Does extrinsic mortality accelerate the pace of life? A bare-bones approach

Jean-Baptiste André[1] and François Rousset[2]

[1]Institut Jean Nicod, Département d'études cognitives, ENS, EHESS, PSL Research University, CNRS, Paris France
[2]ISEM, Univ Montpellier, CNRS, EPHE, IRD, Montpellier, France.

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1 Abstract

It is commonly asserted that when extrinsic mortality is high, individuals should invest early in reproduction. This intuition thrives in the literature on life-history theory and human behavior, yet it has been criticized repeatedly on the basis of mathematical models. The intuition is indeed wrong; but a recent theoretical criticism has confused the reason why it is wrong, thereby obscuring earlier and sounder criticisms. In the present article, based on the simplest possible model, we sought to clarify these issues. We confirm earlier findings that extrinsic mortality can affect the evolution of pace of life, not because it leaves little time to reproduce, but through its effects on density-dependent competition. This result highlights the importance of accounting for density-dependence in theoretical models and data analyses. Further, we find little support for the recent claim that the direction of selection on a reaction norm in a variable environment cannot be easily inferred from models made in homogeneous environments. In conclusion, although life-history theory is still imperfect, it has provided simple results that deserve to be understood.

2 Introduction

Life history theory is widely used to interpret in an adaptive way a number of inter-individual differences observed within the human species, that can be linked to variations in individuals' environments. In particular, consistent data show that people living in harsh environments (poor, dangerous, and/or uncertain environments; the notion of harshness being partly ambiguous) invest more, and earlier, in reproduction, and less in the growth and maintenance of their biological capital (see Pepper and Nettle 2017 for a review). In the context of life history theory, this observation is interpreted as the plastic expression of a "fast" strategy supposed to be adaptive in harsh environments.

Not all the dimensions of the physiology and behavior of people living in harsh conditions are well characterized empirically and, above all, not all have a clear adaptive explanation. Life history approaches still require many developments to account for the whole logic of human intra-specific variability (see Mathot and Frankenhuis 2018 for a review). But there is at least one intuition in this literature, that has (bibliometrically if not scientifically) survived previous discussions: when extrinsic mortality is high, selection favours a fast strategy, that is a strategy with a relatively high investment in reproduction and low investment in survival. When extrinsic mortality is high (i.e., in a harsh environment), individuals have a greater chance of dying before they have had time to reproduce and therefore, so the intuition goes, they must invest early and intensively in reproduction, to increase their chances of reproducing before they die. That is, their pace of life must be fast. Conversely, when extrinsic mortality is low, individuals are unlikely to die young, hence they can afford to invest in growth and survival and to delay their reproduction. That is, their pace of life must be slow. This intuitive understanding of the effect of extrinsic mortality was initially spelled out by Williams (1957), and is commonly recalled in the evolutionary biology literature, as well as in the literature on evolution and human behavior (see e.g., Ellis et al., 2009; Nettle, 2010; Belsky et al., 2010; Griskevicius et al., 2011; Frankenhuis et al., 2013; Mell et al., 2018).

However, a preprint posted on bioRχiv (Baldini, 2015a) has introduced great concern into the literature by stating, based on a mathematical model, that this effect of extrinsic mortality would be false, or at least much more fragile than previously thought. In principle, Baldini's article deals with all the effects of harshness on life history, but in practice his main critique of the literature does focus on the effect of extrinsic mortality. More specifically, Baldini (2015a) makes two claims. First, he claims that high extrinsic mortality need not favor a fast life history strategy. Second, he claims that the evolutionarily stable reaction norm in the case of a plastic species facing a variable environment cannot be inferred from the finding of hard-wired evolutionarily stable strategies in distinct homogeneous environments. Consequently, life history approaches cannot be used, as they are currently, to explain the intra-specific variability of human life history strategies if this variability is based on phenotypic plasticity. Baldini's preprint has had a fairly strong influence in the community of evolutionary psychologists and evolutionary anthropologists and is also cited in the recent review of the theoretical literature by Mathot and Frankenhuis (2018), as an integral part of the theoretical articles available in the field.

