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Review

The Conundrum of Heterogeneities in Life History Studies

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What causes interindividual variation in fitness? Evidence of heritability of latent individual fitness traits has resparked a debate about the causes of variation in life histories in populations: neutralism versus empirical adaptationism. This debate about the processes underlying observed variation pits neutral stochastic demographic processes against evolutionarily relevant differences among individual fitness traits. Advancing this debate requires careful consideration of differences among inference approaches used by proponents of each hypothesis. Here we draw parallels between several disciplines focusing on processes generating variation in individuals' life-course, and we contrast methodologies to disentangle these processes. We draw on other disciplines to clarify terminology, risks of flawed inference, and expand the panel of hypotheses and formalizations of processes generating variation in life histories.

What Is at Stake in the 'Heterogeneity versus Stochasticity' Debate?

Longitudinal studies of vertebrates show that individuals differ in observed longevity and number of offspring [1,2] with a minority contributing most offspring to future generations. Is this difference among individual fates mostly due to chance, or do individuals differ in fitness traits? Although most biologists would consider that the truth lies somewhere in the middle, the debate is unresolved when the fitness traits are not directly observable. Large interindividual differences in lifetime fitness prompted biologists to assume an underlying variation in 'quality' between the individuals, although this is an old terminology which remains unclear [3–5]. An alternative formulation is that populations are heterogeneous [5]. Heterogeneity can manifest itself through a positive relationship between reproductive success and survival probabilities [6]. The idea of a ranking of individuals according to values of demographic parameters such as longevity and reproductive success (fitness traits) goes back to the 1980s in wildlife studies [7,8], but is called by several names: '**fixed heterogeneity**' [5], **persistent** '**demographic heterogeneity**' [9], or '**individual heterogeneity**' [8] (see Glossary). Heterogeneity in fitness traits is a cornerstone of evolutionary ecology, yet identifying all the observable individual characteristics that can explain heterogeneity is impossible. **Hidden Persistent Demographic Heterogeneity (HPDH)** describes unobserved individual characteristics that are fixed after individuals entered the study. In statistical models of mortality risk, HPDH controls for unobserved differences across individuals caused by factors that have not been, or cannot be measured [10]. The original motivation was to quantify unexplained heterogeneity [10] and to avoid flawed inferences in studies of senescence [11–17]. Indeed, in heterogeneous populations the pattern of variation in survival probability throughout life expressed in the population differs from that expressed at the level of groups of 'frail' and 'robust' individuals, for instance [11,14]; this disjunction illustrates '**heterogeneity's ruses**' in demography [14]. HPDH has historically been used as a tool of statistical convenience to account for unexplained differences

Trends

Evidence of heritability of individual fitness traits in wild populations has reopened a debate about the relative contribution of neutral, stochastic demographic processes to observed variations in life histories.

There are conceptual differences among published studies documenting heterogeneity in life histories; differences so fundamental that they led to misunderstandings between schools of thought.

The question of the processes generating heterogeneity in longitudinal trajectories has stimulated a large body of work in econometrics, political, social and biomedical sciences, which have highlighted risks of flawed inference; these risks have been overlooked in biology.

Other disciplines offer useful frameworks for future work on life histories in three areas: terminology, the characterization of the diversity of processes underlying variation in life histories, and the methods of statistical inference to disentangle these processes.

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among individuals in 'quality', while leaving it unclear how precisely this elusive 'quality' relates to evolutionary theory [4,5]. Although studies of heterogeneity in mortality risk have historically been focused on people, animals, and machines [14]), this framework has been extended to other organisms, for example, plants and insects [12,18].

A conjunction of factors recently propelled HPDH to the forefront in life history studies [19–21]:

(i) A methodological breakthrough in longitudinal data analysis allows estimation of HPDH when the detectability of individuals is imperfect, a typical situation in wildlife studies [22,23]. Previously only a handful of studies with quasiperfect detectability could address HPDH, for example [15–17,24–29].

(ii) Support for HPDH [22,23,30] and heritability of unobserved 'latent' traits *in natura* [30–32], in the situation of imperfect detectability.

(iii) The emergence of the neutral theory for life histories [33–35], which aims at evaluating the contributions of stochastic demographic processes and HPDH to lifetime fitness. This theory has been tested using data from animals, plants, and humans [35].

These developments used different approaches and reached conflicting conclusions about the processes underlying variation among individual life histories and lifetime fitness [30–35]; but the debate is essentially about empirical adaptationism versus neutralism [36,37]. The proponents of the neutral theory for life histories contend that large differences in lifetime reproductive success are erroneously interpreted as 'adaptive, resulting from and driving evolution by natural selection' [34], and that empirical studies have underestimated the contribution of stochastic processes to lifetime reproductive success and longevity. In other words, biologists might have erroneously attributed the diversity of life histories to unobserved, possibly heritable interindividual differences in fitness traits, whereas chance alone in homogeneous populations is sufficient to explain most of the life history variation in populations. Yet quantitative genetics studies of lifetime reproductive success or lifespan have provided evidence of heritability in these important parameters for evolutionary studies [38]. As in demography [11], behavioral ecology [39], and wildlife studies [40,41], quantitative genetics often relies on latent parameters to estimate heritability in life history traits [42,43], although such latent variables (i.e., genetic additive variance) are informed by the pedigree, unlike HPDH. The current controversy about the contribution of unobserved individual traits to the diversity of life histories in populations is twofold: the reality of **latent traits** is challenged, as well as the possible contribution of natural selection to life-course variation [36,37].

The relative importance of HPDH and other processes generating variation in longitudinal trajectories has stimulated a large body of work in econometrics, political and social sciences [44–49]. Studies in these disciplines have highlighted risks of flawed inference about these processes, which have been overlooked in biology. There are fundamental differences among methodologies used to address HPDH in biological studies [21–23,27,30–35,50], and failure to identify the consequences of methodological choices led to misunderstandings among biologists. In addition, despite identical questions (e.g., do latent traits matter in shaping an individual's life-course?), some methodologies used in biology clearly differ from those used in disciplines focusing on humans. We review and contrast the methods used to tease apart the contributions of different processes to interindividual variation in life histories. We draw parallels between disciplines to clarify the different views of heterogeneity, the statistical formalizations of heterogeneity, and the approaches available to address variation in longitudinal data. This paper highlights the conceptual differences among studies of heterogeneity in life histories, and unveils misunderstandings about latent traits across schools of thought. We outline that other disciplines offer useful frameworks for future work on life histories. We emphasize three areas in which other disciplines can bring new perspectives to life history studies: terminology, the diversity of processes underlying variation in life histories, and the methods of statistical inference to disentangle these processes.

