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SYNTHESIS

Demystifying individual heterogeneity

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Abstract

Among-individual variation in vital rates, such as mortality and birth rates, exists in nearly all populations. Recent studies suggest that this individual heterogeneity produces substantial life-history and fitness differences among individuals, which in turn scale up to influence population dynamics. However, our ability to understand the consequences of individual heterogeneity is limited by inconsistencies across conceptual frameworks in the field. Studies of individual heterogeneity remain filled with contradicting and ambiguous terminology that introduces risks of misunderstandings, conflicting models and unreliable conclusions. Here, we synthesise the existing literature into a single and comparatively straightforward framework with explicit terminology and definitions. This work introduces a distinction between potential vital rates and realised vital rates to develop a coherent framework that maps directly onto mathematical models of individual heterogeneity. We suggest the terms "fixed condition" and "dynamic condition" be used to distinguish potential vital rates that are permanent from those that can change throughout an individual's life. To illustrate, we connect the framework to quantitative genetics models and to common classes of statistical models used to infer individual heterogeneity. We also develop a population projection matrix model that provides an example of how our definitions are translated into precise quantitative terms.

KEYWORDS
dynamic condition, fixed condition, individual heterogeneity, individual stochasticity, life-history theory, population dynamics, vital rates

INTRODUCTION

Individual vital rates, such as mortality rates and birth rates, determine how individuals survive, develop and reproduce throughout their life. Historically, population models built around life tables assumed that all individuals within a given age or stage class have equal vital rates (e.g. Lefkovitch, 1965; Leslie, 1945; Lewis, 1942). For example, Leslie and Lefkovitch matrix models are designed to characterise the rich details of age- or stage-specific changes in life history, but they assume that all individuals within a given age or stage class are identical. In practice, these models generate theoretical predictions in which all individuals have equal expected fitness because they follow the same life-history schedule. While the assumption of identical vital rates greatly simplifies the construction of population models and analyses of population dynamics (Barfield et al., 2011; Barks & Laird, 2020; Gomes, 2019; Vindenes et al., 2012), accumulating empirical evidence reveals that among-individual variation in vital rates exists in nearly all populations, even when the individual members are

genetically identical (Cressler et al., 2017; Jouvet et al., 2018). This individual heterogeneity[1] in vital rates can be measured directly (e.g. Cressler et al., 2017; Dahlgren et al., 2016; Jouvet et al., 2018); however, it is typically characterised by measuring individual heterogeneity in other phenotypic traits that are thought to be important causal drivers of differences in vital rates (Coulson et al., 2006a; Ellner et al., 2011; Lande, 1982). Individual heterogeneity can result in major variation among individual life histories that scales up to have substantial ecological and evolutionary consequences for populations, including impacts on population growth rates (Cressler et al., 2017; Doak et al., 2005; Guillemain et al., 2014; Kendall et al., 2011; Plard et al., 2016; Russell et al., 2011; Stover et al., 2012; Zuidema et al., 2009), rates of evolution by natural selection (Barks & Laird, 2020; Bolnick et al., 2011; Cressler et al., 2017; Gillespie et al., 2013; McGuigan & Blows, 2009; Saito et al., 2013) and population persistence (Acker et al., 2014; Coulson et al., 2005; Demetrius et al., 2004; Krieger et al., 2020; Singh et al., 2004; Vindenes & Langangen, 2015; Vindenes et al., 2012).

Despite the extensive list of studies that repeatedly find individual heterogeneity across a wide variety of species and environments (see Table S1 in Supporting Information for a review), researchers have yet to agree upon a unified framework for the field. Studies of heterogeneity in populations, therefore, remain filled with contradicting and ambiguous terminology that often leaves their contributions inaccessible to researchers outside the field. As a result, multiple terms have been used in the literature to refer to equivalent concepts, leading to a web of terminology including "fixed heterogeneity" (Chambert et al., 2013; Jouvet et al., 2018; Tuljapurkar et al., 2009), "permanent heterogeneity" (Cam et al., 2016; Vindenes et al., 2012), "persistent heterogeneity" (Brooks et al., 2017; Kendall et al., 2011), "constant heterogeneity" (Brooks et al., 2017) and "consistent heterogeneity" (Fay et al., 2018; Vindenes et al., 2008), just to name a few. Moreover, many studies of individual heterogeneity do not define relevant terms transparently, and some studies do not define them at all. The emerging problem, therefore, is that the consequences of individual heterogeneity cannot be fully understood when some researchers are studying the same biological phenomena using different terminology, while others are studying different biological phenomena using the same terminology. These fundamental differences between definitions introduce the risk of misunderstandings, inconsistent modelling and unreliable conclusions that will persist until a universal framework of heterogeneity is accepted.

Previous reviews have recognised and tried to clarify the ambiguities that surround defining heterogeneity in populations (Authier et al., 2017; Bergeron et al., 2011; Cam et al., 2016; Gimenez et al., 2018; Kendall & Fox, 2003; Wilson & Nussey, 2010), yet the list of new terminology and interpretations continues to grow. A recent example of a study that attempts to resolve these ambiguities is from van Daalen and Caswell (2020), who argue that individual heterogeneity can be defined using differences in demographic rates, and that this is distinct from the chance events that result in individual stochasticity. In this study, we build on their work and the work of others to examine existing definitions, observations and theoretical advances in the literature on individual heterogeneity. We establish a distinction between potential and realised vital rates that allows us to synthesise previous works into a single, and comparatively straightforward framework with explicit terminology and definitions. This integrative framework is meant to reduce the confusion attached to current terminology and to clearly delineate a distinction between individual heterogeneity and individual stochasticity. In addition to clarifying the range of possible influences of heterogeneity at the individual and population levels, this synthesis aims to improve consistency between theoretical models (e.g. Boyce, 1977; Deere et al., 2017; Noonburg et al., 2015; Plard et al., 2016; Vindenes et al., 2008) and empirical studies (e.g., Dierickx et al., 2019; Ducros et al., 2020; Letcher et al., 2011; McLean et al., 2019; Vedder & Bouwhuis, 2018). To reduce ambiguity further, we also introduce a simple population projection matrix model that illustrates the definitions of heterogeneity in precise quantitative terms. This example helps to connect the framework to quantitative genetics models and to the major classes of statistical models used to infer individual heterogeneity from data.

DEFINING HETEROGENEITY

Individual vital rates

The framework (Box 1; Figure 1) begins at the level of individual vital rates (e.g. mortality, birth and development rates). First, suppose that an individual's lifetime can be broken into n time steps. We consider an individual's vital rate to be drawn from a distribution at each time step, such that a sequence of n draws represents the realisation of one potential lifetime for that individual. At each time step, an individual's *potential vital rate* is defined by a distribution of possible values of the vital rate, and this might be influenced by extrinsic and intrinsic factors such as the individual's habitat, its genotype or its age. An individual's *realised vital rate* is defined as the vital rate that it actually experiences over a given time

[1]Throughout this paper, the term "individual heterogeneity" will refer to individual heterogeneity in vital rates. This approach follows previous definitions in the field (e.g. Vindenes et al., 2008; Bonnet & Postma 2016; Cressler et al., 2017; Jenouvrier et al., 2019), but contrasts with broader definitions that allow individual heterogeneity to refer to any among-individual variation (e.g. Hamel et al., 2018; Jolles et al., 2020). We use a narrower definition of individual heterogeneity to help eliminate any ambiguities from the framework presented here.