Baldini's results are partly based on classical papers in evolutionary biology (e.g. Abrams 1993; Williams et al. 2006; Caswell 2007), and a further paper published since then studies the effects of extrinsic mortality along the same lines but in greater detail (Dańko et al. 2017, and see Moorad et al. (2019) and Del Giudice, 2019 for a review; note that Baldini also published an article later, which partly reflects the same ideas; Baldini 2015b). Unfortunately, while Baldini (2015a) is right to affirm that the classic intuition is false, he is right for a wrong reason. In particular, his paper is based on a definition of extrinsic mortality that differs from the definition usually used in the evolutionary literature, which leads to misleading results (see also Del Giudice, 2019 in this special issue). In part for this very reason, he does not present an alternative way of understanding the effect of extrinsic mortality, which leaves non-theoreticians in a hopeless situation, doubting the whole theory because they cannot understand it with enough confidence. This is all the more problematic as several theoretical works have developed more careful criticism of the classical intuition. The key issue, as emphasized by Dańko et al. (2018), is to take into account how density-dependence affects fitness.

Here our objective is to clarify this issue and expose in the simplest possible way the main points that need to be understood, by scholars in evolutionary psychology and evolutionary anthropology, regarding the effect of extrinsic mortality on the evolution of pace of life. Our analysis is simpler than previous works, which rested on the mathematics of age-structured populations which may obscure some simple messages. However, we concur with the conclusions of these previous works and in particular with Dańko et al. (2018) who stressed that the importance of taking density dependence into account is still under-appreciated in broad discussions of life-history evolution (although accounted in a number of specific models) and that too little effort is put to document pathways of density-dependence in natural populations. The same problems have been met in the study of spatially structured populations (Leturque and Rousset, 2004).

Our aim is thus to build a minimal model that captures the key findings of this literature in the simplest possible way (in particular the results of Dańko et al. 2017). In a nutshell these findings are the following. (1) Extrinsic mortality does *not* directly affect the evolution of life history traits. (2) Extrinsic mortality only affects the evolution of life

history *indirectly* through its effect on the intensity of competition. A higher extrinsic mortality reduces the intensity of competition whereas a lower mortality leads to more competition. (3) This modification of the intensity of competition can affect in turn the evolution of life history and this effect can be understood in a principled manner (4) *Quantitatively* speaking, the effect of extrinsic mortality on life history in a plastic species living in a variable environment may be different from its effect in a hard-wired species living in a constant environment. (4) But, *qualitatively*, the direction of the effects are the same in both cases.

3 Modelling approach

3.1 Measuring the effect of selection in a homogeneous environment

The objective of the following analysis is to measure the effect of natural selection on a quantitative trait that controls the pace of life of individuals, or more generally a quantitative trait that affects both their fecundity and survival. For example, it could be a trait that increases fertility at the expense of life expectancy, or it could be a trait that consists in investing into survival at the expense of fertility. Our analysis uses an approach developed by Day and Gandon (2005). We start by considering a species living in a homogeneous environment, and we will see afterward how this approach can be generalized to account for the case of a plastic species living in a variable environment.

We consider a population consisting of n different genotypes. At time t the genotype i is in density $N_i(t)$ with $i \in [\![1, n]\!]$. Total population density at t is therefore $N(t) = \sum_{i=1}^{n} N_i(t)$. In what follows, for the sake of simplicity, we will sometimes drop the explicit dependency on time, but it always remains implicitly present. Each genotype is characterized by the value of a quantitative trait z_i that potentially affects both mortality and fertility. In fact, the model is general in the sense that z could really represent *any* trait.

1. We assume that an individual with a trait value z has a constant mortality rate over his lifetime, given by the function $d(z, N) = \mu + m(z, N)$ where μ is the extrinsic mortality, defined as the minimum mortality rate below which individuals cannot go (see Dańko et al. 2017), and $m(z, N)$ represents the intrinsic mortality rate that depends both on the individual's strategy, z, and also potentially on the intensity of competition in the population (hence on the total density N). Note that, in the sake of simplicity, we assume that density-dependent competition only affects the intrinsic mortality of individuals. Extrinsic mortality is supposed to be a fixed constant independent of demography.