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The Common Methodological Basis: Markovian Models for Series of Reproductive States and Survival Events

The standard framework in vertebrate or plant studies considers structured populations, where demographic parameters vary according to such state variables as age, developmental stage, or reproductive state (success, number of offspring, failure; Box 1). Age is a deterministic state variable: knowledge of state at time t permits full knowledge of state at time $t + 1$. Other state variables, such as reproductive or developmental state, are stochastic: the state of an individual

Box 1. Life Cycle, Longitudinal Trajectories and Associated Demographic Parameters

An individual history is a sequence of states which describe the breeding activity and survival, generally on a yearly basis for long-lived animals (Figure IA). In this example based on kittiwakes (*Rissa tridactyla*) [53], seven states are considered: PB, prebreeder; N, nonbreeder; F, breeding failure; 1 C, 1 chick fledged; 2 C, 2 chicks fledged; 3 C, 3 chicks fledged; D, dead. Following [51,52], the series of states can be modeled using a multistate Capture–Mark–Recapture model including survival probability and transition probability among states. S_t^r: survival probability of an individual in state r at time t. Ψ_t^{rs}: probability that an individual that was in state r at time t is state s at time $t + 1$, given that it survived from t to $t + 1$. If the individual survives in two consecutive years, the sequence of states in the individual's life is accounted for by a first-order Markov process, where state in year $t + 1$ depends on state in year t (Figure IB). The two broken boxes correspond to the part of individual histories that is considered as the result of stochastic processes in multistate models focusing on the segment of life starting at recruitment. Transition probability between states in year t and $t + 1$ can also depend on other observable covariates such as sex, age, year, age of first breeding, cohort, year, cumulative number of offspring produced at age a [20,34,68].

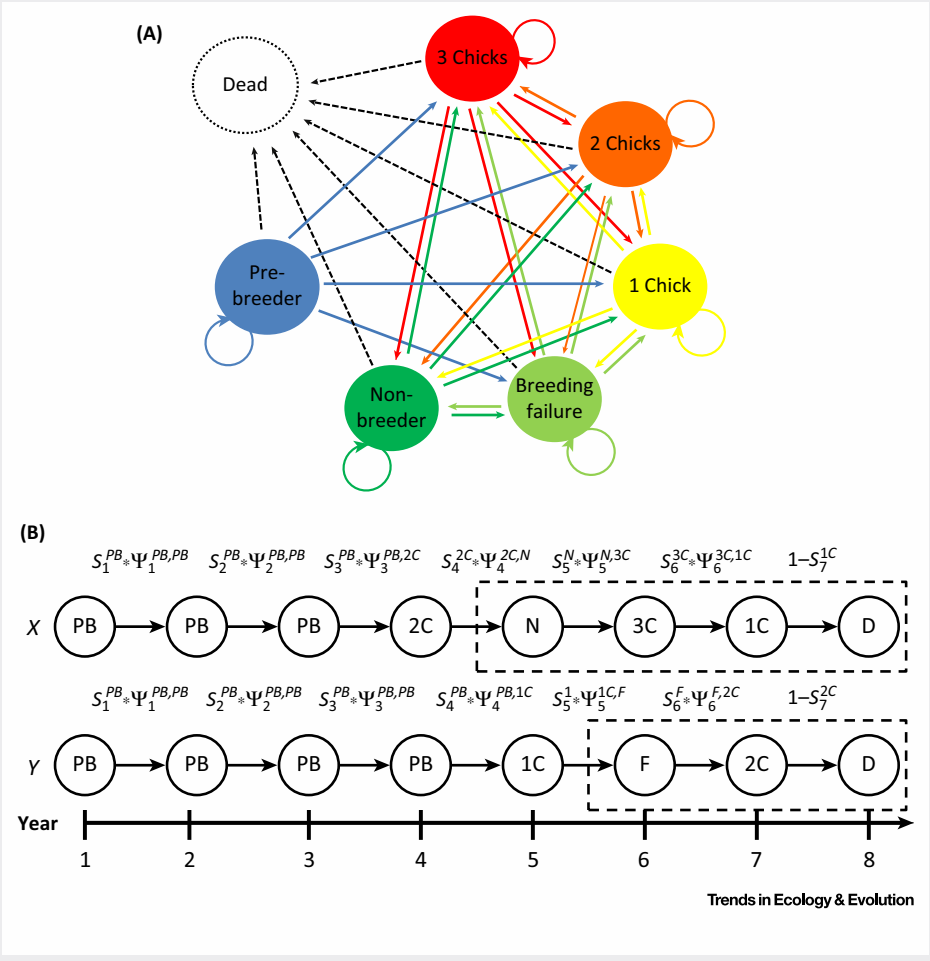

Figure I. Possible Transitions among States in Individual Trajectories. (A) Possible state transitions. (B) Two possible longitudinal trajectories of equal length and associated demographic parameters.

Glossary

Demographic heterogeneity: *sensu* [9], 'Among-individual variation in vital parameters such as birth and death rates that is unrelated to age, stage, sex, or environmental fluctuations.' Demographic heterogeneity refers to interindividual differences in demographic parameters.

Demographic stochasticity: the process behind variations in the realized fates of individuals under specified values of demographic parameters [9]. This concerns all the demographic parameters underlying longitudinal trajectories: survival, reproduction, or transition probability among reproductive states. In age-structured populations, or in populations where survival probability does not vary with age, demographic stochasticity creates a distribution of longevities.

Dynamic heterogeneity: the pattern of variation in realized life histories (e.g., sequences of reproductive states) that arises solely from the realization of Markovian binomial or multinomial processes. This variance exists in populations (or groups) composed of individuals characterized by identical demographic parameters [33].

Fixed heterogeneity or persistent demographic heterogeneity: permanent differences among individuals in survival probability or fecundity (breeding and success probability, or number of viable offspring). The interindividual differences in demographic parameters are assumed to be constant throughout life, or at least during the life stage under investigation (e.g., after recruitment) [5,9].

Frailty: in survival analysis, including all the important risk factors is usually beyond reach, sometimes because investigators do not have all the relevant information at the individual level [56]. For example, in the biomedical field, it might not be possible to measure all relevant covariates related to the disease of interest, either because the importance of some covariates is still unknown, or because collecting such data would be prohibitively expensive. In such situations, it is useful to consider heterogeneity caused by unknown covariates or factors [56]. 'Frailty [. . .] is a general concept that does not distinguish

in year t is a random variable. At age a, there is a distribution of individual states; if individuals survive until age $a + 1$, they will change state in a stochastic manner. Populations structured by age or state are heterogeneous, but here heterogeneity is measured in aggregates of individuals, not at the individual level. In longitudinal studies, where the same individual is ideally followed from birth to death, statistical models can account for the relationship between state and survival probability. The succession of states until death–that is, the **trajectory**–can be accounted for by Markovian models where reproductive output in year $t + 1$ depends on previous state(s) (Box 1).

