# Stratégies fast-slow dans les populations humaines : appliquer les principes de la théorie des traits d'histoire de vie pour expliquer la variation interindividuelle

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# Fast-slow strategies in human populations. Applying insights from life history theory to explain patterns of interindividual variation


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# Abstract

Social gradients in behavior have been documented across various domains of people's lives. In western countries, low SES individuals tend for instance to invest less in their education, to smoke more, are more subject to overweight and are more willing to take risks in financial settings. Being exposed to deprivation therefore seems to elicit a constellation of behaviors that appear to covary in a systematic fashion. This behavioral constellation of deprivation has been mostly interpreted as the product of poor decision making abilities, of a general failure of willpower. In this dissertation we explore a different interpretation that is rooted in adaptive explanations of human behavior. Instead of viewing the behaviors of low SES individuals as suboptimal deviations from a global optimum, they are seen as adjustments of people's overall life strategies that are, from an evolutionary point of view, adaptive in the particular context of a deprived ecology. Indeed, we will explore the idea that deprived environments select for strategies that put more weight on present outcomes over uncertain future outcomes, and that this present orientation in low SES individuals propagates across a range of decision domains, giving rise to the constellation. To this aim, we first use structural equation models on observational data from a diversity of samples, to analyze the covariation between peoples' behaviors in several relevant domains (health, reproduction, social trust) and their exposure to deprivation during childhood and/or adulthood. Overall, we find that a lower somatic effort tends to covary with a more short-term reproductive strategy, as well as lower social trust. This pattern is associated with a higher exposure to deprivation, with unique effects of early life conditions. In addition to this empirical work, we further investigate the theoretical underpinnings of our working hypotheses, from an adaptationist perspective. Specifically, we build a formal life history model to predict optimal changes in discounting within and between individuals. This allows us to highlight that the extent to which individuals prefer short-term rewards, should vary depending on two main parameters: 1) the uncertainty around their ability to actually collect delayed rewards, and 2) the opportunity costs of not having the reward during the delay. Finally, we conclude by discussing the promising perspective of further integrating the approach adopted in the present thesis, with more traditional social and behavioural sciences.

# Résumé

Des gradients sociaux ont été documentés pour une variété de comportements individuels. Au sein des pays occidentaux par exemple, les personnes ayant un statut socio-économique plus bas ont tendance à investir moins dans leur éducation, à fumer davantage, sont plus susceptibles de souffrir d'obésité et plus enclines à prendre des risques dans des contextes économiques. Ainsi, être exposé à des conditions de vie précaire semble engendrer une série de comportements, covariant de manière systématique. Ce syndrome comportemental a été principalement interprété comme le produit de déficits cognitifs ayant traits à la prise de décision et/ou à un manque de volonté. Dans ce manuscrit, nous explorons une approche différente, qui s'ancre dans les explications adaptationnistes des comportements humains. Au lieu d'appréhender les comportements observés chez les individus en situation de précarité comme des anomalies, ceux-ci sont perçus comme des ajustements des stratégies individuelles d'histoire de vie, adaptés à la vie dans des environments précaires. En effet, nous approfondirons l'hypothèse selon laquelle un environment précaire sélectionne des stratégies qui accordent davantage de poids aux bénéfices immédiats plutôt qu'aux investissements de long terme, affectant dès lors toute une gamme de comportements. Dans un premier temps, nous analyserons par le biais de modèles d'équations structurelles, différents jeux de données afin de mesurer le degré de covariation de comportements ayant trait à la reproduction, à l'investissement dans la santé et au niveau de confiance, ainsi que leur association éventuelle avec le niveau de précarité éprouvé pendant l'enfance ou à l'âge adulte. Globalement, nos résultats indiquent qu'une réduction de l'investissement dans la santé co-varie avec une stratégie de reproduction plus court-termiste, ainsi qu'un plus faible niveau de confiance. De plus, ce pattern est surreprésenté chez les personnes en situation de précarité, avec des effets persistants des conditions pendant l'enfance. En parallèle de ces travaux empiriques, nous nous sommes par ailleurs intéressés aux fondements théoriques de nos hypothèses de travail. Précisément, nous avons développé un modèle formel de stratégie d'histoire de vie prédisant les changements de préférences temporelles intra- et interindividuelles. Celui-ci nous a permis de mettre en évidence l'importance de deux facteurs distincts pour déterminer le degré optimal avec lequel des individus doivent préférer les récompenses de court-terme: 1) le niveau d'incertitude sur la probabilité de collecter une récompense délayée dans le temps, et 2) le coût d'opportunité à ne pas bénéficier de la récompense pendant la période de délai. Enfin, nous concluons ce travail en discutant des perspectives particulièrement intéressantes offertes par une intégration plus poussée de l'approche développée dans cette thèse, avec d'autres sciences sociales et sciences du comportement plus traditionnelles.

# Contents

# Chapter 1

# General introduction

## 1.1 A tale of two guppies

On the island of Trinidad, guppies (*Poecilia reticulata*) can be found in abundance in the numerous freshwater streams and rivers. Due to the complex topography of the landscape, these small tropical fishes experience important gradients in environmental conditions. In particular, Trinidadian guppies live in two broad categories of local habitats, that differ markedly with respect to predation risk (Endler 1995; Gordon et al. 2009; Reznick 1997). The 'low-predation' habitats usually correspond to small upstream portions of the water streams, above barrier waterfalls, and where mainly small, immature guppies are preyed upon by the killifish, *Rivulus harti*. The 'high-predation' habitats are found in the downstream portions of the rivers and are inhabited by a diversity of stronger predators that target preferentially large, mature individuals (Endler 1978; Haskins et al. 1961). These local differences in the composition of the predator community have been maintained through time by the geographical barriers that prevent larger predators from colonizing the upstream parts. Thus, except for a few individuals that might occasionally transfer from the upper to the lower sections of the rivers, the two populations are basically kept in isolation from one another, and individual guppies will almost always encounter only one type of predation regime during their lifetime.

In addition to the criterion of predation pressure, the two populations actually differ

by whole suites of physiological, morphological, demographical and behavioural traits, to the point that they are often considered as two distinct ecotypes of guppies (Sullam et al. 2015). For instance, in comparison to the high-predation populations, female guppies in the low-predation sites tend to mature at later ages, have fewer, larger offspring and invest less in reproduction relative to their body size (Gordon et al. 2009). Both males and females show more conspicuous color patterns and shorter but wider bodies (Endler 1995). Behaviourally, the courtship styles of the males include more displays and less sneaking, made at longer distance and with less intensity. Schooling is also less important and the level of agression between conspecifics is higher (Seghers et al. 1991). These are just a few examples of a long list of typical differences that have been carefully documented over the years by evolutionary ecologists (Magurran 2005).

Thus, even though individuals from both populations actually belong to the same species, there are considerable and systematic variations with respect to a multitude of aspects of their phenotypes. Such differences seem to map with particular environmental conditions and appear in a coordinated fashion. Indeed, genetic analyses conducted on several populations of Trinidadian guppies suggest that the low-predation populations originated from indepent events of colonization of the upstream sections, from various high-predation sites (Crispo et al. 2005). Yet, although independent, these phases of expansion always co-occured with a deviation from the phenotypic distribution of the ancestral population and a stabilization on similar trait values for the various low-predation sites. Such a phenomenon has been supported by experimental evidence gathered from artificially introducing individuals from an ancestral population to new sites, with either a similar high-predation regime or a low-predation one (Gordon et al. 2009). Only ten years after the manipulation, the populations of newly introduced guppies already exhibited phenotypic distributions closer to the ecotype that matched their habitat type.

Hence, the natural history of the Trinidadian guppies raises a number of questions that are really at the heart of evolutionary ecology. For instance, why do we observe important differences between the traits express by guppies in the low *vs* high

predation habitats? How is such variation generated? Why should independent populations colonizing new sites characterized by a low predation pressure converge on similar phenotypic distributions? And why should a variety of morphological, physiological and behavioural traits covary in a systematic fashion? In the following section, I introduce life history theory, a body of theoretical and empirical work in evolutionary biology built to adress these fundamental problems.

## 1.2 Finding regularities in the vertiginious diversity of organisms' life cycles

The previous case study illustrated the potential existence of substantial phenotypic variation between populations of the same species and the need to provide some evolutionary explanation for it. Yet, a quick look at the variation in life cycles across the tree of life will surely make the task of formulating any general theory appear quite daunting. Indeed, we can observe in nature both blue whales which weigh more than a hundred tonnes and measure up to 30 meters, become sexually mature after five to ten years of age, give birth to one large offspring every two to three years, and might live until eighty years; and our Trinidadian guppies, that on average will measure less than 10 centimeters, mature after 10 to 20 weeks, reproduce 2 or 3 times in a year by producing until 20 eggs each time, and might typically live two years or less. Then, how can we explain this diversity in fundamental traits such as age and size at maturity, birth rates or lifespan?

Classic life history theory is the subfield of evolutionary biology that concentrates precisely on explaining the evolution of these basic demographic traits (which are called life history traits), that, combined together determine the lifetime reproductive success of an organism (Roff 2002; Stearns 1992). It provides a unified framework to predict patterns of variation in life history traits based on the principles that, 1) limited resources available to an organism will have to be traded off between several biological functions, and 2) different ecologies will favor different allocation strategies. The first point refers to the idea that there are no 'Darwian demons' in nature (Law 1979), that is, no organism that can maximize all components of

fitness simultaneously. A unit of energy can be use either for immediate reproduction, maintenance or growth, but never for all domains at the same time. This leads to the most fundamental life history trade-off, which opposes current and future reproduction (Stearns 1992). Indeed, at an abstract level, an organism's life cycle could be conceptualized as a succesion of allocation decisions, that choose at each point in time between a short-term option, which directly converts resources to fitness through reproduction, and a long-term option, which uses resources to increase future reproductive prospects at the cost of immediate reproduction. Then, a significant part of the variance observed in life histories should originate from the way different ecologies will affect the optimal resolution of the trade-off between current and future reproduction.

Going back to the case study of the Trinidadian guppies, the main ecological factor that distinguishes the two types of habitat is the composition of the predator community. Life history theory therefore drives us to ask in what way such differences in predation exposure might affect the trade-off between current *vs* future reproduction. As described in the previous section, in the low-predation sites, guppies are mainly preyed upon by a relatively small species of killifish, selectively targetting immature individuals; while in the high predation sites, larger guppies are the preferred preys of a variety of stronger predators. Thus, between the two types of habitat, the mortality rates experienced by the various age classes that structure the guppy populations will differ. In the low predation sites, juveniles suffer from high rates of predations due to the presence of the killifish but once individuals manage to reach their reproductive size, they are exposed to relatively low mortality rates. In the high predation habitats on the other hand, juveniles might be more likely to survive due to the absence of the killifish (but see Reznick et al. (1996)), whereas mortality rates for adults will tend to increase as they become exposed to more predation pressure as they grow.

Consequently, in high predation environments where adult mortality is important, investing in future reproduction will be comparatively risky due to the higher probability of dying before reaping the benefits of any long-term investment. Then, all else being equal, individuals should adopt a life history strategy that yield benefits

at shorter time horizons. This predicts that higher adult mortality should bring individuals to mature at earlier ages and smaller sizes, and to invest more into reproduction during early reproductive bouts, even though this means sacrificing offspring production at later ages (Gadgil and Bossert 1970; Michod 1979).

The previous pattern captures a key part of the life history differences between the two ecotypes of guppies and is predicted by the impact of different levels of mortality rates on the trade-off between current and future reproduction. Furthermore, as described previously, the above predictions are consistent with the experimental data obtained from the artificial introduction of hundreds of guppies into various new habitats (Gordon et al. 2009). Going beyond Trinidadian guppies, the generality of the phenomenon is also supported by another experiment of artificial evolution conducted in laboratory conditions with the fruit fly, *Drosophila melanogaster* (Stearns 2000). During several years, tens of thousands of flies divided into distinct lines were subjected to one of two treatments. In the first treatment, which corresponded to a high mortality condition, twice a week 90% of the flies were killed by the experimenter. In the low mortality treatment on the other hand, only 10% of them were killed, twice a week as well. Densities of larvae and adults were matched for all lines, and food, temperature, humidity and light cycle were kept identical in both conditions. After four years, as predicted by the theory, flies exposed to the high mortality treatment were smaller at eclosion, grew and matured faster, invested more into reproduction early in life and had a shorter lifespan than the flies that evolved under low extrinsic mortality.

Thus, as exemplified by the Trinidadian guppies natural history and the fruit fly experiment, age-specific extrinsic mortality rates are a fundamental aspect of an organism's environment that will greatly affect which type of allocation strategy is optimal. In particular, when adult mortality is high, organisms should invest less in future reproduction. This will affect an array of life history traits in a systematic fashion (Stearns 1992), as seen with age/size at maturity and reproductive efforts in both guppies and fruit flies. In addition to age-specific extrinsic mortality, resource availability is the other basic ecological paremeter that will profoundly affect

organisms' life history traits (van Noordwijk and de Jong 1986). Indeed, the amount of resources individuals can extract from their environment will directly condition their potential reproductive rates at any age. In a resource restricted environment, levels of inter- and intra-specific competition will usually increase, and traits that allow organisms to capture a greater share of the resource pool will be at a selective advantage. This will affect the optimal rates of growth and sizes of individuals at various ages. Such predicted effects of resource availability on individuals' life histories can shed light on another set of differences between the two guppies ecotypes. Indeed, low-predation habitats are also usually characterized by lower light levels which restrain primary productivity and increase food limitation for the local guppy population (Reznick, Butler Iv, and Rodd 2001). Competition is therefore generally increased in low-predation habitats, and, as predicted by life history models, guppies inhabiting those sites produce fewer but larger offspring.

## 1.3 Responding to ecological factors - the role of adaptive phenotypic plasticity

Hence, organisms' life cycles are profoundly shaped by the interplay between diverse environmental factors that affect survival and reproductive rates. Life history models use optimality assumptions to predict the best allocation strategy in a given ecology and under a set of trade-offs, by identifying how a change in any particular life history trait would affect the overall fitness of the life cycle (Stearns 2000). This procedure will then tell (based on the model assumptions), which exact value for the trait into consideration is expected to evolve by natural selection.

Yet, individuals within a same species do not all experience identical environmental conditions. And with these ecological differences come variation in life history traits, as seen with the Trinidadian guppies. Thus, instead of a single value in a phenotypic space, in the case of within species variation, the theory actually needs to predict the whole sequence of trait values that an organism should produce to match a range of environmental conditions. This has been done in the field by modelling the evolution of reaction norms for particular life history traits (Kawecki and Stearns

1993; McNamara and Houston 1992; Stearns and Koella 1986). A reaction norm describes the set of phenotypes produced by a single genotype across a range of environmental conditions. It is tightly linked to the concept of phenotypic plasticity, which is the ability of a single genotype to produce various phenotypes (*i.e.*, reaction norms are the outputs of mechanisms of phenotypic plasticity).

To illustrate the concept with an example, the reaction norm for age at maturity with respect to extrinsic mortality will be a function, that tells at what age an organism will mature for each mortality rate of a particular range. In life history models, the goal is to find the optimal reaction norm, *i.e.*, the function that yields the highest overall fitness for the lifecycle. Then, when looking at variation within and between populations, if an organism modifies its phenotype such that, once moved into a different environment it gets closer to the value predicted by the optimal reaction norm, the underlying process of phenotypic plasticity will be called adaptive.

In the case of the Trinidadian guppies, the mapping between the habitat type and the life history traits measured is due both to processes of local adaptation (*i.e.*, changes in the genotypic structure of the populations in response to the local selective pressures) and to mechanisms of adaptive phenotypic plasticity (Reznick and Yang 1993; Reznick and Bryga 1996). Indeed, female guppies raised under conditions of food restriction will produce larger offspring on average. This reflects a process of adaptive phenotypic plasticity, because the change in phenotype (here the size of the offspring) occurs within a single generation and larger offspring have a higher lifetime reproductive success in resource-limited environments. Therefore, female Trinidadian guppies possess one or several mechanisms, designed to modify the level of energetic investment they provide to their offspring, depending on internal and/or external cues that reflect resource availability in their particular environment.

In some cases, the nature of these cues allowing adaptive phenotypic plasticity has been discovered. For instance, in the tiny crustacean Daphnia (Daphnia longicephala), individuals will develop large spiny helmets relative to their body size only when chemical cues (kairomones) from its natural predator are present in the environment (Weiss, Leimann, and Tollrian 2015). This type of inducible defense allows an organ-

ism both to increase its survival by carrying a helmet in predator rich environment, and make a better use of the resources (*e.g.*, to produce additional eggs) when there is no need for such protection. Hence, provided that specific mechanisms for adaptive phenotypic plasticity are not too costly (and that several other conditions are satisfied, see Nettle, Frankenhuis, and Rickard (2012)), they will significantly increase individual fitness in populations living in heterogeneous environments and are expected to evole. Therefore, mechanisms of adaptive phenotypic plasticity could be responsible for a significant part of the variation in life history traits observed for a large number of organisms.

## 1.4 Extending the classic theory to include physiology and behaviour

Thus, life history theory and the concept of an optimal reaction norm allow us to advance adaptationist explanations for the variation in life history traits observed both between and within species. However, as the case study of the Trinidadian guppies illustrates, systematic phenotypic differences between populations living under distinct ecological regimes are not restricted to classic life history traits (*i.e.*,, age at maturity, lifespan, etc.). Indeed, recall for example that guppies from low-predation populations exhibit more conspicuous color patterns, have different courtship styles and schooling behaviours, and are more agressive towards conspecifics (Endler 1995). Hence, the suite of traits that distinguish the two ecotypes also involve physiological and behavioural characteristics. Therefore, it has been argued that between and within species variation in life history traits should be integrated with differences in morphology, physiology and behaviour (Réale et al. 2010). This claim relies both on empirical and theoretical grounds.

From a theoretical point of view, the integration of life history traits with morphology, physiology and behaviour can be justified based on two key ideas. First, life history traits are actually complex traits that depend on the occurrence of particular physiological, morphological and behavioural processes. Second, certain combinations of traits will be maladaptive, while others will increase fitness. Thus, correlational

selection pressures will maintain favorable associations and eliminate the one that are deleterious, leading to the evolution of syndromes of traits. To illustrate this idea with a concrete example, an organism with a life history characterized by both high levels of boldness when foraging in the presence of predators, and a very late age at maturity is very likely to be counterselected, because the probability that it dies before reproducing is too high.

Applying the same general idea to the current *vs* future reproduction trade-off has led to the concept of the Pace of Life Syndrome (Réale et al. (2010); Ricklefs and Wikelski (2002); Figure 1.1). The latter states that both between and within species, coordinated suites of behavioural, physiological and life history traits sould map on a fast-slow continuum of strategies. Fast strategies involve traits that favor short-term benefits (*i.e.*, current reproduction) and translate into lower somatic effort (*i.e.*, less investment into the growth and maintenance of the body) and greater reproductive effort in the early period of life. Slow strategies on the other hand aim at long-term benefits and will consist of traits that increase future reproductive prospects.

The concept of the Pace of Life Syndrome initiated an impressive wave of empirical research in behavioural ecology during the last decade. It built on a preexisting litterature documenting significant covariation between life history traits, a large portion of which was captured by a fast-slow continuum (*e.g.*, Bielby et al. (2007); Sæther (1988)). Regarding the extended version of the fast-slow continuum advocated by the pace of life hypothesis, the evidence accumulated over the years has been equivocal overall (Montiglio et al. 2018; Royauté et al. 2018). Yet, the core idea still has an interesting theoretical foundation (although somewhat underspecified, see Mathot and Frankenhuis (2018)) and remains central in behavioural ecology.

## 1.5 From Trinidadian guppies to human behaviour

Evolutionary ecology therefore provides a set of theoretical principles, as well as a rich body of empirical data, shedding light on the determining factors behind patterns of variations, for a vast array of phenotypic traits. Specifically, we have seen that depending on the environmental conditions and the state of an organism, different

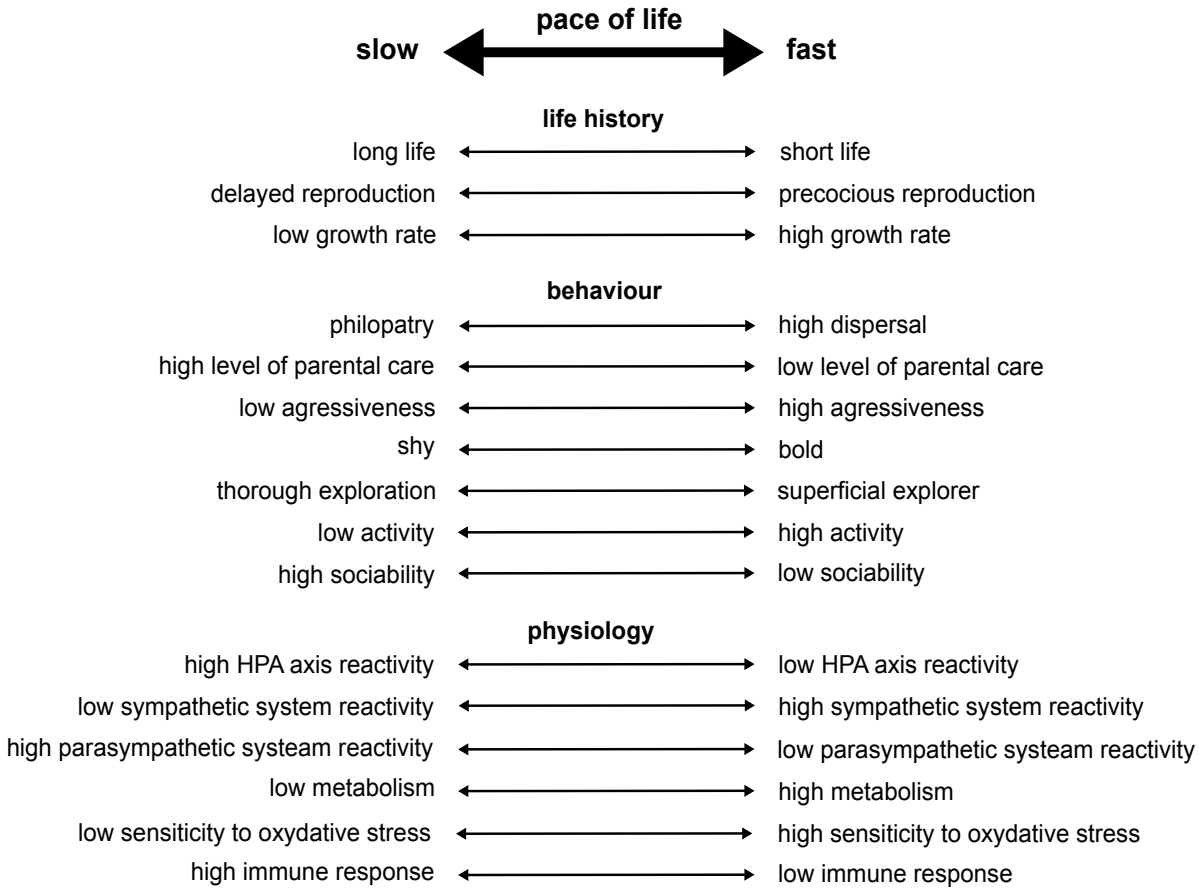


Figure 1.1: Schematic representation of the pace-of-life continuum (reproduced from Réale et al., 2010).

suites of traits are expected to be favored. In particular, in environments where the future is uncertain due to high rates of extrinsic mortality and morbidity, individuals should exhibit traits that favor short term gains. At its core, this phenomenon is thought to reflect the fundamental trade-off between current *vs* future reproduction, and to give rise to complex suites of interdependent traits, that broadly map onto a fast-slow continuum of life history strategies.

The approach described so far is very general in its scope. In the present thesis, we aim to show that it can lead to important insights, well beyond the illustrative case of the Trinidadian guppies. In particular, we would like to argue that the two core ideas that 1) harsh environments select fast strategies and 2) mechanisms of adaptive phenotypic plasticity allow individuals to match their local environmental conditions during development, can improve significantly our understanding of human interindividual variation for a variety of traits. Importantly, this perspective is not new, but instead constitutes the foundation of a rapidly growing field, that applies and extend evolutionary developmental theories to the study of human physiology, cognition and behaviour (Belsky, Steinberg, and Draper 1991; Chisholm et al. 1993; Del Giudice 2009; Ellis et al. 2009; Ellis and Del Giudice 2019; Figueredo et al. 2006; Frankenhuis, Panchanathan, and Nettle 2016). In the following section, I introduce some of the main results obtained to this day from such an approach. Then, I describe how the present thesis builds on this earlier work and outline how it attempts to adress some of the remaining questions.

## 1.6 Fast-slow strategies and psychosocial acceleration theory

The work reported in the present thesis arguably falls under the scope of the so-called psychosocial acceleration theory (Belsky, Steinberg, and Draper 1991; Nettle, Frankenhuis, and Rickard 2012). It is a developmental theory that draws on the life history principles reviewed in the previous sections, to predict that individuals developing under stressful, adverse conditions, will eventually adopt fast life history strategies. The theory is evolutionary in the sense that adverse events experienced

throughout ontogeny are viewed as cues that can be used by mechanisms of phenotypic plasticity to produce locally adpative phenotypes (Brumbach, Figueredo, and Ellis 2009; Ellis et al. 2009).

Indeed, they are thought to provide information to an individual about the survival and reproductive rates that are characteristic of its developing environment. Particularly severe or frequent adverse events would indicate an environment where large future gains are unlikely to be realized, gearing the organism towards the development of a fast life history strategy. Like in the case of the Trinidadian guppies, a fast strategy involves both a decrease in somatic effort, *i.e.*, a lower fraction of the individual's resources is devoted to guarantee its continued survival, and an increase in reproductive effort that focuses more on immediate reproduction (Figueredo et al. 2006).

Interestingly, adverse events can both play the role of external or internal cues (Nettle, Frankenhuis, and Rickard 2013). In the first case, early adversity provides a 'weather forecast' of the state of the environment in which the individual is likely to mature and reproduce. In the second case however, it is by having a direct impact on the state of the organism itself that early adversity affects its allocation decisions. Indeed, if the stressful conditions experienced in early life produce irreversible damages to an individual's soma, its future prospects are likely to be comparatively worse and a faster strategy should be adopted even though the quality of the environment might eventually improve (Rickard, Frankenhuis, and Nettle 2014). The two mechanisms are not mutually exclusive and might both contribute to the acceleration of life history strategies in response to early adversity.

Starting by investigating the effects of adverse conditions during childhood on the timing of maturity in girls (Belsky, Steinberg, and Draper 1991), the field has since generated a large body of empirical work that tests the association between harsh environments and traits thought relevant to the fast *vs* slow spectrum of life history strategies. In the reproductive domain, it has been shown multiple times that more family conflict and the absence of the father during childhood is associated with an early age at menarche in girls (Ellis 2004; Gaydosh et al. 2018; Moffitt et al. 1992;

Tither and Ellis 2008; but see also Sear, Sheppard, and Coall 2015). Somatic cues such as a low birthweight, or slow postnatal growth have also been found to predict earlier age at menarche (Adair 2001; Cooper et al. 1996; Sloboda et al. 2007). In addition to sexual maturity, birthweight and paternal involvment have also been found to predict age at first birth (Nettle, Coall, and Dickins 2009). Other aspects of reproductive strategies such as the number of offspring, sexual debut and focusing more on short *vs* long term relationships have also been investigated (*e.g.*, Guégan et al. (2001); Simpson et al. (2012); Xu, Norton, and Rahman (2018)).

Beyond the reproductive domain, somatic effort also seems to vary with the degree to which individuals have been exposed to adversity. Indeed, they are well known gradients in health preventive behaviours (Daniel Nettle 2011; Pepper and Nettle 2014; Stringhini 2010), with individuals with a low socioeconomic status being more likely to engage in behaviours that are detrimental to their health. Moreover, some of these differences in health related behaviours, have been linked to indidivduals' perception of the extrinsic mortality risk attached to their environment (Pepper and Nettle 2014). Other social gradients in behaviours not directly related to health or reproduction, such as financial decisions (Haushofer and Fehr 2014) or investment in education (Blanden 2004) have also been documented (see Pepper and Nettle (2017) for a review). Eventually, placing some of these empirical findings on an axis representing a continuum of fast-slow strategies (Figure 1.2), leads to a pattern of covariation that echoes strikingly with the Pace of Life Syndrome documented in the animal litterature (Figure 1.1; Réale et al. (2010)).

Thus, there is already a rich body of empirical and theoretical work that closes the gap between our understanding of life history variation in Trinidadian guppies and life history variation in human populations. Yet, open questions remain and several directions can be explored to further improve the value of the approach. In the present thesis, we focus on three of them: first, strengthening the empirical basis for the existence of coordinated life history strategies in humans; second, testing further the association of non health related or reproductive behaviours with the fast-slow continuum; and third, refining the theory of the adaptive value of fast life history

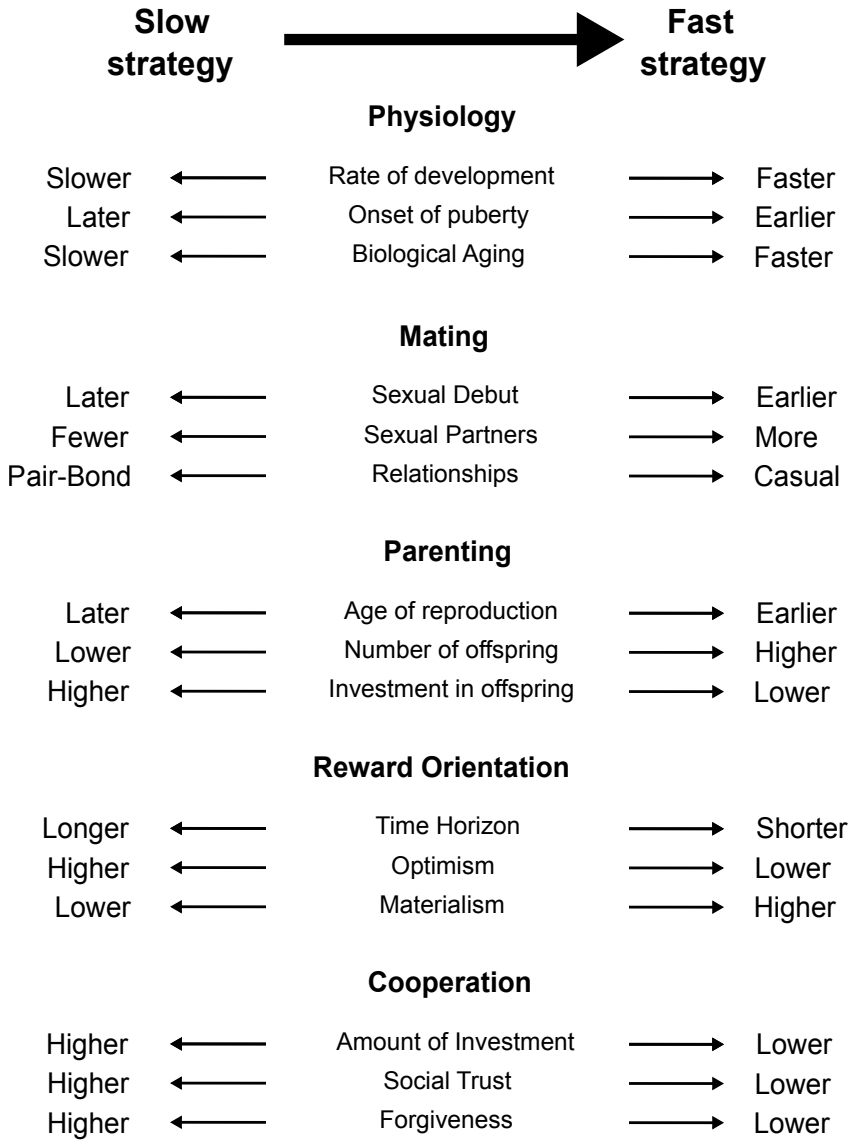


Figure 1.2: Schematic representation of the fast-slow continuum of human life history strategies (adapted from Griskievicius et al., 2013).

strategies that discount future gains.

The second chapter of this manuscript addresses the first point. Indeed, although taken altogether findings from individual studies suggest that humans do form coherent allocation strategies that map onto a fast-slow continuum, this basic premice has only rarely been tested on a single sample. Moreover, studies that assessed the covariation between traits across domains mostly did so using a psychometric approach on samples of adolescents or young adults (*e.g.*, Brumbach, Figueredo, and Ellis (2009); Figueredo et al. (2005)). Yet, the psychometric approach to human life history strategies has come under some scrutiny (Copping, Campbell, and Muncer (2014) ; Gruijters and Fleuren (2018)). Hence, we report a study which used more direct measures of health and reproductive bahaviours in a representative sample of the French population. Our main goal was to test the core hypothesis that higher reproductive effort covaries with less somatic effort, and that in accordance to psychosocial acceleration theory, individuals that had been exposed to more adversity during childhood would also have adopted faster life history strategies.

In the third chapter, we investigate how interindividual differences in prosociality might relate to the fast-slow continuum and psychosocial acceleration theory. Specifically, we focus on interindividual variation in social trust and its association with socioeconomic status and other life history indicators. The issue of whether greater prosociality should be expected in affluent or harsh environments is still debated both on empirical and theoretical grounds (*e.g.*, Amir, Jordan, and Rand (2018); Daniel Nettle, Colléony, and Cockerill (2011); Robinson and Piff (2017)). On one hand, some authors argue that to cope successfully with the harshness of their environment, people are forced to increase their level of cooperation. On the other hand, because harsh environments favor strategies geared towards short term benefits, cooperation should decrease as it corresponds to a long term strategy which foregoes the immediate benefits of defection. Here, we report a series of studies on social trust that tend to support the latter hypothesis.

Lastly, in the fourth chapter we develop a formal life history model that allows us to explore variation in delay discounting. Indeed, the distinction between fast-

slow strategies suggests that most of the observed covariation between traits might reflect differences in the degree at which future outcomes are discounted. Although verbal explanations of the fast-slow continuum in terms of delay discounting are common, formal models are still largely lacking (Mathot and Frankenhuis 2018). Furthermore, present oriented strategies are generally predicted to be favored under harsh conditions, because in such environments future outcomes are more uncertain. Yet, our model highlights that changes in opportunity costs associated to differences in capital, should be considered along variation in uncertainty levels to explain delay discounting and the origins of fast-slow strategies.

# Chapter 2

# Fast-slow strategies in humans: reproductive behaviour, somatic effort and early adversity

## 2.1 Objectives and summary

Numerous studies have shown an association between some measure of adversity and reproductive or health-related behaviours. Overall, they tend to find that exposure to higher levels of adversity predicts a decrease in health protective behaviours and a faster reproductive strategy, often highlighting unique effects of the conditions experienced early in life. Yet, as mentioned in the general introduction, such studies only rarely look at the broad pattern of covariation between somatic and reproductive effort, although it is arguably the core feature of the fast-slow constinuum (Figure 1.2).