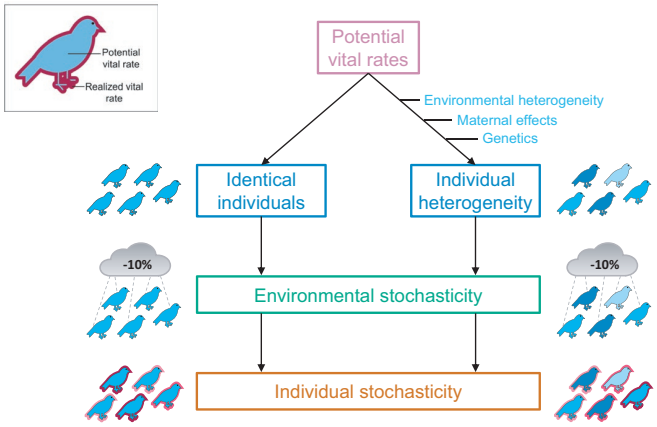

FIGURE 1 A conceptual framework for individual heterogeneity, shown for a single snapshot in time. Individuals are characterised by both a potential vital rate and a realised vital rate. At each time step, an individual's potential vital rate is described by a distribution of possible values. An individual's realised vital rate is the vital rate that it actually experiences over a given time step, which can be viewed as a particular draw from its potential vital rate distribution. Individuals with identical potential vital rates (identical individuals) are distinguished from individuals that differ in their potential vital rates (individual heterogeneity). Individual heterogeneity may arise from any process that causes potential vital rates to differ among individuals, such as environmental heterogeneity or maternal effects. Groups of identical individuals and groups with individual heterogeneity are both subject to environmental stochasticity and individual stochasticity. Environmental stochasticity refers to random variation in environmental conditions that similarly affects the *potential* vital rates of all individuals in a given condition. Individual stochasticity refers to the random chance events (e.g. living or dying, breeding or failing to breed, recruiting or failing to recruit) that change an individual's *realised* vital rates

step, which can be viewed as a particular draw from its potential vital rate distribution. Thus, two individuals with identical potential vital rates will typically have different realised vital rates simply due to chance events. If an individual were able to relive its life, its sequence of potential vital rates would be the same each time, while its sequence of realised vital rates might differ.

Individual heterogeneity

In the simplest case, we say that a population consists of identical individuals if all individuals have the same potential vital rates (Box 1; Figure 1, left side). However, the assumption of identical individuals is unrealistic as real organisms differ (often substantially) in their potential vital rates. Individual heterogeneity is defined as among-individual variation in potential vital rates (Box 1; Figure 1, right side). Thus, individual heterogeneity occurs when the distribution of possible vital rates at a given time differs between individuals within a population. One way to think about this is to suppose that individuals differ in some underlying quality that we refer to as condition. The term "condition" is deeply entrenched in the evolutionary ecology literature (Hill, 2011; McNamara & Houston, 1996; Rowe & Houle, 1996; Wilson & Nussey, 2010), and here we use this term (*sensu* Ronget et al., 2017) to represent a measure of overall individual quality. High condition individuals are expected to perform better than low condition individuals because their higher quality means that they will have a higher value of at least one potential vital rate. For example, an individual with high condition might have a high birth rate and be expected to produce more offspring than an individual with low condition. If present, trade-offs between potential vital rates are expected to reduce among-individual differences in condition (Bruijning et al., 2019; Dahlgren et al., 2016; Gould et al., 2018; Lemaître et al., 2015), as the benefits that individuals gain from a high value of one vital rate will be counteracted by a low value of another vital rate. Trade-offs are not ubiquitous; however, many studies find a positive correlation between vital rates that structures the population into groups of high- and low-quality individuals (e.g. Fay et al., 2018; Moyes et al., 2011; Olijnyk & Nelson, 2013; Vedder & Bouwhuis, 2018).

Among-individual differences in potential vital rates generate a hierarchy in which individuals can be ranked by their condition (Badger et al., 2020; Cam et al., 2016; Cam et al., 2013; Chambert et al., 2013). An individual's position in this hierarchy provides information on its own relative fitness, and on how it may influence population dynamics. Individuals with higher vital rates may experience fewer trade-offs (Hamel et al., 2009a; Moyes et al., 2011; Oosthuizen et al., 2019), enjoy superior social ranks and competitive success (Hamel et al., 2009b; Hart et al., 2016; Lemaître et al., 2018; Lloyd et al., 2020) and contribute more to population growth than lower condition individuals (Coulson et al., 2006b; van de Pol et al., 2006; Zuidema et al., 2009; Jansen et al., 2012). However, these benefits only last as long as an individual's position in the condition hierarchy is maintained.

When individual heterogeneity exists in a population, the persistence of individual condition (and thus the distribution of potential vital rates) throughout an individual's lifetime can have a large influence on the ecological

Box 1 A framework for individual heterogeneity

Potential vital rate

The distribution that defines the set of possible vital rates for an individual at a given time. These vital rates are typically expressed as the probability of experiencing an event per unit time (Kohyama et al., 2018). Examples include potential mortality rates, birth rates and development rates.

Realised vital rate

The vital rate that is actually expressed by an individual. An individual's realised vital rate is determined as a draw from its potential vital rate distribution. In the absence of individual stochasticity, an individual's realised vital rate is equal to its expected potential vital rate.

Identical individuals

Individuals with equal potential vital rates. Individual heterogeneity will never be present among identical individuals; however, individual stochasticity will be present if the individuals vary in their realised vital rates (Authier et al., 2017; van Daalen & Caswell, 2020).

Individual heterogeneity

Among-individual variation in potential vital rates. This individual heterogeneity occurs when the distribution of possible vital rates at a given time differs between individuals. Processes that generate individual heterogeneity could include environmental heterogeneity (Ducros et al., 2020; Fay et al., 2018), maternal effects (McLean et al., 2019; Ofstad et al., 2020) or genetics (Jouvet et al., 2018; Nepoux et al., 2015), although this list is not exhaustive. The resulting individual heterogeneity forms a hierarchy in which individuals can be ranked by their condition (Chambert et al., 2013).
Individual heterogeneity can produce differences in the expected fitness of individuals within a population because individual vital rates directly impact individual fitness. The amount of variation among individual fitness values that is produced will depend on (i) the strength of individual heterogeneity and (ii) correlations among vital rates. While positive associations between vital rates are expected to enhance fitness differences, negative associations may equalise fitness among individuals.

Environmental heterogeneity

Environmental differences (biotic and abiotic, temporal and spatial) between two or more locations (Heino et al., 2015; Stein et al., 2014). Environmental heterogeneity may act as a source of individual heterogeneity because it contributes to determining individual ranks in the condition hierarchy. For example, if an individual develops to an adult in a location with plentiful resources, it may enjoy higher potential vital rates as an adult than a similar individual that develops in a location with fewer resources.

Fixed condition

Individual heterogeneity in which potential vital rates are fixed throughout an individual's life. An individual's relative rank in the condition hierarchy may only change as new individuals are recruited into the population through births or immigration, or as current individuals leave the population through deaths or emigration.

Dynamic condition

Individual heterogeneity in which potential vital rates, and therefore individual ranks in the condition hierarchy, may change at any time throughout an individual's life.

Environmental stochasticity

Random variation in the environment that similarly affects the potential vital rates of all individuals in a given condition (Acker et al., 2014; Engen et al., 2007; Sæther & Engen, 2015). Unlike environmental heterogeneity, environmental stochasticity cannot act as a source of individual heterogeneity because it modifies

the potential vital rates of all individuals in the same way, such that it does not impact the condition hierarchy. Environmental stochasticity contrasts with individual stochasticity because it influences large populations equally as strongly as small populations (Melbourne & Hastings, 2008), and because it changes potential vital rates while individual stochasticity results from variation in realised vital rates.