Markovian models are useful to address whether reproduction at time t carries costs on survival probability between t and $t + 1$, or on reproduction at time $t + 1$ in survivors [51]. If so, raising numerous offspring at time t will be associated with a decrease in the probability of raising numerous offspring at time $t + 1$. This reflects a trade-off between current and future reproduction. This approach is central to empirical evaluations of the fundamental concept of trade-offs in wildlife studies [51]: trade-offs are expected to lead to specific arrangements of states in trajectories. Alternatively, successful reproduction at time t can be associated with a high probability of success at time $t + 1$ [6]. In both cases, past reproduction will influence future state: this is called 'state-dependence' in other disciplines [47,48].

Longitudinal Studies of Repeated Events: Cross-disciplinary Parallels

To understand the processes governing arrangements of states in trajectories, biologists can draw on other disciplines. When we ask: 'Does past reproduction modify the probability of future outcomes?', economists would ask: 'Does unemployment cause future unemployment?' [46]. Such questions actually have a long history in research focused on employment trajectories of humans [44–49].

Hypotheses and statistical models for longitudinal data

Two hypotheses were proposed in the 1980's to explain repeatability of human behavior in econometrics [47]:

(i) *State-dependence.* This is the process generating correlation between past and future in individuals' lives; it reflects how experiencing an event alters the probability of experiencing the event again. For example, experiencing unemployment might lead to a higher probability of experiencing unemployment again in the future, by erosion of human capital [49]. In biology, current state is influenced by past history in different ways: trade-offs translate into negative state-dependence, and the spiral of failure into positive state-dependence. Economists use Markov models to address state-dependence [44,47], as do biologists. For the latter, however, the **Markov processes** are usually 'hidden' in the sense that imperfect detection can obscure knowledge of animal states at each sampling occasion. **Multistate Capture–Mark–Recapture (CMR) models** are used to alleviate the issue [51] (Box 1). State is observable if the animal is recaptured or resighted, but multievent models can also account for unobservable states or state uncertainty [52].

(ii) *Unobserved heterogeneity.* This hypothesis attributes the persistence in observed outcomes to differences across individuals in a baseline propensity to experience the events [46–48]. Borrowing from economics models once again, persistent unemployment may result from low education or other unfavorable characteristics that can be hard to measure in practice [47]. In biology, repeatability of states might reflect differences in individual propensities to breed successfully, or latent fertility rates *sensu* [53]. This situation is consistent with the hypothesis of differences in 'individual quality' [3–5]. Generally, '**unobserved heterogeneity**' is equated to 'fixed heterogeneity' in life history studies (HPDH), though unobserved individual differences can also change during life [47,54]. The existence of hidden sources of variation in mortality or reproduction is often a legitimate concern in analyses of longitudinal data [55,56], even when

between acquired weakness, lifestyle factors, environmental risks, and innate biological frailty. It combines in a single measure all the factors that operate to increase or decrease a given individual's mortality risk, regardless of the source of heterogeneity' [10]. Individual heterogeneity is usually described by a random effect with a specified distribution [8,22,56]. HPDH can be modeled this way (Table 1).

Heterogeneity's ruses: 'As a cohort of people, animals, or machines ages, the individuals at highest risk tend to die or exit first. This differential selection can produce patterns of mortality for the population as a whole that are surprisingly different from the patterns for subpopulations or individuals. [. . .] because patterns at the individual level may be simpler than composite population patterns, both theoretical and empirical research may be unnecessarily complicated by failure to recognize the effects of heterogeneity' [14].

Hidden persistent demographic heterogeneity HPDH: the factors causing differences in survival, breeding, or success probabilities are unobservable or not observed. Unobserved heterogeneity in survival probability is also referred to as 'frailty' [10–14,56].

Individual heterogeneity: *sensu* [8], unobserved interindividual variation in demographic parameters (fitness traits). Differences in demographic parameters among individuals are assumed to be constant throughout life, or at least during the life stage under investigation (e.g., after recruitment).

Individual stochasticity: *sensu* [70], 'The movement of an individual through its life cycle is a random process, and although the eventual destination (death) is certain, the pathways taken to that destination are stochastic and will differ even between identical individuals.' Individual stochasticity is a specific category of pattern observed in individual trajectories in stage- or age-structured populations, and is intrinsically linked to demographic stochasticity. Assuming a multistate (Capture–Mark–Recapture) model [51], it is the variation in series of states created by (i) the successive realizations of stochastic transition processes governed by specified transition probabilities, and (ii) the

investigators cannot explain such variation. Heterogeneity might for example reflect genetic differences [38,57], or individual characteristics molded during development before recruitment in studies starting at recruitment [9]. For evolutionary ecologists, variation at the individual level can be relevant to natural selection if heritable [58,59]. In fact, this variation is relevant to population viability, irrespective of heritability [7–9,60,61].

In econometrics, state-dependence and unobserved heterogeneity lead to different policy recommendations [48,62–64]. In the case of state-dependence, a short-term policy helping unemployed workers move to employment will cause a persistent increase in employment [47]. Conversely, if persistence in unemployment is due to unobserved heterogeneity [49], then the policy is unlikely to succeed [47].

Importantly, HPDH and state-dependence are not mutually exclusive. It is possible to include both components in statistical models [30,32,47,65] (Box 3, Model I). Economists consider both hypotheses simultaneously [47,64,66], whereas evolutionary ecologists first considered hypotheses separately [24–26,67]. Only recently have they used Markovian models with HPDH [30–32]. HPDH is often modeled using individual random effects [22,30]. This statistical operationalization of heterogeneity is consistent with the concept of **frailty** in biomedicine and demography [10,13,55].

Where the Parallels between Methodologies End

In biology, several studies have recently turned to neutral models [33–35,68,69] to address the amount of variation in individual trajectories attributable to stochasticity in a Markovian process. The neutral theory for life histories posits that differences among individual trajectories only reflect the randomness in the realization of biological processes: reproduction and mortality. These processes are governed by demographic parameter values (survival and transition probabilities) of a Markov model with an absorbing state: death. The parameters can change throughout life, but are shared by individuals of the same age or state in year t. Even in populations where survival probability varies only by age, or does not vary at all, realizations of the survival process generate trajectories of different lengths, that is, a distribution of longevities. In stage-structured populations, the Markov process on which the neutral theory for life histories is based generates a pattern of '**dynamic heterogeneity**' in trajectories [33]. Caswell [70,71] named the resulting variation '**individual stochasticity**'.