In this chapter, we report the results from a cross-sectional study that aims to fill part of this gap, using data from a survey of a representative sample of the French population. Specifically, we fitted on this data a structural equation model, including in the measurement part a latent factor that captured the common variance between indicators of somatic investment (proportion of life spent smoking daily, body mass index, self-reported health status and self-reported efforts in looking after one's health), and reproductive strategy (age at first birth, sexual debut, number

of offspring and number of short-term relationships). The latent factor was itself regressed on a general measure of the level of adversity experienced during their childhood. The latter was calculated from participants' answers to a questionnaire, composed of a list of items gathered from the litterature, all previously used to capture several dimensions of the harshness of people's environment. This measure of environmental harshness was computed using two distinct methods: 1) by summing the z-scores obtained for the different scales, and 2), by calculating a weighted sum, where the weights were determined explicitly from the data. The latter approach allowed us to compare the relative contributions of different adverse events to the association with the latent life history factor. Overall, we found that lower somatic effort covaried with a more short-term and early reproductive strategy (but not with a larger number of children). This pattern was also associated with a higher level of harshness experienced during childhood. However, measuring harshness using weights computed explicitly from the data only highlighted the importance of having been exposed to violence during childhood. Despite some important limitations inherent to the design of the study, we conclude that the evidence obtained in this study is consistent with the existence of coordinated life history strategies in humans.

The remainder of this chapter corresponds to a paper that has been published in the journal *Evolution and Human Behaviour*.

# 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**

## 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.**

## 2.2 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 and Sassi 2008). Adolescent childbearing has also been a major concern for policymakers because of the potential health costs and loss of education and labor opportunities for teenage mothers (Hoffman, Foster, and 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 et al. 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 trade-offs among traits and environmental factors that affect mortality and fertility rates (Stearns 2000). Drawing

on these fundamental insights, evolutionary psychologists started to investigate how specific adversity events occurring during ontogeny, could be used by individuals as cues to adjust their strategies (Belsky, Steinberg, and Draper 1991; Chisholm et al. 1993). This led to a fruitful body of research often designated by the name 'psychosocial acceleration theory' (Nettle, Frankenhuis, and 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, Frankenhuis, and 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, Frankenhuis, and Nettle (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, and 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 et al. 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 et al., n.d., 2010; Moffitt et al. 1992; Tither and Ellis 2008) and more sexual partners (Simpson et al. 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, and 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 and 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 (Daniel Nettle 2010). Indeed, subjective socioeconomic standing is associated with reported effort in looking after one's health in a crosssectional sample of the American population, and the effect of subjective socioeconomic position is

fully mediated by perceived extrinsic mortality risks (Pepper and 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 2012; Belsky et al. 2010; Brumbach, Figueredo, and Ellis 2009; Hartman et al. 2017; 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, Steinberg, and Draper 1991; Figueredo et al. 2005, 2006; Griskevicius et al. 2011; Réale 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, and Robson 2003). To our knowledge, only one study (Brumbach, Figueredo, and Ellis 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 and 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 2012; Ellis et al. 2009; Frankenhuis, Panchanathan, and Nettle 2016).

## 2.3 Methods

### 2.3.1 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 nonresponse. 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.

### 2.3.2 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).

#### 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 et al. 2012; Daniel Nettle and 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 (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.

## Reproductive strategy

Participants' reproductive strategy was assessed using four items from the literature (Nettle, Coall, and Dickins 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.

Participants' somatic strategy was also assessed using four items previously used in the literature (Pepper and 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.

### 2.3.3 Analysis

All statistical analyses were carried out in R 3.0.3 (https://www.rproject. 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 and 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 and 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, Figueredo, and Ellis (2009); Figure 2.1 A), harshness was also modelled as an unknown weights composite in a subsequent SEM to gain these inferential benefits (Figure 2.1 B).

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 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 et al. 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 Figure 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).

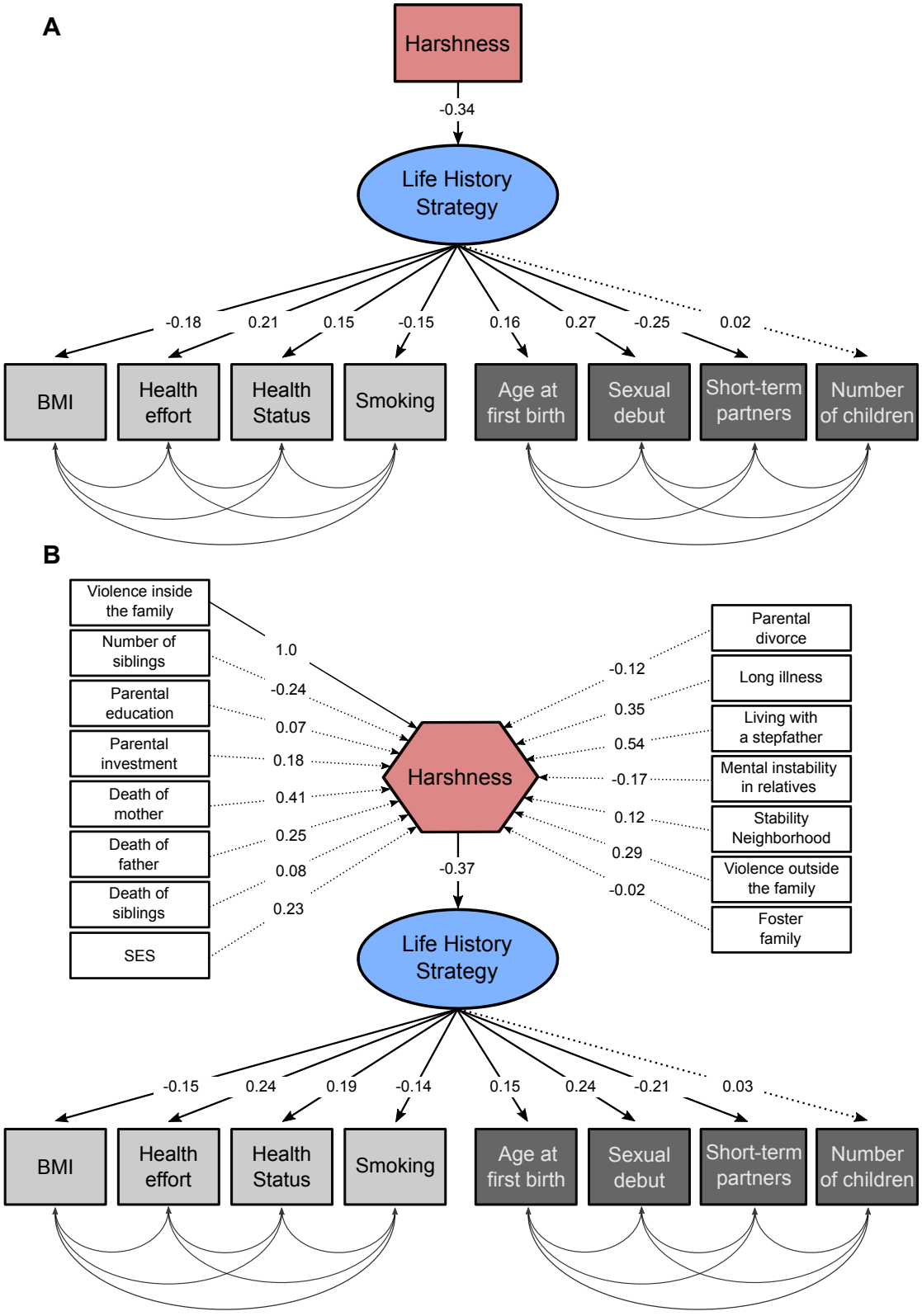


Figure 2.1: Path diagrams of the structural models fitted showing for clarity only the structure of the residual covariances in the measurement model and standardized regression weights for harshness and reflective indicators. Significant paths at the 5% level are represented with a plain arrow **A.** Model 1- Harshness is modelled as a weighted sum **B.** Model 2 - Harshness is modelled as a latent composite.

Table 2.1: Descriptive statistics and correlations from self-report data (n=1015).

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) | (10) |
|---|---|---|---|---|---|---|---|---|---|---|
| BMI (1) | - | | | | | | | | | |
| Health status (2) | -0.2 | - | | | | | | | | |
| Health effort (3) | -0.12 | 0.18 | - | | | | | | | |
| Smoking (4) | 0.04 | -0.09 | -0.12 | - | | | | | | |
| Number of children (5) | 0.13 | 0 | 0.01 | -0.05 | - | | | | | |
| Age at first birth (6) | -0.09 | 0.13 | 0 | -0.05 | -0.37 | - | | | | |
| Sexual debut (7) | -0.03 | 0.04 | 0.06 | -0.2 | 0.04 | 0.18 | | | | |
| Short-term partners (8) | 0.04 | 0 | -0.1 | 0.19 | -0.07 | 0.1 | - | - | | |
| Harshness (9) | 0.05 | -0.14 | -0.07 | 0.12 | 0.04 | -0.1 | 0.04 | 0.04 | - | |
| Age (10) | 0.11 | -0.14 | 0.21 | 0.04 | 0.17 | -0.15 | 0.07 | 0.07 | -0.06 | - |
| Mean | 26.04 | 2.75 | 68.98 | 0.21 | 2.15 | 25.66 | 6.21 | 6.21 | -0.06 | 52.52 |
| SD | 5.15 | 0.7 | 19.01 | 0.25 | 0.96 | 4.86 | 12.42 | 12.42 | 12.36 | 14.33 |
| Range | 13.6–58.8 | 1–4 | 0–100 | 0–0.91 | 1–7 | 14–56 | 0–160 | 0–160 | -17.2–66.6 | 19–87 |

## 2.4 Results

### 2.4.1 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 2.1 reports descriptive statistics and the correlation matrix for the variables included in the first SEM (Figure 2.1 A). An extended table including the individual harshness indicators used in the second SEM (Figure 2.1 B) 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 Figure 2.1 on the data. Fit indices and parameters estimates are reported separately for each model in the following sections.

Table 2.2: Statistical and practical fit indices for the structural equation models.

| Model | $\chi^2$ | df | p-value | RMSEA | SRMR | CFI |
|---|---|---|---|---|---|---|
| Model1 | 58.6 | 15 | ¡0.001 | 0.046 | 0.03 | 0.93 |
| Model2 | 191 | 113 | ¡0.001 | 0.026 | 0.028 | 0.83 |

### 2.4.2 SEM with harshness as a sum of z-scores

## Model fit

Table 2.2 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, and 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.

## 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' (Figure 2.1 A; 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.

#### 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.

### 2.4.3 SEM with harshness as a latent composite

#### 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.

## 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 (Figure 2.1 B). 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).

## 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 $c$=$-0.37$, $p < 0.001$; Figure 2.1 B, 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%.

## 2.5 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 and 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 and 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 (W. E. Frankenhuis and Panchanathan 2011b, 2011a). 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 and 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 2012; Belsky and Pluess 2009). For instance, there is empirical evidence that the effects of harshness on female pubertal development are genetically moderated (Hartman, Widaman, and 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 crosssectional 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, Figueredo, and Ellis 2009; Hartman et al. 2017; 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 and Liu 2014), attention deficits (de Rooij et al. 2010), anti-social behaviours (Neugebauer 1999), lower educational level (Lavy, Schlosser, and Shany 2016), or lower probability

of being married and getting a job (Almond et al. 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, Panchanathan, and Nettle 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, and 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, Avery, and Trenell 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.

## 2.6 Supplementary information

# Appendix A – Questionnaire

## General instructions

You will now fill out the last questionnaire in session 1. Some of the questions are personal. You are asked these questions so that participants' responses can be analyzed according to their individual profile and history. We wish to remind you that all of the responses we collect will be analysed completely anonymously. If you would prefer to not respond to some of the questions, you will simply have to tick the box « I don't want to answer ».

## Your Childhood (1/2)

**1. Sex :**
Female / Male

**2. Birth year of your mother?**
Please select one of the following answers: (*Answers from 1908 to 1990)*

**3. Is your mother still alive?**
Yes / No

**4. Year of death**
***Answer this question only if you answered 'No' to « Is your mother still alive? »***
Please write your answer here:

**5. Birth year of your father?**
Please select one of the following answers: (*Answers from 1908 to 1990)*

**6. Is your father still alive?**
Yes / No

**7.Year of death**
***Answer this question only if you answered 'No' to « Is your father still alive? »***
Please write your answer here:

**8. How many children did your mother have?**
Please select one of the following answers:
I do not know 1 2 3 4 5 6 7 8 9 10 and more

**9. Among your mother's children you are the:**
Please select one of the following answers:
I do not know ◯ 1st ◯ 2nd ◯ 3rd ◯ 4th ◯ 5th ◯ 6th ◯ 7th ◯ 8th ◯ 9th ◯ 10th or more

**10. How many children did your father have?**
Please select one of the following answers:
I do not know 1 2 3 4 5 6 7 8 9 10 and more

**11. Among your father's children you are the:**
Please select one of the following answers:
I do not know ○1st ○2nd ○3rd ○4th ○5th ○6th ○7th ○8th ○9th ○10th or more

**12. Did your parents get divorced or separated?**
Yes / No

**12b. How old were you?**
***Answer this question only if you answered 'Yes' to « Did your parents get divorced or separated? »***
Please write your answer here:

**13. Have you ever lived with a step-father?**
***Answer this question only if you answered 'Yes' to « Did your parents get divorced or separated? »***
Please select one of the following answers:
Yes / No

**13b. From what age on?**
***Answer this question only if you answered 'Yes' to « Have you ever lived with a step-father? »***
Please write your answer here:

**14. Were you ever placed in an institution or in a foster family?**
Yes / No

**15. My parents always seemed to care about what I was doing.**
*Scale from 1 to 100*

**16. My father was always there when I needed him.**
*Scale from 1 to 100*

**17. My mother was always there when I needed her.**
*Scale from 1 to 100*

# Your Childhood (2/2)

**18. When I was growing up, someone in my house was always yelling at someone else.**
*Scale from 1 to 100*

**19. Some of the punishments I received when I was a child now seem too harsh to me.**
*Scale from 1 to 100*

**20. I guess you could say that I wasn't treated as well as I should have been at home.**
*Scale from 1 to 100*

**21. I consider that during my childhood, I was a victim of:**
Please select all appropriate answers :
Physical abuse
Sexual abuse
Psychological abuse
No form of abuse
I don’t want to answer

**21b. These episodes were caused by:**
***Answer this question only if you answered 'Physical abuse' or 'Psychological abuse ' or 'Sexual abuse' to the question «* I consider that during my childhood, I was a victim of *»***
Please select all appropriate answers:
One or several people in my family
One or several people outside my family

**22. My family usually had enough money for things when I was growing up.**
*Scale from 1 to 100*

**23. I grew up in a relatively wealthy neighborhood.**
*Scale from 1 to 100*

**24. I felt relatively wealthy compared to the other kids in my school.**
*Scale from 1 to 100*

**25. During your childhood, did you live with one or several people who were:**
Please select all appropriate answers:
alcoholic
violent
depressed
who suffered from a mental disorder
who regularly took street drugs
who sometimes had issues with the judicial system
who had spent time in prison
none of the above
I don’t want to answer

**26. From your birth until you were 7 years old, did you suffer from a long disease that required a hospitalisation?**
Please select one of the following answers:
Yes / No

**27. From your birth until you were 7 years old, how many times did you move?**
Please write your answer here:

**28. From your birth until you were 7 years old, how many times did you change schools?**
Please write your answer here:

**29. From your birth until you were 7 years old, how much did your father take care of you?**
Please select one of the following answers:
He left my mother taking care of us.

He took care of us, but less than my mother.
He took care of us as much as my mother.
He took more care of us than my mother.
This question does not apply to me.

**30. From your birth until you were 7 years old, did one of your sibling die?**
Yes / No

# Your adult life and family

**31. How tall are you (in cm)?**
Please write your answer here: *(number between 65 and 220)*

**32. How heavy are you (in kg)?**
Please write your answer here:

**33. Have you had children?**
Yes / No

**33b-1. How many:**
***Answer this question only if you answered 'Yes' to « Have you had children? »***
Please write your answer here: *(number between 1 and 19)*

**Date of Birth for each child: (***number between 1920 and 2014)*

# Your health and safety

**34. How much effort do you make to look after your health and ensure your safety these days?**
*Scale from 1 to 100*

**35. How is your health in general?**
Please select one of the following answers:
Bad
Acceptable
Good
Excellent

**36. In total, during how many years did you smoke daily or almost daily?**
Please write your answer here:

**We are now going to ask you some questions about your voluntary sexual experiences (these questions do not apply to non consensual experiences you may have had):**

**37. Have you ever had a sexual intercourse?**
Please select one of the following answers:
Yes / No / I don’t want to answer

**38. How old were you when you had your first sexual intercourse?**
***Answer this question only if you answered 'Yes' to « Have you ever had sexual intercourse? »***
Please write your answer here:

**39. With how many different partners have you had sexual intercourse without having an interest in a long-term committed relationship with this person?**
***Answer this question only if you answered 'Yes' to « Have you ever had sexual intercourse? »***
Please write your answer here:

# Appendix B

**Fig. B.1** Distribution of self-reported general health status (N=1015)

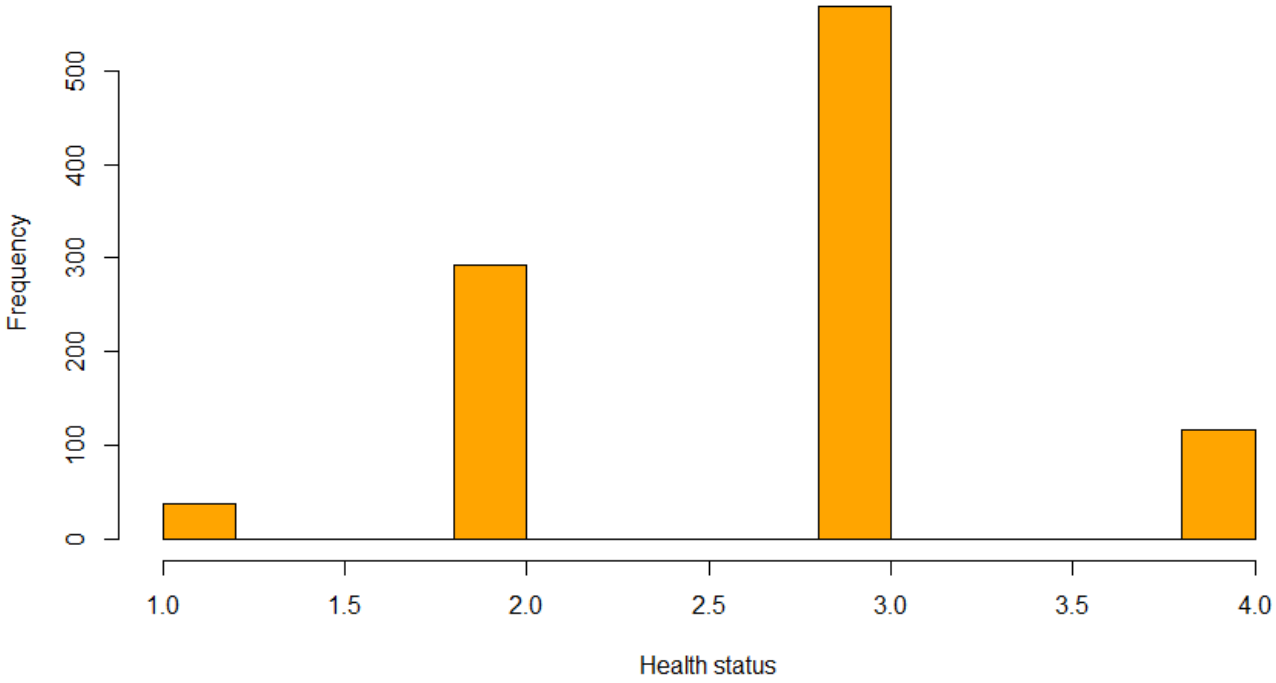

**Table B.1** Descriptive statistics and correlations from self-reported data (N=1015).

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) | (10) | (11) | (12) | (13) | (14) | (15) | (16) | (17) | (18) | (19) | (20) | (21) | (22) | (23) | (24) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Smoking (1) | 1.00 | | | | | | | | | | | | | | | | | | | | | | | |
| Health status (2) | **-0.09** | 1.00 | | | | | | | | | | | | | | | | | | | | | | |
| Health effort (3) | **-0.12** | **0.18** | 1.00 | | | | | | | | | | | | | | | | | | | | | |
| BMI (4) | 0.04 | **-0.20** | **-0.12** | 1.00 | | | | | | | | | | | | | | | | | | | | |
| Number of children (5) | -0.05 | -0.00 | 0.01 | **0.13** | 1.00 | | | | | | | | | | | | | | | | | | | |
| Age at first birth (6) | -0.05 | **0.13** | -0.00 | **-0.09** | **-0.37** | 1.00 | | | | | | | | | | | | | | | | | | |
| Sexual debut (7) | **-0.20** | 0.04 | **0.06** | -0.03 | 0.04 | **0.18** | 1.00 | | | | | | | | | | | | | | | | | |
| Short-term partners (8) | **0.19** | -0.00 | -0.10 | **0.04** | **-0.07** | **0.10** | **-0.22** | 1.00 | | | | | | | | | | | | | | | | |
| Death of mother (9) | 0.08 | -0.04 | 0.01 | 0.02 | -0.01 | **-0.11** | -0.03 | -0.03 | 1.00 | | | | | | | | | | | | | | | |
| Death of father (10) | 0.01 | -0.02 | -0.04 | **0.09** | 0.07 | **-0.06** | -0.04 | **0.07** | **0.10** | 1.00 | | | | | | | | | | | | | | |
| Death of siblings (11) | 0.03 | -0.03 | 0.04 | 0.03 | 0.01 | -0.02 | 0.10 | -0.01 | -0.01 | -0.03 | 1.00 | | | | | | | | | | | | | |
| Parental divorce (12) | **0.07** | 0.02 | -0.04 | -0.03 | **-0.07** | 0.01 | **-0.12** | 0.02 | 0.01 | 0.02 | 0.02 | 1.00 | | | | | | | | | | | | |
| Living with a stepfather (13) | **0.08** | -0.06 | -0.05 | 0.01 | **-0.08** | -0.01 | **-0.09** | -0.03 | -0.02 | 0.02 | 0.00 | **0.64** | 1.00 | | | | | | | | | | | |
| Foster family (14) | **0.08** | -0.01 | 0.01 | 0.04 | -0.02 | -0.02 | -0.03 | 0.02 | **0.13** | **0.06** | -0.01 | **0.12** | **0.16** | 1.00 | | | | | | | | | | |
| Violence in the family (15) | **0.13** | **-0.18** | **-0.07** | 0.01 | 0.05 | **-0.05** | -0.09 | 0.03 | 0.05 | -0.03 | 0.03 | **0.14** | **0.17** | **0.22** | 1.00 | | | | | | | | | |
| Violence outside the family (16) | **0.07** | -0.05 | -0.02 | -0.04 | -0.04 | 0.02 | -0.03 | 0.04 | 0.04 | 0.02 | **0.07** | **0.13** | **0.11** | **0.24** | **0.16** | 1.00 | | | | | | | | |
| Mental instability in relatives (17) | **0.04** | **-0.04** | 0.00 | -0.03 | -0.02 | -0.03 | **-0.11** | 0.04 | 0.03 | 0.00 | **0.05** | **0.24** | **0.24** | **0.17** | **0.39** | **0.16** | 1.00 | | | | | | | |
| Long illness (18) | -0.00 | -0.04 | -0.03 | **0.07** | 0.00 | -0.01 | **-0.10** | **0.14** | 0.03 | 0.05 | 0.01 | -0.02 | -0.04 | -0.03 | 0.04 | -0.03 | **0.05** | 1.00 | | | | | | |
| SES (19) | 0.00 | **-0.08** | **-0.06** | 0.06 | **0.05** | **-0.10** | 0.02 | **-0.05** | 0.01 | **0.06** | 0.01 | 0.01 | -0.00 | 0.01 | 0.06 | 0.02 | **0.12** | 0.02 | 1.00 | | | | | |
| Parental investment (20) | **0.07** | **-0.10** | **-0.14** | 0.06 | 0.05 | **-0.08** | **-0.07** | 0.02 | **0.09** | **0.08** | 0.01 | **0.31** | **0.27** | **0.22** | **0.43** | **0.17** | **0.31** | 0.03 | **0.20** | 1.00 | | | | |
| Parental education (21) | 0.05 | **-0.17** | **-0.08** | 0.03 | 0.05 | **-0.07** | -0.04 | 0.00 | 0.03 | -0.00 | 0.02 | **0.13** | **0.15** | **0.16** | **0.59** | **0.19** | **0.35** | 0.02 | **0.16** | **0.52** | 1.00 | | | |
| Stability neighborhood | 0.05 | -0.04 | -0.04 | -0.01 | 0.02 | -0.04 | -0.02 | 0.05 | 0.05 | 0.02 | 0.05 | **0.22** | **0.20** | **0.11** | **0.17** | 0.07 | **0.12** | **0.04** | -0.04 | **0.23** | **0.17** | 1.00 | | |

| | | | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| (22) | | | | | | | | | | | | | | | | | | | | | | | | |
| Number of siblings (23) | 0.01 | 0.01 | **0.08** | 0.05 | **0.14** | **-0.10** | **0.07** | -0.01 | 0.02 | 0.07 | **0.18** | -0.07 | -0.07 | 0.04 | -0.01 | 0.02 | 0.01 | -0.02 | **0.20** | 0.07 | **0.06** | **-0.04** | 1.00 | |
| Age (24) | 0.04 | **-0.14** | **0.21** | **0.11** | **0.17** | **-0.15** | **0.17** | 0.07 | -0.00 | 0.01 | 0.03 | **-0.19** | **-0.14** | 0.02 | -0.04 | -0.03 | **-0.13** | 0.03 | **0.06** | -0.06 | -0.01 | -0.03 | **0.15** | 1.00 |
| Mean | 0.21 | 2.75 | 69.0 | 26.0 | 2.15 | 25.7 | 18.6 | 6.21 | 0.03 | 0.07 | 0.05 | 0.12 | 0.06 | 0.02 | 0.14 | 0.03 | -0.11 | 0.03 | -43.0 | -75.2 | 29.4 | 0.97 | 3.42 | 52.5 |
| SD | 0.25 | 0.70 | 19.0 | 5.15 | 0.96 | 4.86 | 3.13 | 12.4 | 0.17 | 0.26 | 0.22 | 0.33 | 0.23 | 0.15 | 0.34 | 0.17 | 3.57 | 0.18 | 24.0 | 24.6 | 25.4 | 1.15 | 1.83 | 14.8 |
| Range | 0-0.91 | 1-4 | 0-100 | 14-59 | 1-7 | 14-56 | 10-61 | 0-160 | 0-1 | 0-1 | 0-1 | 0-1 | 0-1 | 0-1 | 0-1 | 0-1 | -1.3-37.6 | 0-1 | -100--1 | -100--1 | 1-100 | 0-11.5 | 1-10 | 19-87 |

Values in bold mean $p<0.05$

**Table B.2** Parameter estimates, standard errors and their significance for measurement and structural coefficients of model 1.

| Parameter | value | se | z | *p* | standardized |
|---|---|---|---|---|---|
| LHS → BMI | -0.85 | 0.34 | -2.48 | 0.01 | -0.18 |
| LHS → Health effort | 3.76 | 1.33 | 2.83 | 0.005 | 0.21 |
| LHS → Health status | 0.21 | 0.05 | 4.16 | <0.001 | 0.15 |
| LHS → Smoking | -0.14 | 0.02 | -6.54 | <0.001 | -0.15 |
| LHS → Number of children | 0.03 | 0.04 | 0.62 | 0.54 | 0.02 |
| LHS → Age at first birth | 0.79 | 0.23 | 3.50 | <0.001 | 0.16 |
| LHS → Sexual debut | 0.92 | 0.14 | 6.48 | <0.001 | 0.27 |
| LHS → Short-term partners | -2.92 | 0.83 | -3.52 | <0.001 | -0.25 |
| Harshness → LHS | -0.06 | 0.01 | -5.94 | <0.001 | -0.34 |
| Age → Number of children | 0.01 | 0.002 | 5.03 | <0.001 | 0.11 |
| Age → Age at first birth | -0.05 | 0.01 | -4.57 | <0.001 | -0.14 |
| Age → Sexual debut | 0.04 | 0.007 | 5.29 | <0.001 | 0.14 |
| Age → Short-term partners | 0.07 | 0.03 | 2.70 | 0.007 | 0.08 |
| Age → Health status | -0.007 | 0.001 | -4.85 | <0.001 | -0.07 |
| Age → BMI | 0.04 | 0.01 | 3.50 | <0.001 | 0.12 |
| Age → Health effort | 0.28 | 0.04 | 6.8 | <0.001 | 0.21 |
| Age → Smoking | 0.001 | 0.001 | 1.77 | 0.08 | 0.014 |
| $COV_{BMI/Health\ effort}$ | -10.437 | 3.70 | -2.82 | 0.005 | -0.11 |
| $COV_{BMI/Health\ status}$ | -0.47 | 0.14 | -3.29 | 0.001 | -0.14 |
| $COV_{BMI/Smoking}$ | -0.09 | 0.06 | -1.47 | 0.14 | -0.09 |
| $COV_{Health\ effort/Health\ status}$ | 1.89 | 0.57 | 3.31 | 0.001 | 0.16 |
| $COV_{Health\ status/Smoking}$ | 0.02 | 0.01 | 1.73 | 0.08 | 0.13 |
| $COV_{Health\ effort/Smoking}$ | -0.02 | 0.25 | -0.08 | 0.94 | -0.01 |
| $COV_{Number\ of\ children/Age\ at\ first\ birth}$ | -1.64 | 0.16 | -10.05 | <0.001 | -0.36 |

| | | | | | |
|---|---|---|---|---|---|
| $COV_{\text{Number of children/Sexual debut}}$ | -0.002 | 0.01 | 0.02 | 0.99 | 0.001 |
| $COV_{\text{Number of children/Short-term partners}}$ | -0.86 | 0.47 | -1.82 | 0.07 | -0.07 |
| $COV_{\text{Age at first birth/Sexual debut}}$ | 2.29 | 0.57 | 4.03 | <0.001 | 0.16 |
| $COV_{\text{Age at first birth/Short-term partners}}$ | 9.18 | 2.62 | 3.50 | <0.001 | 0.16 |
| $COV_{\text{Sexual debut/Short-term partners}}$ | -6.17 | 1.61 | -3.84 | <0.001 | -0.17 |

**Table B.3** Parameter estimates, standard errors and *p*-values for the weights of Model 2.

| parameter | value | se | Z | *p* | standardized |
|---|---|---|---|---|---|
| Harshness → LHS | -0.25 | 0.07 | -3.88 | <0.001 | -0.37 |
| Violence inside the family → LHS[a] | -0.73 | 0.19 | -3.88 | <0.001 | -0.24 |
| SES → Harshness | 0.23 | 0.22 | -1.07 | 0.29 | 0.23 |
| Parental investment → Harshness | 0.18 | 0.25 | -0.72 | 0.48 | 0.18 |
| Stability neighborhood → Harshness | 0.12 | 0.21 | 0.57 | 0.57 | 0.12 |
| Parental education → Harshness | 0.07 | 0.26 | 0.28 | 0.78 | 0.07 |
| Number of siblings → Harshness | -0.24 | 0.20 | -1.19 | 0.24 | -0.24 |
| Death of father → Harshness | 0.25 | 0.20 | 1.24 | 0.21 | 0.25 |
| Death of mother → Harshness | 0.41 | 0.21 | 1.92 | 0.06 | 0.41 |
| Death of siblings → Harshness | 0.08 | 0.21 | 0.36 | 0.72 | 0.08 |
| Living with a stepfather → Harshness | 0.54 | 0.29 | 1.88 | 0.06 | 0.29 |
| Parental divorce → Harshness | -0.12 | 0.28 | -0.41 | 0.69 | -0.12 |
| Mental instability in relatives → Harshness | -0.17 | 0.19 | -0.92 | 0.35 | -0.17 |
| Violence outside the family → Harshness | 0.29 | 0.23 | 1.27 | 0.20 | 0.29 |
| Long illness → Harshness | 0.35 | 0.23 | 1.53 | 0.13 | 0.35 |
| Foster family → Harshness | -0.016 | 0.17 | -0.09 | 0.93 | -0.02 |

[a] All values for this effect are computed from the partially reduced form of model 2

**Table B.4** Summary statistics for the cross-validation analysis (n=1000, 50/50).

| | **Standardized coefficient (mean)** | **Standardized coefficient (sd)** | **z-statistic** |
|---|---|---|---|
| Harshness | -0.19 | 0.08 | -2.51 |
| Death of mother | 0.20 | 0.16 | 1.55 |
| Death of father | 0.15 | 0.14 | 1.07 |
| Death of siblings | -0.00 | 0.17 | 0.01 |
| Parental divorce | -0.05 | 0.22 | -0.31 |
| Living with a stepfather | 0.27 | 0.22 | 1.53 |
| Foster family | -0.03 | 0.12 | -0.35 |
| Violence in the family | 0.49 | 0.18 | 3.12 |
| Violence outside the family | 0.13 | 0.15 | 0.91 |
| Mental instability in relatives | -0.05 | 0.18 | -0.48 |
| Long illness | 0.22 | 0.21 | 1.49 |
| SES | 0.08 | 0.19 | 0.60 |
| Parental investment | 0.09 | 0.19 | 0.54 |
| Parental education | 0.05 | 0.20 | 0.34 |
| Stability neighborhood | 0.07 | 0.13 | 0.48 |
| Number of siblings | -0.11 | 0.15 | -0.82 |

# Chapter 3

# Extending the fast-slow continuum: childhood harshness and social trust

## 3.1 Objectives and summary

In the previous chapter, we have found evidence that indicators of people's somatic and reproductive strategies covary to some extent, and that individuals' position on the spectrum of life history strategies is associated to the level of adversity experienced during childhood. As discussed in the general introduction however, a more diverse array of behaviours is thought to map onto the fast-slow continuum (Figure 1.1; Figure 1.2). In particular, individual differences in prosociality have been argued to be linked to people's broader life history strategies. However, there is not a strong consensus yet about the direction of the association. This is in part because plausible theoretical arguments can be advanced to support both an increase or a decrease in prosociality in harsher environments, and because the empirical evidence on the link between cooperation and deprivation is not conclusive.

Nevertheless, recent work in economics has provided strong support for a causal link between environmental harshness, particularly when experienced during childhood and a reduction in reported levels of social trust in adulthood. The best evidence of this relationship comes from a natural experiment in post World War 2 Germany,

that allowed economists to quantify the impact of exogenous variations in caloric input during childhood on self-reports of social trust later in life. Other evidence in the psychology literature also demonstrates that resource scarcity experienced in utero also has an impact on adult social trust. Hence, a clearer picture is starting to emerge, at least once the focus is restricted to social trust.

In this chapter, we first report a series of studies that aimed to extend this work by further testing the external validity of this relationship between early deprivation and levels of social trust in adulthood, and by looking at the association between the fast-slow continuum and individual differences in social trust. To this aim, we first analyzed data from a large independent European survey (European Values Study), going beyond a single country approach to test the robustness of the association. Then, we conducted an online experiment to assess whether the relationship between early deprivation and lower social trust would arise not only in self-reports, but also when measuring people's actual behaviours in a trust game. We also tested directional hypotheses about the idea that depending on their socioeconomic status, individuals would not exhibit the same sensitivity to the stakes of the games and to the probability that they would see their prosocial acts reciprocated. Thirdly, we used the data available from our representative sample of the French population to test the idea that diminished social trust is part of a broader behavioural constellation that appears in response to environmental harshness.