Individual stochasticity

Random variation in the realised vital rates of individuals (Caswell, 2009). Even in populations where all individuals experience the exact same values of potential vital rates, there will be random variation in the events realised by each individual. The effects of individual stochasticity are stronger in smaller populations than in larger populations where many of the random differences in realised vital rates cancel each other out (Vindenes et al., 2012). Individual ranks in the condition hierarchy are not impacted by individual stochasticity, since individual stochasticity arises from variation in realised vital rates.

and evolutionary dynamics of the population (Coste & Pavard, 2020; Jenouvrier et al., 2018; Plard et al., 2012; Snyder & Ellner, 2018; Steiner & Tuljapurkar, 2012; Vindenes & Langangen, 2015). The degree of persistence of these vital rates is typically classified as fixed or dynamic. Fixed and dynamic condition lie at opposite ends of a continuous spectrum, and real organisms generally fall somewhere between these two extremes (Coulson et al., 2006b; Orzack et al., 2011; Plard et al., 2012; Plard et al., 2018). This classification scheme separates types of individual heterogeneity according to the pattern of potential vital rates throughout the lifetime of individuals, without the need to directly account for the biological mechanisms that generate or maintain individual heterogeneity in populations (Figure 2). Empirical research shows that there are countless processes underlying both fixed and dynamic condition (e.g. Bishop et al., 2019; Bowen et al., 2015; Gould et al., 2018; Paoli et al., 2020), although some of these processes contribute more often to one type of individual heterogeity than the other. For example, among-individual variation in genotypes will generally produce fixed condition in vital rates; however, mutations in the genome may lead to dynamic condition. Despite the fact that some processes tend to align more with fixed versus dynamic condition, there is no clear one-to-one mapping between biological processes and the type of condition or the population-level outcome. Accordingly, we believe it is better to form a conceptual understanding of individual heterogeneity and its structure in populations using the theoretical extremes of fixed and dynamic condition, rather than using the underlying biological mechansims.

Fixed condition refers to individual heterogeneity in which potential vital rates are set at birth and persist throughout life (Box 1). Thus, fixed condition describes individual life histories where the distribution of possible vital rates is permanent over an individual's lifetime. An individual's relative rank in the condition hierarchy may only change as new individuals are recruited into the population through births or immigration, or as current

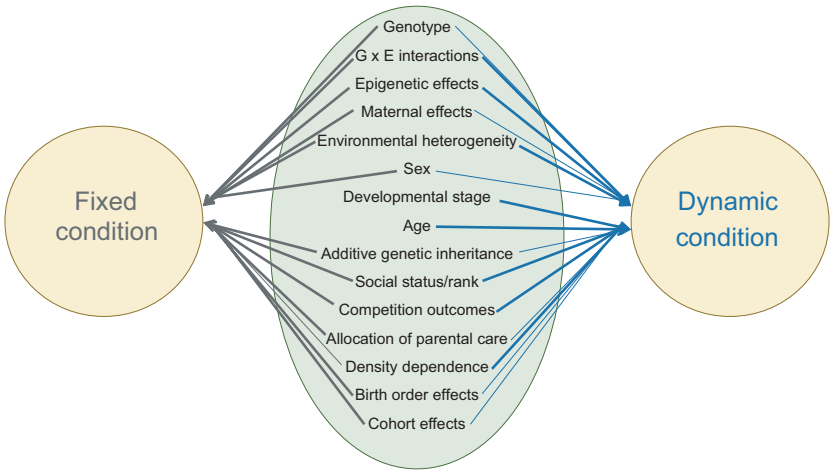

FIGURE 2 Many biological mechanisms generate and maintain both fixed (grey) and dynamic (blue) condition. These processes often contribute more to one type of individual heterogeneity (thick lines) than the other (thin lines). Taking a theoretical perspective allows individual heterogeneity to be separated into just two types, whereas classifying based on the biological mechanisms would require an inordinate number of heterogeneity categories

individuals leave the population through deaths or emigration. Fixed condition has been found across a wide range of taxa, including insects, birds and fish (Table S1). It is expected to be especially common in plants and other sessile organisms, where spatial environmental heterogeneity drives differences among individual vital rates (Dahlgren et al., 2016; Hesse et al., 2008; Kendall et al., 2011). These potential vital rates may be observable or unobservable, and the framework that we present here makes no assumptions regarding the heritability of vital rates (Figure 3a, b).

At the other extreme, dynamic condition refers to individual heterogeneity in which potential vital rates may change at any point throughout an individual's life (Box 1). Dynamic condition, therefore, describes individual life histories where the distribution of possible vital rates can shift over an individual's lifetime. This type of individual heterogeneity has been detected in taxa ranging from seabirds to ungulates, and bacteria (Table S1). For example, capture–mark–recapture studies show that many bird species switch between high, medium and low reproductive stages throughout their life (Jenouvrier et al., 2018; Orzack et al., 2011; Steiner et al., 2010). As with fixed condition, the potential vital rates generated by dynamic condition may be observable or unobservable, and there are no assumptions regarding their heritability. Moreover, dynamic condition includes situations where an individual's potential vital rates depend on its previous vital rates (Figure 3c) and situations where potential vital rates at one time are independent of any previous values (Figure 3d). The former case accounts for state dependence; if an individual's current vital rate depends on its past vital rate(s), then that vital rate is deemed state dependent (Authier et al., 2017; Cam et al., 2016). State dependence in potential vital rates may be selected for when, for example, the environment that an individual experiences at one time provides cues about future environmental conditions (Descamps et al., 2008; Douhard et al., 2013; Fay et al., 2018; Hamel et al., 2009b).

Previous works generally refer to fixed condition as "fixed heterogeneity" and dynamic condition as