The neutral theory for life histories posits that populations are composed of phenotypically identical individuals [33–35], a condition fulfilled by having a single transition matrix for the entire population (Box 2; Box 3, Model II). There is, however, variation among individuals in realized longevity and successions of reproductive states during life (Box 2), but this variation is evolutionarily neutral [35]. Empirical tests of the neutral theory for life histories concluded that 'nonselective' stochastic demographic processes are sufficient to explain observed distributions of longevities, lifetime reproductive success or fitness (but see [19,20]). Conversely, the HPDH hypothesis assumes that the observed fate of individuals is the realization of stochastic processes governed by parameter values of a model including unobserved and potentially unobservable variables [44]. This statistical formalization accounts for persistent individual-specific parameter values: survival, breeding, and success probabilities (Box 3, Model III). HPDH assumes that populations are phenotypically heterogeneous, that is, models rely on individual-specific transition matrices, which is a necessary condition for natural selection to occur. This allows addressing whether latent fitness traits are heritable [31].

In the neutral theory for life histories, the realizations of stochastic demographic processes provide a null hypothesis for observed life history variation [33–35,68]. To assess the baseline

variation in longevities resulting from successive realizations of stochastic survival processes governed by specified state-specific survival probabilities [70,71]. In age-structured populations, individual stochasticity manifests itself by the distribution of longevities [71].

Latent traits: conceptual constructs used when the observed outcome of interest, for example, alive at age a, is assumed to depend on unmeasurable individual traits: here, the individual survival probability [53]. This probability cannot be measured using data from the individual only: the sample size is 1 and death occurs only once. Individuals are then assumed to be characterized by an unmeasured, latent propensity to survive. In longitudinal data analysis, latent variables are mathematical constructs used to represent the effect of unobservable (or unmeasured) factors on a response, and are also used to account for the unobserved heterogeneity between subjects (HPDH). Latent traits can remain unchanged during life, or not [47,78,79]. If investigators were able to measure all the relevant individual factors and covariates influencing the outcome of interest, 'latent' variables would not be needed. In quantitative genetics, latent traits have a specific interpretation: 'The expected (additive) effect of an individual's genotype on phenotype, usually expressed relative to the population mean phenotype' [43].

Markov process: a sequence of random (state) variables indexed by time with serial dependence in the outcomes. The state of the process at $t + 1$ depends only the recent past state(s).

Multistate Capture–Mark–Recapture (CMR) models [51,52]: in their first-order Markovian version, **s**equences of states in individual histories are governed by S_t^r: survival probability of an individual in state r at time t, and Ψ_t^{rs}: the probability that an individual in state r at time t and that survives from t to $t + 1$ is in state s at time $t + 1$. Such models account for state-dependence. In studies from wild animals, an additional parameter accounts for the probability of detecting an individual in state r at time t, given that the individual is alive and present in the study area (p_t^r).

Neutral Theory for Life Histories, null and neutral models: this theory has first been developed for stage-

variation in trajectories expected with state-dependence only, neutral models *a priori* exclude the alternative process: HPDH. The transition matrix is used to assess the expected amount of variation among trajectories under fully random expectation. Individual trajectories are also simulated using parameter estimates (Box 2). The one-sided alternative hypothesis is that variation in observed trajectories is larger than that expected with state-dependence only. The predictive performance of the neutral model is evaluated by comparing actual and simulated data (e.g., lifetime reproductive success). This is the 'sufficiency view' of null models [69]. If the null hypothesis is not rejected, other processes (including HPDH) are not considered, meaning that the diversity of life histories reflects neutral variation in a homogeneous population. This approach based on neutral models to draw inferences about HPDH differs from that used in longitudinal data analysis [26,30,50,63,64,66,72,73]. In the latter, the focus lies on the discrimination among hypotheses by subjecting data to formal inference models incorporating the different processes of interest [74,75].

The question of how best to tease apart the contributions of state-dependence and HPDH to the observed diversity of trajectories in populations [19] has stimulated a large body of work in econometrics and social sciences [44–49,62–64,66,76]. This question is also central to tests of the Neutral Theory of Life Histories. The fact that these hypotheses have different implications in terms of social policy provided a strong incentive to develop robust approaches to disentangle the contributions of both processes to longitudinal trajectories in humans. Methodological development in these disciplines concerns longitudinal data analysis techniques, more specifically approaches to obtain unbiased estimates of Markovian processes and HDPH, and to test hypotheses about both processes.

Box 2. Decomposition of the Variation in Longitudinal Trajectories in Phenotypically Homogeneous Populations

The Neutral Theory for Life Histories aims at evaluating the variation in life history trajectories expected under the hypothesis that populations are phenotypically homogeneous. The phenotype is defined by a transition matrix in a first-order Markovian model. Assume that there are two reproductive states: success (1) and failure (2), plus an absorbing state: 'dead'. The probability of an individual being in state s in year $t+1$ is governed by the survival and transition matrices:

$$\begin{pmatrix} S^1 & 0 & 1-S^1 \\ 0 & S^2 & 1-S^2 \\ 0 & 0 & 1 \end{pmatrix} \times \begin{pmatrix} \Psi^{11} & \Psi^{12} & 0 \\ \Psi^{21} & \Psi^{22} & 0 \\ 0 & 0 & 1 \end{pmatrix}$$

Here survival probability (S^r) and transition probability (Ψ^{rs}) are assumed to be constant over time, but this simplifying assumption can be relaxed. S^r is the survival probability of an individual in state r. The probability that an individual is in state s in year $t+1$ exclusively depends on its state r in year t (Ψ^{rs}), and the sequence of states in the individual's life is accounted for by a first-order Markov process [51]. The total variation in lifetime reproductive success reflects the variation among trajectories due to the arrangement of states, and to the time of death (Figure IA; values used for simulations: $\Psi^{11} = 0.65$, $\Psi^{21} = 0.55$. $\Psi^{12} = 1 - \Psi^{11}$, $\Psi^{22} = 1 - \Psi^{21}$, $S^1 = 0.92$, $S^2 = 0.845$, success probability in the first reproductive event: 0.55). With real data, longitudinal data analysis techniques [44,47,65,72], and multistate capture–mark–recapture (CMR) models [51,52] can be used to address the sources of variation in both mortality and the sequence of reproductive states.

In studies of the Neutral Theory for Life Histories, the total variation in lifetime reproductive success is decomposed into two parts. First the variation among trajectories due to 'reproductive dynamics' exclusively [33]: mortality is not considered (Figure IB; trajectories were truncated at 30 years of age). The probability of the individual being in state s in year $t+1$ is governed by a transition matrix: with only two states, success and failure: $\begin{pmatrix} \Psi^{11} & \Psi^{12} \\ \Psi^{21} & \Psi^{22} \end{pmatrix}$. Second, the 'within-trajectory' variation due to mortality only, assuming that the reproductive trajectory is predefined. A realized reproductive trajectory is first simulated without considering mortality (Figure IC, upper left panel). The survival matrix is mapped *a posteriori* and creates variation due to mortality occurring at different ages within the trajectory [33,34]. The 'within-trajectory' variance *sensu* [34] is only accessible via simulations. With real data, trajectories cannot be first realized and then *a posteriori* truncated; actual trajectories are not defined after death. Longitudinal data analysis techniques or CMR models cannot be used to estimate this variance.

structured populations, where series of states (e.g., reproductive outcomes) in longitudinal trajectories are accounted for by Markovian processes (Box 1). In stage-structured populations, the neutrality assumption is that all genotypes produce a single life-history phenotype (the same transition matrix [33]; Box 2), or that there is only one life history genotype (one transition matrix) in the population. The transition matrix defines the phenotype. 'Null' or 'neutral' models are used interchangeably in the neutral theory for life histories [33–35].