2. We assume that an individual with trait z produces offspring at a constant fertility rate over his lifetime, given by the function $b(z, N)$, which also potentially depends both on the individual's strategy, and on the intensity of density-dependent competition. The form of the functions $b(\cdot)$ and $d(\cdot)$ need not be specified.

The dynamics of the n genotypes in this population are given by the following system of

differential equations

$$\frac{1}{N_i}\dot{N}_i = r(z_i, N) \quad \text{for all } i$$

where $r(z, N) = b(z, N) - d(z, N)$ is the net growth rate of each genotype. The frequency of each genotype being $q_i \equiv \frac{N_i}{N}$, the dynamics of genotypic frequencies are given by the system

$$\dot{q}_i = \frac{\dot{N}_i}{N} - q_i\frac{\dot{N}}{N} = q_i(r_i - \bar{r})$$

where the dot accents denote time derivatives (that is, for any variable x, $\dot{x} = \frac{dx}{dt}$), and $\bar{r}$ is the average r_i over the genotype distribution. The average value of z at time t is $\bar{z} \equiv \sum_{i=1}^{n} z_i q_i$, which therefore changes due to natural selection at a rate given by

$$\dot{\bar{z}} = \sum_{i=1}^{n} z_i \dot{q}_i = \sum_{i=1}^{n} z_i q_i (r_i - \bar{r}) = \text{cov}[z, r]. \tag{1}$$

At each instant, the average value of z changes, under the effect of natural selection, at a rate given by the covariance between z and the instantaneous growth rate of genotypes, r. The trait z increases to the extent that is is associated with a larger growth rate, and vice versa. Equation 1 is a simple version of the so-called Price equation (Price, 1970).

To simplify the analysis, we now consider the case where the variability of z is small. In this case, we write the trait value of each allele as a deviation from the average trait value, i.e. $z_i = \bar{z} + \zeta_i$. We then express the change in $\bar{z}$ due to selection (Eq. 1), to the second order in ζ_i. Assuming that all allelic effects scale as a common factor ζ, in the Appendix we show that, for small ζ, it simplifies into

$$\dot{\bar{z}} = \frac{\partial r(z, N)}{\partial z}\,\text{var}(z) + O(\zeta^3) \tag{2}$$

where $\text{var}(z) = \sum_{i=1}^{n} q_i \zeta_i^2$ is the variance of z in the population. In other words, to the second order, the change, due to selection, of the average value of z is simply proportional to the derivative of the growth rate r with respect to z. In what follows, we simply call this value the selection gradient on z, given by

$$S(z, N) \equiv \frac{\partial r(z, N)}{\partial z} = \frac{\partial b(z, N)}{\partial z} - \frac{\partial m(z, N)}{\partial z}. \tag{3}$$

This expression is general. It does not imply any specific form of density-dependent competition (in fact it is valid even in the total absence of density-dependent competition) and it does not even assume that the population is at a demographic equilibrium. From this expression, our objective will be to understand how, and under what conditions, the selection gradient may depend on the extrinsic mortality of individuals.

3.2 Measuring the effect of selection in a variable environment

We now turn to Baldini (2015a)'s second statement that the evolutionarily stable reaction norm in the case of a plastic species facing a variable environment cannot be inferred from the hard-wired evolutionary stable strategies in different homogeneous environments. To do so, we extend the above approach to consider the case where the environment is

variable, either in space or time. For example, in some places or at certain times, extrinsic mortality is high, while it is low in others. We assume that individuals are plastic in the expression of their life history trait z. In an environment of type ε they express a trait value z^ε. For simplicity, we neglect (i) the cost of plasticity and (ii) the possible existence of a delay in the expression of plasticity. We simply assume that, in each type of environment, individuals are able to instantaneously express a life history strategy specific to that environment, by modifying their allocation to survival and reproduction. Importantly, we also assume that the fecundity and mortality of individuals living in an environment of type ε only depend (i) on the properties of ε, and (ii) on the density N^ε of competitors in this very environment. That is, individuals are not affected by the environments in which they do not live (or only indirectly via N^ε). Under these assumptions, our objective is to evaluate the direction of natural selection on each value of z^ε.