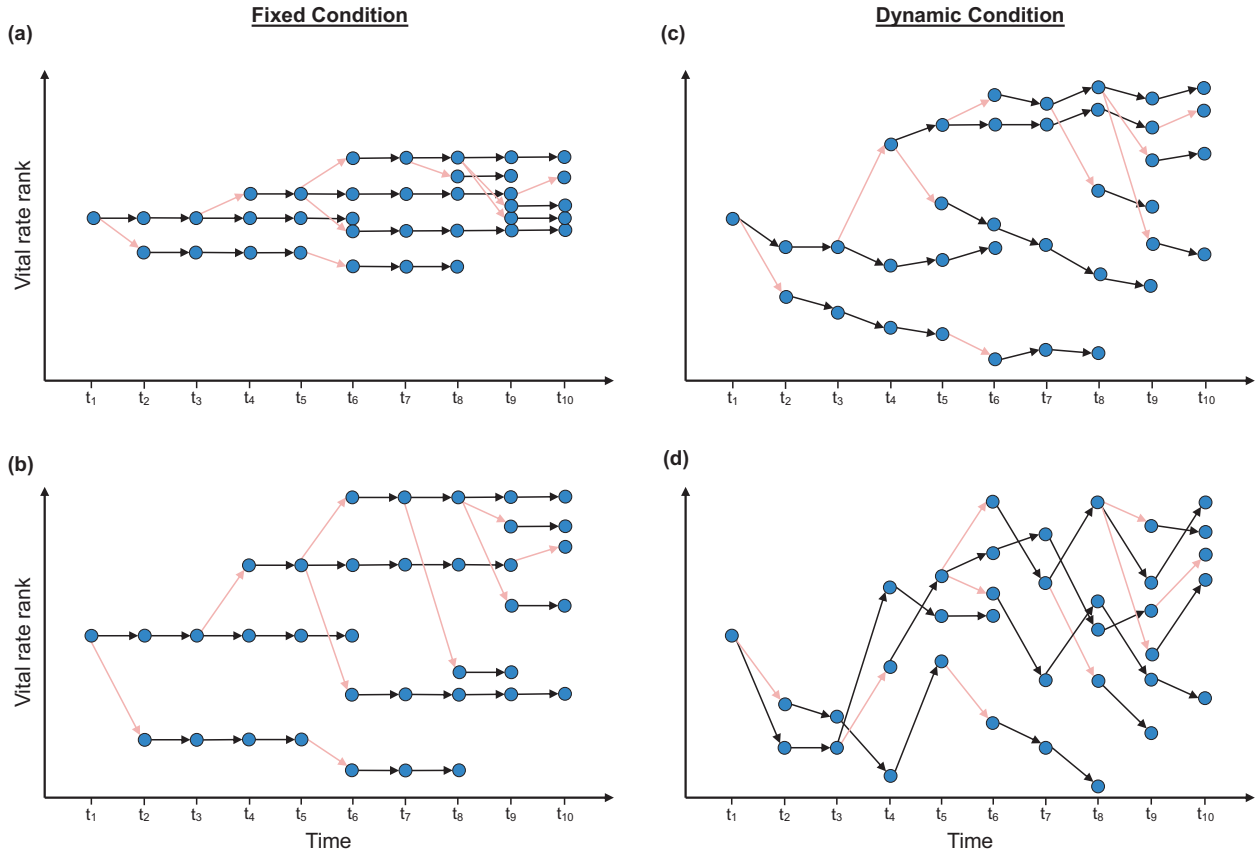

FIGURE 3 Condition hierarchies generated by individual heterogeneity. Each blue dot represents an individual at a particular time (x-axis). Individual vital rate ranks (y-axis) depend on time, and on the type of individual heterogeneity: fixed condition in (a) and (b), or dynamic condition in (c) and (d). As time progresses, individuals can survive (black arrows) and/or give birth to new individuals (pink arrows). (a) A population with fixed condition and heritable vital rates. (b) A population with fixed condition and no heritability in vital rates. (c) A population with dynamic condition and state dependence limiting the possible vital rate transitions between time steps. (d) A population with dynamic condition and no state dependence, leaving individuals free to transition to any potential vital rate phenotype at any time. Potential vital rates can also be heritable or non-heritable under dynamic condition

"dynamic heterogeneity" (e.g. van Daalen & Caswell, 2020; Plard et al., 2018; Tuljapurkar et al., 2009). The challenge with existing terminology, however, is that it can be interpreted in at least two ways: (1) fixed and dynamic heterogeneity are sometimes used in reference to *phenotypes* that are permanent or changing over the lifetime of individuals (e.g. van Daalen & Caswell, 2020; Tuljapurkar & Steiner, 2010; Vindenes et al., 2012) and (2) fixed and dynamic heterogeneity are sometimes used in reference to *among-individual differences* in phenotypes that are permanent or changing over the lifetime of individuals (e.g. Badger et al., 2020; Cam et al., 2013; Chambert et al., 2013). These different definitions for existing terminology create ambiguity and can cause misidentification. For example, consider a population where the survival probability of individuals changes over time, but the differences among individuals in their survival do not change. One interpretation using existing terminology would label this example as dynamic heterogeneity because individual phenotypes are changing over time, whereas the other interpretation would label this as fixed heterogeneity because among-individual differences do not change over time. The framework that we present here would classify this example as dynamic condition because potential vital rates can change over time.

The lack of consensus among definitions has resulted in an explosion of confusing terminology. For example, fixed heterogeneity has also been referred to as permanent heterogeneity (Cam et al., 2016; Vindenes et al., 2012), persistent heterogeneity (Brooks et al., 2017; Kendall et al., 2011), consistent heterogeneity (Fay et al., 2018; Vindenes et al., 2008), constant heterogeneity (Brooks et al., 2017), unobserved heterogeneity (Cam et al., 2016; Caswell & Vindenes, 2018; Jenouvrier et al., 2018), HPDH (hidden persistent/permanent demographic heterogeneity) (Authier et al., 2017; Cam et al., 2016; Oosthuizen et al., 2019), latent heterogeneity (Bonnet & Postma, 2016; Caswell & Vindenes, 2018; Chambert et al., 2013; Fay et al., 2018), latent traits (Bergeron et al., 2011; Bonnet & Postma, 2016; Cam et al., 2016; Paterson et al., 2018; Snyder & Ellner, 2018; Steiner et al., 2010) and static traits (Bergeron et al., 2011; Fay et al., 2018; Hamel et al., 2018; Orzack et al., 2011; Vindenes & Langangen, 2015). Likewise, dynamic heterogeneity has been referred to as transient heterogeneity (Brooks et al., 2017), state dependence (Authier et al., 2017; Cam et al., 2016; Snyder & Ellner, 2018), variable heterogeneity (Badger et al., 2020; Chambert et al., 2013), correlated variable heterogeneity (Badger et al., 2020), latent traits (Cam et al., 2016; Gimenez et al., 2018), labile traits (Brooks et al., 2017; Childs et al., 2016), dynamic frailty (Cam et al., 2016; Gimenez et al., 2018), dynamic traits (Bergeron et al., 2011; Vindenes & Langangen, 2015) and luck (Snyder & Ellner, 2018; Tuljapurkar et al., 2020). This dizzying array of existing terminology reduces clarity in the literature and ultimately impedes progress in the field.

Environmental heterogeneity and stochasticity

One of the most common mechanisms generating individual heterogeneity is environmental heterogeneity (Figure 1), which is defined as environmental differences between two or more locations (Box 1). Individuals may attain higher or lower potential vital rates depending on their environment, and so environmental heterogeneity contributes to determining individual ranks in the condition hierarchy. Environmental heterogeneity may generate fixed condition if, for example, one environment consistently provides more nutrients than another, or it could generate dynamic condition if the environment that provides more nutrients changes over time or if individuals can move between locations.

Unlike environmental heterogeneity, environmental stochasticity does not act as a source of individual heterogeneity because it is assumed to modify the potential vital rates of all individuals in the same way, such that it does not impact the condition hierarchy (Figure 1). Taking an approach similar to the existing literature (Acker et al., 2014; Engen et al., 2007; Sæther & Engen, 2015), the framework that we present here defines environmental stochasticity as random variation in the environment that similarly affects the potential vital rates of all individuals in a given condition (Box 1). This definition recognises that random environmental events (e.g. droughts, floods, rain storms) can sometimes influence individuals of low condition more strongly than individuals of high condition, or vice versa. However, the rank of potential vital rates (i.e. which individuals are high and low condition) will not change, and so environmental stochasticity cannot generate individual heterogeneity on its own.

Heterogeneity versus stochasticity: a lingering debate

Individual heterogeneity refers to variation among individuals in their potential vital rates, and it is important to distinguish this variation from individual stochasticity. Individual stochasticity refers to the differences among individual *realisations* of the same underlying, potential, vital rate (Box 1; Figure 1; see also Caswell, 2009; Hartemink & Caswell, 2018; Jenouvrier et al., 2019). The resulting variation may resemble that which is produced by dynamic condition because both processes allow individual vital rates to take on new values at any time. Indeed, dynamic condition at its most extreme allows potential vital rates to change randomly at any time. As such, we expect that dynamic condition may often be empirically indistinguishable from the random changes in realised vital rates occurring due to individual stochasticity. These similarities prompted previous studies to use the term "dynamic heterogeneity" (dynamic condition in this framework) synonymously

with "individual stochasticity" (e.g. Authier et al., 2017; Badger et al., 2020; Caswell & Vindenes, 2018; Gimenez et al., 2018; Snyder & Ellner, 2018; Tuljapurkar et al., 2020; Vindenes et al., 2012). However, the two processes differ in the underlying cause of among-individual variation in vital rates: individual stochasticity arises from random changes in realised vital rates, whereas dynamic condition requires systematic changes in potential vital rates. Individual stochasticity could be modelled in a population of identical individuals, while models of dynamic condition must incorporate among-individual variation in potential vital rates. Recent works have made a similar distinction by treating individual stochasticity and "dynamic heterogeneity" as separate processes (Coste & Pavard, 2020; van Daalen & Caswell, 2020; Hartemink & Caswell, 2018; Jenouvrier et al., 2019).