Trajectory: (i) Observed trajectory. In longitudinal data analysis [72] and for analysis of data from wild animals with Capture–Mark–Recapture models [22,30,51,52], trajectories are data. Data consist of sequences of states, possibly partially observed (i.e., including missing data). In studies of wild animals, data can be missing because of incomplete detection of individually marked animals [51,52], or because of difficulties in assigning a state to an individual [80]. In studies with perfect detection of individuals, age at death is observed if trajectories are uncensored [72], and in studies with imperfect detection, age at death can sometimes be observed when dead recoveries are available [94]. (ii) The trajectory *sensu* [33] considered in the neutral theory for life histories is by nature a hypothesis which depends on the form of the neutral model specified *a priori*: a Markov process whose parameters are estimated using a multistate Capture–Mark–Recapture approach [51]. (iii) The reproductive trajectory is a possible realized sequence of reproductive states following a specified transition matrix [34]. The trajectory is defined independently of when death occurs: the sequence is truncated at an age exceeding the largest observed age at death (Box 2). A sequence of state-specific survival probabilities is *a posteriori* associated with each possible series of states, which leads to a distribution of age at death for each trajectory (Box 2). Assuming that phenotypically identical individuals follow the same transition matrix and exhibit the same realized sequences of states, the within-trajectory variance is the variance in realized

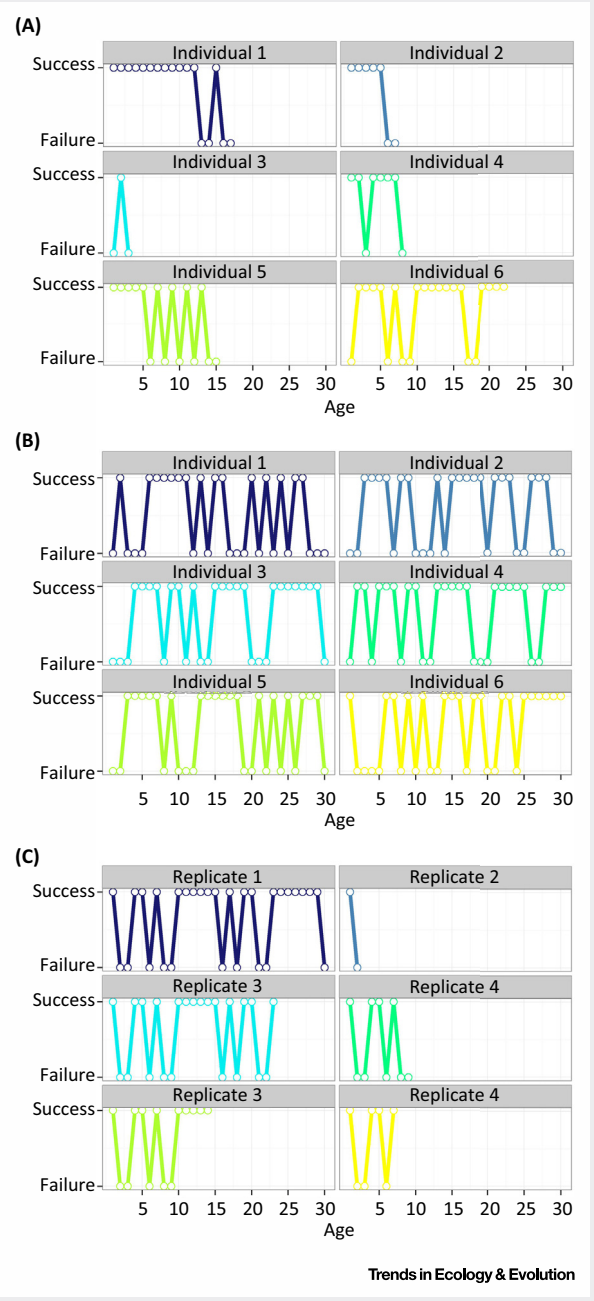

Figure I. Decomposition of the Processes Generating Variation in Longitudinal Trajectories. (A) Six realized longitudinal trajectories. (B) Six realized reproductive trajectories based on a transition matrix; mortality is not considered. (C) Within-trajectory variation in realized trajectories due to mortality occurring at different ages.

age at death along this realized sequence of states.

True state-dependence: the definition in econometrics is that '[. . .] past experience has a genuine behavioral effect [on future outcomes] in the sense that an otherwise identical individual who did not experience the event would behave differently in the future than an individual who experienced the event' [47].

Unobserved heterogeneity: in econometrics this hypothesis states that '[. . .] individuals may differ in certain unmeasured variables that influence their probability of experiencing the event but that are not influenced by the experience of the event [. . .]' [47]. 'If individual differences are stable over time, individuals who experience the event in the past are likely to experience the event in the future, even though the actual experience of the event has not modified individual behavior' [48].

True versus Spurious State-dependence: Insights from Non-ecological Fields

Economists and biologists want to evaluate the contribution of different processes to the diversity of trajectories. For this purpose, it is crucial to obtain unbiased estimates of state-dependence and HPDH. Because policy efficiency depends on the sign and the size of the state-dependence effect [76], economists were mostly concerned about overestimating state-dependence [46,47,64], that is heterogeneity giving the illusion of state-dependence. When there is

Box 3. Does the Past Influence the Future? State-dependence and Unobserved Heterogeneity in Longitudinal Data Analysis

The question of the processes generating the arrangement of 'states' (e.g., reproductive states) in individual trajectories is shared by many disciplines focusing on longitudinal data. For example 'Does infant death alter the risk of infant death of the subsequent sibling (a scarring effect)?' [45] Empirical studies in econometrics have provided evidence that the conditional probability that an individual will experience the event in the future is a function of past experience [47,48]. There are two explanations for this regularity: true state-dependence, and unobserved heterogeneity [47]. True state-dependence accounts for the modification of the probability of future outcome by past outcome (the 'scarring effect'). Another hypothesis is that individuals differ in unmeasured variables that influence their probability of experiencing the event, for example, environmental characteristics [45]. This hypothesis corresponds to hidden heterogeneity in econometrics, hidden persistent demographic heterogeneity in biology (HPDH). Both processes can be included in a single statistical model for longitudinal data.