Overall, supporting the previous findings obtained in economics, we found repeatedly across studies a negative association between deprivation and individual differences in social trust. Importantly, we always found a unique association between childhood socioeconomic status and adult social trust. Furthermore, a higher level of social trust was also associated with indicators of a slow strategy, adding new evidence on its link with the fast-slow continuum. Regarding participants' behaviours in the trust games however, we did not find any differences in participants' sensitivity to the parameters of the games across levels of SES.

All these findings are reported in a paper in preparation, that constitutes the first part of the present chapter.

The second part of the chapter on the other hand, corresponds to the preliminary results of a preregistered study on the association between harshness in the first years of life and children's level of social trust at 11 years old. Since the association between early life conditions and social trust in adulthood appears quite robust across different datasets, we were interested in testing whether the same relationship would be already present in late childhood.

Our sample came from the EDEN mother-child cohort, which provides longitudinal data on numerous aspects of children's environment, as well as the level of parental care they received at various point in time during their childhood. This gave us the opportunity to look at the role of parental investment in mitigating or accentuating the potential effects of a harsh environment. Indeed, we hypothesized that the level of parental care received by the child would be both a mediator of the effect of harshness (harsher environments decreasing the level of care received, which would in turn decrease social trust) and a moderator (more care received would attenuate the impact of the harshness of the environment on the child's level of trust).

Interestingly, at this stage of the analysis we found no evidence for a decrease in social trust with harsher conditions during the first years of life. Furthermore, we found no support either for our hypotheses on the impact of parental care.

These preliminary results are briefly introduced and discussed in the form of a short note.

# Converging evidence for the lasting association between childhood harshness and social trust

**Hugo Mell, Lou Safra, Perline Demange, Yann Algan, Nicolas Baumard, Coralie Chevallier**

## Abstract

**Social trust, i.e. the general expectation that people usually do not try to exploit each other, is a belief central to social decision-making processes. The degree to which it is held can vary considerably between individuals and societies, deeply affecting a range of prosocial behaviours. Across empirical studies, higher socioeconomic status (SES) has been identified as an important predictor of greater social trust at the individual level. Although this association has mostly been reported for measures of SES taken in adulthood, recent studies in psychology and economics have found unique effects of harshness experienced during childhood on social trust assessed decades later. Some of the evidence reported shows strong internal validity, suggesting a causal link between childhood deprivation and adult social trust. Yet, studies assessing whether this relationship holds across settings remain relatively rare, leaving the issue of its external validity comparatively open. Here, we report a series of three studies, providing overall support for a robust association between childhood deprivation and social trust, beyond the influence of current SES. The first study tested whether higher childhood and current SES are uniquely associated with greater social trust, based on a selection of items from the European Values Study (EVS), an independent large-scale survey of 46 European countries (N=66281). The second study investigates whether the same relationship extends to an experimental measure of trust (will-**

**ingness to play in a modified trust game). Lastly, looking at variations in social trust from the perspective of psychosocial acceleration theory, the third study examines whether it covaries with markers of fast-slow life history strategies in response to harshness experienced during childhood.**

## 3.2 Introduction

The degree to which people trust others in a society can lead to a range of important political, economic and social outcomes. For example, individual differences in social trust are reflected in different attitudes towards the welfare state (Algan, Cahuc, and Sangnier 2016). There is also converging evidence in the economic literature that an increase in social trust can boost a country's economic growth (Bjørnskov 2017). Furthermore, social trust is increasingly seen as an important determinant of overall well-being, for instance by making people more resilient to adversity and by reducing well-being inequalities (Helliwell, Huang, and Wang 2016). Hence, from a policy-making point of view, there is a salient need to uncover the roots of social trust.

Far reaching consequences of individual variations in trust should however not come as a surprise, since any prosocial interaction will be conditioned on the level of trustworthiness individuals are willing to attribute to one another. Indeed, social trust can be viewed as an estimate of the uncertainty around cooperation in a given environment. Untrustworthy social partners are susceptible to prefer the immediate benefits of defection over delivering a promised reward or paying a cost to achieve any particular outcome. Thus, differences in social trust are expected to have a strong impact on cooperative tendencies. The more someone declares believing others to be trustworthy, the more likely they should be to invest resources into them in order to collect the long-term benefits of cooperation. Such a causal effect of social trust on people's willingness to invest in others has been documented experimentally both in adults and children (Michaelson et al. 2013; Michaelson and Munakata 2016).

Because of this conceptually pivotal role in explaining how people navigate the

social world, levels of generalized trust have been extensively measured in social science studies and a convergent pattern has started to arise from the data. In particular, significant differences in social trust map with individual variations in socioeconomic status (SES), such that high-SES individuals place more trust in others than people of lower SES. Furthermore, exposure to deprivation during childhood has also been found to uniquely contribute to differences in social trust observed later in life (Petersen and Aarøe 2015; Hörl et al. 2016; Sheehy-Skeffington et al. 2017). The most striking illustration of the long-lasting detrimental effect of childhood harshness on social trust in adulthood has been put forward by Hörl and colleagues (2016) who used a natural experiment in post World War II Germany. The authors demonstrated that an exposure to low caloric rations as a child led people to judge others as less trustworthy when they were adults (Hörl et al. 2016). Similarly, in a Danish sample, Petersen and Aarøe (Petersen and Aarøe 2015) found that low birth weights (conceived as a forecast cue of a harsher future environment), predicted reduced social trust in adulthood, even after controlling for a range of confounding factors.

Therefore, higher levels of deprivation appear to elicit greater distrust in others' prosocial intentions, with early life conditions playing a key causal role. Yet, why this particular pattern should be expected to arise and why beliefs measured in adulthood should be sensitive to early life conditions remains unclear. One possible way to interpret such patterns is to consider them as the product of developmental mechanisms that shift individuals' traits and beliefs to optimize their behavior in their local environment (Frankenhuis and de Weerth 2013; Frankenhuis, Panchanathan, and Nettle 2016). In particular, drawing on psychosocial acceleration theory (Belsky 2012; Nettle, Frankenhuis, and Rickard 2012), it has been argued that greater levels of social trust should be observed in more affluent and stable environments, where the short-term cost incurred by investing in others is more likely to be offset by the long-term benefits of cooperation. As a consequence, this type of behavior could be expected to covary with a broader cluster of traits characteristic of slow life history strategies (Petersen and Aarøe 2015).

A life history strategy refers to the sequence of allocation decisions made by an organism throughout its lifetime (Stearns 1992; Ellis et al. 2009). Variation in life history strategies has been shown to be partly captured by a broad axis that reflects the tradeoff between present and future orientation. At one end of the continuum we found fast life history strategies, that aim towards smaller but more certain short-term benefits, and at the other end slow strategies that focus on greater but more uncertain long-term benefits (Bielby et al. 2007; Del Giudice 2014). Faster strategies are thought to be adaptive in harsh environments characterized by high levels of extrinsic mortality-morbidity (Ellis et al. 2009), where the benefits from long-term investments are unlikely to be collected. Psychosocial acceleration theory further predicts that during development, individuals will adjust their allocation decisions towards the optimal life history strategy based on cues of the quality of their local environment.

Hence, psychosocial acceleration theory offers a compelling framework to investigate the links between an individual's SES and social trust throughout its lifetime. Since individuals' propensity to trust others might reflect their tendency towards delay gratification, we expect it to correlate with other attributes of a fast-slow strategy. Specifically, higher social trust should be more frequently observed among individuals that experienced lower levels of deprivation both during childhood and as adults. Such a perspective on variations in social trust echoes other lines of research on the effect of deprivation on allocation strategies. Indeed, a low SES in adulthood has already been associated with decision making processes biased towards short-term benefits, with manifestations measurable on a wide range of outcomes (for reviews see Pepper and Nettle (2017); Sheehy-Skeffington et al. 2017). In addition to current effects of SES, associations between harsh early life conditions and faster strategies have also already been documented for a number of traits. For instance, in a representative sample of the French population, a broad measure of childhood harshness predicted both an early investment in reproduction and a lower investment in the maintenance of the soma (Mell et al. 2017). Effects of participants' SES during childhood have also been reported for risk-taking behaviors (Jordan, Amir, and Rand 2017), with individuals coming from lower-SES background being more risk-averse, as

well as for social preferences such as leader choice, with individuals from lower-SES background favoring more authoritarian leaders (Safra et al. 2017).

Interestingly however, it should be noted that in the case of prosocial tendencies measured based on the actual behaviours of participants in economic games, the evidence for a link with socioeconomic status is rather mixed. Indeed, using various measures of SES, some studies found that a lower SES was associated with less investment in cooperation (Nettle, Colléony, and Cockerill 2011; Korndörfer, Egloff, and Schmukle 2015; Safra et al. 2016); while others found a positive relationships (Piff et al. 2010, 2012; Jordan, Amir, and Rand 2017; but see Francis 2012) or no relationship at all (Wu et al. 2017). Thus, assessing the relationships between SES and prosociality using both survey data and experimental measures would allow for instructive comparisons in this context. Additionally, using actual behaviours in economic games, makes it possible to test finer grained predictions about the impact of childhood and current SES on cooperative behaviors. Indeed, in our framework, cooperation is viewed as any investment in social interactions with benefits and costs distributed across time with some degree of uncertainty around these outcomes. The magnitude of the costs and benefits determine the stakes associated to the interaction (the size of potential gains and losses) and the uncertainty will come from the probability of being exploited by one's social partner (the probability of exploitation). Depending on an individual's SES we might expect different sensitivities to the values of these two parameters. Assuming marginally decreasing returns of the amounts of resources possessed, the prospect of losing even a small quantity of resources for a low-SES individual with almost no capital will be dramatic compared to a rich individual with comfortable amount of resources to buffer the losses. Hence, in addition to being less willing to cooperate overall, we can make the additional predictions that low-SES individuals should be particularly sensitive both to an increase in the probability of exploitation and the size of potential losses compared to high-SES individuals.

Thus, building on previous studies highlighting a potential causal influence of childhood harshness on social trust, the current paper reports a series of three studies

with the following respective goals: 1) testing the basic relationship on a large sample spanning across multiple countries; 2) assessing whether SES predicts variations in an experimental measure of social trust and whether it is associated with different sensitivities towards the stakes and uncertainty surrounding cooperative interactions; 3) examining the covariation of social trust with investments in health and reproduction, as markers of individuals life history strategies. The next three sections detail the protocol and results obtained for each study individually, and the paper ends with a general discussion of the main findings.

### 3.2.1 Study 1

To assess the robustness of the relationships between social trust with childhood and current SES, we took advantage of available data from the European Values Study. This independent large-scale sociological survey was conducted on 66281 respondents living in 46 different European countries (the European values survey, Wave 4, European Values Study Longitudinal DataFile 1981-2008 (EVS 1981-2008), 2015).

## Materials and Methods

#### Study sample and selected variables

The analysis reported here was performed on Wave 4 of the European Values Study (between years 2008 and 2010) for a total number of 66281 respondents distributed in 46 countries (mean number of respondents per country: 1441 ± 94 sd). The EVS questionnaires include the following three questions on individuals levels of generalized trust which are routinely used in national and international surveys on social values: 'Generally speaking, would you say that most people can be trusted or that you can't be too careful in dealing with people?', 'Would you say that people usually only take care of themselves or that they try to be helpful most of the time?' and 'Do you think that most people would try to take advantage of you if they had the opportunity or that they would try to be fair?'. Answers available to the participants

for the first question were 'most people can be trusted', 'can't be too careful' or 'don't know', whereas a 1-10 scale was used for the last two items. Individual responses for the three questions served as indicators for a latent social trust variable. Regarding SES indices, answers to the questions 'Parent(s) had problems to make ends meet' and 'Parent(s) had problems replacing broken things' (ranging from 'Yes' to 'Not at all') were summed to provide a single proxy for individuals' childhood SES, whereas self-reported participants' income was used as a measure of current SES. In addition to participants' age and own level of education, the level of education of their parents as well as their parents' political involvement, interest for news and interest in books were included as controls for the effect of SES indices on social trust.

**Analysis**

The impact of SES indices on social trust was assessed through a simple structural equation model, childhood SES, current SES and the control variables. Missing values were imputed using the mice R package by generating 19 complete datasets. This technique allows a flexible implementation of multiple imputation that can accommodate both the continuous and categorical nature of our data. The SEM analysis was conducted with the lavaan package, using the WLSMV estimator, which provides more robust parameters estimates and standard when categorical latent indicators are present (Rosseel 2012). Results obtained for the 19 imputed datasets were pooled using the function runMI of the package semTools. Parameter estimates and standard errors were pooled using Rubin's rule (Rubin 2004).

Eventually, the model was fitted separately on each of the 46 countries with participants' parents' control variables in addition to their own age and level of education. Standardized coefficients were then extracted for each relationship and their significance was assessed at the group-level.

## Results and discussion

The analysis of the EVS data yielded a positive association between childhood SES and social trust in adulthood (average $\beta$=0.04, p <.001). Similarly, individuals with higher income also showed greater social trust as adults ($\beta$=0.06, p<.001). Thus, the pattern observed across a panel of 46 European countries is consistent on average with the hypothesis that both higher childhood and current SES uniquely contribute to greater levels of social trust in adulthood. The relationship however might be moderated by local factors, since the effect sizes were relatively spread and in some more marginal cases the direction of the relationship changed.

### 3.2.2 Study 2

In this section, we report results from an online experiment that further investigated the links between childhood/current SES, social trust and an experimental proxy for prosociality. Incorporating a measure of actual behaviour in an economic game, allowed us to extend the results obtained for the first study to a non-subjective indicator of prosociality. Furthermore, more fine-grained influences of deprivation on cooperative tendencies could be examined. Indeed, by means of a modified trust game, we were able to manipulate both the probability of reciprocation and the stakes involved in an act of cooperation. This experimental setup therefore allowed us to test whether individuals coming from a low SES background showed higher sensitivity to the stakes and probability of reciprocation as discussed in the introduction.

## Materials and Methods

#### Overall design and procedure

75 English-speaking participants (27 female, 48 male) aged 18 to 65 years old (M=34.27, SD=12.62), recruited via the online plateform Prolific.ac2, participated in this 15-minutes study for £0.8 (and a bonus up to £1). The experiment was programmed on Qualtrics and consisted of two independent behavioral tasks: a trust game and a socio-economic questionnaire. The trust game was always presented first,

followed by the socioeconomic questionnaire.

### Variables of interest

*Childhood and current SES, Social trust*

Childhood and current SES were assessed using the 3-items scales developed by Griskevicus and colleagues (2011). Specifically, for the index of childhood SES, participants had to grade their degree of agreement on a 1-100 scale to the statements '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 others kids in my school'. Analogously, the index for current SES consisted on the average level of agreement to the adult equivalent of the previous items, namely, 'I have enough money to buy things I want', 'I don't need to worry too much about paying the bills', and 'I don't think I'll have to worry about money too much in the future'.

Social trust was measured using the general trust question used in Study 1, i.e. 'Generally speaking, would you say that most people can be trusted or that you can't be too careful in dealing with people?' answered on a 1-10 scale.

*Trust game*

Sixteen independent trust games were played with a different partner each time. At the beginning of the experiment, participants were informed that the tokens used during the game would be transformed into a real money bonus at the end of the game (1 token=£0.1). Participants were informed of the probability of reciprocation of each partner (65, 70, 75 or 80%) and of the amount of tokens (1, 2, 3 or 5) required to play the game (i.e., what they had to give to interact with this particular socia partner). This number corresponded to the number of tokens the participant would lose or win, whether their partner chose to reciprocate or not, i.e. to the stakes of the cooperative act (cf Figure 3.1).

At the beginning of each game, participants received 5 tokens, corresponding to a bonus payment of 50 cents and had to indicate their willingness to play with

the presented partner by answering the question 'How much do you want to play with this partner?', using a 9-point scale ranging from 'not at all' to 'extremely'. Individual scores in 'willingness-to-play' were then used as our dependent measure of prosociality, to be explained by variations in childhood and current SES.

Importantly, in order to control for the probability of reciprocation and the stakes associated with each partner, the partners were actually simulated by a computer program, which was never specified to the participants. Moreover, participants did not get feedback about the partner's choice to reciprocate or not. Instead, only once participants had indicated their willingness-to-play for the whole sequence of 16 trust games, one game was randomly selected to calculate the bonus that would be added to the participants' payment. To do so, a random number between 1 and 9 was drawn; if this number was below the participant's willingness-to-play, the game was played and the partner's decision to reciprocate was simulated based on her reciprocation probability. If the random number was above the participant's willingness-to-play, the game was not played and the participant kept his/her 5 tokens. Finally, the resulting number of bonus tokens and the corresponding amount in £ that would be added to the participant's payment was revealed on the final screen.

**Analysis**

Social trust, childhood SES, current SES, stakes and probabilities were normalized. The self-reported measure of trust was analyzed using a simple linear regression model taking childhood and current SES as regressors. On the other hand, due to the repeated measurement of willingness-to-play, individual scores were analyzed using a mixed linear model taking stakes and probability as a within-subject regressor, with participant ID as the grouping variable (random intercept). This model integrated both random intercept and random slopes on the effect of stakes and probabilities. Childhood, current SES and social trust were incorporated as between-subject regressors with participant ID as the grouping variable (random intercept), while stakes and probability were within-subject regressors. The model integrated both a random intercept, with participant ID as the grouping variable, and random slopes

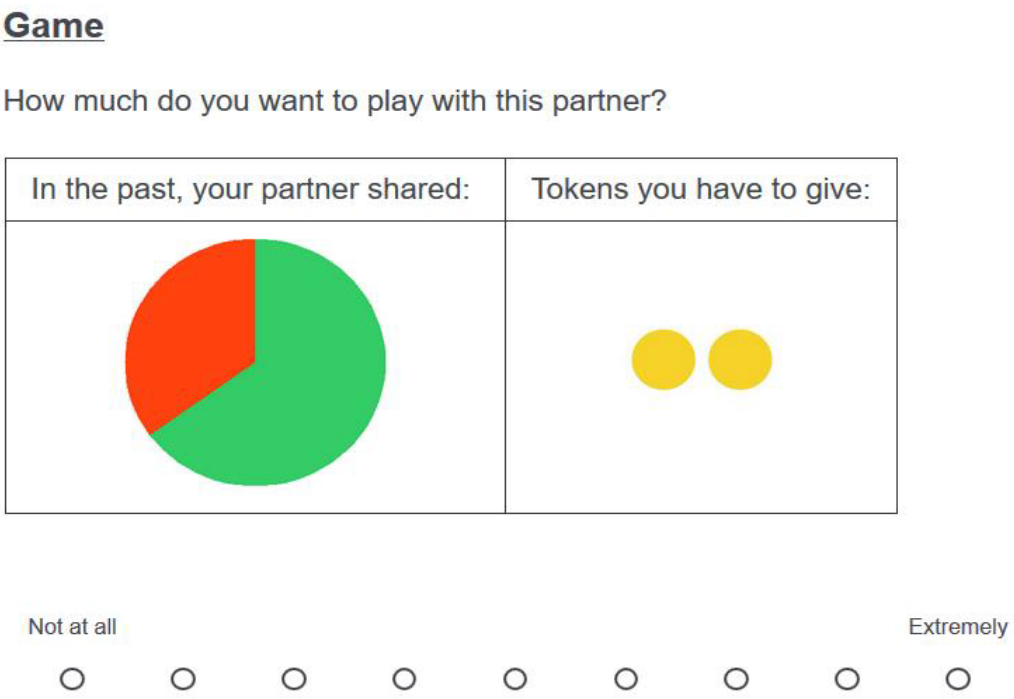


Figure 3.1: Trust game decision's screen.

on the effect of stakes and probabilities. Interaction terms for the within and between regressors were also included in the model.

## Results and discussion

In line with the results reported in study 1, childhood SES was positively associated with social trust ($b$=0.16 ± 0.05, t=3.20, p= .002). However, current SES was not associated with social trust once childhood SES was accounted for ($b$=0.06 ± 0.05, t=1.243, p= .219). Similarly, childhood SES had a positive main effect on willingness to play the trust games ($b$=0.32 ± 0.16, t(73)= 2.01, p=.048; Figure 3.2), whereas current SES did not ($b$=-0.13 ± 0.16, t(73)=-0.83, p>.250).

Furthermore, no interaction between childhood or current SES and stakes or reciprocation probability were found: childhood SES*stakes ($b$=0.02 ± 0.09, t(1119)=0.21, p>.250), childhood SES*reciprocation probability ($b$=0.01 ± 0.08, t(1119)=0.17, p>.250), childhood SES*stakes*reciprocation probability ($b$=-0.02 ± 0.03, t(1119)=-0.76, p>.250), current SES*stakes ($b$=-0.02 ± 0.09, t(1119)=-0.22, p>.250), current

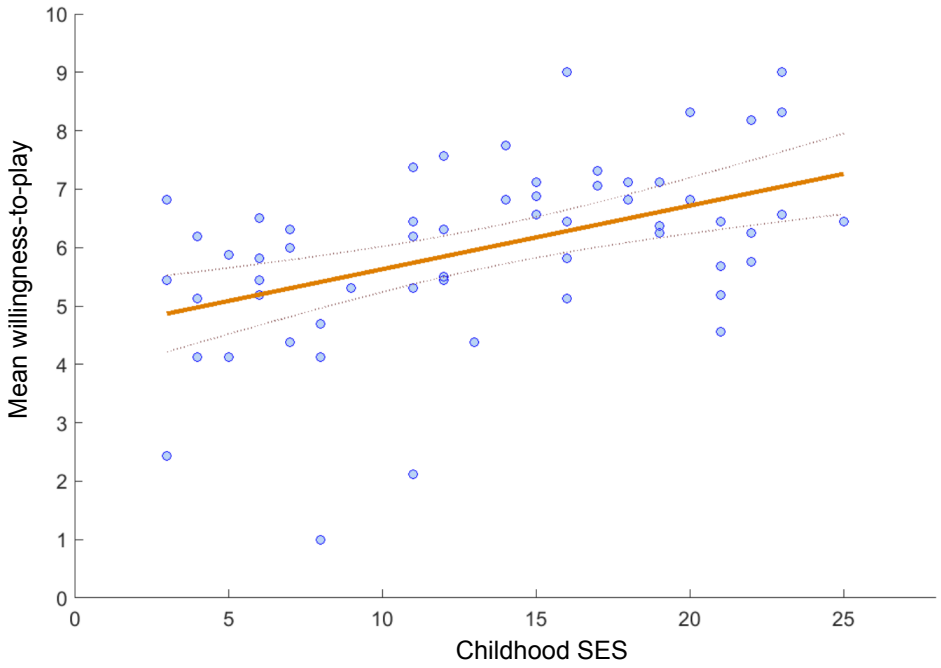


Figure 3.2: Participants' average willingness-to-play plotted against childhood SES. Dotted lines correspond to the lower and upper bounds of the 95% confidence interval of the regresssion.

SES*reciprocation probability ($b$= -0.02 ± 0.08, t(1119)=-0.22, p>.250), current SES*stakes*reciprocation probability ($b$=-0.01 ± 0.03, t(1119)= -0.35, p>.250). The two measures of trust were also associated, with a main positive effect of social trust on willingness-to-play ($b$=0.41 ± 0.14), t(73)=2.80, p=.007.

### 3.2.3 Study 3

The last study investigated whether indicators of social trust covary with other markers of individuals' life history strategies, and more specifically if lower childhood SES predicts faster life history strategies. To investigate these links, we exploited survey data gathered from a cross-sectional sample of the French population, including information about participants health and reproductive behaviours, childhood SES and social trust.

## Materials and Methods

#### Sample and procedure

Our sample consisted of 899 French adults (474 females) aged 16 to 83 (mean: 47.8 ± 16.3 sd) recruited online via the French polling institute IPSOS. Initially, a total of 11000 participants were invited to answer a demographic survey and were then selected based on age, gender, geographical region, urban vs rural and occupation to constitute a representative sample of the French population using quota sampling. Selected participants were then asked to fill a series of questionnaires about the quality of their rearing environment during childhood and their current situation in adulthood; about their investment into their own health and reproduction; as well as a series of items assessing their level of social trust. 1691 participants eventually completed the whole series of questionnaire. Participants with a number of improper responses and/or missing values exceeding three standard deviations above the sample's average were excluded from the analysis (N=48), yielding our final sample size of 899 participants.

### Variables of interest

*Childhood and current socioeconomic status*

Participants' both childhood and current SES were measured using the 3-items scales (Griskevicius et al. 2011) described in the previous study. For each type of SES, average values over the thee items are computed in both cases to obtain a single index.

*Life history indicators*

As shown in a previous work based on the same study sample (Mell et al. 2017), measures of individuals' investments in health and reproduction co-vary in a pattern consistent with the fast-slow continuum. Namely, on average participants with higher body mass index spent a greater proportion of their life smoking, reported a poorer health in general and making fewer efforts to preserve it, started their sexual life earlier, had more short-term sexual relationships and had their first child earlier (but note that 'number of offspring' was not part of the general pattern). Here we used the same life history indicators in order to examine whether variations in social trust would also be captured by a more general axis consistent with the fast-slow continuum. To preserve our sample size however, we did not consider measures of actual events of reproduction, i.e. age at first birth and number of offspring in the main analysis.

*Measures of social trust*

As for study 1, social trust was measured using participants answers to the questions 'Generally speaking, would you say that most people can be trusted or that you can't be too careful in dealing with people?', 'Would you say that people usually only take care of themselves or that they try to be helpful most of the time?' and 'Do you think that most people would try to take advantage of you if they had the opportunity or that they would try to be fair?'. In addition to the previous variables, an additional index of social trust was computed based on a series of six 5-point

Likert scales asking participants how much trust they put in various circles of people (*e.g.*, 'How much do you trust your neighbors?'). Responses to the six questions were averaged to obtain a complementary index of individuals' level of social trust.

### Analysis

All statistical analyses were carried out in R 3.0.3 (https://www.rproject.org/) with Rstudio v0.99. First, Structural Equation Modelling (SEM) was used to test our theoretical model, namely that higher social trust co-varies with slow life history indicators along a latent fast-slow continuum, and, slower strategies are associated with higher childhood SES. More precisely, our measurement model included a first latent factor for the indicators of social trust, as well as a second latent variable accounting for the shared variance between the two indicators of reproductive strategies (sexual debut and number of short-term relationships) and the four indicators of investment in health (BMI, health status, health efforts and years spent smoking).

For scaling and identification purposes, the variance of the latent variables was set to one. Furthermore, because correlations between respective indicators of the reproductive or health domains might not be fully captured by the single life history factor, our model also allowed for residual correlations within each domain only. Also, due to the wide age range characterizing our sample (16 to 83), participants' age was also used as an auxiliary variable to control for its effects on life history indicators. Finally, the structural part of the model included a childhood SES as a predictor for the life history strategy factor, and both childhood SES and life history strategy for the latent social trust variable (Figure 3.3 A).

In addition to the previous SEM, we also fitted a simpler model similar to the one reported in study 1 for the associations between the childhood and current SES and the social trust factor (Figure 3.3 B). Both SES indices were again used as predictors of the latent scores while controlling for participants' age and level of education. This second model allows for more direct comparisons with the results of studies 1

and 2.

Prior to running the analysis, we also performed multiple imputation of missing values by chained equations using again the R package mice (Buuren and Groothuis-Oudshoorn 2011) in order to preserve our sample size and reduce bias in the estimation of model parameters. A set of 10 imputed dataset was thus generated using the same packages mentioned in the methods section of the first study. Lastly, for the first model, chi-square statistics and related fit indices were pooled using the method described in Li, Meng, Raghunathan, and Rubin (1991). The fit of the model to our data was assessed both using the chi-square statistics and a couple of commonly used approximate indices (Kline 2016), namely the Comparative Fit Index (CFI) and the Root Mean Square Error of Approximation (RMSEA). Indeed, models fitted on large samples are known to be frequently rejected based on the chi-square test even when they contain no severe misspecifications (Schermelleh-Engel, Moosbrugger, and Mäüller 2003). Hence, approximate indices are generally useful to avoid the problem of size dependency (Kline 2016).

## Results and discussion

Based on our hypothesis drawn from psychosocial acceleration theory, we expected: 1) a positive association between a latent life history factor contrasting fast-slow strategies and the latent social trust variable, and 2) negative relationships of childhood SES with both slow life strategies and social trust. Table 3.1 reports a set of descriptive statistics for the various measures and Figure 3.3 the main standardized $\beta$ coefficients associated to each SEM. Regarding the first model, the p-value associated to the chi-square statistics was significant but values for the RMSEA (0.06) and the CFI (0.91) did not indicate severe misspecifications.

Overall, the patterns of covariation obtained support the above hypotheses (Figure 3.3 A). First, as expected, indicators of health and reproductive strategies covaried in a manner consistent with the fast-slow continuum. Indeed, standardized coefficients reported on Figure 3.3 show that high scores on the life history factor reflect slower strategies, by correlating with later sexual debut ($\beta$=0.078, p<.036), fewer

Table 3.1: Descriptive statistics and correlations from self-report data (n=899).

| | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) | (10) | (11) | (12) | (13) | (14) |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Smoking (1) | 1.00 | | | | | | | | | | | | | |
| Health Status (2) | -0.11 | 1.00 | | | | | | | | | | | | |
| Health Effort (3) | -0.17 | 0.23 | 1.00 | | | | | | | | | | | |
| BMI (4) | 0.06 | -0.27 | -0.10 | 1.00 | | | | | | | | | | |
| Sexual Debut (5) | -0.20 | -0.03 | 0.03 | 0.04 | 1.00 | | | | | | | | | |
| Short-term partners (6) | 0.18 | -0.04 | -0.07 | 0.03 | -0.16 | 1.00 | | | | | | | | |
| General Trust (7) | 0.00 | 0.09 | 0.12 | 0.01 | 0.10 | 0.07 | 1.00 | | | | | | | |
| Others Prosociality (8) | -0.04 | 0.04 | 0.16 | -0.02 | -0.02 | -0.01 | 0.30 | 1.00 | | | | | | |
| Score Trust (9) | -0.03 | -0.03 | -0.14 | 0.01 | -0.02 | -0.02 | -0.47 | -0.39 | 1.00 | | | | | |
| cSES (10) | -0.02 | 0.15 | 0.08 | -0.08 | -0.03 | 0.05 | 0.10 | 0.11 | -0.04 | 1.00 | | | | |
| SES (11) | -0.07 | 0.18 | 0.27 | -0.11 | 0.10 | -0.06 | 0.18 | 0.17 | -0.21 | 0.23 | 1.00 | | | |
| Age (12) | 0.17 | -0.26 | 0.19 | 0.21 | 0.19 | 0.09 | 0.15 | 0.17 | -0.25 | -0.10 | 0.18 | 1.00 | | |
| Education (13) | -0.08 | 0.11 | -0.01 | -0.18 | 0.09 | 0.02 | 0.11 | 0.00 | -0.05 | 0.19 | 0.19 | -0.16 | 1.00 | |
| Gender (14) | -0.15 | 0.02 | 0.07 | -0.17 | -0.09 | -0.18 | -0.06 | 0.03 | -0.02 | 0.03 | -0.07 | -0.10 | 0.01 | 1.00 |
| Mean | 0.20 | 2.80 | 67.85 | 25.54 | 18.65 | 6.64 | 0.30 | 5.25 | 2.37 | 43.32 | 49.53 | 47.83 | 4.87 | 0.53 |
| SD | 0.24 | 0.72 | 19.24 | 5.18 | 3.38 | 13.80 | 0.46 | 1.84 | 0.71 | 23.60 | 24.26 | 16.27 | 1.87 | 0.50 |
| Min | 0.00 | 1.00 | 0.00 | 13.63 | 12.00 | 0.00 | 0.00 | 0.50 | 1.00 | 1.00 | 1.00 | 16.00 | 1.00 | 0.00 |
| Max | 0.88 | 4.00 | 100.00 | 47.97 | 61.00 | 200.00 | 1.00 | 10.50 | 5.00 | 100.00 | 100.00 | 83.00 | 8.00 | 1.00 |

short-term sexual relationships ($\beta$=-0.105, p<.024), smoke less ($\beta$=-0.378, p<.001), greater efforts put into maintaining a good health ($\beta$=0.582, p<.001) and a better health status in general ($\beta$=0.664, p=.001) and lower bmi ($\beta$=-0.258, p=0.043). In parallel, the indicators for social trust were all strongly correlated to the latent factor (General trust: $\beta$=0.603, p<.001; Others' prosociality: $\beta$=0.497, p<.001; Index Trust: $\beta$=0.685, p<.001). Furthermore, greater levels of social trust were associated with scores reflecting a slower strategy ($\beta$=0.222, p=.002). Lastly, individuals that experienced a higher SES during childhood also exhibited both slower life strategies ($\beta$=0.154, p=0.003) and greater social trust in adulthood ($\beta$=0.132, p=0.005). Results from the second model on the other hand provided further support for the pattern of associations already found in study 1 and 2 (Figure 3.3 B). Indeed, lower childhood and current SES were again uniquely associated with less social trust (respectively, $\beta$= 0.09, p=0.03; $\beta$=0.20, p<.001).