An individual placed in an environment of type ε has fecundity $b^\varepsilon(z^\varepsilon, N^\varepsilon)$ and mortality $\mu^\varepsilon + m^\varepsilon(z^\varepsilon, N^\varepsilon)$, where N^ε measures the intensity of density-dependent competition in the environment of type ε. As in the case of a homogeneous environment (Equation 3), the instantaneous direction of selection on z^ε is given by

$$S_\varepsilon(z^\varepsilon, N^\varepsilon) = \frac{\partial b^\varepsilon(z^\varepsilon, N^\varepsilon)}{\partial z^\varepsilon} - \frac{\partial m^\varepsilon(z^\varepsilon, N^\varepsilon)}{\partial z^\varepsilon} \tag{4}$$

4 Results

4.1 Selection in a constant environment

The first thing we observe in equation 3 is that μ does not appear in the expression of S, hence that the selection gradient on the trait z cannot depend on extrinsic mortality (except through an effect on the other parameters in this equation; "indirect effect", see below). In other words, the level of extrinsic mortality experienced by individuals, in itself, has no effect on selection for *any* trait, which is a standard result obtained by several classic papers (Abrams, 1993; Williams et al., 2006; Caswell, 2007) and see Moorad et al. (2019) for a review. However, the selection gradient on the trait z can depend *indirectly* on extrinsic mortality. All other things being equal, if extrinsic mortality is higher then the population density, N, is always lower since individuals live shorter lives on average. Extrinsic mortality can therefore affect the evolution of the life history strategy indirectly via its effect on N (since N appears in eq. 3).

Here we find a discrepancy with Baldini (2015b) who observes that, even in the absence of density-dependent regulation (thus without any effect of demography), what he calls "extrinsic mortality" does affect the evolutionarily stable life history, and this in a direction opposite to the standard view. This result, however, is actually a consequence of using a definition of extrinsic mortality that is inconsistent with previous works. Extrinsic mortality is standardly defined as the minimum mortality below which individuals cannot fall (see Dańko et al. 2018), and it corresponds to the notion of a truly extrinsic source of mortality (independent of the individual's actions). By contrast, Baldini's extrinsic mortality parameter is a maximal mortality rate: it is the rate affecting individuals who do not invest any resource at all in their survival. Thus defined, "extrinsic mortality" increases the marginal return on investment in survival, forcing individuals to invest

more in survival and reproduce later, thus giving the impression of an effect opposite to the standard predictions. On the contrary, if defined properly, in the absence of density-dependent regulation extrinsic mortality merely has no effect on the evolution of life history traits.

4.1.1 If pace of life and competition have independent effects, then extrinsic mortality plays no role in the evolution of pace of life

Let us first assume that the trait z and density-dependent competition affect fertility and mortality independently from each other, i.e., that they have additive effects. We write the fertility of a genotype z as $b(z, N) = b_0(z) - \alpha N$, and its mortality as $m(z, N) = \mu + m_0(z) + \beta N$, where α and β measure the intensity of the effect of competition, respectively, on fertility and survival.

In this case, the selection gradient on z is simply given by $S(z, N) = b_0'(z) - m_0'(z)$ and is therefore independent of demography. This is logical since, in this situation, density-dependent competition affects all genotypes identically. As a result, extrinsic mortality cannot have any effect on the evolution of z. A particular case of this situation occurs in the total absence of density-dependent regulation ($\alpha = \beta = 0$; which is nothing more than a specific form of an absence of interaction). In this case, extrinsic mortality also plays no role in the evolution of z (see also Moorad et al. 2019).

4.1.2 If pace of life interacts with competition, then extrinsic mortality affects its evolution

Let us now assume that the trait z and density-dependent competition interact with one another. We thus write the fertility of a genotype z as $b(z, N) = b_0(z) - \alpha N - \gamma b_0(z)N$, and its mortality as $m(z, N) = \mu + m_0(z) + \beta N + \delta m_0(z)N$, where γ and δ measure the *interaction* effects between z and N, respectively on fertility and mortality. If $\gamma > 0$, then competition reduces more strongly the fertility of an individual who invests a lot in reproduction (if $\gamma < 0$, it is the opposite). If $\delta > 0$, then competition increases more strongly the mortality of an individual who invests little in survival (if $\delta < 0$, it is the opposite).