Suppose $y_{i(t+1)} \sim \text{Bernoulli}\left(p_{i(t+1)}\right)$, where $y_{i(t+1)}$ stands for a binary outcome ($y_{i(t+1)} = 1$: successful reproduction; $y_{i(t+1)} = 0$: failure) of individual i at time $t + 1$, and $p_{i(t+1)}$, for its probability of success. The model accounting for both state-dependence and HPDH is

$$\log\left(\frac{p_{i(t+1)}}{1-p_{i(t+1)}}\right) = \beta_0 + \gamma y_{it} + \alpha_i \qquad \text{(I)}$$

where β_0 is an intercept and $\alpha_i \sim N(0, \sigma_{individual})$ is a random individual effect.

γ is the parameter quantifying true state-dependence.

If an individual i experienced the event at time t, the log-odds of experiencing the event at time t+1 is:

$$\log\left(\frac{p_{i(t+1)}}{1-p_{i(t+1)}}\right) = \beta_0 + \gamma + \alpha_i$$

The counterfactual corresponds to what would be the odds of experiencing the event at time $t + 1$ had individual i not experienced the event at time t:

$$\log\left(\frac{p^*_{i(t+1)}}{1-p^*_{i(t+1)}}\right) = \beta_0 + \alpha_i$$

Thus $\gamma = \log\left(\frac{p_{i(t+1)}}{1-p_{i(t+1)}}\right) - \log\left(\frac{p^*_{i(t+1)}}{1-p^*_{i(t+1)}}\right)$, that is γ quantifies how having experienced the event affects the probability of experiencing it again relative to not having experienced it.

State-dependence is a reduced form of (I):

$$\log\left(\frac{p_{i(t+1)}}{1-p_{i(t+1)}}\right) = \beta_0 + \gamma y_{it} \qquad \text{(II)}$$

Likewise, HPDH is a reduced form of (I):

$$\log\left(\frac{p_{i(t+1)}}{1-p_{i(t+1)}}\right) = \beta_0 + \alpha_i \qquad \text{(III)}$$

Models (II) and (III) are not simplified versions of each other.

The relationship between the state-dependence model (Model II) and the transition matrix $\begin{pmatrix} \Psi_t^{11} & \Psi_t^{12} & 0 \\ \Psi_t^{21} & \Psi_t^{22} & 0 \\ 0 & 0 & 1 \end{pmatrix}$ used in stage-structured population models is the following:

$$p_{i(t+1)} = \begin{cases} \Psi_t^{21} \text{ if } y_{it} = 0 \\ \Psi_t^{11} \text{ if } y_{it} = 1 \end{cases}$$

The model including HPDH and state-dependence relies on individual-specific matrices $\begin{pmatrix} \Psi_t^{11} & \Psi_t^{12} & 0 \\ \Psi_t^{21} & \Psi_t^{22} & 0 \\ 0 & 0 & 1 \end{pmatrix}_i$. This model assumes a distribution of individual transition matrices, which is accounted for by random individual effects (α_i) in Model (I).

Similarly, for longevity, under the HPDH hypothesis, individual-specific survival matrices are used $\begin{pmatrix} S_t^1 & 0 & 1-S_t^1 \\ 0 & S_t^2 & 1-S_t^2 \\ 0 & 0 & 1 \end{pmatrix}_i$.

true state-dependence (for example, a scarring effect whereby a spell of unemployment leads to more unemployment irrespective of an individual's working skills), large-scale interventions are possible to favor movement to employment and promote persistent employment. However, if this state-dependence is spurious and due to heterogeneity, such interventions will miss the target. Two seminal papers in econometrics have addressed the consequences of ignoring HPDH [46,48]. In heterogeneous populations, improper treatment of HPDH creates a conditional relationship between future and past events, that is 'spurious' state-dependence [47,48]. If the true data-generating process includes HPDH, Markovian models based on observable or partially observable states will yield upwardly biased estimates of transition probabilities and true state-dependence (Box 4). In such a situation, biologists would overestimate support for the neutral theory for life histories. Conversely, state-dependence is properly estimated when HPDH is also taken into account (Box 4).

The communicating vessels phenomenon (Box 4), whereby two processes can replace each other to account for state persistence across time [76] can explain contradictory findings in the same data [24,34]. Ignoring HPDH results in inflated estimates of state-dependence [50,76], and the latter decrease when HPDH is taken into account [50], which is consistent with economists' results [76]. Moreover, to address whether consideration of HPDH changes conclusions concerning the neutral theory for life histories [34], investigators are tempted to try to mimic HPDH in *post-hoc* simulations by adding an arbitrary level of interindividual variation to transition probabilities estimated with Markovian models. The implicit assumption is that the estimated state-dependence effect remains unchanged regardless of whether it is estimated using a model including HPDH, or not. However, this assumption does not hold [76] because of the communicating vessels phenomenon.

Teasing Apart the Processes Underlying Variation in Individual Life Histories: Where to Next?

Identifying and Developing Methods for Robust Inference

To address the contribution of stochastic processes to variation in lifetime fitness, the neutral theory for life histories specifies a single hypothesis, that of state-dependence. In the context of longitudinal data analysis, this approach can lead to spurious support for neutrality. The

Box 4. Communicating Vessels: Inference about True State-dependence and Hidden Heterogeneity

A major result from econometrics is how state-dependence and hidden heterogeneity (HPDH) can bias estimation in statistical models of longitudinal data [47,76]. To illustrate this phenomenon, suppose that only state-dependence or hidden heterogeneity is operating (Box 3). A very simple binomial model with only state-dependence is:

$$\log\left(\frac{p_{i(t+1)}}{1-p_{i(t+1)}}\right) = \beta_0 + \gamma y_{it} \quad (\text{state} - \text{dependence})$$

A very simple binomial model with only HPDH is:

$$\log\left(\frac{p_{i(t+1)}}{1-p_{i(t+1)}}\right) = \beta_0 + \alpha_i \quad (\text{HPDH})$$

(i) Suppose HPDH is the true data-generating process, but life histories are analyzed with a model with state-dependence and ignoring HPDH. Spurious state-dependence is detected: the estimate is biased upward depending on the true amount of HPDH (Figure IA).

(ii) Suppose state-dependence is the true data-generating process, but life histories are analyzed with a model with HPDH and ignoring state-dependence. Spurious HPDH is detected: the estimate is biased upward depending on the true amount of state-dependence (Figure IB).

There is a communicating vessel phenomenon between state-dependence and HPDH in statistical models used to analyze life-history trajectories. Ignoring one process may lead to overestimating the other [44,46,47]. Both state-dependence and HPDH should be considered in tandem to obtain unbiased parameter estimates and accurate inferences [44–49,62–66,76]. If either HPDH or state-dependence is a priori chosen as a null model, this null model will not be rejected because of the positive bias in parameter estimates.