### 3.2.4 General discussion

The series of studies reported in the previous sections investigated the relationship between social trust and SES in early life and adulthood. A robust pattern was found across study 1 and 3 that used survey data on relatively large samples: individuals that experienced higher SES showed greater social trust, with a unique contribution of SES during childhood. In Study 2, which used a smaller online sample, the same

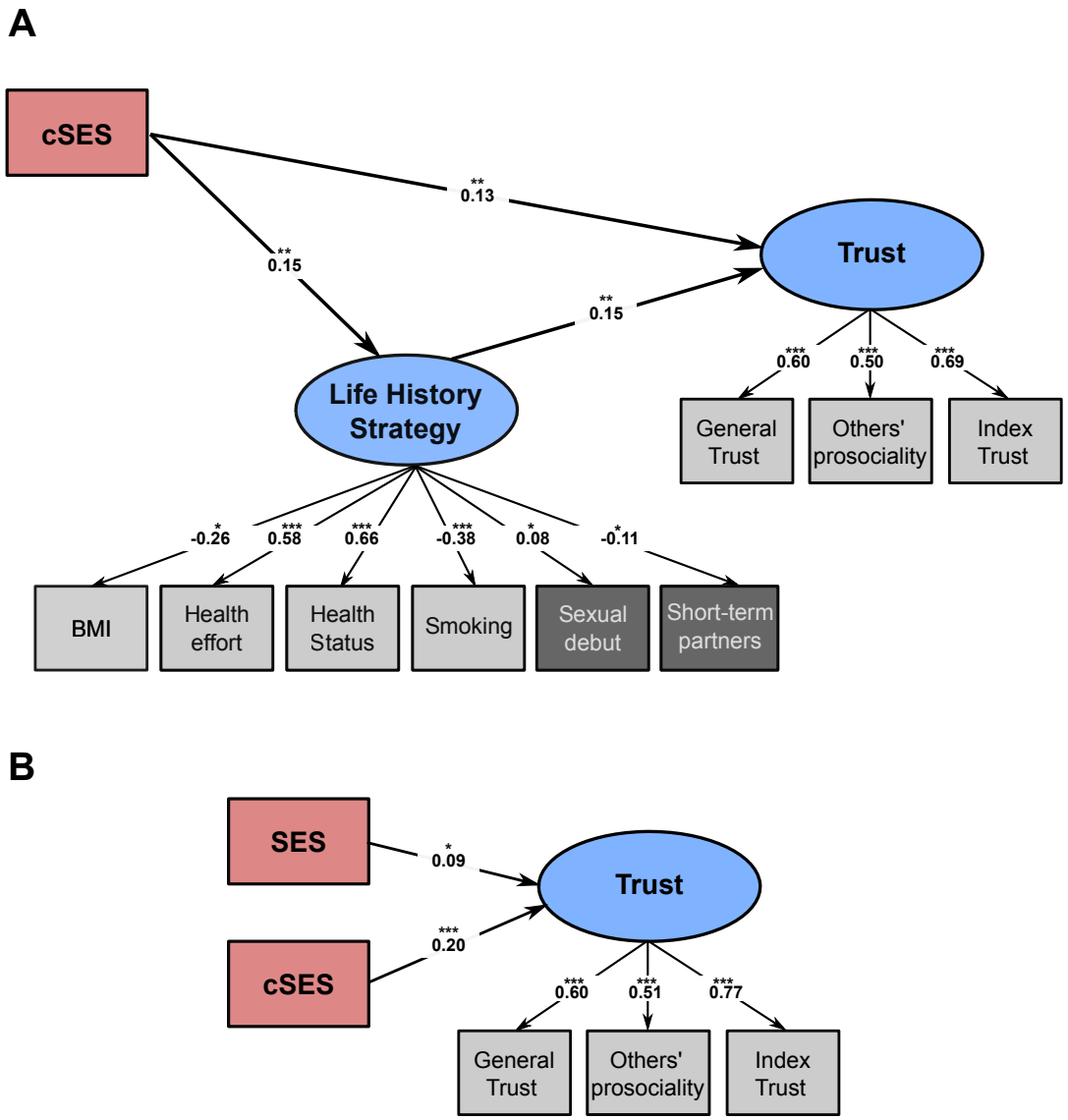


Figure 3.3: Path diagrams of the structural models. **A.** Model 1 - Childhood SES, life history strategies and social trust **B.** Model 2 - Replication of the negative relationship between childhood and current SES with social trust.

effect of childhood SES was found both on social trust and participants' willingness to play in a trust game, an experimental proxy for prosociality. Hence, the results obtained overall are consistent with the hypothesis that people use their degree of affluence during ontogeny to calibrate their level of prosociality later in life. Furthermore, study 3 highlighted the fact that individuals' levels of social trust might be integrated in broader patterns of life history strategies. Indeed, greater social trust positively correlated with both greater investment in health and long-term oriented reproductive behaviors. This finding therefore supports the claim that social trust is an indicator of long-term orientation and consequently of slow life history strategies (Petersen and Aarøe 2015). In the experimental study however, participants' current SES did not correlate significantly with either self-reported social trust or willingness to play in the trust games. Finally, contrary to our initial hypothesis participants' SES did not seem to affect their sensitivity to the stakes and probability of reciprocation during the economic games.

The remainder of the paper further discusses these main findings.

Across the three studies reported above, we found that experiencing lower levels of affluence during childhood was associated on average with lower self-reported social trust in adulthood. This link between deprivation and social trust is consistent with several studies already available in the literature. In a longitudinal study of a UK sample, participants' parental SES at age 11 predicted their general trust in others at age 33 (Sheehy-Skeffington er al. 2017). In a study investigating the effects of birth weight on social trust (Petersen and Aarøe 2015), the SES of the mother at birth appeared to have a distal impact on levels of trust during adolescence. And in the natural experiment exploiting evidence from hunger episodes in post war Germany (Hörl et al. 2016), lower caloric rations imposed by the occupying forces had long lasting effects on individuals' level of trust decades later. Thus, our results further support these earlier findings, extending the external validity of the proposed link between individual differences in self-reported general trust and early-life circumstances. As a note of caution however, it should be noted that, while an average effect of childhood SES on trust was present in the EVS survey, it did not

seem to hold across all countries. Further cross-cultural studies might therefore be useful to uncover how this relationship could potentially be moderated by additional factors in various regional settings.

Our second study also found an association between childhood socioeconomic status and prosociality, assessed through participants' investments in a trust game. This finding suggest that relationships observed between childhood and social trust measured via subjective questionnaires are readily extendable to actual behaviours in cooperative interactions. As mentioned in the introduction however, similar studies about the effects of SES during childhood on prosocial behaviour in economic games has yielded conflicting results so far. For instance, young American males who grew up in harsher environments where found to be more likely to use exploitative strategies and retaliatory defection in an iterated prisoner's dilemma (McCullough et al. 2012). Such a pattern is in line with our own findings and with the idea that early adversity promotes lesser prosociality. Yet, in a more recent online study, childhood SES had either no or the opposite effect on a composite score of cooperation calculated across thirteen types of economic games (Wu et al. 2017). Similarly, in a series of online experiments where participants had to play both a dictator and an ultimatum game (Jordan, Amir, and Rand 2017), subjects coming from lower SES background were found to make higher offers to their partners. However, this effect held for one particular 2-items measure of childhood SES but vanished once the analysis was conducted with the same 3-items childhood SES measure used in study 2.

The evidence for the effect of current SES on prosociality is weaker than for childhood SES since no association was found in study 2, but still globally support the hypothesis that higher deprivation decreases prosociality. As stated in the introduction, the broader literature is largely consistent with a negative relationship between deprivation and social trust (Steijn and Lancee 2011; Eric M. Uslaner and Mitchell Brown 2005; Brandt, Wetherell, and Henry 2015). However, as for childhood SES the picture becomes less clear for individual behaviours in economic games. Indeed, although we found no effect in the context of a trust game, some studies have found

that people with lower SES were less prosocial in economic games (Nettle, Colléony, and Cockerill 2011; Gomes and McCullough 2015) but other studies found opposite results (Piff et al. 2010; Jordan, Amir, and Rand 2017; but see also Francis 2012). For instance, Nettle and colleagues (2011) found a huge reduction in the average offers in a dictator game among people coming from deprived neighborhoods, while Jordan and collaborators (2017) found higher offers from low-SES individuals in both a dictator and ultimatum game in large online experiments.

Hence, such discrepancies in the empirical literature using economic games suggest that effects found for both childhood and current SES on investments in trust games should be interpreted with some degree of caution. This holds also true for the lack of significant interactions in study 2 between childhood SES and trust game parameters (i.e. stakes and probability of reciprocation). Indeed, we hypothesized that individuals with low-SES would be less willing to engage in high stakes interactions due to a lesser ability to absorb losses in general. The lack of supporting evidence for this hypothesis is however not very conclusive in the case of our experiment, since the stake of the games had no main effect on participants' willingness to play. Because the design of our experiment did not seem to elicit an appropriate response to this particular parameter, we could not properly address these more subtle predictions on SES levels and investment in cooperation.

Thus, more work is required to assess the generalizability of the relationships obtained. In particular, using more ecologically valid measures of cooperation in parallel to the more artificial economic games might help to clarify some of the current confusion.

In addition to the strict relationships between SES and social trust, we also found in study 3 that higher social trust correlates with markers of a slower life history strategy (i.e. higher investment in health and more long-term reproductive behaviours). Thus, in contrast to the findings recently reported by Wu and collaborators (2017), our study supports the proposal that life history strategies could account for part of the individual variation in prosocial tendencies. The differences between the two studies are not easily interpretable however since Wu and colleagues used psychometric

indicators as proxies for their participants' life history indicators, whereas we had more direct indicators of health and reproductive strategies. Moreover, prosociality was restricted to self-reported social trust in our analysis whereas Wu et al. measured different outcomes in a range of economic games to construct an experimental measure of cooperation. Indeed, it has already been shown that psychometric indicators of life history strategies such as the High-K scale do not always adequately correlate with more objective indicators like sexual debut (Copping, Campbell, and Muncer 2014; Gruijters and Fleuren 2018). On the other hand experimental measures of cooperation might better reflect prosocial behaviors in real life (but see Palminteri and Chevallier (2015)). Hence, more comprehensive studies integrating elements of both designs are probably necessary to settle the general relationship between prosociality and fast-slow life history strategies.

Overall, the current work therefore supports the hypothesis that experiencing socioeconomic hardship can have both present and lasting negative effects on social trust and cooperation. Negative effects that would be part of a more general switch to a faster life history strategy, aiming at securing more immediate rewards. Nonetheless, in regard to the currently rather mixed results available in the literature, more empirical work is required to assess the robustness of these relationships. Furthermore, even though the findings reported above were quite consistent across the three studies, they suffered from several limitations. Indeed, all the estimates are correlational in nature, come from cross-sectional samples and could be biased by unobserved variables affecting childhood, current SES and social trust altogether. In particular, it has been argued that much of the individual variation in behaviours that researchers try to explain through developmental mechanisms that calibrate individuals' phenotype to certain cues gathered from the environment, could in reality be due to individual genetic differences (Zietsch 2016). In the case of social trust however, we know through the analysis of natural experiments from post-war Germany (Hörl et al. 2016) that deprivation in early life can have substantial and lasting effects on an individual's phenotype. Hence at least part of the effects of SES on prosociality might capture such plastic responses to the environment. Nevertheless, it is undeniable that using such natural experiments or more genetically sensitive

designs are a crucial step forward to test and refine this broader developmental framework. Indeed, it is the proper identification of the mechanisms underlying individual variation in prosociality that might eventually lead to socially important public interventions.

# Childhood harshness, parental investment and social trust in late childhood.

**Hugo Mell, Ava Guez, Lilas Gurgand, Frank Ramus, Coralie Chevallier**

## 3.3 Introduction

The series of studies reported in the previous section of the manuscript provided further support for a lasting impact of early childhood deprivation on social trust. Yet, the question of how this calibration might occur during development and whether the effects of deprivation already manifest themselves during childhood was not addressed. From a policy making point of view however, uncovering at which point in life differences in social trust arise, and, by which aspects of the child's environment they might be moderated is an important step towards the design of effective interventions.

Indeed, if variations in social trust triggered by differential exposure to deprivation are already expressed during childhood, they might reinforce themselves by further conditioning the social experience of individuals throughout adolescence. Early differences in exposure to harsness could then have cumulative effects on the emergence of trust beliefs, which might explain the discrepancies observed between adults that have similar socioeconomic status. Futhermore, although two individuals might be exposed to equivalent levels of harshness as they grow up, there will be some variation in the amount of support they receive from their parents. Hence, one could expect that increases in parental investment would have important buffering effects on the impact of early exposure to deprivation. Nevertheless, the relationships between harshness, parental investment and trust might be complicated by the fact that levels of parental investment are themselves likely to be endogenous to the quality of the environment. Indeed, lower parental investment is also thought to be a marker of a fast life history strategy triggered by a harsh environment(Ellis et al. 2009),

and some empirical evidence supporting this link is already available (see Cabeza de Baca and Ellis (2017) for a review). Thus, parental investment might eventually play a role in the emergence of trust beliefs both as a moderator and a mediator of the effects of environmental harshness.

To our knowledge, studies on the role of parental investment in this particular matter are still lacking, whereas some research has been conducted on the impact of deprivation on children's prosocial tendencies in general. Although the evidence remains relatively scarce, a few studies have tested the association between children behaviour in economic games and some measure of their exposure to socioeconomic deprivation. For instance, in a recent study measuring offers in a 'Quality Dictator Game' made by 6-7 years old children, coming either from a very deprived neighborhood, or a middle-SES neighborhood from the same Romanian city (Safra et al. 2016); the authors found that children living in the harsher neighborhood behaved less prosocially. These findings extended similar results obtained for a British sample of primary school students (Benenson, Pascoe, and Radmore 2007). On the other hand, high SES children have been found to offer less than their low SES counterparts in a similar dictator game, when information about the status of the recipient was available (*i.e.*, the unknown child was described as a 'sick child'). Similarly, Chen an collaborators (2013) found that 4 years old children living in low SES households were the more prosocial in an dictator game involving a friend, but differences vanished once the recipient was an unknown stranger. Hence, the previous findings suggest that differences in prosocial behaviours related to socioeconomic status in children might actually vary across cooperation contexts, and/or that a non linear relationship between prosociality and SES might actually be more accurate (Safra et al. 2016).

Yet, all the studies mentionned above relied on economic games to quantify variation in children's prosociality. Such experimental measures however introduce a variety of important parameters, such as the actual type of game used, the stakes and the kind of rewards involved, or the information available about the partner. Although this ability to recruit different facets of the decision-making processes at play will

usually constitute an attractive methodological feature (*cf* study 2 of the previous paper), the resulting variation measured in children's offers will inevitably reflect more than just individual differences in trust beliefs. Furthermore, in our work, survey items repeatedly appeared more robust to measure individual differences than behavioural tasks. Interestingly, this phenomenon fits in a more general pattern starting to emerge from the experimental literature on risk tasking, where standard questionnaires seem to better capture individual differences. Hence, to bear on the research in adults exemplified by the series of studies reported previously, more specific measures of social trust would seem better suited.

Below, we briefly report preliminary results from a preregistered study (see the General Appendix for a copy of the preregistration), designed to adress some of the issues raised above. Specifically, the aim of the study was first, to measure social trust in children in a way that allows for the comparison with the data collected with adult subjects; second, to investigate the role of parental care in modulating the relationship between deprivation and trust; third, to test the association between deprivation and social trust without relying on participants' retrospective assessment, often decades later, of the harshness of their childhood environment. Indeed, even though several studies have found encouraging evidence for the accuracy of retrospective measures of childhood circumstances (*e.g* Havari and Mazzonna (2015); Osypuk, Kehm, and Misra (2015)), synchronous evaluations of environmental harshness should still reduce measurement error and yield more precise estimates.

## 3.4 Study design and data transformations

The research reported here took advantage of an ongoing study of the EDEN mother-child cohort (see Heude et al. (2016) for a detailed description of the cohort profile). The main objective of the EDEN cohort is to provide longitudinal data for the exploration and testing of various hypotheses on the impact of prenatal and early postnatal factors on child health and development. EDEN was set up in 2003 in two university maternity clinics (in Nancy and Poitiers, France), by the local clinical teams in collaboration with research teams from the National Institute of Health

and Medical Research (Inserm). Exclusion criteria for the study included having a personal history of diabetes, twin pregnancy, intention to deliver outside the university hospital or to move out of the study region within the following 3 years, and the inability to speak French. Originally, 2002 pregnant women joined the cohort but due to severe attrition rates in the follow-up years, only 425 children aged 11 years old remained in the cohort at the time of the current study. Importantly, the initial cohort did not form a representative sample of the French population. Instead, urban, well-educated and high-income households are over-represented among the EDEN mothers. Moreover, this characteristic has been accentuated over the years due to differential attrition with regard to socioeconomic status (Heude et al. 2016).

For the purpose of the current study, a selection of items relating to the harshness of a child's environment and to the amount of parental care received, were gathered from parental questionnaires, children questionnaires and observational data collected during visits to the research centers (see section 'Variables' of the preregistration for a list of the variables retained and the frequency at which they were collected). Since the EDEN cohort was not initially designed to study children's social trust, the latter was only assessed during the last wave, when we had the opportunity to add a scale for social trust to the questionnaires used until then. Therefore, levels of trust have only been collected once children were eleven years old.

In order to obtain comparable data with the studies involving adult participants, we assessed social trust by rephrasing the three questions previously used in a way more suitable for children. Specifically, they had to give their answers by telling whether they agreed a little or completely with one of two opposite statements from a duo of fictional characters, each statement corresponding to the extremes of the corresponding scale used with adults: 'Who do you agree most with?' 1) Fred : 'Generally most people can be trusted', Nico : 'You can't be too careful in dealing with people'; 2) Fred : 'Most of the people try to be fair', Nico : 'Most of the people take advantage of me'; 3) Fred : 'Most of the time people try to be helpful', Nico : 'Most of the people are mostly looking out for themselves'.

For all type of variables, missing values were imputed according to the procedure

described in the previous study. Then, answers for each of the trust question were transformed into z-scores separately, before being summed together in order to obtain a global index. Due to the longitudinal nature of the study, multiple measures at different points in time were available for harshness and parental investment items. For each variable, values obtained for a particular wave were first transformed into z-scores, which were then used to compute the average value for each item across years. Finally, a global harshness indicator and an index of parental investment were calculated respectively from the means of the corresponding items.

Thus, based on the individual scores obtained for the Trust, Harshness and Parental investment indices, we could explore the following set of questions: 1) *What is the effect of environmental harshness on trust for 11 year-old children?*, 2) *Is the effect of environmental harshness buffered by parental investment?*, and 3) *To what extent is the effect of environmental harshness mediated through parental investment, and to what extent is parental investment affected by environmental harshness?*. To do so, we fitted a series of three linear regression models, each associated to one of the above question. The next section reports a short summary of the estimates obtained for each model and offers a brief discussion of the result.

## 3.5 Results and discussion

The first regression model can be described with the following equation:

$$Trust = \alpha + \sum_{i=1}^{4} \beta_i Control_i + \beta_5 Harshness + \epsilon$$

The second term refers to a set of control variables that was used for all regression models and that included the level of education of the mother, the level of education of the father, the gender of the child and the location of the hospital where the mother went (either Nancy of Poitiers). Our prediction was that children that had been exposed to higher levels of harshness would have lower scores on the trust index.

Table 3.2: Regression coefficients and significance tests for Model 1.

| | est | se | t | df | Pr(¿—t—) |
|---|---|---|---|---|---|
| Harshness_score | 0.01 | 0.02 | 0.77 | 412.79 | 0.44 |
| controlecentre | 0.06 | 0.08 | 0.70 | 416.92 | 0.48 |
| controlesexe | 0.11 | 0.08 | 1.35 | 416.65 | 0.18 |
| controleetudesm | -0.21 | 0.09 | -2.36 | 402.30 | 0.02 |
| controletudesp | -0.14 | 0.09 | -1.61 | 246.23 | 0.11 |

As shown in Table 3.2, none of the coefficients estimated in model 1 were significant. Thus, we found no evidence for an impact of levels of deprivation experienced during childhood on social trust expressed at age 11.

The second regression model included an additional parameter for the level of parental investment (PI), as well as an interaction term between Harshness and Parental investment:

$$Trust = \alpha + \sum_{i=1}^{4} \beta_i Control_i + \beta_5 Harshness + \beta_6 PI + \beta_7 PI * Harshness + \epsilon$$

Our additional prediction for this model was that higher levels of parental care received during childhood would both have a direct positive effect on trust and an indirect one by mitigating the negative effect of harshness.

Table 3.3: Regression coefficients and significance tests for Model 2.

| | est | se | t | df | Pr(¿—t—) |
|---|---|---|---|---|---|
| Harshness_score | 0.01 | 0.02 | 0.79 | 411.10 | 0.43 |
| PI_score | 0.01 | 0.01 | 0.99 | 401.22 | 0.32 |
| controlecentre | 0.07 | 0.08 | 0.87 | 414.68 | 0.39 |
| controlesexe | 0.11 | 0.08 | 1.37 | 414.68 | 0.17 |
| controleetudesm | -0.21 | 0.09 | -2.32 | 401.02 | 0.02 |
| controletudesp | -0.14 | 0.09 | -1.56 | 247.58 | 0.12 |
| Harshness_score:PI_score | 0.00 | 0.00 | -0.33 | 395.06 | 0.74 |

Table 3.4: Regression coefficients and significance tests for Model 3.

| | est | se | t | df | Pr(¿—t—) |
|---|---|---|---|---|---|
| Harshness_score | -0.09 | 0.14 | -0.61 | 338.33 | 0.54 |
| controlecentre | -2.76 | 0.65 | -4.21 | 414.54 | 0.00 |
| controlesexe | -0.26 | 0.65 | -0.40 | 330.67 | 0.69 |
| controleetudesm | -0.48 | 0.73 | -0.66 | 390.55 | 0.51 |
| controletudesp | -0.19 | 0.74 | -0.26 | 187.43 | 0.79 |

Again, none of the coefficients obtain were significant (Table 3.3). Variation in parental investment therefore did not seem to explain trust scores and logically, no significant interaction between harshness and parental caring was found.

Lastly, the third regression model adds an equation for the relationship between Harshness and Parental investment, in order to assess whether parental investment might mediate the effect of harshness on trust:

$$PI = \alpha + \sum_{i=1}^{4} \beta_i Control_i + \beta_5 Harshness + \epsilon$$

Here we predicted that higher harshness would be associated to lower levels of parental investment and that the decrease in parental caring would mediate some of the effect of harshness.

Results reported in Table 3.4 show that differences in harshness did not explain variation in parental investing. Surprisingly however, we found a significant association between the hospital were the mother were recruited for the study and levels of parental caring. This effect could therefore reflect some important average socioeconomic differences between participants coming from the two cities.

Thus, the preliminary results obtained from this analysis of the EDEN cohort provided no support for our initial predictions and failed to replicate the well-known relationship between environmental harshness and parental investment (Cabeza de Baca and Ellis 2017). Specifically, no association was found neither for harshness nor for parental investment with trust measured at age 11 and the global indicator of harshness did not correlate with the index of parental caring. Hence, since no direct

effects were obtained for any of the predicted relationships, logically no evidence was found for a role as a mediator or moderator of parental caring. At this stage therefore, we can conclude that the present data does not support the hypothesis that the effects of early deprivation on social trust are already visible in late childhood.

Yet, the lack of significant associations reported here could also come from the fact that the EDEN cohort does not constitute a representative sample of the French population. Indeed, as stated in the previous section, high-SES households are over represented among mothers in the cohort and this imbalance has been worsened by higher attrition rates over the years for low SES participants. Thus, it is plausible that the range of exposure to deprivation as well as the variation in parental caring are too narrow in this study to explain meaningful differences in social trust.

Nonetheless, if it turns out that the results obtain here have some external validity and that the lasting impact of childhood deprivation on adults' trust documented in the previous studies are robust, this would leave us with the interesting puzzle that the effects of deprivation remain dormant for a significant part of people's development. From a range of experiments in developmental psychology and observations from sociology we know that children can behave prosocially from early ages (*e.g.*, Dunfield et al. (2011); Warneken and Tomasello (2006)). Five years old children also already seem to be able to apply trust selectively based on the information they possess about others' intentions (Liu, Vanderbilt, and Heyman 2013). Moreover, from the studies cited in the introduction, we know that in economic games children share with unknown strangers, can modify their offers depending on the context, and we also have evidence that their behaviour is affected by their socioeconomic status. Thus, it is intriguing that even though they do participate in prosocial interactions at young ages that might sollicitate their trust beliefs, and even though SES is correlated with actual prosocial behaviours in experimental settings, we did not find any association in the case of social trust. Perhaps part of the explanation is the degree to which children of this age rely on cooperative interactions in their everyday life, in particular with complete strangers. However, this remains rather speculative and in the end, we do not have strong *a priori* theoretical reasons to predict such a delay in the

effect of deprivation on trust beliefs. Thus, further research is needed to establish whether the paradox is actually real and explore some potential solutions.

# Chapter 4

# Theoretical foundations of the fast-slow continuum: towards a better understanding of individual differences in delay discounting

## 4.1 Objectives and summary

Chapter 2 and 3 focused on measuring the empirical patterns of covariation between a series of key traits, and how the relationships obtained fitted with the idea of a fast-slow continuum. Overall, the evidence gathered suggests that it should be worth to investigate further the hypothesis that people developing in harsh environments form coordinated strategies geared towards short-term benefits.

So far in our discussion of the fast-slow continuum, the adaptive value of a faster strategy has been justified by the higher adult mortality rates characteristic of a harsh ecology. Under such circumstances, one should be present-oriented because there is more uncertainty around the fact that there will be any future at all. The logic is straightforward and as proved to be powerful in explaining important features of organisms' life cycles. Yet, as one tries to integrate individual differences for a variety of behaviours and beliefs (such as social trust) with other life history indicators on the fast-slow continuum, it becomes less clear whether differences in

exposure to mortality can still account for the patterns observed. Determining which ecological factors gave rise to the selection pressures for the underlying mechanisms of phenotypic plasticity is however an important issue. Indeed, it provides us with *a priori* hypotheses on the cues that are likely to be the most involved in the calibration of people's life history strategies. Yet, as discussed in the previous chapter however, for certain traits such as people's level of prosociality, the theory seems to lag behind and clear predictions are lacking.

In the present chapter, we describe an ongoing theoretical project which attempts to expand the framework used to interpret life history variation on the fast-slow continuum. In particular, we develop two main arguments. First, that some components of people's fast *vs* slow strategies operate at timescales where differences in mortality are unlikely to account for most of the variation observed. Thus, there is a need to identify factors that could matter at shorter timescales. This leads us to our second main point, namely that there are opportunity costs associated to the time spent waiting for a delayed reward and that such costs might increase for low SES individuals. Then, factors affecting these opportunity costs should be considered alongside differences in mortality and any other factor that makes the future more uncertain.

The chapter is divided into two parts. The first is a short commentary published in the journal *Behavioural and Brain Sciences* in response to the target article published by Pepper and Nettle (2017). It contains a series of verbal arguments on the issues just raised, that provides a basis for the formal life history model developped in the second part. In the latter, we derived a measure of individuals rate of delay discounting from their optimal life history strategy. This allows us to study how discounting should vary within an individual across ontogeny and between individuals across environments. The model highlighted the importance of considering the variation in opportunity costs associated to variations in individuals' state, in order to understand changes in delay discounting. The direction in which discounting should be expected to vary however, will depend on several assumptions about the effects of an organism's allocation decisions on its state. These results are

further discussed in the form of a paper, still in preparation.

# Both collection risk and waiting costs give rise to the behavioral constellation of deprivation

**Hugo Mell, Nicolas Baumard, Jean-Baptiste André**

## Abstract

**Pepper & Nettle explain the behavioral constellation of deprivation (BCD) in terms of differences in collection risk (i.e., the probability of collecting a reward after some delay) between high and low socioeconomic status (SES) populations. We argue that a proper explanation should also include the costs of waiting per se, which are paid even when the benefits are guaranteed.**

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In an experimental study of impulsive decision making in starlings (Bateson et al. 2014), birds showing greater telomere attrition (an integrative marker of a poor biological state) were found to favor sooner-smaller rewards (one pellet of food in 1 second) over larger-later ones (five pellets in $x$ seconds). An interpretation of these results based on differential mortality risks would be as follows: Starlings in a poorer biological state have a greater probability of dying before collecting delayed rewards and should therefore privilege short-term benefits. This interpretation would be undermined, however, by the fact that dying during a choice experiment that did not exceed a few minutes is an extremely unlikely event, even for birds in poor states.

In the target article, the authors provide an explanation for the behavioral constellation of deprivation (BCD) that is mainly based on variation in extrinsic mortality. However, as in the starling example above, average differences in mortality are unlikely to account for socioeconomic status (SES) specific discounting rates when rewards are delayed over short periods (*e.g.*, weeks, months, or even a few years). This point can be illustrated in humans with the study by Ramos et al. (2013)

cited by Pepper & Nettle (P&N), which reports that slum-dwelling youth discounted future rewards more than university students. In this study, the delay used in the questionnaire did not exceed 75 days. Thus, the estimated cumulative probability of dying during the following 75 days would have had to be very high to justify the preference of sooner but smaller rewards. Such a situation, though, is not expected to hold across the majority of populations where the BCD is observed.

Hence, a gap seems to emerge once one tries to explain present orientation with differences in mortality whenever decision making is affected during short timescales. One way to address such cases in line with the target article would be to examine other factors underlying variation in collection risks (*e.g.*, individuals' social capital, population level of cooperation). However, a complementary approach that does not follow from P&N's framework would rely on factors independent of collection risk.

We see at least one corresponding source of time discounting that ought to be considered: the cost of waiting for a reward per se (i.e., the cost paid by an individual even when the benefits are guaranteed). But why should there be a cost of waiting in the absence of a collection risk? After all, in a population at a demographic equilibrium, $x$ fitness units now are strictly equivalent to $x$ fitness units later. Delaying a reward is costly, however, if this reward can be invested into an individual's capital to increase his or her future ability to exploit the environment. In such a case, delaying the reward entails an opportunity cost corresponding to the additional fitness units that would have been gained with the increased level of capital during the delay. This principle can be illustrated with a thought experiment: Imagine a farmer who participates in an economic study in which he is offered a choice between receiving \$1,000 now or \$2,000 in a month. Because this particular farmer does not own any expensive agricultural equipment, he is only able to sow half of his fields simultaneously. However, \$1,000 now would allow him to buy new equipment and exploit his whole farm. This would yield him an expected \$2,500 increase in revenue by the end of the month. Hence, our farmer should prefer the smaller-sooner reward, even though the collection risk in our example could be close to zero and the larger

reward is only delayed by a month. Instead, the fact that his current level of capital is associated with a particularly high opportunity cost in productivity determines his choice. Conversely, imagine a farmer who already owns sophisticated agricultural machines taking part in the same study. For him, $1,000 is not enough to upgrade his equipment. Rather, he is currently trying to save $15,000 by the end of the month to buy some extra land. In this case, waiting a month for the larger reward more greatly reduces the amount of money he has to save.

Such effects of the current amount of capital are likely to be pervasive. Indeed, in addition to increased productivity, as in the above example, an individual's capital can also yield a reduction in mortality risk (*e.g.*, by buying a house in a town's safest neighborhood) or protect against capital depreciation (*e.g.*, by investing in fire insurance). Crucially, the effect of capital should also directly map SES differences in temporal discounting. Although a formal treatment is needed here, we expect that when people have almost no capital, even the smallest amount of resources are likely to drastically improve their productivity or reduce their mortality. Therefore, they should generally favor sooner rewards even during shorter timescales. The more capital one already has, however, the larger the amount of resources that will be required to significantly increase it further, and the less steeply that future rewards should be discounted.

As an illustration, compare the cost one might pay for living in a small apartment rather than a house to the cost of living on the streets. In the first case, it might be noisy neighbors, the lack of a garden, or the inability to host many relatives for dinner. In the second case, however, it includes physical degradation from being exposed to climatic hazards, lack of hygiene or assaults from others, the inability to collect welfare support, social and economic exclusion in general, and so on. Therefore, someone living on the streets is likely to prefer any basic accommodation now over an individual house in 6 months, whereas someone living in a small flat might be willing to wait 6 months for an even better house.

In conclusion, ultimately, the interactions between waiting costs per se and collection risk will determine individuals' temporal discounting. Hence, by adding this novel

class of factors to P&N's framework, we can expect to deepen our understanding of the BCD.

# Delay discounting and the opportunity costs of waiting.

**Hugo Mell, Félix Geoffroy, Nicolas Baumard, Jean-Baptiste André**

## 4.2 Introduction

In western countries, individuals living in more deprived environments tend to show clusters of behaviors characteristic of a fast life history strategy. For instance, they usually have children earlier (*e.g.*, Nettle (2011)), invest less in their health (*e.g.*, Stringhini (2010)) and their education (*e.g.*, Blanden (2004)), or save a lesser portion of their financial resources for the future (Haushofer and Fehr (2014)). Rather than reflecting a lack of willpower or cognitive dysfunction, it has been argued that this so-called *behavioural constellation of deprivation* (BCD; Pepper and Nettle (2017)) and fast life histories in general reflect adaptive processes that tailor individuals' psychology and behaviour to the particular needs of their ecology (*e.g.*, Ellis et al. (2009); Frankenhuis, Panchanathan, and Nettle (2016)). The constellation includes a variety of traits that might relate to physiology (*e.g.*, sexual maturity, Webster et al. (2014)), demography (*e.g.*, age at first birth, Nettle (2011)), beliefs and social preferences (*e.g* attitudes towards authoritarian leaders, Safra et al. (2017)) or behaviour (*e.g.*, prosocial behaviour, Nettle, Colléony, and Cockerill (2011)). Thus, from an adaptationist stance, a theoretical challenge arises in identifying the selective forces and mechanisms that could explain the covariation of such a diversity of traits.

Interestingly, patterns of covariation observed in human populations often bear striking similarity to those measured for a wide range of animal species. Indeed, a fast-slow continuum has been shown to capture a significant part of life history variation and contrasts at the fast end, species with high reproductive rates, fast development and short lifespan, with the opposite suite of traits for species at the slow end (*e.g.*, Bielby et al. (2007); Sæther (1988)). More recently, based on a wave of empirical and theoretical work mostly from the field of animal personality, the

life history fast-slow continuum has been extended to the more general concept of a Pace of Life Syndrome (POLS; Réale et al. (2010); Ricklefs and Wikelski (2002)). In addition to the covariation of classic life history traits such as age at maturity, number of offspring or lifespan, the POLS inculdes as well on the fast-slow continuum traits that relate to physiology and behaviour. Moreover, the idea of a pace of life is used to capture both between and within species variation. Hence, evolutionary explanations are once again needed to explain the broad covariation of traits described empirically in non human species, which might at the same time give important insighs on the parallel phenomenon of the BCD found in human populations.

Although formal models of the pace of life syndrome are still largely lacking (Mathot and Frankenhuis 2018), most of the verbal explanations given in the litterature have conceptualized fast-slow strategies as opposite ways to resolve the tradeoff between current and future reproduction. In the case of slow strategies, an organism foregoes the benefits of immediate reproduction by delaying its age at maturity, but will develop in a way (*e.g.*, increase body size) such that benefits obtained from reproduction at older ages will overcompensate the immediate losses. As a consequence, some associations between traits are predicted to be suboptimal (*e.g* a late age at maturity and very bold juveniles), and correlational selection pressures will ensure that values for interdependent traits are fine tuned to one another (Bell and Sih 2007; Réale et al. 2010). Overall then, slow traits should share the property of favoring larger-later gains over smaller-sooner ones. Thus, the distinction between fast and slow strategies would have its roots in differences in delay discounting reflecting the degree of intensity of the current *vs* future reproduction tradeoff. A similar interpretation could be advanced for the BCD as well (Pepper and Nettle 2017). Indeed, empirical observations show that significant variation in discounting rates exists both at the country and individual levels in human populations (Wang, Rieger, and Hens 2010, 2016), and that some of this variation correlates with indicators of deprivation (country GDP or level of income). Thus, the theoretical challenge now becomes to explain why different environments should elicit different rates of delay discounting.

The explanation classically offered states that a short-term strategy should be selected in ecologies where the probability to get a reward at the end of a delay is low (*i.e.*, the future is uncertain). Borrowing the terminology used in behavioural ecology (Fawcett, McNamara, and Houston 2012; Houston, Kacelnik, and McNamara 1982), the last statement is equivalent to say that delay discounting should increase when the collection risk associated to the delayed option is high. Indeed, in a situation where the delayed reward will be almost certainly impossible to collect, the delayed option should be chosen only if the differences in quantity or value between the two rewards is extremely large. Hence, this predicts that we should be able to identify specific factors in deprived ecologies that increase collection risk and that are causally linked to the development of faster life history strategies.