Studies that assume equivalence between dynamic heterogeneity and individual stochasticity typically view dynamic heterogeneity as an evolutionarily neutral process (Authier et al., 2017; Steiner & Tuljapurkar, 2012). These studies assume that random differences throughout an individual's life that do not change its potential vital rates are non-heritable (Bonnet & Postma, 2016; Giaimo et al., 2018). Hence, the variation that is generated cannot be passed on to the next generation and it is neutral with respect to evolution. On the other hand, the framework presented here (Figure 1) emphasises that dynamic condition requires genuine differences in potential vital rates, and these potential vital rates may be transmitted from parents to offspring (Figure 3c, d). Moreover, potential vital rates that improve individual fitness may be favoured by selection. Dynamic condition can, therefore, play a role in evolution by natural selection, unlike individual stochasticity.

A THEORETICAL APPROACH TO INDIVIDUAL HETEROGENEITY

To demonstrate an application of the framework, we develop an explicit example of how individual heterogeneity in survivorship and birth rate fits into a simple age-structured population projection matrix. This example closely follows previous work from Plard et al., (2018), who used an integral projection model to estimate the age- and sex-dependent influences of fixed heterogeneity (morphology) and dynamic heterogeneity (body condition) on individual fitness and population growth rates. Here, we take a simpler approach that considers a single sex with discrete traits to illustrate how fixed and dynamic condition can generate structural differences in a population model.

The goal of this mathematical model is to clarify the framework and definitions that we present here. Accordingly, the model uses a minimal number of age classes and potential vital rates for the purposes of illustration. While it does not incorporate the full breadth of biological complexity that occurs in real populations, this example provides a basis from which additional complexity can be incorporated. It assumes that the values and degree of persistence of potential vital rates in a population are known, such that they can be used to predict ecological and evolutionary outcomes (i.e. forward modelling approach). We will expand this model to accommodate more advanced population structures in future works.

Consider an age-structured population with no density dependence and two ages: juvenile (age 1) and adult (age 2). Juveniles survive and develop to adults with probability s and adults give birth to β offspring on average (Figure 4a). Here, we adopt a pre-breeding census representation, and so β also accounts for the survival of newborns to age 1 (e.g. Kendall et al., 2019; Okuyama, 2019). There is no reproduction from juveniles, and no survival of adults after reproduction. The number of individuals in each age class at time t is given by

$$n_t = \begin{bmatrix} J_t \\ A_t \end{bmatrix}$$

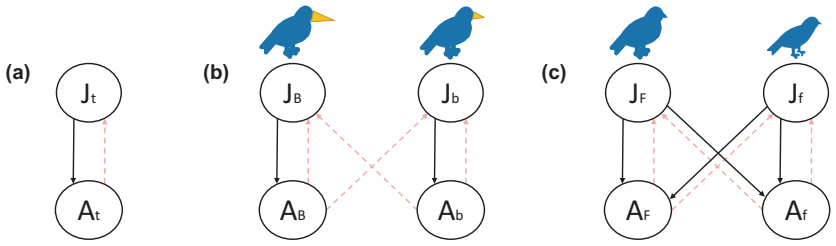

FIGURE 4 Flow diagrams for a two-age population. Solid lines represent survival of juveniles, and dashed lines represent births of offspring for (a) a population of identical individuals, (b) a population with fixed condition and (c) a population with dynamic condition. In (a), individuals are represented by an average survival probability and birth rate that applies to the entire population. In (b), there are two beak sizes that determine an individual's potential vital rates: long and short. In (c), there are two body fat phenotypes that determine an individual's potential vital rates: high fat and low fat. An individual's potential survival probability and birth rate are permanent throughout life with fixed condition as in (b), whereas these potential vital rates can change throughout life with dynamic condition as in (c). Individual stochasticity influences realised vital rates in all three populations, while individual heterogeneity is only present in populations (b) and (c)

where J_t is the number of juveniles and A_t is the number of adults. The expected number of juveniles and adults at time *t+1* is given by

$$n_{t+1} = Ln_t$$

where L is the Leslie (age-structured) matrix

$$L = \begin{bmatrix} 0 & \beta \\ s & 0 \end{bmatrix}$$

This matrix assumes that all individuals share the same potential vital rates, s and β, and so individuals can only differ due to individual stochasticity.

To incorporate individual heterogeneity in this two-age model, suppose that individuals vary in some trait that directly influences their potential vital rates. Although these vital rates can sometimes be directly estimated (e.g. Tuljarpurkar et al. 2009; Gimenez et al., 2012; Olijnyk & Nelson, 2013), previous frameworks often take a similar approach that maps among-individual variation in vital rates to differences in phenotype (e.g. Coulson et al., 2010; Hairston et al., 2005; Knight et al., 2008; Vindenes & Langangen, 2015). For example, a bird's beak size might determine how much food it can acquire, and so birds with an optimal beak size would have more energy to allocate to increasing survival and/or reproductive success compared to birds with other beak sizes. Here, we assume that an individual's beak size category is permanent throughout life and determines its potential vital rates. Accordingly, beak size acts as a source of fixed condition in individual life histories. Plard et al., (2018) classify this example as a measure of "fixed heterogeneity" or "individual quality."

At the opposite extreme, dynamic condition allows individuals to transition between potential vital rates at any time. Here, we will take body fat as an example of a trait generating dynamic condition among individual life histories. An individual's body fatness may depend on several factors that change over its lifetime, such as nutrient availability in the environment, competitive abilities and/or social status. Viewed in this way, Plard et al., (2018) consider body fat a measure of "dynamic heterogeneity" or "condition."

As a simple illustration, assume that individuals can be categorised into one of two types of beak sizes, long (*B*) and short (*b*), and one of two body fat levels, high fat (*F*) and low fat (*f*). Birds with long beaks are assumed to have higher fitness, thus the potential vital rates of long-beaked individuals are greater than the potential vital rates of short-beaked individuals. Another way to conceptualise this difference in condition is to imagine the hierarchy, where long-beaked individuals rank higher in terms of their overall condition than short-beaked individuals. Likewise, individuals with high body fat have higher potential vital rates, and thus a higher condition, than individuals with low body fat.

Assuming that any beak size (*B* or *b*) can be found with any body fatness (*F* or *f*), there are four possible phenotypes in the population: individuals can have a long beak and a high body fat (potential vital rates s_{BF} and β_{BF}), a long beak and a low body fat (potential vital rates s_{Bf} and β_{Bf}), a short beak and a high body fat (potential vital rates s_{bF} and β_{bF}) or a short beak and a low body fat (potential vital rates s_{bf} and β_{bf}). Individuals with a long beak and high body fat will have the highest condition in the population because they have the highest potential vital rates. Similarly, individuals with a short beak and low body fat will have the lowest condition in the population because they have the lowest potential vital rates. The other two phenotypes will rank somewhere in the middle because they have one high valued and one low valued trait influencing their potential vital rates.

Considering these four types of individuals gives the population vector

$$n_t = \begin{bmatrix} J_{BF} \\ J_{Bf} \\ J_{bF} \\ J_{bf} \\ A_{BF} \\ A_{Bf} \\ A_{bF} \\ A_{bf} \end{bmatrix}$$

where J_{ij} and A_{ij} are the number of juveniles and adults with beak size i and body fatness j.