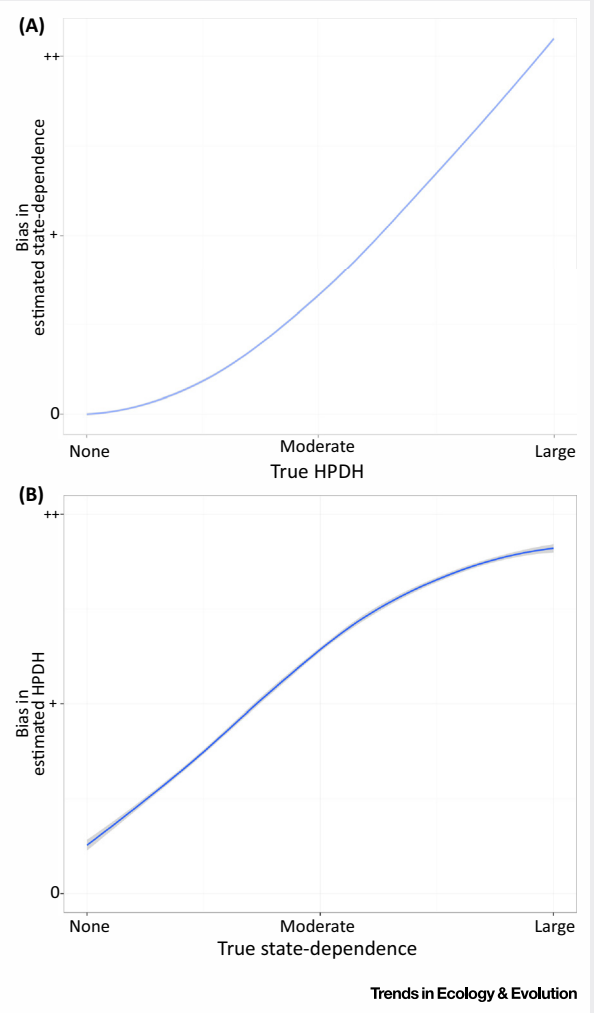

Figure I. Bias in Estimates of State-dependence or Hidden Heterogeneity According to the Model Used to Analyze Data. (A) HPDH used as a data-generating process but data analyzed with a statistical model that only includes state-dependence. (B) State-dependence used as a data-generating process but data analyzed with a statistical model that only includes HPDH.

estimated parameters used to specify the phenotype (transition matrix) in the neutral theory for life histories entirely depend on the statistical model chosen for estimation. To identify the relevant sources of variation in parameters, a natural approach is to define a set of candidate models and to identify the models that do a better job at accounting for the data than others. This requires evaluating the strength of evidence for non-exclusive hypotheses (state-dependence, HPDH) with, for example, information criteria [74]. The contribution of state-dependence to variation in observed trajectories can only be addressed with estimates unbiased by HPDH [44,47]. Accurate parameter estimation might not be possible outside a multimodel inference framework.

HPDH and state-dependence account for processes operating at the level of demographic parameters: yearly survival and transition probabilities between breeding states. Longitudinal analysis techniques focus on this level. Conversely, simulation-based tests of the neutral

theory for life histories use patterns in entire trajectories for inference, that is, the end product of successive realizations of survival and breeding processes. When the interest lies in entire trajectories, a renewed emphasis on partitioning the variance in longevity into individual stochasticity and HPDH [70,71] components opens perspectives for accurate tests of the neutral theory for life histories. This approach is based on estimation of the moments of random variables relevant to entire trajectories in stage-structured populations: longevity, and the number of visits to nonabsorbing states [70]. Here the first step is accurate estimation of model parameters, the approach favored by economists. With this method, it is possible to include the state-dependence model in a set of candidate models accounting for different combinations of processes and to estimate the strength of evidence for these models.

Clarifying Terminology

Classically 'fixed' heterogeneity evokes fixed differences among individuals due to genetic background [38], maternal effects or ontogeny [9], and is viewed as unobservable [21–23,33–35]. 'Dynamic' heterogeneity refers to partially observable discrete states exclusively, and is accounted for by Markovian models [33–35]. This creates confusion because (i) fixed heterogeneity can be accounted for by observable traits (demographic parameters associated with observable individual covariates) [77], (ii) models considering dynamic latent traits exist (dynamic frailty) [78,79], (iii) and Markov processes can entail unobservable states [47,80–82]. It is noteworthy that economists also consider that unobserved components can be modeled using a Markov process, or time-varying effects [64], and that initial differences in unmeasured variables can be eliminated with the passage of time [47]. We believe that the development of a wide range of models for longitudinal data has rendered the usual 'fixed versus dynamic heterogeneity' framework too narrow and imprecise. A more precise terminology would endorse the statistical formalization of differences among individuals in demographic parameters; this would help clarify what 'heterogeneity' covers in different studies and promote cross-pollination between disciplines. A taxonomy of models is reported in Table 1; note that different features can be combined to create more complex models (e.g., state-dependence plus HPDH). The choice of the statistical models to use depends on answers to the following questions:

(i) Are parameter values individual-specific? This situation corresponds to individual effects taking real values from continuous distributions (individual covariates, random effects). Parameters are individual-specific if they cannot be estimated using data from individual B to completely characterize individual A, even if A and B have the same age, are in the same state in the same year for example.

(ii) Is the hierarchy among individuals in survival or fecundity maintained with the passage of time? Individuals can differ in baseline fecundity rate for example, which corresponds to a random intercept in random effects models. Individual-specific fecundity can also change during life, which is modeled using an individual slope for the effect of age on reproduction. In both cases intercepts are constant, as well as slopes in the latter situation. However, with random intercept and slope models, individuals with the largest fecundity when they entered the sample might not necessarily be those with the largest fecundity as time passes [26,39], because the curves describing variation of fecundity with age in two individuals can intersect. Permanent heterogeneity corresponds to a situation where the hierarchy among individuals does not change during the study.

(iii) Do we assume that interindividual variation in demographic parameters can be accounted for by observable variables only (body condition, physical or physiological characteristics, etc.), do we consider a 'mathematical construct' to account for hidden heterogeneity [83], or do we consider both sources of variation in tandem?