Arguably, extrinsic mortality (and morbidity) is the factor most frequently mentioned to explain differences in life history strategies for human populations (Ellis et al. 2009; Nettle, Frankenhuis, and Rickard 2012). Its theoretical relevance to explain variation in delay discounting is undoubted, as it captures the simplest form of collection risk. Indeed, the risk of dying (or being incapacitated in the case of morbidity) during the waiting period is one reason why the reward might never be collected. Although the extrinsic aspect of mortality is often hard to assess, empirical studies consistently show that higher mortality rates correlate with various fast life history attributes in both human populations and non-human species (*e.g* Charnov (1991); Gordon et al. (2009); Quinlan (2010); Stearns (2000)).

Yet, the magnitude of the differences in extrinsic mortality in most developed societies calls into question its explanatory power for certain components of the BCD (Mell, Baumard, and André 2017; Riis-Vestergaard and Haushofer 2017). Indeed, some traits might involve decisions about rewards that are delayed over relatively short timescales (*e.g.*, days, months, a few years). In contrast, differences in life expectancies between people coming from deprived vs favorable environments are usually of a few years in western countries, which suggests limited variations in extrinsic mortality rates. Nevertheless, a small initial increase in mortality due to extrinsic factors could actually decrease the incentives for individuals to invest in

their own health, such that disparities in mortality might eventually grow larger as people get older (Nettle 2010). Although interesting and quite plausible, the idea of feedback loops accentuating mortality gradients is not necessarily a straightforward prediction when mortality is modelled with an intrinsic component (Shokhirev and Johnson 2014). Furthermore, whether these reinforcing effects would lead to sufficient increases in discounting to explain the empirical data is unknown.

Thus, considering other collection risk factors is likely to allow for a better account of the data. For example, in highly competitive environments, waiting for a delayed reward might be a risky strategy as the probability to see it disputed by a conspecific increases. Sooner-smaller rewards would therefore also tend to be preferred in such an environment, independently of any differences in mortality rates. Similarly, the average degree of trustworthiness of local social partners should affect delay discounting, since the uncertainty around others' intentions will generate some risk that rewards at stakes in cooperative interactions will not be collected.

Hence, it seems that shifting the focus from extrinsic mortality, to collection risk factors that are still salient at shorter timescales, has the potential to produce new insights on the origins of fast-slow strategies. Yet, in this paper we argue that, even a theory of delay discounting including every imaginable sources of collection risk, would still be insufficient to fully account for the variance measured in individual discount rates. But why should we expect two individuals that are exposed to the exact same degree of collection risk, to discount future gains differently, beyond random genetic and developmental effects? As highlighted in a short commentary recently plubished (Mell, Baumard, and André 2017), to predict whether a larger-later reward should be preferred over the smaller-sooner one, requires to know the actual cost of not having the smaller reward during the delay. In other words, there is a cost of waiting for the reward *per se* due to the loss of investment opportunities. Indeed, 100$ now should be preferred over 200$ in a month as long as they can be invested in ways that are expected to yield more than 200$ within the same time frame.

The importance of considering the loss of opportunities in situations of intertemporal

choice is not a new one (*e.g* Soman et al. (2005); Urminsky and Zauberman (2015)) and in particular the disctinction between collection risk and opportunity costs associated to the waiting period has been clearly drawn in the field of behavioural ecology (see Stevens and Stephens (2010)). Yet, formal models incorporating this type of cost to further explain the links between delay discounting and the fast-slow continuum are lacking. Below, we described an attempt to build a mathematical model that allows us to explore variations in delay discounting, in the context of an organism's life history. The core of the model is based on the verbal arguments exposed in our previous commentary (Mell, Baumard, and André 2017). Indeed, by modelling the incremental process by which an organism might acquire more capital to increase its productivity or have more control over its mortality risk, we want to show that changes in an organism's state will yield differences in delay discounting. In particular, we want to highlight the fact that the accumulation of capital can generate changes in discounting even when collection risk remains constant. To this aim, we represent the life cycle of an organism as a sequence of allocation decisions distributing energy between growth and reproduction. The following section describes in details such a life cycle and the associated optimal allocation problem.

## 4.3 Allocation model

### 4.3.1 Biological scenario

Consider the following stylized life cycle, where an organism extracts resources from its environment at a certain rate, dedicates part of these resources to maintenance and divide the surplus between growth and reproduction. At each instant, the organism may die with a certain probability from various sources of mortality. Energy allocated towards reproduction directly contributes to the organism's fitness, whereas resources invested in growth increase its survival and productivity at subsequent ages.

Growth occurs through the incremental acquisition of various *features* with specific effects on mortality protection and resources extraction. Examples of *protective features* include the production of toxins and repulsive substance against herbivores by numerous plant species, the shell of a turtle or the alarm system of a house.

Similarly, examples of *productive features* include the long, thin adhesive tongue of an ant-eater, the ability of jays to remember the location of seeds burried months earlier or the mechanical tractor that allows a farmer to labour several hectars of fields in a day. More generally, a *feature* might be any physical structure or skill that an organism can acquire during its development to improve its survival or productivity.

Importantly, in the case of social species, individuals' resource acquisition rate and exposure to mortality might largely depend on the *features* of their conspecifics. It is particularly apparent in human societies where extensive parental care as well as transfers between non kin are vital and ubiquitous (Gurven 2004; Hill and Hurtado 2009; Kaplan et al. 1985). Similarly, good housing conditions during childood or the use of medical innovations such as vaccins are examples of *features* coming from an individual's social environment that will also greatly reduce mortality risk. Thus, adopting an extended phenotype perspective, the *state* of an organism will encompass both features acquired individually and provided by the social environment.

For clarity, the amount of energy accumulated for each *feature* is referred to as the *size of the feature*, while the total energy invested across *features* constitutes the organism's *capital*. When an organism invests energy to further develop a *feature*, it is assumed that it will subsequently devote the appropriate amount of resources to maintain its new state. Hence, the size of a feature can only grow or remain constant in this scenario (*i.e.*, there is no depreciation of capital).

Finally, the net energy available for allocation at each time point is completely determined by the current size of the organism's *productive capital*.

Eventually, the biological scenario described above captures the central problem of life history theory, namely *"Which proportion of the net energy available now should be used for growth, to the detriment of current reproduction, in an attempt to increase future reproductive prospects?'*. This dynamic allocation problem has been formalized and studied on multiple occasions in the life history litterature from the 1970's onwards (León 1976; Perrin and Sibly 1993; Taylor et al. 1974). The following section, builds on this previous body of work to provide a mathematical

formulation of our stylized life cycle (see Perrin (1992) in particular for an analogous model).

### 4.3.2 Formalizing the allocation model

Throughout the life cycle of the organism, time $t$ is measured continuously and starts at $t_0 = 0$, which marks the birth of the organism. There is no maximum lifespan associated to the life cycle, instead the expected life expectancy depends on the amount of energy an organism might invest throughout its life to mitigate mortality risk $\mu$.

For each $t$, the state of the organism is fully characterized by two variables: its capital $\mathbf{x}(\mathbf{t})$ and the probability $\ell(t)$ that it is still alive at time $t$. The organism's capital $\mathbf{x}(\mathbf{t})$ is a vector composed of the size $x_i$ of each feature at time $t$:

$$\mathbf{x}(\mathbf{t}) = \begin{bmatrix} x_1(t) \\ x_2(t) \\ \vdots \\ x_N(t) \end{bmatrix},$$

where $N$ is the number of unique features that an organism might acquire during its lifetime (*i.e.*, the size of the *feature set*). The total size of an organism's capital can consequently be defined as $X(t) = \sum_{i=1}^{N} x_i(t)$.

As described in the previous section, the net energy available for allocation at each $t$ is a function of the organism's capital at that time, noted $P(\mathbf{x}(\mathbf{t})) = P(x_1(t), x_2(t), \ldots, x_N(t))$. The organism's energy surplus at birth is given by $P_0 = P(\mathbf{x}(\mathbf{0}))$, which depends on the initial state of the organism's capital at $t_0$. The initial capital endowment $\mathbf{x_0}$ is exogenous and comes mainly from the features provided by its social environment. For simplicity, the fraction of the organism's energy surplus due to the social environment is assumed to be constant throughout the life cycle.

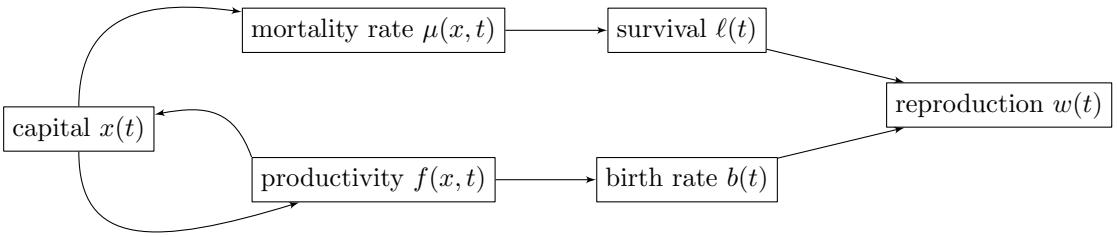


Figure 4.1: Schematic diagram of the allocation model.

### 4.3.3 Monitoring changes in state

The state of the organism can be tracked by a set of $N$ differential equations that record the dynamic of the size $x_i(t)$ of each feature, as well as a differential equation that records changes to the organism's probability $\ell(t)$ to be alive at each $t$.

The state equations associated to the organism's *capital* are thus defined for $i \in \{1, \ldots, N\}$ as:

$$\frac{dx_i}{dt} = u_i(t) \cdot P(\mathbf{x}(\mathbf{t})), \tag{4.1}$$

where $u_i(t) \in [0, 1]$ and refers to the fraction of the energy surplus $P(\mathbf{x}(\mathbf{t}))$ that is used to increase the size of feature $i$ at time $t$. Hence, $u(t) = \sum_{i=1}^{N} u_i(t) \leq 1$ gives the proportion of the available resources allocated to growth at time $t$.

Note that the dynamic of the *capital size* can be obtained directly from the state equation for the size of the individual features:

$$\frac{dX}{dt} = \sum_{i=1}^{N} \frac{dx_i}{dt} = u(t) \cdot P(\mathbf{x}(\mathbf{t})) \tag{4.2}$$

Finally, the state equation associated to the probability $\ell(t)$ to reach any particular age is:

$$\frac{d\ell}{dt} = -\mu(\mathbf{x}(\mathbf{t})) \cdot \ell(t) \tag{4.3}$$

Like the productivity function $P$, the instantaneous mortality rate $\mu$ is a function of

the organism's capital $\mathbf{x(t)}$. However, to lighten the notation $P(\mathbf{x(t)})$ and $\mu(\mathbf{x(t)})$ are hereafter simply written as $P(t)$ and $\mu(t)$ respectively.

### 4.3.4 Current reproduction and fitness

As stated previously, all resources that are not consumed to increase capital size (*i.e.* not allocated to growth) are used for reproduction and contribute directly to fitness. The instantaneous birth rate of the organism, $b(t)$, can therefore be expressed as:

$$b(t) = (1 - u(t)) \cdot P(t) \tag{4.4}$$

Hence, the allocation policy of the organism's life cycle is the matrix $\mathbf{U} = (u_{i,t})_{i\in\{1,\dots,N\},t\geq 0}$, which specifies at each age the proportion of the available resources to be steered towards the growth of each feature rather than immediate reproduction. It captures all the allocation decisions made across the organism's lifetime and will evolve by natural selection depending on its overall impact on the life cycle's fitness, $W$. The latter is defined here as the organism's lifetime reproductive success, given by:

$$W = \int_0^\infty \ell(t)b(t)dt \tag{4.5}$$

Thus, the optimal allocation policy is defined as the matrix of allocation decisions $\mathbf{U}^* = (u^*_{i,t})_{i\in\{1,\dots,N\},t\geq 0}$, wich divides resources between individual features and between growth and reproduction such that the value of $W$ associated to the life cycle is maximized. The whole life history of the organism can then be deduced from the optimal policy $U^*$ by computing for each age the size of each feature $x_i(t)$, the mortality risk $\mu(t)$ with the associated probability of being alive $\ell(t)$, and the instantaneous birth rate $b(t)$.

### 4.3.5 Optimal control and Pontryagin's Maximum Principle

As recognized early by life history theorists, this formulation of the general life history problem makes it fall into a wider class of dynamic optimization problems that can be analyzed using optimal control theory (León 1976; Taylor et al. 1974). In particular, Pontryagin's Maximum Principle (Pontryagin, Neustadt, and Pontryagin 1986) states that for $\mathbf{U}^*$ to constitute an optimal policy implies that it maximizes at each t the function $H$ defined as follows:

$$H(\mathbf{x}(\mathbf{t}), \mathbf{u}(\mathbf{t}), \lambda(\mathbf{t}), t) = \ell(t) \cdot b(t) + \lambda_i(t) \cdot \frac{dx_i}{dt} + \lambda_{N+1}(t) \cdot \frac{d\ell}{dt} \tag{4.6}$$

The function $H$ is called the *Hamiltonian* and depends on four types of variables: time $t$, the vector of state variables $\mathbf{x}(\mathbf{t})$ (note that $\ell$ is actually another state variable and could be written as $x_{N+1}$), the vector of control variables $\mathbf{u}(\mathbf{t})$ and the vector of socalled *costate variables* $\lambda(\mathbf{t})$. To simplify the notation, $H(\mathbf{x}(\mathbf{t}), \mathbf{u}(\mathbf{t}), \lambda(\mathbf{t}), t)$ is written hereafter as $H(t)$. The Hamiltonian function thus introduces an additional type of variable, the costates $\lambda$, which measure the marginal effect on fitness $W$ of a small change in each of the state variable (Dorfman 1969; Iwasa and Roughgarden 1984).

The dynamic of all costates $\lambda$ is directly linked to the Hamiltonian $H(t)$ through the following relationship:

$$\frac{d\lambda_i}{dt} = -\frac{\partial H}{\partial x_i}, \tag{4.7}$$

with the associated limit condition on each $\lambda$:

$$\lim_{t \to \infty} \lambda_i(t) = 0 \tag{4.8}$$

The necessity of the above limit conditions can be understood intuitively in the case of a biological life cycle, since as time goes to infinity the probability $\ell(t)$ that the

organism remains alive reaches zero. Therefore, the impact on fitness of further changes to the organism's state will eventually be cancelled by the death of the organism.

Similarly, an intuitive biological justification can be given for the Hamiltonian equation and Pontryagin's Maximum Principle in the context of life history problems (Iwasa and Roughgarden (1984); Perrin and Sibly (1993); Taylor et al. (1974); see Dorfman (1969) for an economics based interpretation). The following section further outline some of these insights.

### 4.3.6 Biological interpretation of the Hamiltonian equation and Pontryagin's Maximum Principle

#### The costates $\lambda$ as marginal values

In order to grasp the biological meaning of the hamiltonian equation and the logic behind Pontryagin's maximim principle, first recall that the costates $\lambda_i(t)$ associated to each of the $N$ organs can be viewed as the marginal values of a unit increase of the organ size at time t. Figure 4.2 illustrates this interpretation for an hypothetical life cycle with a *bang bang* life history strategy (*i.e.*, ontogeny consists of a pure growth phase followed by a pure reproductive phase after maturity).

Consider a simple scenario where there is a single feature in the feature set with effects either on productivity only (Figure 4.2 A) or mortality only (Figure 4.2 B). The black curves represent the trajectories of the feature size $x(t)$ and instantaneous reproduction $\omega(t)$ for a reference life cycle with allocation policy $\mathbf{U}$. The red and green curves show the effects on these trajectories of a unit perturbation $\Delta$ of the feature size $x$ at different time points (respectively $t_1^*$ and $t_2^*$).

Since the life history strategies are *bang-bang*, the general structure of the life cycle is always the same. The size of the feature $x(t)$ first increases continuously until it reaches the value $x_\alpha$ at a particular time $t_\alpha$ when the organism matures. After maturity, $x$ remains constant at the value $x_\alpha$ and instantaneous reproduction $\omega$ starts. As the switch to reproduction occurs, $\omega$ starts at its peak value $\omega_\alpha$, before

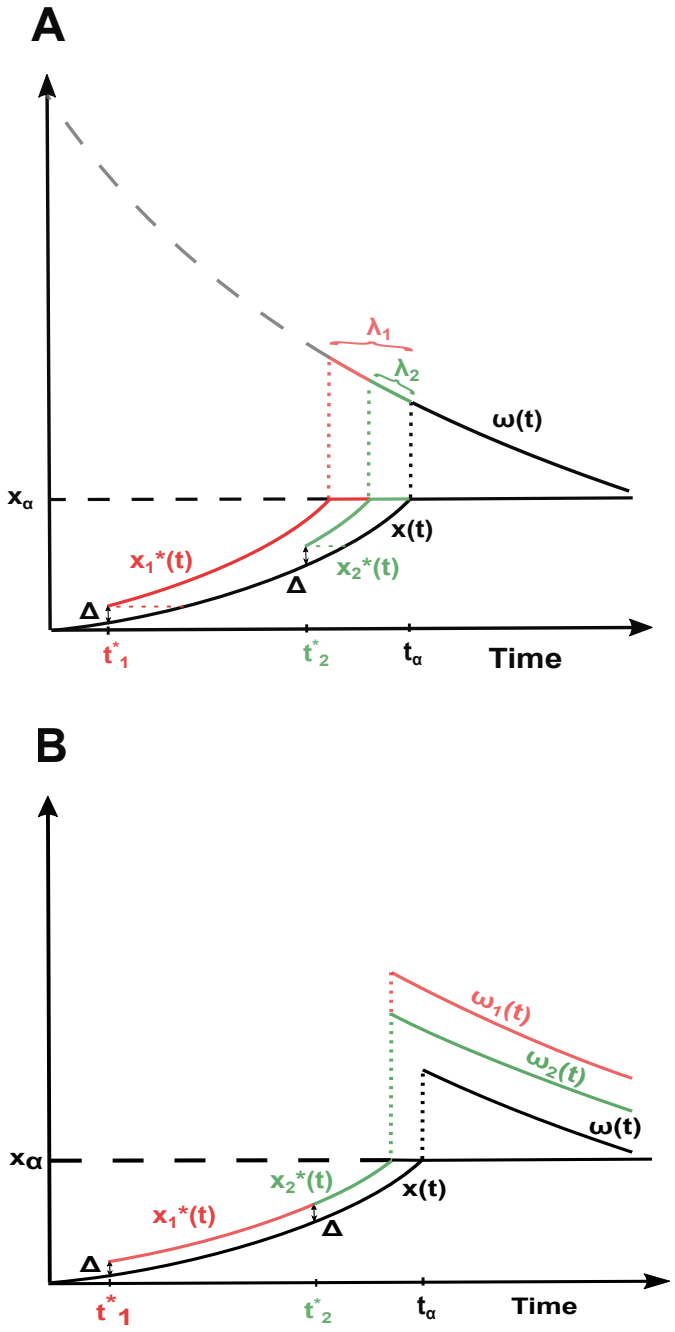


Figure 4.2: Marginal interpretation of the costate lambdas. **A**. The feature set is only composed of a single *productive* feature. **B**. The feature set is only composed of a single *protective* feature.

decreasing gradually towards zero as the probability $\ell(t)$ to stay alive does.

To understand the effects introduced by a unit pertubation $\Delta$, consider first a case where the feature only affects energy acquisition (Figure 4.2 A). The red curves capture the effect of a perturbation $\Delta$ that occurs relatively early during the organism's life cycle, at time $t_1^*$. Before $t_1^*$, the trajectories of the feature size are identical for the two life cycles. At $t_1^*$, $x_1^*(t_1^*) = x(t_1^*) + 1$ as a result of the unit perturbation $\Delta$. A larger feature yields a higher productivity, which during the growth phase will be used to further increase feature size. As a consequence, the perturbed trajectory $x_1^*(t)$ will increase at a faster rate for all subsequent times. Indeed, right after the perturbation the organism can grow at a rate $P(x(t_1^*)+1)$, that would otherwise have been attained only at a later stage of its development.

Thanks to the growth acceleration induced by the perturbation, the value $x_\alpha$ at which the organism switches to reproduction will be reached earlier. Therefore, the perturbed life cycle includes an additional period of reproduction from $t_{\alpha_1}$ to $t_\alpha$, when $P(x_\alpha)$ units of energy are invested into reproduction at each time. To obtain the instantaneous reproduction $\omega(t)$ during this period however, the birth rate must be discounted by the probability that the organism is still alive, $\ell(t)$ ($\omega(t) = b(t)\ell(t)$). $b$ is constant and equals to $P(x_\alpha)$ throughout the reproductive phase for both the perturbed and the reference life cycles. On the other hand, $\ell(t)$ decreases steadily at a pace determined by the mortality risk $\mu$. Hence, the instantaneous reproduction of the perturbed life cycle starts at a higher peak value $\omega_\alpha^*$ and decreases to the value $\omega_\alpha$ at $t_\alpha$. Finally, from $t_\alpha$ onwards, $\omega^*$ and $\omega$ will follow the same trajectory. Thus, the additional reproductive output between $t_{\alpha_1}^*$ and $t_\alpha$ is the marginal value $\lambda_1$ of the perturbation $\Delta$ performed at time $t_1^*$, which measures the expected rise in fitness ultimately produced by a unit increase of the feature size $x$ at $t_1^*$.

Furthermore, the marginal value interpretation of the costates *lambda* also makes apparent some property of their dynamics throughout the life cycle. Specifically, a similar reasoning shows that the value of $\lambda$ must decrease with time. Indeed, consider another unit perturbation $\Delta$ of the feature size $x$ that would this time be performed at a later time $t_2^*$ during the organism's life cycle. The green curves

in Figure 4.2 capture the effects triggered by the perturbation on $x$ and $\omega$ from which $\lambda_2$ is reconstructed. Because of the feedback loop between feature size and productivity (cf Figure 4.1), $\lambda_2$ ought to be smaller than $\lambda_1$. Indeed, in the case of a productive feature, it is the reduction in the time necessary to reach the size at maturity $x_\alpha$ that determines the marginal value of the perturbation. The speed $\frac{dx}{dt}$ at which growth occurs is the lowest at the beginning of the organism's life cycle and increases continuously until maturity. Hence, the time to grow an extra unit of feature spared by the perturbation $\Delta$ will become comparatively shorter as $\frac{dx}{dt}$ increases during develoment. Thus, the effect on fitness of later perturbations will always be smaller and the costate $\lambda$ must decrease monotonously with time.

The same conclusion can be drawn for a feature that would only reduce mortality risk (Figure 4.2 B). In this case, the effect of the perturbation $\Delta$ on the age at maturity $t_\alpha$ is relatively small. No matter when the perturbation is done, $t^*_\alpha$ will always correspond to the time at which the value $x(t) = x_\alpha - 1$ is reached alongside the reference trajectory. Indeed, there is no feedback loop between productivity and feature size $x$ in this scenario, so the rate $\frac{dx}{dt}$ is unaffected by the perturbation $\Delta$. The main effect of the perturbation comes from the reduction of the mortality rate, which follows the increase in $x$ and slows down the pace at which the survival probability $\ell(t)$ gradually falls. Thus, the earlier the perturbation, the higher the probability that the organism survives until maturity. Consequently, $\omega^*(t)$ will necessarily start at a higher value for earlier perturbations (Figure 4.2). After maturity, $\ell$ decreases at the same the rate $\mu(x_\alpha)$ for all trajectories, such that $\omega^*$ will remain comparatively higher for the earliest perturbation.

Thus, the decrease in marginal values $\lambda$ over time must hold both for productive and protective features.

## Biological intuition behind Pontryagin's Maximum Principle

Recall that using Pontryagin's Maximum Principle, the optimal allocation policy has been specified as the one that maximizes the Hamiltonian equation for each instant $t$:

$$\begin{aligned} H &= \ell(t) \cdot b(t) + \lambda_i(t) \cdot \frac{dx_i}{dt} + \lambda_{N+1}(t) \cdot \frac{d\ell}{dt} \\ &= \ell(t) \cdot (1 - \mathbf{u(t)}) \cdot P(t) + \lambda_i(t) \cdot u_i(t) \cdot P(t) - \lambda_{N+1} \cdot \mu(t) \cdot l(t) \end{aligned}$$

$$H(t) = \ell(t) \cdot b(t) + \lambda(t) \cdot \frac{dx}{dt} + v(t) \cdot \frac{d\ell}{dt}$$

Based on the intuitive interpretation of the costates built up in the previous section, H(t) can now be seen as the sum of the effects on the life cycle's fitness $W$, of all events that occured at time $t$. Indeed, the first term of the equation corresponds to $\omega(t)$, that is the immediate fitness contribution obtained by reproducing at that age, discounted by the probability to reach that age. From the marginal value interpretation of $\lambda_i$, the second term of the equation sums the gains in fitness resulting from the energy used to grow the features at time t (which are eventually collected through the increase of $\omega$ at some older ages). Finally, the third term captures the loss in reproductive prospects due to the decrease in survival probability $\ell$.

In this particular model, only the first two terms are affected by the allocation decisions $u(t)$. Hence, Pontryagyn's Maximum Principle tells us that the values of u(t) should be chosen such that their sum is always maximized. Since the first term is the immediate contribution to fitness at $t$, and the second the fitness benefits ultimately obtained through the growth of the features, the necessary condition that the optimal policy satisfies Pontryagin's Maximum Principle now becomes biologically intuitive.

### 4.3.7 Deriving the optimal allocation schedule

As shown in Perrin (1992) for an analogous model, the general structure of the optimal allocation schedule can be deduced from the Hamiltonian and costate equations. The next section briefly reports the main analytical results regarding the allocation schedule that are needed for the treatment of temporal discounting. A more detailed account of their derivation is given in the supplementary information.

Consider a simple version of our biological scenario which involves only two features, each of them having potential effects on both productivity and mortality risk. Productivity and mortality are assumed to be strictly concave functions of the features sizes, and at each time the organism must choose the proportions $u_1(t)$ and $u_2(t)$ of the energy surplus to be allocated to feature 1 and feature 2 respectively. For any baseline value of $u_1$, a one percent rise in the resources allocated to feature 1 should be made only if the following two conditions are satisfied: 1) a direct investment in reproduction increases the Hamiltonian by a lesser amount and 2) the same is true for an investment in feature 2.

These conditions can be checked by taking the derivatives of the Hamiltonian (see Equation 4.6) with respect to $u_1$ and $u_2$:

$$\begin{cases} \frac{\partial H}{\partial u_1} = P \cdot (\lambda_1 - \ell) \\ \\ \frac{\partial H}{\partial u_2} = P \cdot (\lambda_2 - \ell) \end{cases}$$

The first condition is then equivalent to $\frac{\partial H}{\partial u_1} > 0$, which is true whenever $\lambda_1 > \ell$. The second condition is satisfied when $\frac{\partial H}{\partial u_1} > \frac{\partial H}{\partial u_2}$, which means that $\lambda_1 > \lambda_2$. Conversely, increasing the investment in feature 2 is advantageous whenever $\lambda_2 > \lambda_1, \ell$.

Since the above conditions do not depend on the values of the controls $u_1$ and $u_2$, they must lead to the exclusive allocation of the whole energy surplus to one of three options: either all resources are allocated to reproduction and $u_1, u_2 = 0$, all resources are allocated to feature 1 ($u_1 = 1$, $u_2 = 0$), or inversely all resources are allocated to feature 2 ($u_1 = 0$, $u_2 = 1$).

Yet, simultaneous allocation of resources between the two features or between growth and reproduction might still arise if the marginal values of the best options equalize over a non-zero period (Perrin 1992). In the case of simultaneous allocation between features 1 and 2, this would mean that at some time during the life cycle, $\lambda_1(t) = \lambda_2(t)$ and $\frac{d\lambda_1}{dt} = \frac{d\lambda_2}{dt}$. For all $t$ before the age at maturity $t_\alpha$, the value of the costate $\lambda_i(t)$ of any feature in acquisition is given by the expression (see Supplementary information for more details):

$$\lambda_i(t) = \frac{\mu(t)}{P(t)} + \frac{P_0\lambda_0 - \mu 0}{P(t)} \tag{4.9}$$

Then, it can be shown that the conditions for simultaneous allocation between the features implies that the following equation is verified:

$$\frac{\partial \frac{P}{\mu+k}}{\partial x_1} = \frac{\partial \frac{P}{\mu+k}}{\partial x_2}, \tag{4.10}$$

where $k$ is a constant defined as $k = \lambda_0 P_0 - \mu_0$.

Furthermore, since $\lambda_1(t)$ and $\lambda_2(t)$ decrease monotonously with time, they will eventually reach the value of $\ell$ at which point the organism should mature and start reproducing. From the conditions imposed by Pontryagin's Maximum Principle at the age at maturity, it can be shown that the switch to reproduction occurs when:

$$\frac{\partial \frac{P}{\mu}}{\partial x_1} = \frac{\partial \frac{P}{\mu}}{\partial x_2} = 1 \tag{4.11}$$

Equation 4.11 therefore tells us that no energy should be allocated to growth once returns no longer exceed investments in terms of the ratio of productivity over mortality, $\frac{P}{\mu}$.

Thus, it is now possible to sketch the general structure of the optimal life cycle for such a scenario. First, the organism will start by allocating all of its energy to the feature whose costate $\lambda$ has the highest value. In pratice, it will be equivalent to choosing the

feature which yields the greatest increase of the ratio, $\frac{P}{\mu+k}$. The value of $\lambda$ associated with this feature will decrease with time and if at some point before maturity it equalizes with the other costate, the organism will then invest simultaneously between the two features. Such simultaneous allocation occurs whenever during the growth phase investing energy in feature 1 or 2 produces the same increase of $\frac{P}{\mu}$. Because of the concavity of $P$ and $\mu$, the effects of the features on both productivity and mortality will eventually decrease to the point where equation (number) is satisfied and returns from growth no longer exceed investments. At this time the values of both costates equal $\ell(t)$ and the organism matures. Once maturation has occured, a purely reproductive phase begins during which the organism produces offspring at a constant rate $b = P(x_\alpha)$, while its probability to remain alive $\ell$ gradually decreases towards zero.

## 4.4 Measuring time discounting

Formally, time discounting can be defined via a differential equation that tracks the decrease in the value $v(t)$ of any outcome with time:

$$\frac{dv}{dt} = -\delta(t)v(t), \tag{4.12}$$

where $\delta$ is the *instantaneous discount rate* and measures the intensity at which an individual discount future outcomes.

In the context of our model, $\delta$ can be similarly characterized as the rate at wich the value of a resource unit decreases with time. Let us note $\gamma(t)$ the value of a resource unit at any point of the organism life cycle. Then, we can rewrite Equation 4.12 as: $\frac{d\gamma}{dt} = -\delta(t)\gamma(t)$, which gives for the discount rate $\delta$:

$$\delta(t) = -\frac{\frac{d\gamma}{dt}}{\gamma(t)} \tag{4.13}$$

$\gamma(t)$ should reflect the additional fitness units that can be obtained by using a unit of resource at time $t$ to either grow or reproduce. As seen in the previous sections,

in the case of growth these quantities are given by the value of the costates $\lambda_i$ for each feature in the available set. The marginal value for direct reproduction on the other hand always equals one, since one unit invested into reproduction now yields an equivalent unit increase in fitness according to our model definition.

Then, during the growth phase the marginal value $\lambda(t)$ will be equal to the feature's costate $\lambda_i$ with the highest value:

$$\lambda(t) = max\ \lambda_{i\in\{1,\dots,N\}}(t) \tag{4.14}$$

Note that during a period of simultaneous investment between features, the costates $\lambda_i$ of all the features that receive some energy must be equal and $u(t) = \sum_i^N u_i(t) = 1$, which means that the previous equation still holds. After maturity, all available energy is used for direct reproduction so that we simply have $\lambda(t) = 1$.

Yet, to properly measure the value of a resource at $t$, the probability that the organism is still alive at that point in time must be taken into account. Hence, the expected fitness benefits given by $\lambda(t)$ ought to be discounted by the survival probability $\ell(t)$, giving the following formula:

$$\gamma(t) = \lambda(t)\ell(t) \tag{4.15}$$

Reinjecting Equation 4.15 into Equation 4.13 gives:

$$\delta = -\frac{\frac{d(\lambda\cdot\ell)}{dt}}{\lambda\ell} = -\frac{\frac{d\lambda}{dt}\ell + \lambda\frac{d\ell}{dt}}{\lambda\ell} = -\frac{\frac{d\lambda}{dt}}{\lambda} + \mu(t) \tag{4.16}$$

, which characterizes an organism's discount rate at each point of its life cycle. Since the costates $\lambda$ decrease monotonously with time, $\frac{d\lambda}{dt}$ is always negative and conversely the discount rate $\delta$ will always be positive. Individuals therefore prefer at each stage of their life cycle a resource now over the same quantity of resource obtained later. However, even though the sign of $\delta$ implies that an organism will always discount future rewards, the intensity of the discounting will vary during the

organism's lifetime according to the changes in $\frac{\frac{d\lambda}{dt}}{\lambda(t)}$ and $\mu(t)$. Hence, the following section describes how the discount rate $\delta(t)$ is expected to change across ontogeny for various biological scenarios.

### 4.4.1 Time discounting across ontogeny

The optimal life cycle is divided into two phases, a growth period followed by a pure reproductive period were productivity and mortality remain constant at the value $P_\alpha$ and $\mu_\alpha$ respectively. Starting with the end, the discount rate $\delta(t)_{t>t_\alpha}$ during the reproductive period equals $\mu(\mathbf{x}_\alpha)$, since $\lambda(t) = 1$ and $\frac{d\lambda}{dt} = 0$. This means that during the reproductive period, the rate at which immediate resources are preferred reflects entirely the risk of dying before collecting the resources an instant later.

During the growth phase however, the discount rate will depend on the relative variations of $\frac{\frac{d\lambda}{dt}}{\lambda(t)}$ and $\mu(t)$. Further specifications of the way features affect productivity and mortality are therefore needed to gain additional insights about the fluctuations of $\delta$ before maturity.

#### Discounting during the growth phase with a single feature

Consider a basic scenario where there is a single feature available to the organism, that might have effects on both mortality and productivity. Before growth, the differential equation for the costate is given by (see Supplementary information):

$$\frac{d\lambda}{dt} = -\frac{\partial P}{\partial x}\lambda(t) + \frac{\partial \mu}{\partial x} \tag{4.17}$$

Reinjecting Equation 4.17 into the expresion for the discounting $\delta(t)$ yields:

$$\delta(t) = \frac{\partial P}{\partial x} - \frac{1}{\lambda(t)}\frac{\partial \mu}{\partial x} + \mu(t) \tag{4.18}$$

From equation 4.18, it is now possible to discuss the expected variations in discounting across ontogeny under various scenarios.

First, if the feature only affects resource extraction, then the previous expression

reduces to:

$$\delta(t) = \frac{\partial P}{\partial x} + \mu(t) \tag{4.19}$$

Hence, when there is a single productive feature, the discount rate depends on two terms: $\mu$ which remains constant and captures the collection risk associated with mortality, and $\frac{\partial P}{\partial x}$ which measures the sensitivity of the production function to an increase in the feature size. Hence, the trajectory of the discount rate during the first stage of the life cycle will depend entirely on the form of the production function. Indeed, whether $P$ shows increasing, constant or diminishing returns with respect to feature size will determine the trend in discounting. At one end, increasing returns lead to individuals that discount more as they grow (*i.e. fast* individuals), while the opposite will be true in the case of diminishing returns (*i.e. slow* individuals).