In this model, adults can give birth to offspring with either beak size or body fat level. The probability that an adult gives birth to long-beaked offspring is σ_B and the probability that an adult gives birth to short-beaked offspring is σ_b. Further assume that $\sigma_B + \sigma_b = 1$, so that all offspring must be born into one of the two beak size phenotypes and there is no death of newborns. These transition probabilities are independent of the parental phenotype (e.g. Figure 3b); however, they could be extended to depend on the phenotype of both offspring and adults to provide a model with inheritance (e.g. Figure 3a).

To allow for movement among body fat phenotypes within a generation, we apply a second set of transition probabilities not only to newborn offspring, but also to surviving juveniles. Hence, it is the transition probabilities that are used to distinguish fixed and dynamic condition. Here, α_F is the probability that any individual, newborn or surviving juvenile, transitions to a high body fat by the next time step and α_f is the probability that an individual transitions to a low body fat by the next time step. This assumption may be relaxed in future models to allow different transition probabilities for offspring and survivors (e.g. Figure 3c). Assume further that $\alpha_F + \alpha_f = 1$, so that there

is no death of newborns or surviving individuals from the transition process.

In this example where beak size is permanent throughout life (i.e. fixed condition) but body fat levels can change (i.e. dynamic condition), the Leslie matrix is

$$L_{FD} = \begin{bmatrix} 0 & 0 & 0 & 0 & \sigma_B\alpha_F\beta_{BF} & \sigma_B\alpha_F\beta_{Bf} & \sigma_B\alpha_F\beta_{bF} & \sigma_B\alpha_F\beta_{bf} \\ 0 & 0 & 0 & 0 & \sigma_B\alpha_f\beta_{BF} & \sigma_B\alpha_f\beta_{Bf} & \sigma_B\alpha_f\beta_{bF} & \sigma_B\alpha_f\beta_{bf} \\ 0 & 0 & 0 & 0 & \sigma_b\alpha_F\beta_{BF} & \sigma_b\alpha_F\beta_{Bf} & \sigma_b\alpha_F\beta_{bF} & \sigma_b\alpha_F\beta_{bf} \\ 0 & 0 & 0 & 0 & \sigma_b\alpha_f\beta_{BF} & \sigma_b\alpha_f\beta_{Bf} & \sigma_b\alpha_f\beta_{bF} & \sigma_b\alpha_f\beta_{bf} \\ \alpha_F s_{BF} & \alpha_F s_{Bf} & 0 & 0 & 0 & 0 & 0 & 0 \\ \alpha_f s_{BF} & \alpha_f s_{Bf} & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \alpha_F s_{bF} & \alpha_F s_{bf} & 0 & 0 & 0 & 0 \\ 0 & 0 & \alpha_f s_{bF} & \alpha_f s_{bf} & 0 & 0 & 0 & 0 \end{bmatrix}$$

Notice that while the transition probabilities for offspring depend on both beak size and body fat, the transition probabilities for surviving juveniles depend only on body fat because beak size does not change with age. This degree of consistency in phenotype means that the individual heterogeneity is structured between fixed and dynamic condition, in the sense that surviving individuals can transition into certain phenotypes (non-zero entries in the lower left quarter), but not others (zero entries in the lower left quarter). The phenotypes that are available to an individual depend on the phenotype that it was born into.

To emphasise the structural differences between fixed and dynamic condition, now suppose that all individuals have high body fat levels, such that differences in beak size are the only source of variation among potential vital rates (Figure 4b). In this model, individuals can belong to one of two phenotypes: long-beaked (potential vital rates s_B and β_B) or short-beaked (potential vital rates s_b and β_b). An individual's beak size is still assumed to be permanent throughout life, and so the Leslie matrix including only fixed condition is

$$L_F = \begin{bmatrix} 0 & 0 & 0 & 0 & \sigma_B\beta_B & 0 & \sigma_B\beta_b & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \sigma_b\beta_B & 0 & \sigma_b\beta_b & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ s_B & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & s_b & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

This model of fixed condition is similar to a classic quantitative genetics model of beak sizes. In both approaches, there can be a degree of inheritance in beak size, but once an individual's beak size is set at birth, it is assumed to be permanent throughout life. Both models also recognise that even if individuals are genetically identical, their beak sizes may still vary due to non-genetic sources of individual heterogeneity. However, the way that this non-genetic variation is incorporated contrasts between the models and this distinction can result in cases where predictions of ecological and evolutionary dynamics are different when using quantitative genetics models versus models of fixed condition (see Appendix S1).

As a contrast for this example, suppose that all individuals have long beak sizes but vary in their body fat levels. This situation generates a model of dynamic condition because body fat levels can change over time, and so surviving individuals can transition between potential vital rates throughout their life. Assume that high fat juveniles survive with probability s_F and high fat adults have birth rate β_F, while low fat juveniles survive with probability s_f and low fat adults have birth rate β_f. The Leslie matrix incorporating dynamic condition is therefore

$$L_D = \begin{bmatrix} 0 & 0 & 0 & 0 & \alpha_F\beta_F & \alpha_F\beta_f & 0 & 0 \\ 0 & 0 & 0 & 0 & \alpha_f\beta_F & \alpha_f\beta_f & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \alpha_F s_F & \alpha_F s_f & 0 & 0 & 0 & 0 & 0 & 0 \\ \alpha_f s_F & \alpha_f s_f & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

This matrix is similar to the matrix with fixed condition; however, dynamic condition allows surviving juveniles to develop to adults of either phenotype (Figure 4c). This additional movement among phenotypes impacts population dynamics because fitness depends on individual survival and reproduction. While an individual's expected fitness may increase or decrease from one time to the next under dynamic condition, that same individual's expected fitness would be constant under fixed condition.

The above-fixed and dynamic condition matrices can be reduced to smaller matrices where individuals are classified only by their age (juvenile or adult) and by the trait producing individual heterogeneity (beak size *or* body fat). The population vectors are

$$n_t = \begin{bmatrix} J_B \\ J_b \\ A_B \\ A_b \end{bmatrix}$$

for fixed condition, and

$$n_t = \begin{bmatrix} J_F \\ J_f \\ A_F \\ A_f \end{bmatrix}$$

Box 2 Applying fixed and dynamic condition to simplify previous models and terminology

To illustrate the contributions of the framework that we have developed, we show how it can be applied to consolidate four previous studies of individual heterogeneity. The first is from Vindenes et al., (2012), who construct a population projection matrix model that includes individual heterogeneity in both survival and fecundity. The term "permanent heterogeneity" is used to describe the situation where individual survival and fecundity persists for life. This matrix model uses the same structure as Vindenes et al., (2008); however, the term "consistent heterogeneity" is adopted in the latter study to refer to individuals that differ in their vital rates and keep these vital rates throughout their life. The framework presented here would label both examples as "fixed condition."

More recently, Badger et al., (2020) used a generalised linear mixed model (GLMM) to test for various types of individual heterogeneity in reproductive rates of female grey seals (*Halichoerus grypus*). "Fixed heterogeneity" was defined as the situation where "individuals vary in a consistent manner across all conditions" (Badger et al., 2020) and it was modelled using a random intercept that shifts the reproductive rate of each individual. Other terms in the model of "fixed heterogeneity" included fixed effects of population density, age, breeding experience in the previous year and a random effect of year. Accordingly, individual vital rates can still change (with age, population density, breeding experience and year) throughout life under the "fixed heterogeneity" hypothesis of Badger et al., (2020), which contrasts with the idea that individual vital rates are constant throughout life in the "consistent" and "permanent" heterogeneity matrix models of Vindenes et al., (2008, 2012). The framework that we introduce classifies the GLMM from Badger et al., (2020) as dynamic condition, since an individual's reproductive rates could change at any time throughout life.