The direction of selection on z is given here by $S = b_0'(z)(1-\gamma N) - m_0'(z)(1+\delta N)$. All other things being equal, selection is of course always positive on fertility and negative on mortality. What matters, however, is to measure the *relative* strength of selection on these two components. If selection is particularly strong on fertility, then this means that fast strategies are favoured. While, if selection is particularly strong on survival, then it means that slow strategies are favoured. Here the relative strength of selection on fertility and mortality is given by the ratio $F = (1 - \gamma N)(1 + \delta N)^{-1}$ which is therefore a measure of the strength of selection in favour of a Fast pace of life. From this expression, we find the following.

Higher extrinsic mortality and therefore less intense competition (lower N) favours a faster strategy, with higher investment in fertility and lower investment in survival (higher F), in two cases:

1. If competition reduces the benefit of investing in reproduction, by impacting especially strongly individuals with high fertility ($\gamma > 0$). This is typically the case if

competition strongly affects immature offspring, by reducing their survival, which has the effect of reducing the benefits of investing in reproduction.

2. If competition increases especially strongly the mortality of individuals who invest little in their survival ($\delta > 0$). This can happen if individuals with a small somatic investment are particularly sensitive to competition, and have a mortality rate that increases sharply in the presence of competitors.

Conversely, higher extrinsic mortality and therefore less intense competition (lower N) favours a slower strategy, with lower investment in fertility and higher investment in survival (lower F), in two cases:

1. If competition increases the benefit of investing in reproduction by impacting less strongly individuals with high fertility ($\gamma < 0$).

2. If competition increases especially strongly the mortality of individuals who invest a lot in their survival ($\delta < 0$). This can happen if the older individuals, and/or those who have invested the most in their biological capital, are more strongly impacted by competition than the younger or smaller ones.

In summary, the key notion to remember is that extrinsic mortality *only* affects the evolution of life history via its effect on competition and *by no other means*. To get an intuitive understanding of the effect of mortality implies to get an intuitive understanding of the effect of competition, nothing more. If a trait is an adaptation to intense competition that allows individuals to better thrive under this circumstance, for example by producing fewer but less fragile offspring, by investing in the ability to survive in a dense population, or by investing in the ability to compete for resources, then this trait is particularly favoured when extrinsic mortality is low. Conversely, if a trait is adapted to an environment with a low level of competition, but makes individuals especially sensitive to the presence of conspecifics, for example by producing numerous but fragile offspring, or by investing little in somatic features that allow to better compete, then this trait is favored when extrinsic mortality is high.

4.2 Selection in a variable environment

In the case of a heterogeneous environment, from equation 4 we see that, in a local environment of any given type ε, extrinsic mortality does *not* directly affect the selection on z^{ε}. But it affects it *indirectly* via its effect on the intensity of competition (measured by N^{ε}). We can therefore qualitatively understand the effect of extrinsic mortality in a heterogeneous environment, as we did in a constant environment. If extrinsic mortality is particularly high in a given type of environment, this affects the direction of selection on the life history strategy expressed in this particular type of environment (i.e. it exerts a selective pressure on the reaction norm at this particular point), only to the extent that this higher mortality leads to a relaxation of competition in *this* type of environment. Conversely, if extrinsic mortality is particularly low in a given environment, it affects the direction of selection, only to the extent that this lower mortality leads to an enhanced competition in *this* type of environment.

We can therefore understand why the effect of selection in a variable environment may not be the same, *quantitatively*, as in a constant environment. When the environment is variable, the intensity of competition in a given type of environment does not depend solely on extrinsic mortality in *this* type of environment. If the migration rate from one environment to another is large, or if environment properties change rapidly through time, then demography in a given type of environment is also affected by demography in other types. If extrinsic mortality is very high in a given environment, for instance, individuals living in this environment may still experience a relatively intense competition if they are surrounded (temporally or spatially) by other environments where mortality is low. As a consequence of this demographic coupling between environments, the difference between the life history strategies expressed by plastic individuals in two different environments is likely to be lower than the difference between the hard-wired strategies expressed by two populations evolving independently in different environments.