As reflected in the *law of diminishing returns*, any production function is expected to saturate with regard to capital input at some point. Even though a starting phase of increasing returns, during which additional units of capital have synergistic effects might be a reasonable assumption in many cases, diminishing returns are eventually expected to appear as more and more units are added, yielding an S-shape production function $P$. Thus, due to the law of diminishing returns, individuals should become tendencially slower as they grow. The decrease in discounting will hold for the whole growth phase if $P$ shows strictly diminishing returns. If $P$ has an S-shape however, individuals will discount more until the inflexion point of the production is reached and then get slower until they reach maturity.

For the opposite scenario in which there is a single protective feature, the discount rate is given by:

$$\delta(t) = -\frac{1}{\lambda(t)}\frac{\partial \mu}{\partial x} + \mu(t) \tag{4.20}$$

Plugging into the previous equation the expression for $\lambda(t)$ before maturity (see Equation 4.9) yields:

$$\delta(t) = -\frac{\frac{\partial \mu}{\partial x}}{\mu(t) + \lambda_0 P_0 - \mu_0} P_0 + \mu(t) \tag{4.21}$$

Under this scenario, the second term $\mu(t)$ is no longer constant but will decrease as the feature develops. The direct reduction of mortality during the growth stage will then logically tend to make individuals slower. However, whether the the first term also makes individuals slower as they grow depends on the variations of the ratio $\frac{\frac{\partial \mu}{\partial x}}{\mu(t)}$ over time (the term $\lambda_0 P_0 - \mu_0$ being constant). Note first, that $\frac{\frac{\partial \mu}{\partial x}}{\mu(t)}$ is the proportional rate of change of $\mu$ with respect to feature size $x$. Since the feature's function is specifically to protect the organism from various sources of mortality, $\frac{\frac{\partial \mu}{\partial x}}{\mu(t)}$ must be negative and conversely $-\frac{\frac{\partial \mu}{\partial x}}{\mu(t)}$ is positive.

Hence, three distinct situations might arise depending on whether $\frac{\frac{\partial \mu}{\partial x}}{\mu(t)}$ increases, remains constant or decreases as the feature grows. An increase of the ratio would mean that as $\mu$ decreases, a unit increase of $x$ yields a smaller percentage reduction of the mortality rate. Such a situation could be seen as a a stricter form of diminishing returns, where the loss of efficiency of the feature is no longer in absolute terms but also in a relative sense. In this case, older individuals get slower both due to the decrease in mortality and the loss in relative efficiency to further reduce it as they grow.

If the ratio stays constant, then a unit increase of $x$ always yields the same percentage reduction in $\mu$. The loss of efficiency this time is therefore true only in an absolute sense. Mortality in this case will show a typical exponential decay with respect to feature size and individuals will get slower as mortality diminishes.

However, if the relative efficiency of the feature in protecting against mortality improves as it gets bigger, the ratio $\frac{\frac{\partial \mu}{\partial x}}{\mu(t)}$ will decrease. Then, the tendency of older individuals to discount less future outcomes will arise only if the reduction of mortality rate $\mu(t)$ is greater than these efficiency gains multiplied by the constant productivity $P_0$.

Lastly, if the feature has both protective and productive effects, the effect of the feature on productivity must be reintroduced which gives the full expression for

$\delta(t)$:

$$\delta(t) = \frac{\partial P}{\partial x} + \mu(t) - \frac{\frac{\partial \mu}{\partial x}}{\mu(t) + \lambda_0 P_0 - \mu_0} P(t) \tag{4.22}$$

Then, as discussed for the previous scenarios, the first two terms are expected to decrease during growth and will therefore make individuals slower. The effect of the last term however will differ from the previous case because productivity is no longer constant. Hence, if the relative efficiency of the feature in reducing mortality increase or remains constant with $x$, the necessary increase in productivity will lead individuals to discount more. Yet, there is still a possibility that the last term produces a decrease in discounting, which requires two conditions to be fulfilled: (i) the relative efficiency of the feature to reduce mortality must decrease, and (ii) it must do so at a rate that is not compensated by the gains in productivity. Thus, in the case where both mortality and productivity are affected by the feature size, whether individuals will tend to get slower or faster as they grow will depend on the relative magnitude of these different effects.

## Increasing the number of features

What happens if the size of the feature set is increased ? The same equations for the discount rate $\delta$ derived above will apply independently of the number of features available to the organism. Yet, the sequence of acquisition of the features needs now to be taken into account to allow any general statement about the variations in discounting across ontogeny. Nonetheless, section (nb) on the optimal schedule reported a set of allocation rules that helps us extrapolate from the single feature case to a $N$ features scenario.

Indeed, if the productivity and mortality functions show strictly diminishing returns to the growth of the features, then the latter are acquired by decreasing order of $\frac{\partial P}{\partial x_i}$ when mortality is state independent, and $\frac{\partial \frac{P}{\mu+k}}{\partial x_i}$ when protective features are included. Therefore, $\frac{\partial P}{\partial x_i}$, as well as $\frac{\partial \mu}{\partial x_i}$ in the second case, are expected to decrease as the organism grows. Consequently, under these assumptions the discussion of the trends

in discounting developed in the previous section extends to life cycles involving larger feature set. Namely, if the set contains only productive features, then discounting will decrease as the organism ages. If on the other hand, some control over mortality risk is available, then the overall trend in discounting will depend on the relative magnitude of the changes in mortality $\mu(t)$, productivity $P(t)$, and their respective derivatives with respect to capital size $\frac{\partial \mu}{\partial x_i}$ and $\frac{\partial P}{\partial x_i}$.

To illustrate the analysis developed so far, Figure 4.3 and Figure 4.4 report the results of the simulations of two example life cycles that include five features (see Supplementary information for detaisl about the implementation). In Figure 4.3, features are only productive, while in Figure 4.4 they affect both productivity and mortality. Simulations were conducted using the MATLAB optimal control software GPOPS-II. GPOPS-II provides numerical methods to solve continuous time optimal control problems, by approximating them as a sparse nonlinear programming problem. Although GPOPS-II is usually mainly deployed to solve optimal control problems in engineering and robotics, it has recently been used to model brain life history evolution (Gonzalez-Forero, Faulwasser, and Lehmann 2016).

As shown in Figure 4.3. and 4.4.), the optimal life cycles simulated with GPOPS conform to the patterns obtained analytically (see Supllementary information for details about the simulations parameters). Indeed, when mortality is state independent (Figure 4.3.), growth starts with the exclusive acquisition of feature 1 which has the highest values for $\lambda_i$ and $\frac{\partial P}{\partial x_i}$. As soon as $\lambda_1 = \lambda_2$ and $\frac{\partial P}{\partial x_1} = \frac{\partial P}{\partial x_2}$, the organism begins investing simultaneously in feature 1 and 2. The same phenomenon occurs when $\lambda_1 = \lambda_2 = \lambda_3$ and $\frac{\partial P}{\partial x_1} = \frac{\partial P}{\partial x_2} = \frac{\partial P}{\partial x_2}$ and so on, until eventually all five features receive a fraction of the energy surplus. Then, when for all features $\lambda_i = \ell$ and $\frac{\partial P}{\partial x_i} = \mu$, growth stops and all energy is invested into reproduction. Lastly, the graph for $\delta(t)$ shows that the organism gets slower as predicted.

In the case where features have an additional protective effect (Figure 4.4.), the optimal life cycle again follows the structure derived analytically. That is, features are acquired in decreasing order of $\frac{\partial \frac{P}{\mu+k}}{\partial x_i}$ and the switch to reproduction occurs when $\frac{\partial \frac{P}{\mu+k}}{x_i} = 1$. Finally, the graph for $\delta(t)$ shows that the discounting decreases with time

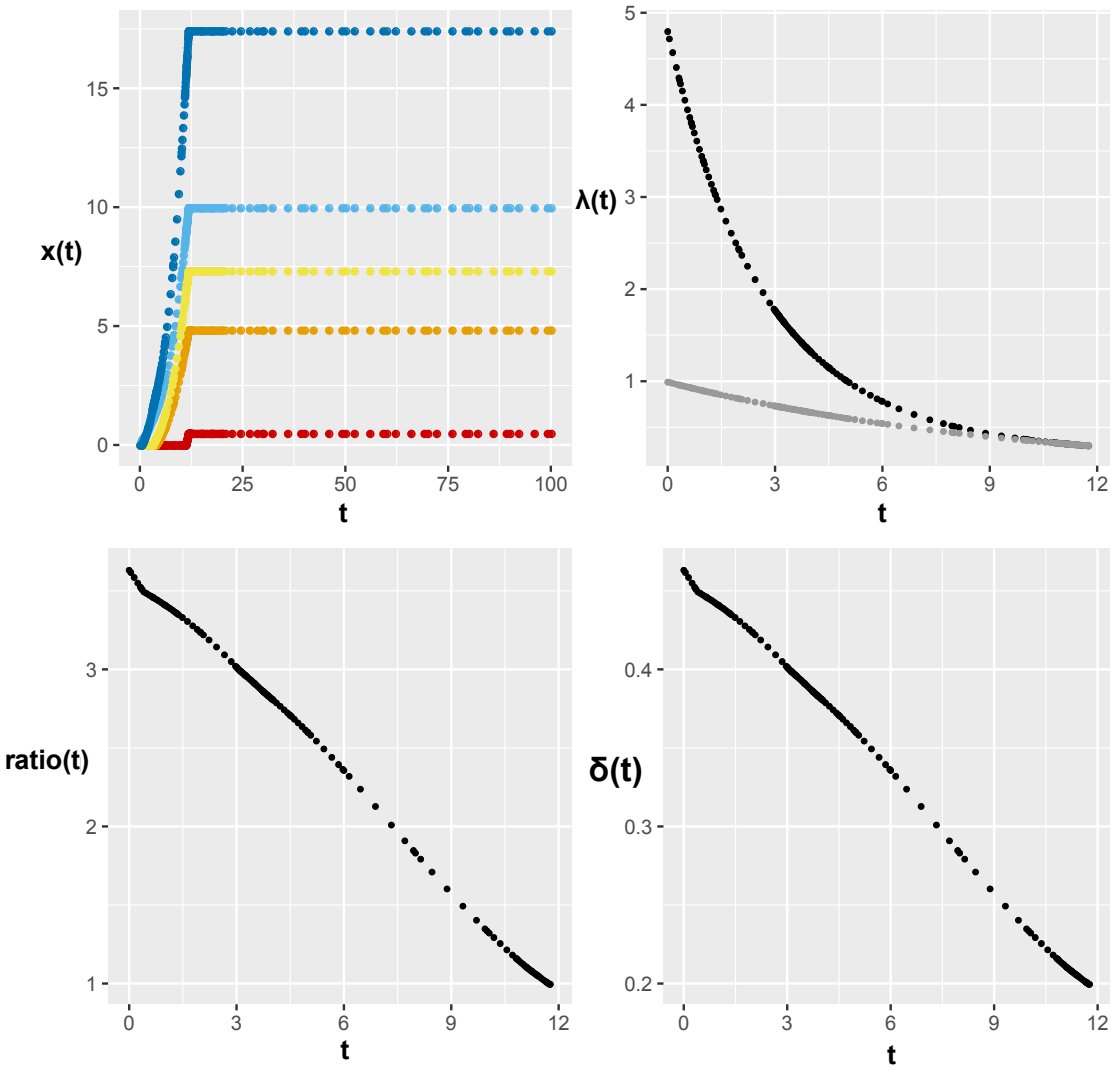


Figure 4.3: **A.** Size of the different features across ontogeny. **B.** Highest $\lambda(t)$ and survival probability $\ell(t)$ from birth until maturity. **C.** Changes in the ratio $\frac{\partial \frac{P}{\mu}}{\partial x}$ from birth until maturity. **D.** Discount rate $\delta(t)$ from birth until maturity

for this set of parameters.

### 4.4.2 Time discounting across environments

The formulation of the allocation model analyzed here permits also to discuss some potential effects of environmental quality on temporal discounting. Indeed, an environment A could be considered of better quality than environment B, whenever $P_{0_A} > P_{0_B}$, that is individuals in environment A are provided with a more productive initial capital $\mathbf{x_0}$ at the start of their lives; and/or $\mu_{0_A} < \mu_{0_B}$, meaning that individuals in environment A are less exposed to or better protected against mortality risk. Several scenarios can then be distinguished with different associated impacts of environment quality on discounting depending on which parameter is varied and what are the effects of the feature.

Recall that the expressions derived in the previous section for the discount rate $\delta$ when features only increase productivity was:

$$\delta(t) = \frac{\partial P}{\partial x} + \mu,$$

whereas once protective features are available it becomes:

$$\delta(t) = \frac{\partial P}{\partial x} + \mu(t) - \frac{\frac{\partial \mu}{\partial x}}{\mu(t) + \lambda_0 P_0 - \mu_0} P(t)$$

Concerning variations in exposure to mortality, when the latter is state independent, a straightforward increase of discounting in environments with higher mortality risk is predicted. This captures the classic effect usually attributed to local variations in mortality risk in verbal explanations of individual and populational differences in discounting and life history strategies. It corresponds to a simple form of collection risk, implying that the higher the chances are to die today, the less one should be willing to focus on rewards collected tomorrow. When mortality risk is reduced via protective features, we expect to find a similar effect of increasing discounting through the term $\mu(t)$. The overall impact of changes in $\mu_0$ on the value of the third

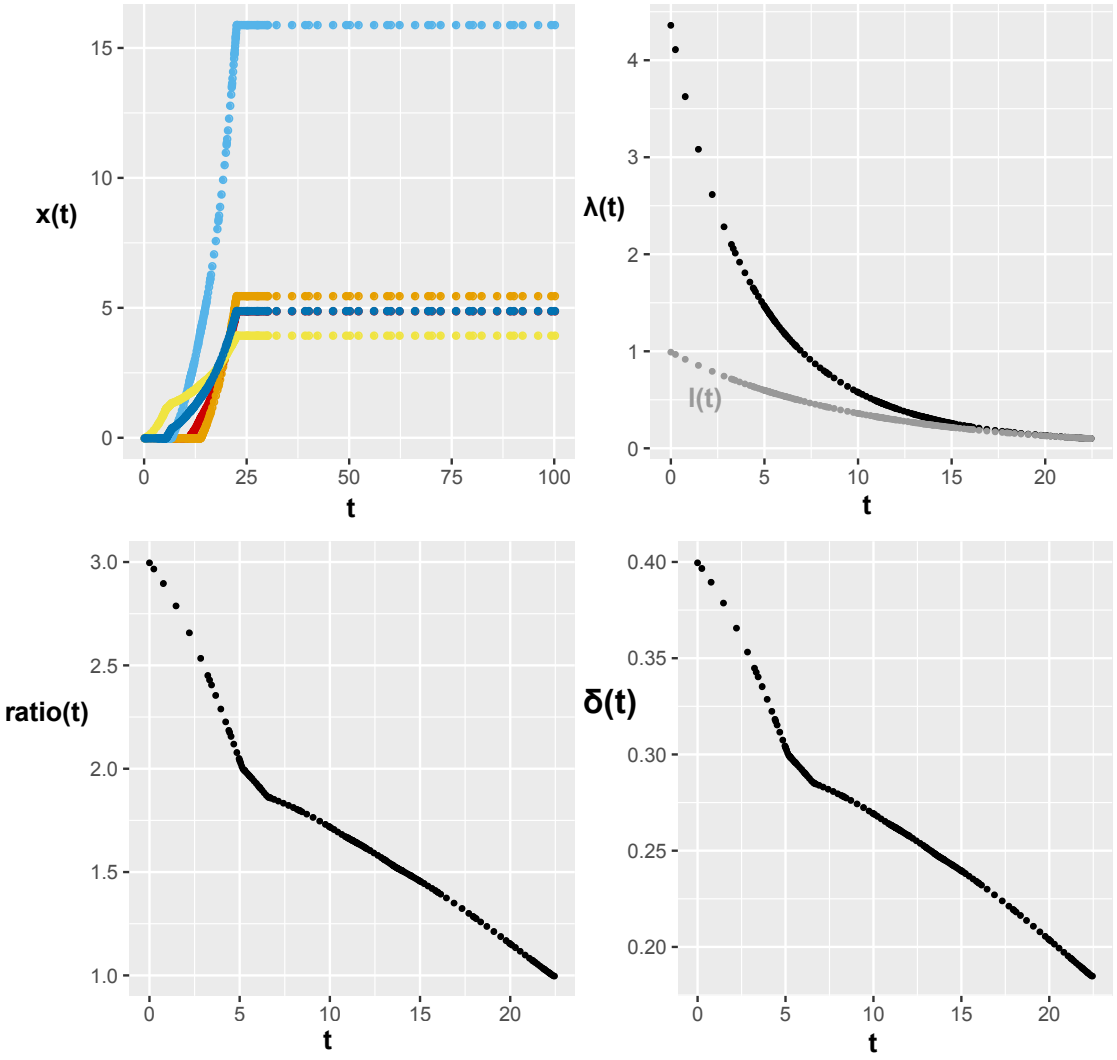


Figure 4.4: Simulation of an optimal life with five features affecting both mortality and productivity. **A.** Size of the different features across ontogeny. **B.** Highest $\lambda(t)$ and survival probability $\ell(t)$ from birth until maturity. **C.** Changes in the ratio $\frac{\partial \frac{P}{\mu}}{\partial x}$ from birth until maturity. **D.** Discount rate $\delta(t)$ from birth until maturity.

term $\frac{\frac{\partial \mu}{\partial x}}{\mu(t)+\lambda_0 P_0-\mu_0} P(t)$ however cannot really be predicted *a priori*.

In a context where environments differ with respect to $P_0$, diverse effects on discounting are also expected. Even though, $P_0$ does not appear in equation (4.4.2), an important indirect effect of local variations in $P_0$ is still to be expected from the way the features are sequentially acquired during the optimal allocation schedule. Indeed, we have seen that at the beginning of its life cycle, an organism invest in the features that yield the highest immediate returns in terms of productivity. As a consequence, temporal discounting is the greatest during this early phase of capital accumulation. The lower the value of $P_0$, the longer the portion of an organism's life cycle will be spent in these stages of high discounting.

The same reasoning applies in the case of state dependent mortality to predict the effect of the first term in equation (4.4.2). Regarding the third term however, the overall effect of a change in $P_0$ is once more difficult to predict *a priori*. Indeed, a higher value of $P_0$ results into a direct increase of the denominator, yet, for reasons invoked above it should also yields higher values of $P(t)$ leading to opposite effects on the overall ratio.

## 4.5 Discussion

In this section, we briefly discuss the main results obtained so far from our model and outline some of its limitations.

By modelling the process of capital accumulation for a developing organism, we were able to highlight the important role of opportunity costs in shaping discount rates. Indeed, our analysis emphasizes the fact that changes in collection risk (implemented as variation in mortality rate in our model), are not necessary to produce differences in delay discounting. In particular, we show that, in a case where mortality is state independent but higher levels of capital increase energy intake, and, under the assumption that capital yields diminishing returns, individuals will have lower discount rates as they possess more capital. More precisely, in our model the decrease in discounting is expected to correspond exactly to the rate at which the marginal

value of a unit of capital diminishes. This leads to two important predictions: 1) older individuals should discount less the future than younger ones, and 2) affluent individuals should also be more willing to wait for larger rewards.

The first prediction is partially supported by the empirical data available on human temporal discounting across the lifespan, which seems to support a U-shape relationship between age and discount rates (Harrison, Lau, and Williams 2002 ; Read and Read 2004; Richter and Mata 2018; but see also Chao et al. 2009 ), meaning that middle aged adults are the most patient. Yet, our model only predicts the initial phase of decrease of delay discounting, not the subsequent regain of impatience as individuals enter the late stages of their life. However, the latter dynamic might actually just mirrors the inevitable increasing in mortality risk as humans grow old (Chao et al. 2009). Then the failure of our model to capture this pattern should not come as a surprise, since no phenomenon of senescence could emerge from our model assumptions.

The second prediction is consistent with the general observation that people living in more deprived environments exhibit fast strategies geared towards short term outcomes (Pepper and Nettle 2017). In our model, this result emerges from the fact that individuals starting their life with a low initial productivity, will spend an important portion of it acquiring the most basic features available to them. Since the optimal schedule consists in investing first in the features that yield the highest returns in terms of productivity, the initial phase of capital accumulation is also the most urgent to complete. On the other hand, an individual starting with a high productivity will rapidly accumulate capital and reach the stages of its development where opportunity costs associated to further productivity gains are relatively low. Thus, introducing variations in productive capital alone is sufficient to recreate the main association between higher capital and lower discounting.

Interestingly however, adding an intrinsic component to mortality risk in the model produces results that are less straightforward to interpret. On one hand, the ability to reduce mortality risk through the acquisition of protective features might reinforce the previous relationships. Indeed, as individuals get older and accumulate more

capital, the associated drop in mortality rate will decrease collection risk and therefore delay discounting. Similarly, individuals that start their life with a high productivity will quickly acquire the most urgent protective features, making them more patient as the future rapidly becomes less uncertain. On the other hand, once mortality also depends on the level of capital, an interaction term between mortality reduction and productivity appears, which might under certain conditions lead delay discounting to increase among older and more affluent individuals. Intuitively, this interaction term seems to capture some process close to the asset-protection principle described in behavioural ecology (Clark 1994). The latter corresponds to the idea that, 'the larger the current reproductive asset, the more important it becomes to protect it'. In the context of our model, this could translate into the fact that the higher the level of capital an individual has accumulated, the more interesting it becomes to invest in mortality reduction. Indeed, for an affluent individual with a lot of productive capital, any additional year of life will eventually translates into the conversion of a large number of resources into fitness. Thus, an equivalent opportunity to reduce mortality risk could actually turn out to increase delay discounting by a larger amount for an affluent individual. This ambivalent effect of mortality obtained once organisms are allowed to exerce some control over it echoes the results obtained for other life history models (Baldini 2015; Shokhirev and Johnson 2014). How important such effects might be to the understanding of fast-slow strategies however is largely unknown.

Despite the potential differences in qualitative predictions from the various scenarios, a common remark can be made, which contrasts with some of the ideas usually advanced in the corresponding litterature. Indeed, as outlined in the introduction, variation in discounting is often thought to arise mainly from differences in collection risk. In particular, people living in more deprived conditions would actually suffer from higher collection risk because of their lack of control over future outcomes (*e.g.*, Pepper and Nettle (2017); Riis-Vestergaard and Haushofer (2017)). Yet, the results detailed above seem to depict a somewhat different picture. In our model, discounting is actually predicted to be higher when individuals' decisions have the higher impact on their situation in terms of productivity and exposure to mortality. On the other

hand, affluent and older individuals have already taken the opportunities that yield the most dramatic impacts on their state. This suggests some interesting additional predictions about delay discounting variation in human populations. Specifically, it predicts that when a vast number of *features* are readily provided by an individual's social environment, the lower it should discount the future. Indeed, in modern social democraties, numerous aspects of people's life are facilitated by public investment such as receiving some form of healthcare, getting an education or using efficient means of transportation. Hence, people already come into society with a myriad of needs already taken care of and the influence of their own actions on their state is reduced. As an hypothesis then, we could predict that people living in societies with an important safety net and a well-developed welfare state would be more patient. On the other hand, when individuals live in countries where they would have a lot of ways to improve there current state (even though most of these opportunities might not be easily accesible), they should display higher levels of delay discounting. Interestingly, some cross-country data on time preferences shows that discounting is higher in countries with higher growth rates (Wang, Rieger, and Hens 2010). More empirical data would however needed to properly assess these claims.

To conclude, some of the limitations of the current model should be mentionned to highlight various ways of improvement. First, the formulation of the allocation problem and the linearity assumption for some of the tradeoffs lead to a 'bang-bang' optimal life history. The latter consists of a period of pure growth, followed by a period of pure reproduction. Yet, such a pattern does not seem particularly faithful to the human life cycle if capital accumulation is assimilated as a form of growth. Second, again due to the 'bang-bang' nature of the optimal strategy, maturity occured as soon as a certain critical size was reached. As a consequence, this actually predicts that more affluent individuals would start reproducing earlier which would usually be considered as a marker of a fast strategy. Although earlier puberty with increased level of resources has been documented for girls in human population (Biro, Greenspan, and Galvez 2012), the same is not true for age at first birth. Adding the possibility for individuals to invest in the quality of their children, might be a way to produce results closer to empirical data. Third, most of

our analysis relied on the assumption of diminishing returns from productive and protective features. Although this is not a controversial assumption as reflected by the law of diminishing returns, S-shape functions for productivity and mortality functions could also be plausible. Many skills might involve an initial learning period during which progress is quite slow before increasing rapidly. The fact that the marginal value of a feature might first increase, however, complicates the derivation of the optimal life cycle. Nonetheless, numerical simulations for a wide range of parameters should provide insights about the importance to consider functions that are not striclty concave. Similarly, we assumed that features would have only additive effects, whereas functions allowing for synergistic or antogonistic effects between features would also be interesting to take into account. Lastly, our analysis relied on the use of the lifetime reproductive success $R_0$ as the measure of the life cycle. Some debate however exists in the theoretical litterature over which fitness measure should be used to predict long-term phenotypic evolution (Mylius and Diekmann 1995; Doebeli, Ispolatov, and Simon 2017), especially in the case of a population evolving in heterogeneous environments (Brommer 2000; Baldini 2015). Indeed, it has been argued in the latter case, that the expected optimal reaction norm should deviate from the phenotypes that would have been expressed by a set of populations evolving each respectively just in one of the corresponding environments. In the supplementary information, we provide a demographic scenario where at the evolutionary equilibrium the optimal reaction norm matches the phenotypes that would evolve in each separate environment, providing that there is no cost associated to plasticity. Nevertheless, the model presented here should be taken mainly as an optimization exercice, build to generate further insights on the forces likely to shape the variation observed in delay discounting.

## 4.6 Supplementary information

# Optimal allocation schedule and expressions for the costates $\lambda$

### General solution for the costates $\lambda$

Let the feature set consist of two independent features, with respective sizes $x_1$ and $x_2$, that have potentially unique effects on both productivity $P$ and mortality $\mu$. We assume that $P$ is a strictly increasing, concave down function of $x_1$ and $x_2$, while mortality $\mu$ is a strictly decreasing concave up function of the features size.

The Hamiltonian equation associated to the dynamic allocation problem described in the main text can be written as:

$$\begin{aligned} H &= \ell b + \lambda_1 \frac{dx_1}{dt} + \lambda_2 \frac{dx_2}{dt} + \lambda_3 \frac{d\ell}{dt} \\ &= \ell(1 - u_1 - u_2)P + \lambda_1 u_1 P + \lambda_2 u_2 P - \mu\lambda_3 \ell \\ &= -\mu\lambda_3 \ell + P[\ell(1 - u_1 - u_2) + \lambda_1 u_1 + \lambda_2 u_2] \end{aligned} \tag{4.23}$$

The associated differential equations for the costate $\lambda$ are therfore given respectively by:

$$\begin{aligned} \frac{d\lambda_1}{dt} &= -\frac{\partial H}{\partial x_1} \\ &= \frac{\partial \mu}{\partial x_1}\lambda_3 \ell - \frac{\partial P}{\partial x_1}[\ell(1 - u_1 - u_2) + u_1\lambda_1 + u_2\lambda_2] \\ &= -\frac{\partial P}{\partial x_1}u_1\lambda_1 + \frac{\partial \mu}{\partial x_1}\lambda_3 \ell - \frac{\partial P}{\partial x_1}[\ell(1 - u_1 - u_2) + \lambda_2 u_2] \end{aligned} \tag{4.24}$$

$$\begin{aligned} \frac{d\lambda_2}{dt} &= -\frac{\partial H}{\partial x_2} \\ &= -\frac{\partial P}{\partial x_2}u_2\lambda_2 + \frac{\partial \mu}{\partial x_2}\lambda_3 \ell - \frac{\partial P}{\partial x_2}[\ell(1 - u_1 - u_2) + \lambda_1 u_1] \end{aligned} \tag{4.25}$$

$$\frac{d\lambda_3}{dt} = \frac{\partial H}{\partial \ell} = \mu\lambda_3 - P(1 - u_1 - u_2) \tag{4.26}$$

$$= \mu\lambda_3 - b \tag{4.27}$$

with the additional limit condition for all costates that:

$$\lim_{t\to\infty} \lambda_i(t) = 0 \tag{4.28}$$

These differential equations are all three of the form $\frac{d\lambda}{dt} + B(t)\lambda(t) = C(t)$. Thus, the solutions they admit are given by the general formula:

$$\lambda_i(t) = e^{-\int_0^t B_i(\tau)d\tau}(K + \int_0^t C_i(s)e^{\int_0^s B_i(\tau)d\tau}ds) \tag{4.29}$$

Evalutating the previous expression for $t = 0$ shows that the constant K actuallys equals the initial value of $\lambda_i(t)$, $\lambda_{0_i}$.

Note that the previous equation can be rewritten as:

$$\lambda_i(t) = e^{-\int_0^t B_i(\tau)d\tau}(\lambda_{0_i} + \int_0^\infty C_i(s)e^{\int_0^s B_i(\tau)d\tau}ds - \int_t^\infty C_i(s)e^{\int_0^s B_i(\tau)d\tau}ds) \tag{4.30}$$

Thus, with the condition that $\lim_{t\to\infty} \lambda_i(t) = 0$, we actually have the expression for $\lambda_{0_i}$:

$$\lambda_{0_i} = -\int_0^\infty C_i(s)e^{\int_0^s B_i(\tau)d\tau}ds \tag{4.31}$$

## The survival costate $\lambda_3$ and reproductive value

Consider first equation (4.26) for the costate associated to survival $\ell(t)$. Based on the previous notation, we have $B(t) = -\mu(t)$ and $C(t) = -b(t)$. Plugging these expressions into the equation {general formula} together with the expression for $\lambda_{0_i}$ we eventually get:

$$\begin{aligned}
\lambda_3(t) &= e^{\int_0^t \mu(\tau)d\tau}\left(\int_0^\infty b(s)e^{\int_0^s \mu(\tau)d\tau}ds - \int_0^t b(s)e^{\int_0^s B_i(\tau)d\tau}ds\right) \\
&= \frac{1}{e^{-\int_0^t \mu(\tau)d\tau}}\int_t^\infty b(s)e^{-\int_0^s \mu(\tau)d\tau}ds \\
&= \frac{1}{\ell(t)}\int_t^\infty b(s)\ell(s)ds \\
&= \frac{V(t)}{\ell(t)}
\end{aligned} \tag{4.32}$$

Thus, as previously shown in the litterature (see for example Goodman (1982); Perrin (1992)), we have the costate for survival that is equal to the ratio of the expected reproduction beyond age $t$ over the probability to reach that age, which is equivalent to Fisher's reproductive value for a population at the demographic equilibrium.

Since the optimal life history is bang bang, there is no reproduction before the age at maturity $t_\alpha$. Therefore, V(t) is constant and equals its initial value $V_0 = 1$ for the whole growth period. At $t_\alpha$, investment in growth stops ($u_1 + u_2 = 0$), such that for the remaining of the life cycle we have $P(t)_{t>t_\alpha} = P_\alpha$ and $\mu(t)_{t>t_\alpha} = \mu_\alpha$. Hence, we can express the value of $V(t)$ at maturity:

$$\begin{aligned}
V(t_\alpha) &= P_\alpha \int_{t_\alpha}^\infty l(s)ds \\
&= P_\alpha \ell_\alpha \int_{t_\alpha}^\infty e^{-\mu_\alpha(s-t_\alpha)}ds \\
&= \frac{P_\alpha \ell_\alpha}{\mu_\alpha}
\end{aligned} \tag{4.33}$$

And so $\lambda_3(t_\alpha)$ must equal:

$$\lambda_3(t_\alpha) = \frac{V(t_\alpha)}{\ell_\alpha} = \frac{P_\alpha}{\mu_\alpha} \tag{4.34}$$

Plugging the previous expression in the expresion for the time derivative of $\lambda_3$ for all $t \geq t_\alpha$, we finally obtain:

$$\frac{d\lambda}{dt} = \mu_\alpha \frac{P_\alpha}{\mu_\alpha} - P_\alpha = 0 \tag{4.35}$$

Thus, during the whole reproductive period, $\lambda_3(t)$ remains constant at the value $\frac{P_\alpha}{\mu_\alpha}$.

## Expressions for $\lambda_1$ and $\lambda_2$ during the growth period

Consider now the costates associated to the changes in features size. Let us assume that at the beginning of the organism's life, we have $\lambda_{0_1} > \lambda_{0_2} > \ell_0$. Then, according to Pontryagin's maximum principle, all resources available to the organism should be invested in feature 1 as long as this condition is verified. Hence, the optimal life cycle starts with an initial period of exclusive investment in feature 1 when $u_1 = 1$. If at some time $t^*$, $\lambda_1 = \lambda_2 > \ell$ and $\frac{d\lambda_1}{dt} = \frac{d\lambda_2}{dt}$, the organism will then enter a phase of simultaneous allocation between both features when $u_1 + u_2 = 1$. Finally, as soon as $\lambda_1 = \lambda_2 = \ell$ will stop growing and redirect all its resources to reproduction (*i.e.*, $u_1 + u_2 = 0$).

From these allocation rules, we can rewrite the differential equation for $\lambda_1$ during each of the three phases.

First, during the initial period of exclusive investment in feature 1, $\frac{d\lambda_1}{dt}$ simplifies to:

$$\frac{d\lambda_1}{dt} = -\frac{\partial P}{\partial x_1}\lambda_1 + \frac{\partial \mu}{\partial x_1} \tag{4.36}$$

Then, during the period of simultaneous investment in both features, since $u_1 + u_2 = 1$ and $\lambda_1 = \lambda_2$, $\frac{d\lambda_1}{dt}$ actually still obeys the previous equation:

$$\begin{aligned}\frac{d\lambda_1}{dt} &= -\frac{\partial P}{\partial x_1}u_1\lambda_1 + \frac{\partial \mu}{\partial x_1} - \frac{\partial P}{\partial x_1}\lambda_1(1-u_1) \\ &= -\frac{\partial P}{\partial x_1}\lambda_1 + \frac{\partial \mu}{\partial x_1}\end{aligned} \tag{4.37}$$

Lastly, during the reproductive $u_1 + u_2 = 0$, so that we obtain:

$$\frac{d\lambda_1}{dt} = \frac{\partial \mu}{\partial x_1}\lambda_3\ell - \frac{\partial P}{\partial x_1}\ell \tag{4.38}$$

Hence, we can see that before maturity $B(t) = \frac{\partial P}{\partial x1}$ and $C(t) = \frac{\partial \mu}{\partial x_1}$, while during the reproductive period $B(t) = 0$ and $C(t) = \frac{\partial \mu}{\partial x_1}\lambda_3\ell - \frac{\partial P}{\partial x_1}\ell$.