The framework of individual heterogeneity that we present establishes a set of terminology that matches distinctions used in mathematical models. In their review of condition dependence, Ronget et al., (2017) define condition as "a generic term to rank individuals within a given population along a continuum, from frail individuals at one end to robust individuals at the other end." This concept exactly matches the approach that we take in the synthesised framework, where condition directly maps onto potential vital rates. The idea of a "continuum" of individual quality corresponds to the hierarchy that we use to rank individuals from low to high condition. High condition individuals will have higher values of at least one potential vital rate. From there, Ronget et al., (2017) focus specifically on condition-dependent mortality, defined as the amount of heterogeneity in mortality risk across individuals. This heterogeneity is also referred to as "frailty," where each individual is assigned a frailty value at birth that persists for life. The framework that we present here recognises that "frailty" is simply a form of fixed condition in mortality rates.

for dynamic condition. The simplified fixed and dynamic condition matrices are given by

$$L_F = \begin{bmatrix} 0 & 0 & \sigma_B\beta_B & \sigma_B\beta_b \\ 0 & 0 & \sigma_b\beta_B & \sigma_b\beta_b \\ s_B & 0 & 0 & 0 \\ 0 & s_b & 0 & 0 \end{bmatrix} L_D = \begin{bmatrix} 0 & 0 & \alpha_F\beta_F & \alpha_F\beta_f \\ 0 & 0 & \alpha_f\beta_F & \alpha_f\beta_f \\ \alpha_F s_F & \alpha_F s_f & 0 & 0 \\ \alpha_f s_F & \alpha_f s_f & 0 & 0 \end{bmatrix}$$

where L_F is the Leslie matrix for fixed condition and L_D is the Leslie matrix for dynamic condition. Although it is beyond the scope of this study, future versions of our model could also account for individual heterogeneity due to age structure by increasing the number of age classes and ensuring that an individual's potential vital rates at least partially depend on its age.

DISCUSSION

Here we have presented a coherent framework for conceptualising individual heterogeneity, which subsumes previous works into straightforward terminology with explicit translations to mathematical models. This framework is built on an approach that envisions individual vital rates as a probability distribution and separates potential vital rates from realised vital rates. An individual's potential vital rates are defined by a probability distribution of possible values, whereas its realised vital rates are particular draws from this distribution. We use this approach to condense the plethora of existing terminology into just two types of individual heterogeneity: fixed condition and dynamic condition (e.g. see Box 2; Table S1). In this discussion, we now draw connections between this framework and the current literature on individual heterogeneity.

One of the more common terms found throughout the literature that we have not yet discussed is "demographic heterogeneity." This term is used to describe the emergent population-level expression of individual heterogeneity (Cressler et al., 2017; Plard et al., 2019) that contributes to ecological and evolutionary outcomes. Individual heterogeneity considers among-individual variation in vital rates, while demographic heterogeneity

focuses on the cohorts of strong and weak individuals that result from individual heterogeneity. Hence, individual heterogeneity necessarily generates demographic heterogeneity in a population, such that one cannot exist without the other. It, therefore, seems redundant to use both terms given that populations with individual heterogeneity will *always* have demographic heterogeneity. We favour the term individual heterogeneity because it clarifies that the differences are ultimately a result of among-individual variation in vital rates.

Historically, empirical studies have used the term "individual heterogeneity" to refer to among-individual differences in condition that could not be explained using measured variables. Hence, among-individual variation in potential vital rates that is produced by conspicuous large-scale physiological differences, such as age, developmental stage, size or sex, has not traditionally been considered as individual heterogeneity (Brooks et al., 2017; Cam et al., 2016; Stover et al., 2012). The reality, however, is that whether among-individual differences in potential vital rates arise from differences among conspicuous physiological classes, or from less obvious sources, is arbitrary as it pertains to the final structure of individual heterogeneity. The result is individuals that differ in their expected vital rates, regardless of the source or how we choose to categorise individuals. Nonetheless, the historical classification is useful for studies that seek to understand the different biological mechanisms generating and maintaining individual heterogeneity in populations. It also separates simple models that incorporate individual differences *among* distinct physiological classes (e.g. Leslie and Lefkovitch matrices) from more complex models that incorporate individual differences among and *within* these classes. Although large-scale physiological classes are typically the most obvious way to categorise individuals, the among-individual differences within these categories are often equally, if not more, important in determining individual fitness and population dynamics (Badger et al., 2020; Fay et al., 2018).

Individual heterogeneity was first studied as fixed condition to account for persistent among-individual variation in vital rates that arises from unmeasured variables. For example, statistical models often use random effects to assign each individual a unique value of latent "quality" that is fixed throughout the individual's life and independent of any measured variables (see Appendix S2; Cam et al., 2013; Chambert et al., 2013; Fox et al., 2006; Gimenez et al., 2018). This traditional approach has prompted some studies to conclude that fixed condition *must* be unobserved (e.g. Authier et al., 2017; Bonnet & Postma, 2016; Tuljapurkar et al., 2009; Vindenes & Langangen, 2015). However, fixed condition also occurs when observed potential vital rates are constant over an individual's lifetime (van Daalen & Caswell, 2020; Dahlgren et al., 2016; Tompkins & Anderson, 2019). In fact, there would be no unobserved individual heterogeneity if researchers were able to measure all of the variables that impact condition and thus the vital rates of interest. As such, the framework proposed here defines fixed and dynamic condition by the degree of persistence in individual vital rates, and not by our ability to observe them.

A number of statistical approaches have been developed to estimate fixed and dynamic condition in real populations (for a review, see Appendix S2; Table S2). Some approaches involve statistical models designed to capture patterns in the data (e.g. van Hamel et al., 2017; de Pol & Verhulst, 2006) and others estimate biological parameters using process-based models (e.g. Tuljapurkar et al., 2009). When choosing which approach to use, it is important to consider the constraints that each model imposes on individual movements among vital rate classes because these movements ultimately determine the type of individual heterogeneity that can be detected (Appendix S2). For example, generalised linear mixed models and capture–mark–recapture analyses typically impose restrictions on the sequences of vital rates that individuals can take throughout their life (e.g. Badger et al., 2020; Brusa et al., 2020; Moyes et al., 2011). Even under dynamic condition, individuals are constrained in their future vital rate paths because the heterogeneity is incorporated using covariates that depend on the individual's vital rate history. Process-based models, on the other hand, are more flexible because an individual's history does not necessarily determine the vital rates that it can attain in the future. For example, the transition matrix approach allows individuals with the same vital rate at one time to follow completely different trajectories of vital rates in the future (e.g. Plard et al., 2012; Steiner et al., 2010; Tuljapurkar & Steiner, 2010).

In practice, direct measurement of individual vital rates can be difficult, if not impossible, to achieve in natural populations. Generally, empirical studies will only be able to observe realised vital rates because these are the rates that are actually experienced by individuals. This constraint will sometimes lead to an overestimation of the among-individual variation in vital rates that can be attributed to individual stochasticity, because what is measured as individual stochasticity is actually individual stochasticity plus any individual heterogeneity that is not specifically accounted for (Cam et al., 2016; Gimenez et al., 2018; Plard et al., 2012). Individual heterogeneity can still be estimated, however, by averaging over many individuals to minimise the effects of individual stochasticity. This approach is well established in the evolutionary literature, where individual genotypes (a common mechanism for fixed condition) are estimated using an average genetic value for the population plus an individual deviation, while controlling for other sources of heterogeneity (e.g. animal models, Kruuk, 2004; Wilson et al., 2010). We emphasise that although it is often challenging, it is critical to empirically distinguish this potential vital rate from the individual's realised

vital rate because the two represent different biological processes. Individuals that vary in their *potential* vital rates have a genuine difference in their underlying condition. However, individuals that vary in their *realised* vital rates may do so because of these differences in potential vital rates *and* because of random events experienced over their life. Accordingly, individuals can have equal potential vital rates and still differ in their realised vital rates (Caswell, 2009; Jenouvrier et al., 2018).

