developmental Psychology*, 44, 1409–1420. <https://doi.org/10.1037/a0013065>.