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Table 1. Some Modeling Options to Account for Variation among Individuals in Longitudinal Trajectories

Distribution of demographic parameters: aggregates (discrete), or individual-specific (continuous)?	Demographic parameter		Model feature accounting for heterogeneity among individuals	Statistical model	Consequence: hierarchy among individuals in demographic parameters during life	Modeling option	Examples
	Time – or age-invariant during the individual's life?	Directly observable or measurable?					
Continuous	Yes	Yes	Fixed effect models	Fixed effect models with time-invariant individual covariates	Fixed	Continuous distribution for observed individual covariate	[77]
Continuous	Yes	No	Random effect models	Random intercept models	Fixed	Continuous distribution for latent individual effects	[22,24]
Continuous	No	Yes	Fixed effect models	Fixed effect models with time-varying individual covariates	Varying	Continuous distribution for observed individual covariate	[98]
Continuous	No	No	Random effect models	Random intercept- and slope models; piecewise random intercept models,[a] time-varying frailty	Varying	Continuous distribution for latent individual effects	[26,30,44,78,79,99,100]
Discrete	Yes	Yes	Fixed effect models	Fixed effect models with time-invariant group variables	Fixed	Finite number of categories for group-specific effects	[77]
Discrete	Yes	No	Mixture models	Latent mixture models	Fixed	Finite number of categories for latent individual effects	[95]
Discrete	No	Yes	Fixed effect models	Markov models (multistate CMR[b] models)	Varying	Finite number of categories for state-specific effects	[34,67,68,90]
Discrete	No	No	Latent growth mixture models	Latent growth mixture models	Varying	Finite number of categories for latent individual effects	[92]
Discrete	No	No	Fixed effect models	Hidden Markov models (multievent CMR models, semi-Markov models, latent Markov models)	Varying	Finite number of categories for state-specific effects	[80–82,95]

[a]Individual trajectories are split into segments and a random effect is used to model observations from each segment; a multivariate distribution can be used for random effects.
[b]Abbreviation: CMR, capture-mark-recapture.

Concluding Remarks

The hypothesis of a hidden distribution of 'individual propensities to experience an event' has received support in other disciplines, but particular difficulties inherent to imperfect detection of individually marked wild animals [22,30,40,41,51,52,80,81,84] have slowed down the convergence towards the approaches used in these areas [45,63,76,85,86]. Thanks to methodological development handling detectability issues, the HPDH hypothesis can be addressed in an enlarged range of taxa [22,23], and the debate about the neutrality of life history variation has been rekindled by recent evidence of heritability of latent traits [31]. This echoes empirical studies from humans documenting heritability of frailty [57,87] and calls for investigations in wild animals [88]. Latent traits are at the heart of a contradiction: (i) in populations with hidden heterogeneity, the genuine pattern of variation in fitness traits with age within individuals cannot be assessed unless unobserved heterogeneity taken into account [16,17,25,27–29,89]. (ii) Nevertheless, in many taxa the observed distribution of lifetime reproductive success can be accounted for by models ignoring HPDH and describing age-related variation in fitness above the individual level, in aggregates [33–35]. This raises the question of whether different data-generating processes can lead to identical distributions of Lifetime Reproductive Success [19]. Moreover, ignoring hidden heterogeneity might have led to biased estimates of state-dependence in previous investigations [76]. This is a cause of concern not only in studies of the neutral theory for life histories, but also in studies of trade-offs among fitness traits using state-dependence models [67,90].

We defend the idea that evaluating the contribution of unobserved heterogeneity and state-dependence to variation in lifetime fitness requires treating these processes as any other hypotheses in a multi-model inference framework [30,32,50,74,75,84]. The debate about the causes of variation in individual life history trajectories will gain in relevance if we consider explicitly the full range of processes and corresponding models available from other disciplines, with the caveat that we need to deal with sampling issues in animal ecology (detection probabilities) that other disciplines can sometimes ignore. Inference approaches should consider *a priori* that observed trajectories can reflect any intermediate position between two extremes: a distribution of individual latent demographic parameters [55], and a situation where aggregates of individuals share the same parameter values, and where variation in observed life histories arise solely from stochastic processes governed by these parameters [35]. We also expect, as in other disciplines, that not all the variables associated with individual variation in demographic parameters can be measured [10,55,56,65,78,79,83]. Interdisciplinary work on unobserved heterogeneity is needed to avoid statistical pitfalls due to model mis-specification in longitudinal data analysis [85,86,91–93].

Accounting for hidden heterogeneity in models is a precaution to avoid flawed statistical inferences [11,76], but latent traits also have direct interpretations in evolutionary ecology [38,42,43,53]. Studies evaluating the extent of hidden heterogeneity, covariation among latent traits [50], clusters in latent traits associated with relatedness among individuals [57,87] or common environment, and the ability of observable variables to account for hidden heterogeneity are needed to assess the significance of latent traits (see Outstanding Questions). The question of the nature, origin and maintenance of such traits remains open [31].

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Outstanding Questions in elucidating the respective roles that different sources of heterogeneity play in the evolution of life histories

How does the imperfect detectability of individually marked animals [40,51,52,94,95] affect our ability to quantify unobserved heterogeneity in fitness traits [22,23]? Knowledge on latent demographic traits in wildlife ecology is lacking, except in unusual situations with high detectability.

How is unobserved heterogeneity distributed in the tree of life? Is there a relationship between longevity and the degree of heterogeneity? In longitudinal studies of plants, when all risk factors affecting mortality cannot be measured, models accounting for unobserved heterogeneity are underrepresented [18].

Can initial differences in unmeasured variables among individuals disappear over time [47,48], or do they persist? Models accounting for the former situation would be consistent with the 'ontogenetic perspective on individual differences' [54], where individual differences can be generated during adulthood, and be reversible.

Do latent fitness traits covary at the individual level? Is the hierarchy among individuals for one fitness trait maintained for the other considered components as well? Answering these questions is tightly linked to technical issues about random effect specifications. Just like trade-offs among fitness traits can be addressed at the level of (partially) observable traits and states [51], the correlation between latent traits [24,30,50,53] can also be addressed if one assumes a continuous distribution for random effects. This correlation can help understand the pattern of covariation detected at the population level [11,14].

How can we best estimate hidden permanent demographic heterogeneity? With a continuous distribution of latent values in the population, or with clusters or groups [92,95]? This choice might also depend on the study context and objectives.

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How do we identify clusters of individuals sharing values of latent traits? Mixture models might be useful for these investigations [92,95]. Models where the number of clusters is estimated from data open new avenues in this area [96].

Is frailty heritable? This question has received interest in human demography [57,87], in wild animal populations with perfect detectability of individuals, but seldom in the typical situation where time of death cannot be observed [88], which leaves the majority of taxa aside. The degree of similarity in frailty might be a decreasing function of the degree of relatedness, which can be addressed using continuous distributions for random effects [38,42,43,88]. This is of particular interest to quantitative geneticists: assuming that natural selection has been acting on most taxa for a long time, is there still heritable variation remaining in any trait closely related to fitness [38,42,43,58]? If so, how is this variation maintained?

Has state-dependence been overestimated in analyses of individual longitudinal data because of improper treatment of unobserved heterogeneity [47]? Alternatively, has unobserved heterogeneity been overestimated because relevant observable factors have been overlooked, or because of random effect misspecification [97]? These non-mutually exclusive processes generating patterns in arrangements of reproductive outcomes in longitudinal data have different interpretations. Either individuals differ in their unobserved propensity to breed successfully or current reproduction depends on the outcome of past reproduction (state-dependence). Ultimately, these processes determine overall fitness.

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