However, from a *qualitative* point of view, the effect of extrinsic mortality is the same in a variable environment than in a constant one, except under some unlikely scenarios. To understand, consider two environments that differ in terms of their level of extrinsic mortality. Say, extrinsic mortality is higher in environment of type ε_1 than in environment of type ε_2. We know from equation 4, that extrinsic mortality in environment ε_1 affects the direction of selection in this environment only via its effect on population density N^{ε_1}. So, for the effect to reverse in the presence of environmental heterogeneity, population density would have to be *larger* in environment ε_2 than in environment ε_1, even though extrinsic mortality is higher. This could only happen in specific scenarios where the migration rate of individuals is so high that the recruitment of dispersing individuals is higher than the recruitment of philopatric ones, or where the environment changes cyclically at a rate close to generation time. But, apart from these specific scenarios, when extrinsic mortality decreases in a given environment, it may increase the intensity of competition less than in a fixed environment taken in isolation, but it does not reduce the intensity of competition in this environment; and conversely, if extrinsic mortality increases in a given environment, it does not increase the intensity of competition, even if it does not reduce it as much as in an environment taken in isolation. Therefore, even though the precise shape of the reaction norm cannot be predicted quantitatively, the direction of its slope can be reasonably well predicted qualitatively from the analysis of evolutionary stable strategies in constant environments.

Here as well, we observe a discrepancy with Baldini's results, who found that an environmental heterogeneity in terms of "extrinsic mortality" could lead to an opposite effect. But, as we pointed above, this result is not based on the standard definition of extrinsic mortality. This matters here because the only quantitative result shown by Baldini in a heterogeneous environment (his Figure 1) concerns the case where density-dependence is absent, which is a situation where extrinsic mortality, if properly defined, has no effect on the evolution of life history anyway.

5 Discussion

In this article we have sought to clarify the effect of extrinsic mortality on the evolution of the ratio of investments in survival vs. reproduction, hereby referred to as the pace

of life. Three intuitions on this point are common in the literature (see e.g. Ellis et al., 2009; Nettle, 2010; Belsky et al., 2010; Griskevicius et al., 2011; Frankenhuis et al., 2013; Mell et al., 2018): (I1) An environment with high extrinsic mortality always leads to the evolution of a fast pace of life (that is, a small investment in survival and a large investment in reproduction). (I2) This effect is due to the fact that, if mortality is high, individuals are likely to die before they have had time to reproduce. (I3) This effect is true both if the environment is constant and the pace of life a hard-wired strategy and if the environment is variable and the pace of life a plastic reaction norm. In the present article, based on a simple model, we have sought to clarify the issue and obtained the following results:

(1) Strictly speaking, the three common intuitions above are false. In particular, intuition I2 is profoundly wrong. When extrinsic mortality does affect the evolution of pace of life, it is not for the intuitive reason that individuals have little time to reproduce, but for another reason related to the effect of extrinsic mortality on competition (see below). This point has already been known in the theoretical literature for a long time (Abrams, 1993; Williams et al., 2006; Caswell, 2007; and see Moorad et al., 2019 for a review).

(2) Theoreticians are not hopeless, however, when it comes to predicting and explaining the effect of extrinsic mortality. With our simple model, we showed that it is possible to explain in a fairly intuitive and principled way the genuine effects of extrinsic mortality on pace of life. Extrinsic mortality has no "direct" effect on the evolution of pace of life. However, it can affect it *indirectly* through its effect on the intensity of competition. Reducing extrinsic mortality always increases the intensity of competition because more individuals can be maintained in the environment. Hence, reducing extrinsic mortality favours slower strategies if these strategies allow to better thrive under intense competition and, on the contrary, faster strategies if these strategies do better under intense competition. Overall, all the traits that are adaptive under intense competition are favoured when extrinsic mortality is low and, conversely, all the traits that are adaptive when competition is relaxed are favoured when extrinsic mortality is high. This is undoubtedly different from intuition I2, but it can be explained to non-theoreticians in a simple manner. In particular, it must be stressed that whereas it is wrong to say that extrinsic mortality *always* favors a faster strategy, in many cases it actually does. This is notably the case in the very frequent situations where density-dependent regulation takes place via the fecondity of individuals (as in virtually all epidemiological models for instance; May and Anderson, 1979). In this case, a reduction in extrinsic mortality leads to a reduction in the marginal return of investing in reproduction relative to survival, which does favour a slower strategy.