Thus, starting with the initial period of exclusive investment in feature 1 (*i.e.*, $t < t^*$), we can write:

$$\lambda_1(t) = e^{-\int_0^t \frac{\partial P}{\partial x1}(\tau)d\tau}(\lambda_{0_1} + \int_0^t \frac{\partial \mu}{\partial x_1}(s)e^{\int_0^s \frac{\partial P}{\partial x1}(\tau)d\tau}ds) \tag{4.39}$$

**A simpler scenario with state independent mortality**

Let us first consider a simpler case where only productivity depends on the features size, such that we have $\frac{\partial \mu}{\partial x_1} = 0$. Hence, the previous equation reduces simply to:

$$\lambda_1(t) = e^{-\int_0^t \frac{\partial P}{\partial x1}(\tau)d\tau}\lambda_{0_1} \tag{4.40}$$

Note that because $u_1 = 1$ during the initial period, we can write according to the chain rule:

$$\frac{dP}{dt} = \frac{\partial P}{\partial x1}\frac{dx_1}{dt} + \frac{\partial P}{\partial x2}\frac{dx_2}{dt} \tag{4.41}$$

$$\frac{dP}{dt} = \frac{\partial P}{\partial x1}u_1 P + \frac{\partial P}{\partial x2}u_2 P \tag{4.42}$$

$$\frac{\frac{dP}{dt}}{P} = \frac{\partial P}{\partial x1} \tag{4.43}$$

This means that for all $t < t^*$, the value of the costate $\lambda_1(t)$ is given by:

$$\begin{aligned} \lambda_1(t) &= e^{-\int_0^t \frac{\frac{dP}{dt}}{P}(\tau)d\tau}\lambda_{0_1} \\ &= \frac{P_0}{P(t)}\lambda_{0_1} \end{aligned} \tag{4.44}$$

At $t^*$, simultaneous investment between features starts and the equations for the features' costates must satisfy the conditions $\lambda_1 = \lambda_2$ and $\frac{d\lambda_1}{dt} = \frac{d\lambda_2}{dt}$. Below, we show that the expression for $\lambda_1(t)$ during the initial period satisfies both conditions required during the period of simultaneous investment.

Indeed, taking its derivative with respect to time gives:

$$\begin{aligned} \frac{d\frac{P_0\lambda_{0_1}}{P}}{dt} &= P_0\lambda_{0_1}[\frac{\partial\frac{1}{P}}{\partial x_1}\frac{dx_1}{dt} + \frac{\partial\frac{1}{P}}{\partial x_2}\frac{dx_2}{dt}] \\ &= P_0\lambda_{0_1}[-\frac{\frac{\partial P}{\partial x_1}}{P}u_1 - \frac{\frac{\partial P}{\partial x_2}}{P}u_2] \\ &= u_1[-\frac{P_0\lambda_{0_1}}{P}\frac{\partial P}{\partial x_1}] + u_2[-\frac{P_0\lambda_{0_1}}{P}\frac{\partial P}{\partial x_2}] \end{aligned} \tag{4.45}$$

On the other hand, recall that the time derivatives of $\lambda_1$ and $\lambda_2$ before maturity are respectively given by:

$$\frac{d\lambda_1}{dt} = -\frac{\partial P}{\partial x_1}\lambda_1 \tag{4.46}$$

$$\frac{d\lambda_2}{dt} = -\frac{\partial P}{\partial x_2}\lambda_2 \tag{4.47}$$

Substituting the proposed solution $\lambda_1 = \lambda_2 = \frac{P_0\lambda_{0_1}}{P}$ into 4.46 and 4.47 therefore gives:

$$\begin{aligned} \frac{d\frac{P_0\lambda_{0_1}}{P}}{dt} &= -\frac{P_0\lambda_{0_1}}{P}\frac{\partial P}{\partial x_1} \\ &= -\frac{P_0\lambda_{0_1}}{P}\frac{\partial P}{\partial x_2} \end{aligned} \tag{4.48}$$

which satisfies equation {dt solution} since $u_1 + u_2 = 1$ during the period of parallel growth of the features. Thus, equation {lambda init} gives the value of both costates $\lambda_1$ and $\lambda_2$ from $t^*$ until the age at maturity $t_\alpha$.

Furthermore, from the equalities $\frac{d\lambda_1}{dt} = \frac{d\lambda_1}{dt}$ and $\lambda_1 = \lambda_2$ we can also deduce that:

$$-\frac{\partial P}{\partial x_1}\lambda_1 = -\frac{\partial P}{\partial x_2}\lambda_1 \tag{4.49}$$

$$\frac{\partial P}{\partial x_1} = \frac{\partial P}{\partial x_2} \tag{4.50}$$

Thus, silmutaneous allocation between features will start as soon as the productivity gains from a small increase in $x_1$ equals those of a small increase in $x_2$. Interestingly, this condition allows to further specify the equation for $\lambda_1$ before maturity by giving an explicit expression for $\lambda_{0_1}$. Indeed, recall that:

$$\lambda_{0_1} = -\int_0^\infty C(s)e^{\int_0^s B(\tau)d\tau}ds \tag{4.51}$$

Since before maturity $C(s) = 0$ and $B(t) = \frac{\partial P}{x_1}$, and after maturity $B(t) = 0$, the pr evious equation can rewritten as:

$$\begin{aligned}\lambda_{0_1} &= \int_{t_\alpha}^{\infty} \frac{\partial P}{\partial x_1}(s)\ell(s)e^{\int_0^{t_\alpha} \frac{\partial P}{\partial x_1} d\tau} ds \\ &= \frac{P_\alpha \frac{\partial P}{\partial x_1}(t_\alpha)}{P_0}\ell_\alpha \int_{t_\alpha}^{\infty} e^{\mu_\alpha(s-t_\alpha)} ds \\ &= \frac{P_\alpha \frac{\partial P}{\partial x_1}(t_\alpha)}{P_0\mu_\alpha}\ell_\alpha\end{aligned} \tag{4.52}$$

Substiting the previous expression in equation {lambda init} gives the following new expression for $\lambda_1$ before maturity:

$$\lambda_1(t) = \frac{P_\alpha \frac{\partial P}{\partial x_1}(t_\alpha)}{P(t)\mu_\alpha}\ell_\alpha \tag{4.53}$$

Lastly, taking into account the fact that at maturity $\lambda(t_\alpha) = \ell_\alpha$, we get the additional equality:

$$\begin{aligned}\frac{\frac{\partial P}{\partial x_1}(t_\alpha)}{\mu_\alpha}\ell_\alpha &= \ell_\alpha \\ \frac{\partial P}{\partial x_1}(t_\alpha) &= \mu_\alpha\end{aligned} \tag{4.54}$$

Thus, we eventually arrive at a simple equation for the value of the costate $\lambda_1$ before maturity:

$$\lambda_1(t) = \frac{P_\alpha}{P(t)}\ell_\alpha \tag{4.55}$$

This result is analogous to the one found in Iwasa and Roughgarden (1984) for a model of plant vegetative growth, with the difference that the life cycle modelled in their paper did not include any extrinsic mortality but a finite lifespan. The same allocation rules yet also apply for the optimal life cycle in our scenario. Namely, all resources should first be allocated to the feature wich yields the highest value of $\frac{\partial P}{\partial x_i}$. Then, the corresponding derivative for the best feature will decrease due to the assumption of diminishing returns, until it eventually equals either the value obtained

with another feature or the mortality rate $\mu$. In the latter case, the organism should mature and enter a purely reproductive period phase, while in the first case the transition to reproduction is preceeded by a phase of simultaneous invesment between features.

**Adding some feature dependent mortality**

The same procedure can be used to analyze the case where features might also reduce mortality risk. Indeed, during the initial period of exclusive investment in feature 1, we already know that $\frac{\partial P}{\partial x_1} = \frac{\frac{dP}{dt}}{P}$. But according to the chain rule we also have:

$$\frac{d\mu}{dt} = \frac{\partial \mu}{\partial x1}\frac{dx_1}{dt} + \frac{\partial \mu}{\partial x2}\frac{dx_2}{dt} \tag{4.56}$$

$$\frac{d\mu}{dt} = \frac{\partial \mu}{\partial x1}u_1 P + \frac{\partial \mu}{\partial x2}u_2 P \tag{4.57}$$

$$\frac{\frac{d\mu}{dt}}{P} = \frac{\partial \mu}{\partial x1} \tag{4.58}$$

Then, using the previous relationships in eq (4.41), we obtain as an expression for $\lambda_1$ before $t^*$ :

$$\begin{aligned}
\lambda_1(t) &= e^{-\int_0^t \frac{\frac{dP}{dt}}{P}(\tau)d\tau}(\lambda_{0_1} + \int_0^t \frac{\frac{d\mu}{dt}(s)}{P(s)} e^{\int_0^s \frac{\frac{dP}{dt}}{P}(\tau)d\tau} ds) \\
&= \frac{P_0}{P(t)}(\lambda_{0_1} + \int_0^t \frac{\frac{d\mu}{dt}(s)}{P(s)}\frac{P(s)}{P_0} ds) \\
&= \frac{P_0}{P(t)}(\lambda_{0_1} + \frac{1}{P_0}\int_0^t \frac{d\mu}{dt}(s) ds) \\
&= \frac{\mu(t)}{P(t)} + \frac{\lambda_{0_1} P_0 - \mu_0}{P(t)}
\end{aligned} \tag{4.59}$$

Then, we can show again that this expression obtained for the initial period also satisfies the conditions imposed during the period of simultaneous allocation.

Let us define the constant variable $k = \lambda_{0_1} P_0 - \mu_0$ such that the previous equation

becomes $\lambda_1 = \frac{\mu+k}{P}$. Taking its derivative with respect to time gives:

$$\begin{aligned}\frac{d\frac{\mu+k}{P}}{dt} &= \frac{\partial\frac{\mu+k}{P}}{\partial x_1}\frac{dx_1}{dt} + \frac{\partial\frac{\mu+k}{P}}{\partial x_2}\frac{dx_2}{dt} \\ &= u_1[\frac{\partial\mu}{\partial x_1} - \frac{\mu+k}{P}\frac{\partial P}{\partial x_1}] + u_2[\frac{\partial\mu}{\partial x_1} - \frac{\mu+k}{P}\frac{\partial P}{\partial x_1}]\end{aligned} \tag{4.60}$$

Recall that the time derivatives of the costates are this time given by:

$$\frac{d\lambda_1}{dt} = -\frac{\partial P}{\partial x_1}\lambda_1 + \frac{\partial\mu}{\partial x_1} \tag{4.61}$$

$$\frac{d\lambda_2}{dt} = -\frac{\partial P}{\partial x_2}\lambda_2 + \frac{\partial\mu}{\partial x_2} \tag{4.62}$$

Hence, substituting $\lambda_1 = \lambda_2 = \frac{\mu+k}{P}$ into equations 4.61 and 4.62 gives:

$$\frac{\partial\mu}{\partial x_1} - \frac{\partial P}{\partial x_1}\frac{\mu+k}{P} = \frac{\partial\mu}{\partial x_2} - \frac{\partial P}{\partial x_2}\frac{\mu+k}{P} \tag{4.63}$$

Thus, the conditions during for the simultaneous allocations are verified and until maturity is reached $\lambda_1 = \frac{\mu+k}{P}$.

Furthermore, from the expression of $\lambda_1$ we can show that at the beginning and for the whole duration of the period of simultaneous allocation, the equality $\frac{\partial\frac{P}{\mu+k}}{\partial x_1} = \frac{\partial\frac{P}{\mu+k}}{\partial x_2}$ is verified. Indeed, for both features we have:

$$\frac{\frac{\partial P}{\partial\mu}}{x_i} = \frac{\frac{\partial P}{\partial x_i}(\mu+k) - P\frac{\partial\mu}{\partial x_i}}{(\mu+k)^2} \tag{4.64}$$

On the other hand, from the equality constraint on the time derivatives of the features costates we have:

$$-\frac{\partial P}{\partial x_1}\frac{\mu+k}{P} + \frac{\partial\mu}{\partial x_1} = -\frac{\partial P}{\partial x_2}\frac{\mu+k}{P} + \frac{\partial\mu}{\partial x_2} \tag{4.65}$$

Multiplying both sides by $-\frac{P}{(\mu+k)^2}$ then gives us:

$$\frac{\partial \frac{P}{\mu+k}}{\partial x_1} = \frac{\partial \frac{P}{\mu+k}}{\partial x_2} \tag{4.66}$$

Finally, we show below that the organism switches to reproduction when $\frac{\partial \frac{P}{\mu}}{\partial x_1} = \frac{\partial \frac{P}{\mu}}{\partial x_2} = 1$. We already know that at maturity $\lambda_1 = \lambda_2 = \ell$ and therefore that $\frac{d\lambda_1}{dt} = \frac{d\lambda_1}{dt} = \frac{d\ell}{dt}$. This allows us to write the following equality:

$$\lambda_3 \ell \frac{\partial \mu}{\partial x_1} - \frac{\partial P}{\partial x_1}\ell = \lambda_3 \ell \frac{\partial \mu}{\partial x_1} - \frac{\partial P}{\partial x_1}\ell = -\mu\ell \tag{4.67}$$

Recalling that during the reproductive period, $\lambda_3 = \frac{P_\alpha}{\mu_\alpha}$, the previous expression reduces to:

$$\frac{P_\alpha}{\mu_\alpha}\frac{\partial \mu}{\partial x_1} - \frac{\partial P}{\partial x_1} = \frac{P_\alpha}{\mu_\alpha}\frac{\partial \mu}{\partial x_2} - \frac{\partial P}{\partial x_2} = -\mu \tag{4.68}$$

$$\frac{\partial \frac{P}{\mu}}{\partial x_1} = \frac{\partial \frac{P}{\mu}}{\partial x_2} = 1 \tag{4.69}$$

Thus, we can finally derive the allocation rules for the optimal life cycle when features might affect both productivity and mortality and yields diminishing returns. First, allocate all resources to the feature that yields the highest increase in terms of the ratio $\frac{P}{\mu+k}$. Then, as this derivative decreases, divide your resources between all features that provide the same returns. When returns no longer exceed investment, that is $\frac{\partial \frac{P}{\mu}}{\partial x_1} = \frac{\partial \frac{P}{\mu}}{\partial x_2} = 1$, switch to reproduction.

# Chapter 5

# General discussion

Throughout this manuscript, we tried to apply insights from evolutionary ecology, and especially life history theory, to explain how individuals might react to environmental harshness. In particular, we adopted a developmental perspective by looking at the way adverse experiences in early life might condition allocation strategies at later ages, impacting a range of phenotypic traits. This general approach is shared between studies at the root of psychosocial acceleration theory. The work reported here built upon this research and provides additional contributions to the field, both empirical and theoretical.

## 5.1 Characterizing the fast-slow continuum

In chapter 2, we investigated the coordinated nature of life history strategies for the fundamental domains of health and reproduction. As hypothesized, we found that in individuals who had been exposed to more adverse early life conditions, lower somatic effort co-occured with higher reproductive effort. These results begin to fill a gap in the litterature, which so far has either focused mainly on the covariation of traits within a single domain of a life history strategy (*e.g.*, either somatic effort, or reproduction), or used a psychometric approach to position individuals on the broader fast-slow continuum. Our study supports the idea that humans adopt coordinated strategies with regard to health and reproduction, which might be tailored in an adaptive way to their particular ecologies.

Yet, it should be noted that the single fast-slow axis of variation only accounts for a small portion of the variance in health and reproductive behaviours. Then, at least in developped nations such as France, other factors matter and will largely determine the variance observed for any single life history indicator. More studies that test the basic premise of the existence of a fast-slow continuum are therefore needed. In particular, studies conducted on populations that cover a wider range or a different portion of the harshness spectrum. Indeed, in a representative sample of the French population, the number of individuals that experienced really high levels of deprivation is likely to be relatively small. Studies that contrast more specifically the life history strategies of individuals coming from the opposite extremes of the harshness spectrum in a given country would be a valuable complement to our findings. Such a strategy has been used to compare various aspects of people behaviours in two british neighborhoods, one belonging to the highest decile on an national index of neighborhood deprivation and the other to the lowest decile, providing very insightful results (Nettle, 2015).

Going beyond western societies altogether would also put to test the robustness of the covariation patterns. Indeed, some cross cultural data for individual traits is already available for several life history variables, further supporting psychosocial acceleration theory in some cases (*e.g.*, age at first birth; Low et al. 2008; Walker et al. 2006) and yielding conflicting results in others (*e.g.*, age at menarche; Sear et al. 2018). Yet, again, the more general covariation structure between somatic and reproductive efforts has not been thoroughly investigated across societies.

Nevertheless, it should be noted that even in the case where cross cultural data would show substantial variation around the main fast-slow pattern, this would not necessarily mean that the general approach advocated here should be judged irrelevant. Indeed, some of the relationships are likely to be moderated by additional factors that are not held constant across societies. An important strength of the life history approach however, is that it can also make predictions about factors that are susceptible to play a moderating role and in which contexts.

For instance, depending on the level at which one tries to explain variation in the

timing of puberty, it can both appear as a marker of a fast or a slow life history strategy. Indeed, by comparing high income societies with societies where a large fraction of the population is under nutritional stress, one is likely to find that an earlier age at menarche actually covaries with traits that usually indicate a slow life history, such as a later age at first birth or greater investments in health protective behaviours. The discrepancy in the covariation between life history indicators however should be fully accounted for by the fact that growth rates are lower for individuals in a precarious nutritional status, which mechanistically delays the age at which maturity can be set off. Once the scope of the analysis is restricted to the variation observed within a high income society, this effect of nutritional status is essentially controlled for and the remaining differences (that can be attributed to phenotypic plasticity) are expected to reflect a switch to a faster life history strategy triggered in an uncertain environment.

This example highlights a classic issue in studying life history evolution, which is that trade offs can be masked by differences in individual attributes that can increase or decrease the intensity at which particular traits are negatively correlated. Differences in energy status, as described for age at menarche, are maybe the most pervasive example of this phenomenon. Although two traits might be 'mechanistically' negatively correlated at the individual level, if initial budgets differ between individuals, the correlation measured at the population level might actually turn out to be positive. This is illustrated quite plainly in humans by the fact that people that have expensive cars also tend to have bigger houses. For any particular individual, investing more money into an expensive car will automatically reduce the size of the houses that can potentially be bought. But due to the particular distribution of income and wealth in human populations, bigger houses will also tend to be owned by people that can afford expensive cars at the same time.

This phenomenon is particularly relevant for studies that investigate social gradients in life history traits, like the ones reported in the present manuscript. In particular, it might predict non-linar relationships between socioeconomic status and certain life history indicators. For instance, we could predict that a U-shape curve would

describe best the relationship between SES and number of offspring. Indeed, even if there is a tradeoff between quantity and quality of offspring, which should yield an initial reduction in number of offspring as SES increases, we could nonetheless expect that people might start to have more children after some SES threshold. There would still be a tradeoff between quantity and quality of offspring but its intensity would be significantly lower for affluent individuals. As people possess more and more resources they can mitigate the negative effect of having an additional offspring on the level of care each child receives (by employing other people to attend to the needs of each child for example).

Thus, more complicated relationships are likely to arise as one test the robustness of the fast-slow continuum across a wider range of environmental and social contexts. This has often been the case in the animal personality litterature, where certain behavioural syndromes have been found to emerge only in particular ecological conditions. For example, agressiveness and boldness are positively correlated in the three-spined stickleback (Gasterosteus aculeatus), only when populations are under predation risk (Bell and Sih, 2007; Dingemanse et al., 2007). Therefore, in similar situtations for humans, a deep examination of the particular tradeoffs and ecological pressures facing different populations or individuals should allow us to gain a finer-grained understanding of the coordinated nature of life history strategies.

In chapter 3, we explored the idea that interindividual variation in social trust could also be related to the fast-slow continuum. Specifically, we tested the hypothesis that harsher environments decrease individuals' social trust, with unique effects of early life conditions on adults' beliefs. Contrary to variation in health and reproduction, for which a considerable number of studies had already shown an association between adversity and faster strategies, there was more doubt surrounding the expected relationship between harsh environments and prosociality. Indeed, it has both been argued that surviving in harsh environments requires higher levels of prosociality, and, that the increase in delay discounting expected in harsh environments should reduce cooperation. However, regarding social trust specifically, previous studies

had mainly shown a positive association with SES. Lasting negative impacts of early adversity had also been found under well-controlled but restricted settings (Hörl et al. 2016; Petersen and Aarøe 2015).

In the series of studies reported, overall we found support for these previous findings in a diversity of samples and added evidence for an association between higher social trust and slow life history strategies. This result would therefore favor a delay discounting account of differences in prosocial tendencies. Yet, how variation in social trust translates to actual differences in individuals' investment in cooperation in natural settings remains to be more thoroughly investigated.

Furthermore, surprisingly we did not find the same general association between higher deprivation and lower social trust in a sample of 11 years old children. There are some methodological shortcomings to the associated study (*e.g.*, a high SES biased sample, missingness in the dependent variables), but there is still the possibility that this null result is real and meaningful. Then, how the detrimental effects of harshness on social trust could remain latent during childhood and resurfaces later in adolescents or adults becomes an intriguing developmental puzzle. This result highlights the value of combining both longitudinal and crossectional studies to test psychosocial acceleration theory. Indeed, once the mapping of more behavioural traits with the fast-slow continuum will be better established empirically, studies investigating the developmental processes at play will constitute an important step forward.

In chapter 4 we built a formal life history model to improve the theoretical underpinnings of our understanding of delay discounting in the context of life history strategies. Until now the focus has mainly been on the role of collection risk, that is on the uncertainty around the future state of the environment. However, our work highlights the importance of taking into account the opportunity costs associated to delayed gratification, in order to explain changes in discounting within and between individuals. Eventually both types of factor will interact to determine an individual's discount rate.

Combining them into a single formal framework would therefore provide a more

complete account of delay discounting. The model described in chapter 4 only includes mortality as a source of collection risk. A modified version in which a unit investment in capital would not have purely deterministic effects (*i.e.*, a corresponding unit increase in capital), but also a stochastic element would be one way to move in that direction. This would integrate some uncertainty around the impact of an individuals' decision on its state and might capture the effects of some other interesting sources of collection risk.

Finally, our focus on delay discounting came from a particular interpretation of the fast-slow continuum, as precisely the product of individual differences in time preferences. Yet, others have argued that the fast-slow continuum might actually be better conceived as the reflection of different risk management strategies (Amir et al., 2018). This is an interesting perspective, since both delay discounting and risk preferences are likely to be the main drivers of the assocations between traits on the fast-slow continuum. Their relative importance however is largely unknown at the moment. Thus, a critical extension of our modelling framework would be to derive a measure of risk preferences and analyze its dynamics within and across individuals.

## 5.2 Causal links in a web of correlations

Althoughe empirical findings reported in the first part of the manuscript add to our knowledge of the associations between early adversity and various facets of human behaviour, the correlational nature of this work should be stressed. Psychosocial acceleration theory posits a causal link between the exposure to harsh environments and the adoption of a fast strategy. Yet, no claim about the causal status of these relationships can be made from the studies described in this thesis.

One important confounding factor that might account for at least part of the correlations observed between life history variables and deprivation is genetics. Indeed, some authors have gone as far as claiming that all the variance attributed to mechanisms of phenotypic calibration is actually caused by unmeasured genetic factors (Sherlock and Zietsch, 2018; Zietsch, 2016), that are correlated both with

socioeconomic factors and psychological variables. Although genetic confounds are likely to play an important role in explaining some of the associations obtained, such an extreme position does not seem convincing in face of the current evidence and based on theoretical grounds.

For instance, making participants coming from either a very deprived, or a very affluent neighborhood play several economic games, highlighted huge differences in the average offers made (Nettle et al. 2010). Even though genetic data was not collected in this particular study, our knowledge of the amount of within population genetic variance makes it rather unlikely that such important population differences in behaviour could be explained by shared genetic factors alone. Furthermore, mechanisms of adaptive phenotypic plasticity are widespread and well-documented across the living kingdom (Ghalambor et al. 2007). In a species like *Homo sapiens* which has been recurrently exposed to a wide range of environmental conditions, it is hard to conceive that such mechanisms would not contribute a significant portion of the variance observed.

Yet, without downplaying the role of development in producing human behavioural variation, it is true that more genetically sensitive designs are in need to consolidate and refine the core findings of psychosocial acceleration theory. Examining the likely interactions between genetic and environmental factors behind the associations reported seems particularly promising. Some work has already been conducted in this direction (*e.g* Hartman et al. 2016), but should certainly benefits from a greater focus in the field.

In addition to the effects of genetics, the relationships obtained in our studies could also be confounded by factors more frequently invoked in the social sciences. It could be tempting for example to attribute the covariation between somatic and reproductive effort to factors such as level of education or intelligence. However, whether such factors should be controlled for in studies investigating the link between harshness and fast life histories is not always clear. Indeed, these variables are themselves likely to be to some extent the products of life history strategies. Indeed, how much energy should be devoted to the development of the brain, or how much

effort should be put into one's education, are eventually just other kinds of allocation decisions that should be influenced by the level of harshness in the environment. Similarly, as discussed in our study on social trust in 11 years old children, levels of parental investments are both expected to buffer and mediate the effects of environmental harshness on a child's cognition. Although we did not find evidence for any of these relationships in our data, the importance of such mechanisms remains quite plausible, at least for other traits than social trust.

Still, studies relevant to psychosocial acceleration theory with higher internal validity are desirable. In this domain however, the field as a whole is making significant progress by better integrating research done mainly in two adjacent fields: experimental biology and economics. Regarding experimental biology, using the European starling (*Sturnus vulgaris*) as a model organism, many intersting effects of early deprivation on individuals' state and behaviour throughout life have been uncovered (*e.g.*, Dunn et al., 2018; Nettle et al., 2017; Bateson et al. 2015; Andrews et al., 2015). Similar work using other model species would yield a very rich body of empirical work from which to draw promising comparisons with the data obtained in humans.

On the other hand, directly manipulating the extent to which people are exposed to adversity during the first years of their life would be unethical. Yet, economic studies have started to produce results that support a causal role of early deprivation on adult behaviours. Indeed, economists have excelled in recent years at identifying natural experiments that allow to infer causality for relationships that would otherwise suffer from a lot of potential confoundings in traditional observational settings. Furthermore, they have shown an increasing interest in evaluating precisely the effects of early life conditions on later outcomes (*e.g.*, Lin and Liu, 2014; Lavy et al., 2016; Hörl et al. 2016). Incorporating results obtained from the analyses of such natural experiments will greatly advance the evidentiary value of the basic research underlying psychosocial acceleration theory.

Another way forward can be found in the growing focus on experimental methods in development economics. Indeed, although exposing individuals to more adversity would be unethical, interventions that improve people's conditions can be conducted

in an ethical way (Banerjee and Duflo, 2009). By measuring how such interventions affect people's psychology, predictions of psychosocial acceleration theory can be tested in an randomized controlled way. In particular, individuals benefiting from the interventions would be expected to adopt an overall slower strategies, with repercussions on a whole range of behaviours.

## 5.3 On the value of the evolutionary developmental approach

Throughout the chapters of this manuscript, we have highlighted how insights from life history theory could serve as a basis for the exploration of a wide range of patterns of interindividual variation in human behaviour. In particular, this approach sheds new light in several ways on social gradients that had often already been documented in other social sciences.

First, it provides an overarching framework that ties together otherwise disparate findings. For instance, the idea of a tradeoff between current and future reproduction provides *a priori* theoretical reasons to expect investment into health protective and reproductive behaviours to covary. Thus it takes into account the covariation between traits across domains and predicts how a suite of traits should respond to a change in environmental conditions. This is especially relevant to policy making, for it suggests that interventions that target a particular kind of behaviour or aspect of people's state can have cascading effects on a whole range of traits, but also allows one to make predictions about these indirect effects.

Second, because of its roots in evolutionary biology, it bridges the gap between findings obtained in the animal and human litteratures. This is an important feature of the approach, since it opens the way to an array of experimental methods that can be employed in a comparative framework and would otherwise have been inaccessible for ethical reasons. Furthermore, the approach relies on mechanisms of adaptive phenotypic plasticity that are universally shared among humans, hence the same framework can be applied for any human population across space and time. This

has led to a new promising area of research, which investigates whether particular historical phenomenon could be explained by the effects on people's psychology of changes in living conditions (*e.g* Baumard and Chevallier, 2015; Baumard et al. 2018).

Third, it tackles the issue of the ultimate causes behind the origins of social gradients. Indeed, complementary approaches in the social sciences usually provide important insights into the proximate mechanisms that give rise to interindividual differences in behaviour, but do not address the question of why we should observe this particular pattern of variation in the first place. By stating that individual variation tracks to some extent the reaction norms shaped by ecological factors and tradeoffs among traits, the evolutionary approach makes prediction about the specific associations that are likely to be produced by psychological mechanisms that have evolved by natural selection.

Lastly, it offers a new perspective on present oriented behaviours. Indeed, studies that find a decrease in delayed gratification among low SES individuals would usually attribute it to a failure of willpower, a lack of self control. Under the life history approach however, such SES based individual differences are viewed as contextually appropriate from an evolutionary point of view (Pepper and Nettle, 2018). This does not mean that present oriented behavior do not entail costs to individuals' health or wellbeing, neither that they cannot be maladaptive in some cases, but rather that the general expectation that they are deviations from a global optimum, identical across all ecologies, is ill founded. Furthermore, it is even quite plausible that such calibration mechanisms actually enhance the performance of low SES individuals in certain settings (Frankenhuis and Ellis, 2017). Once again this has important implications for policy making, by changing our prior expectations on the type of interventions that are likely to be succesful in particular contexts. For instance, if we believe that adolescent childbearing has its roots mainly in a lack of knowledge about contraceptive use or a deficit in future planning abilities, we might be tempted to dedicate most of our resources for the funding of information campaigns targeted at young adolescents. On the other hand, if we believe that the same phenomenon could

actually reflect a contextually approriate response to living in a deprived ecology, we might favor a policy that addresses more structural aspects of the environment, instead of focusing directly on the adolescents.

## 5.4 Concluding remarks

Evolutionary theory does not apply only to innate traits, universally shared across members of a species and irresponsive to experience. Variation in human psychology and behaviour is the product of developmental mechanisms that have themselves evolved by natural selection. Hence, the shape of the distributions measured for any trait in natural populations will reflect the intrinsic properties of these mechanisms.

As the optimal phenotype will vary across environmental conditions, individuals are expected to adjust their developmental trajectory to match the particular requirements of their ecology. Fundamental tradeoffs such as the tension between current *vs* future reproduction will then determine in which direction of the phenotypic space an organism should move.

These general principles apply to any living being and can generate predictions about patterns of interindividual variations that we should expect to find in nature. How much knowledge can be gained by applying these insights to the study of human behavioural variation however is still an open question. The present manuscript falls in with a growing body of research that attempts to provide some answers. Our hope is that it might contribute in some way to the further integration of this broad approach with other behavioural sciences.

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# General Appendix

related outcomes like substance use, accidental injury, and unintentional drowning (Steinberg 2013). All of these phenomena are particularly pronounced in adolescent males (Scheidt et al. 1995), in whom patterns of brain development are somewhat delayed relative to those of females (Lenroot & Giedd 2006), and who are also much more likely to engage in violent aggression (Snyder 2012).

The authors have made a strong case that rates of violent aggression rise with geographic proximity to the equator, which is used as a proxy for climate. But the model may require amending to incorporate the mediating or moderating role that the proportion of adolescents across regions may play in geographic variation in violent aggression.

As it happens, more equatorial countries also contain large – sometimes much larger – proportions of adolescents. Almost without exception, those nations with median ages less than the global median age of 29 are equatorial nations of Africa, Asia, and Central and South America. The youngest countries in the world include equatorially proximate African nations like Uganda, Niger, and Mali. By contrast, the oldest countries include Japan, Germany, Monaco, and other European and Asian countries closer to the poles (Central Intelligence Agency 2016b).

So-called "youth bulges" in countries proximal to the equator could explain increased violence in these countries without reference to life history theories or climate. It has been observed that the proportion of individuals within a society who are between the ages of 15 and 24 is predictive of the prevalence of various forms of violence in that society, including homicide, domestic armed conflict, terrorism, and rioting (Bricker & Foley 2013; Mesquida & Wiener 1999; Urdal 2006). In the United States, a significant proportion of changes in violent crime over time can be explained by fluctuations in the proportion of adolescents (Phillips 2006). Globally, the relationship between violence and the proportion of a country's population composed of adolescents has also been found across several investigations (Cincotta & Leahy 2011; Pampel & Gartner 1995; Urdal 2006).

Van Lange and colleagues might argue that countries like Guatemala, Belize, and Honduras are among the most violent countries in the world because of their equatorial climates, which result in the population of these regions adopting relatively fast life history strategies, characterized by "short-term planning, greater risk taking, a focus on immediate gratification for short-term benefits, and more aggression" (sect. 3.1, para 3). But one could just as easily argue that the reason these quintessentially adolescent traits are pervasive in these countries is that their populations are disproportionately composed of adolescents. These are among the world's youngest countries, and are the three youngest nations in the Americas. Nearly a quarter of the population of Guatemala, for example, is between the ages of 15 and 24 (Central Intelligence Agency 2016b).

It is possible, then, that the patterns the authors have observed do not reflect climate-induced variation in life history strategies, but are instead an artifact of geographic fluctuations in the proportion of youths, owing to reasons that are unrelated to climate. Alternately, it is possible that fluctuations in the proportion of youths could be incorporated into the CLASH model. Youth bulges are thought to emerge during the stage of a nation's development when infant mortality has been successfully reduced, yet fertility rates remain relatively high (Bricker & Foley 2013), but no generally accepted explanation exists for why youth bulges currently cluster around the equator.

Any attempt to incorporate the relationship between youth and violence into the CLASH model should reflect the fact that the relationship between youth bulges and violent aggression may not be a simple one. Variables like access to education and jobs are critical influences on the behavior of youths within a society (Bricker & Foley 2013). Likewise, the effect of youth bulges on violent aggression may be mitigated by protective cultural factors like collectivism, which can transform large youth populations into civic opportunities (Pampel & Gartner 1995). Understanding the role of adolescence in societal variation in aggression therefore requires considering not only how many adolescents a society contains, but also how those adolescents are faring: Are they civically engaged? Educated? Impoverished? Optimistic about their future prospects (Bricker & Foley 2013; Hart et al. 2004; Pampel & Gartner 1995)?

The essential fact remains that a model of cultural variation in violent aggression that does not consider the role of adolescence remains an incomplete model.

## Climate is not a good candidate to account for variations in aggression and violence across space and time

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**Abstract:** We agree with Van Lange et al. that climate is likely to affect individuals' social behavior in many ways. However, we suspect that its impact on physiology and psychology is so remote that its predictive power disintegrates almost completely through the causal chain underlying aggression and violence.

Using data about current and past patterns of aggression and human sociality, we show that the causal role of climate vanishes once one switches from a worldwide perspective to a more local one, and that it becomes quasi-irrelevant once a historical dimension is considered. Evolutionary models in biology provide explanations of variations in traits that are generalizable across both space and time. We believe that this criterion of relevance is, however, not met by CLASH.