(3) Intuition I3 is quantitatively wrong but still qualitatively true. Strictly speaking, models of homogeneous environments do not allow to measure the effect of selection in variable environments. But the discrepancy between the two is only quantitative, not qualitative. Provided the cost of phenotypic plasticity is negligible, the *direction* of the effect of extrinsic mortality in a variable environment (but not the force of the effect) can be predicted from a homogeneous environment model. For example, if a particular model of homogeneous environment shows that the evolutionarily stable pace of life increases with extrinsic mortality, then we can deduce that the evolutionarily stable reaction norm in a variable envionment would also cause the pace of life to increase with extrinsic

mortality.

(4) These different conclusions were discussed in his preprint by Baldini (2015a), and our conclusions superficially concur with his, but for different reasons. In his model, Baldini does not use the standard definition of extrinsic mortality which is a source of confusion (see also Del Giudice, 2019 in this special issue). The standard definition of extrinsic mortality is the minimum mortality below which individuals cannot fall (see Dańko et al. 2018). On the contrary, the parameter that Baldini calls extrinsic mortality is the *maximum* mortality, that is the mortality rate of an individual who would not invest in survival at all. This mortality is possibly an interesting parameter but it is certainly not the same thing as extrinsic mortality. Due to this unconventional definition, he obtains results that are often at odds with the standard effects of extrinsic mortality.

In this paper, we have narrowed the scope of our analysis by focusing only (i) on one aspect of environmental harshness (extrinsic mortality) and (ii) on one aspect of life history strategy (pace of life). Scholars interested in using life history theory to understand human variability, however, are interested more broadly (i) in all the distinctive features of harsh ecologies (that is, not only on extrinsic mortality), and (ii) in all the facets of life history strategies (that is, not only on pace of life). One cannot hope to understand all these effects from models that focus only on extrinsic mortality and pace of life. The empirical and theoretical literature is therefore far from having shed light on the whole range of effects of deprivation on life history.

For example, one could imagine that the most distinctive feature of harsh ecologies is not (or not only) the extrinsic mortality experienced by individuals but rather their maximum mortality (i.e. the parameter that Baldini, 2015a calls extrinsic mortality). In this case then, as in Baldini's model, the marginal return of investing in survival would be higher in harsh environments, which would lead individuals to express a slow strategy in these environments.

Or, in fact, as Kaplan and Gangestad (2015) argues, there is no such thing as a truly extrinsic mortality. It is always possible for an individual to invest in protections against any given hazard. One could rather imagine, therefore, that harsh ecologies are not characterized by a constant mortality parameter, but by the amount and type of hazards that they contain and the resulting larger cost of protecting against them. In this case, individuals living in harsh ecologies would on the contrary invest less in their survival since the marginal return to do so would be lower.

There might also be important features of harsh ecologies that have nothing to do with mortality. For example, as suggested by Mell et al. (2017) and Mell et al., the mere fact of being *deprived*, that is of having a low level of personal capital, could select for a faster psychology (where, by "personal capital", we mean both the embodied capital in the sense of Kaplan et al., 2000, and more generally all the assets that determine one's ability to exploit the environment). Mell et al. show that it is adaptive for individuals whose productivity increases steeply with each additional unit of capital to have a short time-horizon because each unit of resource that they can immediately invest into their capital significantly increases their productivity. By contrast, individuals whose productivity saturates with their personal capital should have a long time-horizon. To understand this result, let us think about the effect of demography on pace of life. In growing populations, it is well-known that selection favours fast strategies because the offspring produced early have more time to produce further offspring than the offspring produced late (Caswell,

2001). In other words, offspring are not just units of fitness to be counted, they also constitute a productive *capital* able to produce further units of fitness, and it is therefore adaptive to invest early in this capital. It turns out that the same effect takes place when one's *personal* capital is growing. Assuming that personal capital has diminishing returns on productivity, Mell et al. thus predict that deprived individuals, that is individuals with a low level of personal capital, should discount the future more than wealthy individuals, not because they have a greater risk of dying at every instant, but because each unit of resource invested into their personal capital makes more difference to their productivity.