We start our demonstration by testing whether climate predicts interpersonal violence during a restricted period within geographic Europe (Fig. 1A,B), an area that is similar in size and culture to the United States. Except for Russia, all major European countries with available climatic and homicide data for the 2008–2012 period were included (35 countries, sources: World Bank (The World Bank Group 2016a; United Nations Office on Drugs and Crimes [UNODC] 2016). Following methodological recommendations from studies that inspired the target article (Burke et al. 2015; Hsiang et al. 2013), we ran a series of correlations between interpersonal violence (measured by the homicide rate [Burke et al. 2015]) and either yearly average temperature or seasonal variations in temperature (i.e., the difference between the average temperature for the three summer months and the three winter months). We included only countries where CLASH was applicable (average year temperature under 24°C). Contrary to CLASH's predictions, both year-by-year and period-wise analyses revealed that interpersonal violence did not vary with temperature (all $r$ values < .12, all $t(34)$ values < 0.67, all $p$ values > .25) (Fig. 1A) and, more surprisingly, increased with seasonality (all $r$ values > .39, all $t(34)$ values > 2.49, all $p$ values < .018) (Fig. 1B).

We then tested whether climatic variables predicted homicide rates in elapsed time periods (Fig. 1C,D) using historical records from two geographically distant countries of the temperate zone: Japan (1924–2004 [Statistics Bureau, Ministry of

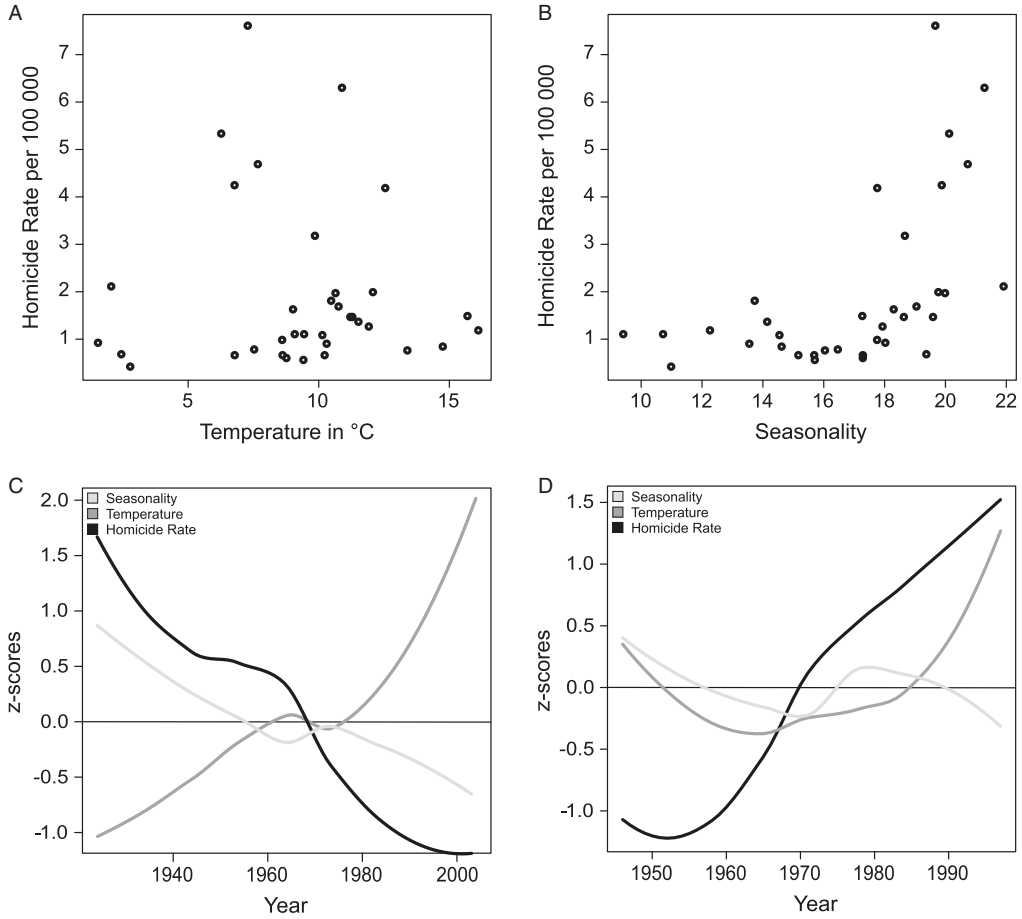


Figure 1 (Mell et al.). (A,B) Homicide rates averaged for the 2008–2012 period for countries (N=35) constituting geographic Europe, as a function of (A) temperature and (B) seasonality. (C,D) Year-by-year evolution of homicide rate, temperature, and seasonal variation for (C) Japan and (D) Great Britain.

Internal Affairs and Communications 2016a; 2016b; The World Bank Group 2016a]) and Great Britain (1951–1999 [Richards 1999; The World Bank Group 2016a]). For representational purposes, all three variables were expressed in units of standard deviations of their respective means over the whole period (*z*-score transformed). Figure 1C and D represent the trend for each variable across the time record, with ranges and standard deviations differing between Great Britain (homicide rate per 100,000: 0.58–2.06, SD = 0.42; temperature: 7.55°C–9.52°C, SD = 0.47; seasonality: 7.54°C–14.01°C, SD = 1.28) and Japan (homicide rate per 100,000: 0.97–4.14, SD = 0.98; temperature: 10.11°C–12.71°C, SD = 0.56; seasonality: 17.5°C–22.95°C, SD = 1.09). The impact of temperature and seasonal variations on homicide rates was the strict opposite of CLASH's predictions for Japan (average temperature: $r = -.52$, $t(79) = -5.43$, $p < .001$; seasonality: $r = .22$, $t(79) = 1.99$, $p = .049$) (Fig. 1C), whereas no effect was observed for Great Britain (both $r$ values < .18, both $t(45)$ values < 1.26, both $p$ values > .215) (Fig. 1D). Figure 1D illustrates the importance of considering historical data to avoid spurious correlations when trying to find determinants of trait variations: during the 1990s, homicide rate and temperature positively covaried in Great Britain, but it is seen by going back further in time that the rise in homicides preceded the temperature increase and that the relationship was actually reversed in the 1950s.

One could argue that testing countries with relatively homogeneous temperate climates is inappropriate because small climatic variance might not include the critical threshold at which more dramatic levels of violence occur. If true, then deviations from the mean could be pure noise. However, we believe that this is not the case. Figure 2 indeed illustrates the difference in murder rates measured for the year 2013 (Federal Bureau of Investigation [FBI] 2013) in all 50 states of the United States and in the five boroughs of New York City (Pediacities NYC) and shows that variations observed at a very local scale (city) can be of similar magnitude as variations observed at a very global scale (continent). We doubt that it is reasonable to posit that climate differences act as a major predictor, at the expense of other explanatory variables such as, for example, differences in income.

In addition to these empirical arguments, we raise a more fundamental concern: CLASH cannot satisfyingly account for major transitions in the evolution of human sociality such as, for instance, the replacement of asocial religions by prosocial ones. Recent work indeed demonstrates that the best explanatory factor of this phenomenon is an increase in affluence (energy capture per capita, urbanization rate, population growth), a variable highly predictive of individuals' level of resources (Baumard et al. 2015). This is in line with the life history framework, which predicts that individuals enjoying higher levels of resources engage in slower life strategies that are characterized by high investments in long-term goals, including cooperative goals. The early emergence of belief systems promoting prosociality can thus be better understood as the consequence of historical changes in the distribution of resources (Baumard & Chevallier 2015), rather than climate. It becomes especially striking when one considers that prosocial religions appeared in different

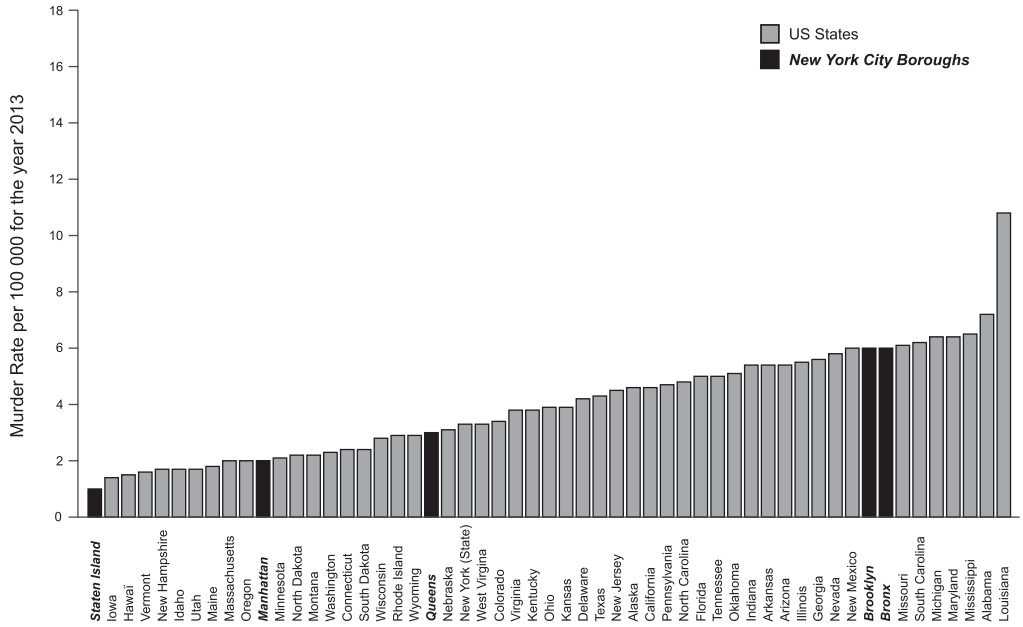


Figure 2 (Mell et al.). Murder rates measured for the year 2013 in all 50 states of the United States and in the five boroughs of New York City.

civilizations located in arid, semi-arid, and tropical zones of the world (Eastern Mediterranean, Mesopotamia, Northern India) long before they emerged in more temperate areas.

In sum, between-individual differences in life strategies – whose acceleration *eventually* leads to greater violence – are more likely to depend on ecological dimensions whose fluctuations matter more than climate for survival and reproduction in complex social worlds. All things being equal, climate differences ought to be part of the general explanation, but we doubt that they should constitute the core feature of evolutionary models of aggressive and violent behaviors.

## The paradoxical effect of climate on time perspective considering resource accumulation

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**Abstract:** Considering purely climate, southern countries are less harsh and more predictable than northern countries. From a historical perspective, freezing winters resulting in fewer available resources contribute to the development of strong future orientation. The paradox is that future orientation contributes to accumulation of resources in the long run, making individuals' immediate living conditions less harsh, leading to slower life strategies.

Predictability and climate can be seen from different points of view. On the basis of Life History Theory (e.g., Hill 1993; Kaplan & Gangestad 2005), it is claimed that fast life strategies (related to reproducing earlier, having higher mortality and morbidity rates, having higher levels of violence) are adopted when life events are unpredictable and harsh (Ellis et al. 2009; Griskevicius et al. 2011), and slow life strategies (the opposite) are adopted when life is more predictable and less harsh. Van Lange et al. connect this to climate and claim that in warmer areas close to the equator, life is more unpredictable and harsher (from the perspective of climate as a result of viruses and natural disasters). Contrary to this argument, if one focuses on climate and its seasonal variation close to the equator, the climate is less extreme and there is less seasonal variation. From the perspective of climate arises the question: What can be more predictable and less harsh than constant warmth with little variation? Contrary to the authors' claim, it is plausible that areas close to the equator are more predictable, and considering purely climate, seasonal changes in areas farther from the equator can result in harsh life conditions such as freezing winters, unexpected summer droughts, and floods.

From a historical perspective, in those regions where the weather was comfortably warm all year, crops could be harvested twice a year and food and shelter were available all year. However, in the past, a second harvest in the winter was not possible in northern regions and it was more difficult to find shelter, which made living conditions harsher and less predictable. In short, they had fewer exposed resources during a certain part of the year. These conditions could have motivated northerners to become more future oriented, that is, to think about the forthcoming winter and its possible negative consequences and, thus, to accumulate and save resources (Ashkanasy et al. 2004). These northerners were forced to accumulate resources to cover the periods when resources were scarce. For them, future orientation was the key to survival (Zimbardo & Boyd 2008). Throughout history, this accumulating behavior driven by future orientation allowed northern societies to reach a higher level of economic development, whereas in southern regions, where resources were available all year long, societies were less focused on the future.

As accumulation of resources reached a point when there were more than enough resources for one harsh winter, the perceived availability of resources changed. If the pantry is full all year, the

## Study Information

1. Title
    1.1. Provide the working title of your study. It may be the same title that you submit for publication of your final manuscript, but it is not a requirement.

Effects of environmental harshness and parental investment on trust

2. Authors

Hugo Mell, Ava Guez, Lilas Gurgand, Franck Ramus, Coralie Chevallier

3. Research Questions
    3.1. Please list each research question included in this study.

- What is the effect of environmental harshness on trust for 11 year-old children?
- What is the relationship between environmental harshness and parental investment? Does harshness influence parental investment? Is the effect of harshness buffered by parental care? Is the effect of harshness mediated by parental investment?
- Exploratory question: What is the effect of environmental unpredictability on trust (and the potential buffering effect of parental investment in that analysis)?

4. Hypotheses
    4.1. For each of the research questions listed in the previous section, provide one or multiple specific and testable hypotheses. Please state if the hypotheses are directional or non-directional. If directional, state the direction. A predicted effect is also appropriate here.

- We expect to see a decrease of trust with increasing environmental harshness (and unpredictability).
- We expect to see a buffering effect of parental investment: the decrease in trust associated with environmental harshness (and unpredictability) will be attenuated by parental investment (interaction).
- We expect to see a decrease of parental investment with rising environmental harshness (and unpredictability).

## Sampling Plan

In this section weÍll ask you to describe how you plan to collect samples, as well as the number of samples you plan to collect and your rationale for this decision. Please keep in mind that the data described in this section should be the actual data used for analysis, so if you are using a subset of a larger dataset, please describe the subset that will actually be used in your study.

5. Existing data
    5.1. Preregistration is designed to make clear the distinction between confirmatory tests, specified prior to seeing the data, and exploratory analyses conducted after observing the data. Therefore, creating a research plan in which existing data will be used presents unique challenges. Please select the description that best describes your situation. Please do not hesitate to contact us if you have questions about how to answer this question (prereg@cos.io).
        5.1.1. Registration prior to creation of data: As of the date of submission of this research plan for preregistration, the data have not yet been collected, created, or realized.
        5.1.2. Registration prior to any human observation of the data: As of the date of submission, the data exist but have not yet been quantified, constructed, observed, or reported by anyone - including individuals that are not associated with the proposed study. Examples include museum specimens that have not been measured and data that have been collected by non-human collectors and are inaccessible.
        5.1.3. Registration prior to accessing the data: As of the date of submission, the data exist, but have not been accessed by you or your collaborators. Commonly, this includes data that has been collected by another researcher or institution.
        5.1.4. Registration prior to analysis of the data: As of the date of submission, the data exist and you have accessed it, though no analysis has been conducted related to the research plan (including calculation of summary statistics). A common situation for this scenario when a large dataset exists that is used for many different studies over time, or when a data set is randomly split into a sample for exploratory analyses, and the other section of data is reserved for later confirmatory data analysis.
        5.1.5. Registration following analysis of the data: As of the date of submission, you have accessed and analyzed some of the data relevant to the research plan. This includes preliminary analysis of variables, calculation of descriptive statistics, and observation of data distributions. Studies that fall into this category are ineligible for the Pre-Reg Challenge. Please contact us (prereg@cos.io) and we will be happy to help you.

6. Explanation of existing data
    6.1. If you indicate that you will be using some data that already exist in this study, please describe the steps you have taken to assure that you are unaware of any patterns or summary statistics in the data. This may include an explanation of how access to the data has been limited, who has observed the data, or how you have avoided observing any analysis of the specific data you will use in your study. The purpose of this question is to assure that the line between confirmatory and exploratory analysis is clear.

We will use data from the Eden mother-child cohort, described by Heude et al. (2015).

7. Data collection procedures.
    7.1. Please describe the process by which you will collect your data. If you are using human subjects, this should include the population from which you obtain subjects, recruitment efforts, payment for participation, how subjects will be selected for eligibility from the initial pool (e.g. inclusion and exclusion rules), and your study timeline. For studies that donÍt include human subjects, include information about how you will collect samples, duration of data gathering efforts, source or location of samples, or batch numbers you will use.

The cohort used in this study is the EDEN mother-child cohort that includes data collected at several time points using parental questionnaires, children questionnaires and data collected during visits to the research centers. The children in the last wave of the EDEN cohort were 11 years old (11.5 years old on average). The main goal of the EDEN study is to determine the relation between prenatal / early postnatal factors and child health / development. EDEN was set up in 2003 in two university maternity clinics (in Nancy and Poitiers, France), by the local clinical teams from the local university hospitals in collaboration with research teams from the National Institute of Health and Medical Research (Inserm). Exclusion criteria included a personal history of diabetes, twin pregnancy, intention to deliver outside the university hospital or to move out of the study region within the following 3 years, and inability to speak French. 2002 pregnant women joined the cohort but there has been important attrition since then and 425 11-year-old children remain in the cohort.It is important to notice that urban, well-educated and high-income households are over-represented among the EDEN mothers compared with the national population. This characteristic is accentuated over the years due to differential attrition.

The study was approved by the Ethical Research Committee (Comité consultatif de protection des personnes dans la recherche biomédicale) of Bicêtre Hospital and by the Data Protection Authority (Commission Nationale de l'Informatique et des Libertés). Informed written consents were obtained from parents for themselves at the time of enrollment and for the newborn after delivery.

8. Sample size
    8.1. Describe the sample size of your study. How many units will be analyzed in the study? This could be the number of people, birds, classrooms, plots, interactions, or countries included. If the units are not individuals, then describe the size requirements for each unit. If you are using a clustered or multilevel design, how many units are you collecting at each level of the analysis?

We will use data from the 425 11 year-old children who remain in the EDEN cohort and who have answered the trust questions.

9. Sample size rationale
    9.1. This could include a power analysis or an arbitrary constraint such as time, money, or personnel.

We will use all the available data.

10. Stopping rule
    10.1. If your data collection procedures do not give you full control over your exact sample size, specify how you will decide when to terminate your data collection.

NA

**Variables**

In this section you can describe all variables (both manipulated and measured variables) that will later be used in your confirmatory analysis plan. In your analysis plan, you will have the opportunity to describe how each variable will be used. If you have variables which you are measuring for exploratory analyses, you are not required to list them, though you are permitted to do so.

11. Manipulated variables
    11.1. Describe all variables you plan to manipulate and the levels or treatment arms of each variable. For observational studies and meta-analyses, simply state that this is not applicable.

Not applicable, as this is an observational study.

12. Measured variables
    12.1. Describe each variable that you will measure. This will include outcome measures, as well as any predictors or covariates that you will measure. You do not need to include any variables that you plan on collecting if they are not going to be included in the confirmatory analyses of this study.

<u>Variables from auto questionnaires :</u>

1) <u>Predictors</u>

a. <u>Environmental harshness</u>

- Has the mother been hospitalized recently? (yes or no, at 1, 2, 3, 5, 8 years old)
- What is the household income? (categorical variable, during pregnancy and at 4 months, 1, 2, 3, 4, 5, 8 and 11 years old)

- Do the parents smoke? (yes or no, frequency, during pregnancy and at 8 months, 1, 2, 3, 5, and 8 years old)
- Do the parents smoke weed ? (yes or no, frequency, at 8 months and 2 and 8 years old)
- Do the parents drink alcohol? (yes or no, amount, before and during pregnancy and at 1, 2, 3, and 8 years old)
- Do the child live near an important road? (yes or no, distance, at 1, 2, 3, and 8 years old)
- Do the mother and the father have a professional activity? (before pregnancy, at 4 months old and at 1, 2, 3, 4, 5, 8 and 11 years old)
- With whom does the mother live ? (during pregnancy, at 8 months old and at 1, 2, 3, 4, 5, 8 and 11 years old
- Are there any difficulties to feed/ provide medical cares/ provide clothes/ in paying rent, heating… (no, yes a little, yes a lot, during pregnancy and at 2, 3, 5, 8, 11 years old).
- Has the mother experienced grueling events? (for various events, how grueling?, at 3, 5 and 8 years old).

b. Parental investment

- At what frequency does the mother / the father do these activities with their child : wash them/ give them their meal / sing with them/ read them a story / play games / take them on a walk / play physical games / listen to music / paint, draw / play with stickers or modeling clay / do cultural journeys / watch TV, play video games / share a meal / put them in bed / pick them up at school or activities / supervise homework / talk about what they did / do shopping? (everyday or almost/3 to 5 times a week/ 1 or 2 times a week/ less than 1 time a week/ never or almost never, at 2, 3 and 8 years old).
- Do the parents encourage their child to tell them about their experiences, or do they take time to listen to them? (yes or no, at 5 years old)
- Does the child take meals with their mother and father (or paternal figure, yes or no, at 5 years old)?
- Is the child authorized to choose some of their food during meals? (yes or no, at 5 years old)
- Do family members take them out? (yes or no, at 5 years old)
- Has the child been to a museum the past year? (yes or no, and at which frequency (once a month/sometimes in a year/ once a year/ never) at 5 years old)?
- Do the parents take the child to the supermarket (yes or no, which frequency(everyday or almost/ once a week/once a month or less), at 5 years old)?
- Can the child choose some foodstuffs he likes when you go to the supermarket?(yes or no, at 5 years old)
- Do the parents teach the child some simple courtesy?(yes or no, at 5 years old)
- Is the child encouraged to read sequences of words? To read numbers? To read letters from the alphabet? To read words? To learn colours? To learn spatial relations? (yes or no, at 5 years old)
- Does the mother use long sentences? Correct grammar and pronunciation? (yes or no, at 5 years old)
- When she talks to the child, does the mother adapt her voice favorably? (does she seems happy to be with her child, does she speak to him in a pleasant and playful manner?) (yes or no)
- Is the TV on during meals? (yes or no, at 5 years old)
- Does the child have toys or musical instruments ? (yes or no, at 5 years old)

- Does the child have toys to learn the animal's names? (yes or no, at 5 years old)
- Are the artistic works of the child kept somewhere in the house? (yes or no, at 5 years old)
- Do the parents try to make the child tidy up their toys after they finish to play? (yes or no, at 5 years old)

c. Controls
- Centre (Nancy or Poitiers)
- Parental education level
- Age of the child

2) Outcomes (trust)
- 3 questions about trust on the 11 years-old questionnaire. :
    1) Who do you most agree with ? Fred : "Generally most people can be trusted", Nico : "You can't be too careful in dealing with people". (I completely agree with Fred/ I agree a little with Fred/ I completely agree with Nico/ I agree a little with Nico)
    2) Who do you most agree with? Fred : "Most of the people try to be fair", Nico : "Most of the people take advantage of me". (I completely agree with Fred/ I agree a little with Fred/ I completely agree with Nico/ I agree a little with Nico)
    3) Who do you most agree with? Fred : "Most of the time people try to be helpful", Nico : "Most of the people are mostly looking out for themselves". (I completely agree with Fred/ I agree a little with Fred/ I completely agree with Nico/ I agree a little with Nico)

13. Indices

13.1. If any measurements are going to be combined into an index (or even a mean), what measures will you use and how will they be combined? Include either a formula or a precise description of your method. If your are using a more complicated statistical method to combine measures (e.g. a factor analysis), you can note that here but describe the exact method in the analysis plan section.

We will combine several measures in order to create the scores of Harshness, Trust and Parental Investment. (See "Transformations")

**Design Plan**

In this section, you will be asked to describe the overall design of your study. Remember that this research plan is designed to register a single study, so if you have multiple experimental designs, please complete a separate preregistration.

14. Study type

14.1. Experiment - A researcher randomly assigns treatments to study subjects, this includes field or lab experiments. This is also known as an intervention experiment and includes randomized controlled trials.
14.2. Observational Study - Data is collected from study subjects that are not randomly assigned to a treatment. This includes surveys, natural experiments, and regression discontinuity designs.
14.3. Meta-Analysis - A systematic review of published studies.
14.4. Other - please explain.

15. Blinding
15.1. Blinding describes who is aware of the experimental manipulations within a study. Mark all that apply.
15.1.1. No blinding is involved in this study.

No blinding is involved in this study. The data collected were initially meant to be used for health studies, we did not made the questionnaires ourselves except for the questions about trust.

15.1.2. For studies that involve human subjects, they will not know the treatment group to which they have been assigned.
15.1.3. Personnel who interact directly with the study subjects (either human or non-human subjects) will not be aware of the assigned treatments.
15.1.4. Personnel who analyze the data collected from the study are not aware of the treatment applied to any given group.

16. Study design
16.1. Describe your study design. Examples include two-group, factorial, randomized block, and repeated measures. Is it a between (unpaired), within-subject (paired), or mixed design? Describe any counterbalancing required. Typical study designs for observation studies include cohort, cross sectional, and case-control studies.

This is a longitudinal cohort survey (during pregnancy, at 4 months, 8 months, 1 year, 2 years, 3 years, 4 years, 5 years, 8 years and 11 years-old).

17. Randomization
17.1. If you are doing a randomized study, how will you randomize, and at what level?
NA

**Analysis Plan**

You may describe one or more confirmatory analysis in this preregistration. Please remember that all analyses specified below must be reported in the final article, and any additional analyses must be noted as exploratory or hypothesis generating.

A confirmatory analysis plan must state up front which variables are predictors (independent) and which are the outcomes (dependent), otherwise it is an exploratory analysis. You are allowed to describe any exploratory work here, but a clear confirmatory analysis is required.

18. Statistical models
    18.1. What statistical model will you use to test each hypothesis? Please include the type of model (e.g. ANOVA, multiple regression, SEM, etc) and the specification of the model (this includes each variable that will be included as predictors, outcomes, or covariates). Please specify any interactions that will be tested and remember that any test not included here must be noted as an exploratory test in your final article.

We will use a regression analysis including Environmental Harshness as a predictor (see "transformations") and Trust as the outcome variable.

Question 1: What is the effect of environmental harshness on trust for 11 year-old children?
Trust = a + b1*H + b2*C + e
With: H=Harshness and C=set of control variables

Question 2a: Is the effect of environmental harshness buffered by parental investment?
Trust = a + b1*H + b2*PI+ b3*PI*H+ b4*C+ e
With: PI=Parental investment

Question 2b: Additional question: To what extent is the effect of environmental harshness mediated through parental investment // To what extent is parental investment determined by environmental harshness?
Trust = a + b1*H + b2*PI+ b3*PI*H+ b4*C+ e
PI = a + b1*H + b2*C + e

19. Transformations
    19.1. If you plan on transforming, centering, recoding the data, or will require a coding scheme for categorical variables, please describe that process.

Transformed variables :

- With whom does the mother live ? We will transform this variable into two binary variables: does the mother live with the father of the child? (yes or no), is there a stepfather living with the child? (yes or no).

- Are there any difficulties to feed/ provide medical cares/ provide clothes/ in paying rent, heating… (no, yes a little, yes a lot). We will transform this variable into a binary variable (yes (yes a little and yes a lot) or no)
- Does the mother and the father have a professional activity ? We will transform this variable into a binary variable (yes or no : the mother is unemployed or not)
- Family income will be divided by the number of children in order to adequately compare economic resources across family types.

Environmental Harshness : every variable at each time point will be z-scored. Variables will be recoded such that for each variable a high score indicates high harshness. Harshness is defined as a composite measure calculated based on the sum of z-scored harshness variables. We will average the z-scores of each variable for all the time points, and then sum up the average z-scores for all the harshness' variables in order to obtain a global score of harshness.

To measure children's trust in others, we will use the answers at 3 questions asked in a questionnaire at 11-years-old. For each question there are 4 possible answers, that we will code by order (1, 2, 3, 4, increase in trust). For each child, we will z-score and then sum up the z-scores at each question in order to obtain a global score of trust.

For the parental investment, we will proceed in the same way as for harshness.

20. Follow-up analyses

20.1. If not specified previously, will you be conducting any confirmatory analyses to follow up on effects in your statistical model, such as subgroup analyses, pairwise or complex contrasts, or follow-up tests from interactions. Remember that any analyses not specified in this research plan must be noted as exploratory.

Additional analyses are not planned, especially as the EDEN cohort is ending.

21. Inference criteria

21.1. What criteria will you use to make inferences? Please describe the information youÍll use (e.g. p-values, bayes factors, specific model fit indices), as well as cut-off criterion, where appropriate. Will you be using one or two tailed tests for each of your analyses? If you are comparing multiple conditions or testing multiple hypotheses, will you account for this?

Two-tailed p-values < .05

22. Data exclusion

22.1. How will you determine what data or samples, if any, to exclude from your analyses? How will outliers be handled?

We will not exclude any data.

23. Missing data

23.1. How will you deal with incomplete or missing data?

Multiple imputation techniques will be used to preserve sample size and avoid biased estimations of model parameters. Following recommendations by Bodner (2008) a number of imputed datasets equivalent to the percentage of missing cases will be generated by fully conditional specifications for categorical and continuous data using the r package mice (Buuren & Groothuis-Oeudshoorn, 2011). This package allows the use of different imputation methods depending on the type of variable with missing entries. Indeed, predictive mean matching will be used for numeric variables, logistic regression imputation for binary data and proportional odds model for ordered categorical variables with more than two levels. All variables used in the analysis models will be included in the imputation model.

24. Exploratory analysis (optional)

24.1. If you plan to explore your data set to look for unexpected differences or relationships, you may describe those tests here. An exploratory test is any test where a prediction is not made up front, or there are multiple possible tests that you are going to use. A statistically significant finding in an exploratory test is a great way to form a new confirmatory hypothesis, which could be registered at a later time.

For the study of Unpredictability, we will measure the median absolute deviation for each variable included in the harshness composite, provided we have a minimum of 3 points. The median absolute deviation is more robust than the standard deviation when few data points are available.

We will use a regression analysis including Unpredictability or Unpredictability and Harshness as predictors and Trust as the outcome variable.

Trust = a + b1*U + b2 *C + e + control for overall level of harshness?
Trust = a + b1*H + b2*PI+ b3*PI*U+ b4 * C + e

**Script (Optional)**

The purpose of a fully commented analysis script is to unambiguously provide the responses to all of the questions raised in the analysis section. This step is not common, but we encourage you to try creating an analysis script, refine it using a modeled dataset, and use it in place of your written analysis plan.

25. Analysis scripts (Optional)
    25.1. (Optional) Upload an analysis script with clear comments. This optional step is helpful in order to create a process that is completely transparent and increase the likelihood that your analysis can be replicated. We recommend that you run the code on a simulated dataset in order to check that it will run without errors.

**Other**

26. Other (Optional)
    26.1. If there is any additional information that you feel needs to be included in your preregistration, please enter it here.

## RÉSUMÉ

Des gradients sociaux ont été documentés pour une variété de comportements individuels. Au sein des pays occidentaux par exemple, les personnes ayant un statut socio-économique plus bas ont tendance à investir moins dans leur éducation, à fumer davantage, sont plus susceptibles de souffrir d'obésité et plus enclines à prendre des risques dans des contextes économiques. Ainsi, être exposé à des conditions de vie précaire semble engendrer une série de comportements, covariant de manière systématique. Ce syndrome comportemental a été principalement interprété comme le produit de déficits cognitifs ayant traits à la prise de décision et/ou à un manque de volonté. Dans ce manuscrit, nous explorons une approche différente, qui s'ancre dans les explications adaptationnistes des comportements humains. Au lieu d'appréhender les comportements observés chez les individus en situation de précarité comme des anomalies, ceux-ci sont perçus comme des ajustements des stratégies individuelles d'histoire de vie, adaptés à la vie dans des environnements précaires. En effet, nous approfondirons l'hypothèse selon laquelle un environnement précaire sélectionne des stratégies qui accordent davantage de poids aux bénéfices immédiats plutôt qu'aux investissements de long terme, affectant dès lors toute une gamme de comportements. Dans un premier temps, nous analyserons par le biais de modèles d'équations structurelles, différents jeux de données afin de mesurer le degré de covariation de comportements ayant trait à la reproduction, à l'investissement dans la santé et au niveau de confiance, ainsi que leur association éventuelle avec le niveau de précarité éprouvé pendant l'enfance ou à l'âge adulte. Globalement, nos résultats indiquent qu'une réduction de l'investissement dans la santé co-varie avec une stratégie de reproduction plus court-termiste, ainsi qu'un plus faible niveau de confiance. De plus, ce pattern est surreprésenté chez les personnes en situation de précarité, avec des effets persistants des conditions pendant l'enfance. En parallèle de ces travaux empiriques, nous nous sommes par ailleurs intéressés aux fondements théoriques de nos hypothèses de travail. Précisément, nous avons développé un modèle formel de stratégie d'histoire de vie prédisant les changements de préférences temporelles intra- et interindividuelles. Celui-ci nous a permis de mettre en évidence l'importance de deux facteurs distincts pour déterminer le degré optimal avec lequel des individus doivent préférer les récompenses à court-terme: 1) le niveau d'incertitude sur la probabilité de collecter une récompense délayée dans le temps, et 2) le coût d'opportunité à ne pas bénéficier de la récompense pendant la période de délai. Enfin, nous concluons ce travail en discutant des perspectives particulièrement intéressantes offertes par une intégration plus poussée de l'approche développée dans ce manuscrit, avec d'autres sciences sociales et sciences du comportement plus traditionnelles.

MOTS CLÉS – *théorie des traits d'histoire de vie, plasticité phénotypique, préférences temporelles, axe fast-slow*

## ABSTRACT

Social gradients in behavior have been documented across various domains of people's lives. In western countries, low SES individuals tend for instance to invest less in their education, to smoke more, are more subject to overweight and are more willing to take risks in financial settings. Being exposed to deprivation therefore seems to elicit a constellation of behaviors that appear to covary in a systematic fashion. This behavioral constellation of deprivation has been mostly interpreted as the product of poor decision making abilities, of a general failure of willpower. In this dissertation we explore a different interpretation that is rooted in adaptive explanations of human behavior. Instead of viewing the behaviors of low SES individuals as suboptimal deviations from a global optimum, they are seen as adjustments of people's overall life strategies that are, from an evolutionary point of view, adaptive in the particular context of a deprived ecology. Indeed, we will explore the idea that deprived environments select for strategies that put more weight on present outcomes over uncertain future outcomes, and that this present orientation in low SES individuals propagates across a range of decision domains, giving rise to the constellation. To this aim, we first use structural equation models on observational data from a diversity of samples to analyze the covariation between peoples' behaviors in several relevant domains (health, reproduction, social trust) and their exposure to deprivation during childhood and/or adulthood. Overall, we find that a lower somatic effort tends to covary with a more short-term reproductive strategy, as well as lower social trust. This pattern is associated with a higher exposure to deprivation, with unique effects of early life conditions. In addition to this empirical work, we further investigate the theoretical underpinnings of our working hypotheses, from an adaptationist perspective. Specifically, we build a formal life history model to predict optimal changes in discounting within and between individuals. This allows us to highlight that the extent to which individuals prefer short-term rewards, should vary depending on two main parameters: 1) the uncertainty around their ability to actually collect delayed rewards, and 2) the opportunity costs of not having the reward during the delay. Finally, we conclude by discussing the promising perspective of further integrating the approach adopted in the present thesis, with more traditional social and behavioural sciences.

KEYWORDS – *life history, phenotypic plasticity, delay discounting, fast-slow continuum*