Lastly, life history is not just pace of life or time discounting. The life history strategy of an individual also includes their investment in various forms of biological capital, and it also includes the temporal variations of their investments over the lifetime, that is their "shape of life" and not only their pace of life (Baudisch, 2011; Jones et al., 2014). Understanding all these features requires still other models. Even more broadly, the concept of life history strategy is sometimes used to encompass also a variety of behaviours (e.g. religiosity, cooperation, etc.) that cannot either be directly understood from models on pace of life or time discounting.

In conclusion, there are many difficulties and still some grey areas in the application of life history theory to human variability, but one must not throw out the baby with the bathwater by rejecting the entire theory on the grounds that it is not yet perfect. Understanding human variation based on resource allocation theory is a research program with an important stake and a solid rationale. We still need to better understand what deprivation really means, better understand the nature of the adaptive responses it has selected for, and better prove empirically the very existence of phenotypic plasiticy, but there is no reason why these shortcomings should lead us to reject, as a matter of principle, the approach as a whole, no more here than in other domains of research.

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Appendix : Measuring the effect of selection to the second order

Here we derive the instantaneous rate of change in the mean value of the trait to the second order in trait variation. We write genotype i's trait value as a deviation relative to the mean, i.e. $z_i = \bar{z} + \zeta_i$, where $\bar{z} = \sum_i q_i z_i$ is the mean trait value, and therefore the mean deviation is zero by definition ($\sum_i q_i \zeta_i = 0$).

The instantaneous rate of change of the trait due to selection is

$$\dot{\bar{z}} = \mathrm{cov}(r, z) = \mathrm{E}[rz] - \bar{r}\bar{z}$$

where $\mathrm{E}[X]$ stands for the expectation of X taken over the entire population.

We have

$$\mathrm{E}[rz] = \sum_i q_i r_i z_i = \sum_i q_i r_i (\bar{z} + \zeta_i) = \bar{z} \sum_i q_i r_i + \sum_i q_i r_i \zeta_i = \bar{r}\bar{z} + \sum_i q_i r_i \zeta_i$$

Hence this gives

$$\dot{\bar{z}} = \mathrm{E}[rz] - \bar{r}\bar{z} = \sum_i q_i r_i \zeta_i$$

Quite generally for a function with a second derivative, for some z_i^{L} between $\bar{z}$ and z_i, one can write $r_i = r + \zeta_i r_{z,1}(\bar{z}) + \frac{\zeta_i^2}{2} r_{z,2}(z_i^{\mathrm{L}})$, with $r_{z,k}(x) = k$th derivative of r with respect to z evaluated for $z = x$ (Taylor's theorem with Lagrange form of the remainder). Then, using $\sum_i q_i \zeta_i = 0$,

$$\dot{\bar{z}} = \sum_i q_i \left(\zeta_i^2 r_{z,1}(\bar{z}) + \frac{\zeta_i^3}{2} r_{z,2}(z_i^{\mathrm{L}}) \right)$$

If the second derivative is bounded in absolute value over the range of all z_i values, then this reduces to

$$\dot{\bar{z}} = r_{z,1}(\bar{z}) \operatorname{var}(Z) + O(\mathrm{E}(|Z^3|)).$$

where we view each ζ_i as a realization of a random variable $Z = z - \bar{z}$. Assuming that all allelic effects scale as a common factor ζ, then $\operatorname{var}(z) = \operatorname{var}(Z) = O(\zeta^2)$ dominates $\mathrm{E}(|Z^3|) = O(\zeta^3)$ for small ζ, so that, to leading order in ζ, the instantaneous change in the mean trait value is given by the first derivative of r with respect to z ($r_{z,1}$), multiplied by the variance of z:

$$\dot{\bar{z}} = r_{z,1}(\bar{z}) \operatorname{var}(z) + O(\zeta^3).$$

which is equation 2 of main text.