Individual heterogeneity was traditionally thought of in a separate paradigm from quantitative genetics. More recently, a handful of studies have recognised quantitative traits as a mechanism generating individual heterogeneity and contributing to evolutionary change in vital rates (Barfield et al., 2011; Childs et al., 2016; Coulson et al., 2017; Janeiro et al., 2017; Rees & Ellner, 2016, 2019). However, yet to be studied (to our knowledge) is the idea that fixed condition and quantitative genetics models are, in some situations, two sides of the same coin. For example, fixed condition is commonly incorporated in quantitative genetics frameworks, which also assume that an individual's phenotype is constant throughout its life (Kruuk, 2004; Lande, 1982). Indeed, models of fixed condition (Appendix S1) and quantitative genetics models can both take an individual's potential vital rates to be determined by an underlying genetic value plus some environmental "noise." This similarity results in situations where models of fixed condition and quantitative genetics are equivalent to one another, yet there are also situations where the two perspectives differ (Appendix S1). Merging individual heterogeneity and quantitative genetics frameworks could provide novel insights to evolutionary consequences of fixed condition in populations.

CONCLUDING REMARKS

Accounting for individual heterogeneity in empirical and theoretical work is crucial for understanding and predicting population dynamics (Kendall et al., 2011; Noonburg et al., 2015; Schindler et al., 2010; Waddle et al., 2019). Yet, existing studies of heterogeneity are often incompatible because of inconsistencies and ambiguities in their terminology and definitions. More than just shortcomings in terminology, these issues highlight the need for a common framework of individual heterogeneity that bridges empirical and theoretical studies.

Our study attempts to fill this gap by synthesising previous approaches into a comprehensive framework of individual heterogeneity (Box 1; Figure 1). The framework provides a set of explicit terminology and definitions that we hope will help to clarify the meaning of individual heterogeneity and individual stochasticity in populations. We also connect the framework to common classes of statistical models that are used to infer individual heterogeneity from data, and we use specific examples to show how this simplified framework can be translated into mathematical models. This translation serves as a direct link between empirical and theoretical studies of individual heterogeneity. Most importantly, the framework presented here allows the focus to shift away from semantics, and towards the substantial impacts of individual heterogeneity on individuals and populations.

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AUTHORSHIP

ABF, TD and WAN designed the synthesised framework. ABF wrote the first draft of the manuscript and all authors contributed substantially to revisions.

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No data were used.

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REFERENCES

Acker, P., Robert, A., Bourget, R. & Colas, B. (2014) Heterogeneity of reproductive age increases the viability of semelparous populations. *Functional Ecology*, 28, 458–468.

Authier, M., Aubry, L.M. & Cam, E. (2017) Wolf in sheep's clothing: model misspecification undermines tests of the neutral theory for life histories. *Ecology and Evolution*, 7, 3348–3361.

Badger, J.J., Bowen, W.D., den Heyer, C.E. & Breed, G.A. (2020) Variation in individual reproductive performance amplified with population size in a long-lived carnivore. *Ecology*, 101, e03024.

Barfield, M., Holt, R.D. & Gomulkiewicz, R. (2011) Evolution in stage-structured populations. *American Naturalist*, 177, 397–409.

Barks, P.M. & Laird, R.A. (2020) Parental age effects and the evolution of senescence. *American Naturalist*, 195, 886–898.

Bergeron, P., Baeta, R., Pelletier, F., Réale, D. & Garant, D. (2011) Individual quality: tautology or biological reality? *Journal of Animal Ecology*, 80, 361–364.

Bishop, A.M., Dubel, A.K., Sattler, R., Brown, C.L. & Horning, M. (2019) Wanted dead or alive: characterizing likelihood of juvenile Steller sea lion predation from diving and space use patterns. *Endangered Species Research*, 40, 357–367.

Bolnick, D.I., Amarasekare, P., Araújo, M.S., Bürger, R., Levine, J.M., Novak, M. et al. (2011) Why intraspecific trait variation matters in community ecology. *Trends in Ecology & Evolution*, 26, 183–192.

Bonnet, T. & Postma, E. (2016) Successful by chance? The power of mixed models and neutral simulations for the detection of individual fixed heterogeneity in fitness components. *American Naturalist*, 187, 60–74.

Bowen, W.D., den Heyer, C.E., McMillan, J.I. & Iverson, S.J. (2015) Offspring size at weaning affects survival to recruitment and reproductive performance of primiparous gray seals. *Ecology and Evolution*, 5, 1412–1424.

Boyce, M.S. (1977) Population growth and stochastic fluctuations in the life table. *Theoretical Population Biology*, 12, 366–373.

Brooks, M.E., Clements, C., Pemberton, J. & Ozgul, A. (2017) Estimation of individual growth trajectories when repeated measures are missing. *American Naturalist*, 190, 377–388.

Bruijning, M., Jongejans, E. & Turcotte, M.M. (2019) Demographic responses underlying eco-evolutionary dynamics as revealed with inverse modelling. *Journal of Animal Ecology*, 88, 768–779.

Brusa, J.L., Rotella, J.J., Garrott, R.A., Paterson, J.T. & Link, W.A. (2020) Variation of annual apparent survival and detection rates with age, year and individual identity in male Weddell seals (*Leptonychotes weddellii*) from long-term mark-recapture data. *Population Ecology*, 62, 134–150.

Cam, E., Aubry, L.M. & Authier, M. (2016) The conundrum of heterogeneities in life history studies. *Trends in Ecology & Evolution*, 31, 872–886.

Cam, E., Gimenez, O., Alpizar-Jara, R., Aubry, L.M., Authier, M., Cooch, E.G. et al. (2013) Looking for a needle in a haystack: inference about individual fitness components in a heterogenous population. *Oikos*, 122, 739–753.

Caswell, H. (2009) Stage, age and individual stochasticity in demography. *Oikos*, 118, 1763–1782.

Caswell, H. & Vindenes, Y. (2018) Demographic variance in heterogeneous populations: matrix models and sensitivity analysis. *Oikos*, 127, 648–663.

Chambert, T., Rotella, J.J., Higgs, M.D. & Garrott, R.A. (2013) Individual heterogeneity in reproductive rates and cost of reproduction in a long-lived vertebrate. *Ecology and Evolution*, 3, 2047–2060.

Childs, D.Z., Sheldon, B.C. & Rees, M. (2016) The evolution of labile traits in sex- and age-structured populations. *Journal of Animal Ecology*, 85, 329–342.

Coste, C.F.D. & Pavard, S. (2020) Analysis of a multitrait population projection matrix reveals the evolutionary and demographic effects of a life history trade-off. *Ecological Modelling*, 418, 108915.

Coulson, T., Benton, T.G., Lundberg, P., Dall, S.R.X. & Kendall, B.E. (2006a) Putting evolutionary biology back in the ecological theatre: a demographic framework mapping genes to communities. *Evolutionary Ecology Research*, 8, 1155–1171.

Coulson, T., Benton, T.G., Lundberg, P., Dall, S.R.X., Kendall, B.E. & Gaillard, J.M. (2006b) Estimating individual contributions to population growth: evolutionary fitness in ecological time. *Proceedings of the Royal Society B*, 273, 547–555.

Coulson, T., Gaillard, J.M. & Festa-Bianchet, M. (2005) Decomposing the variation in population growth into contributions from multiple demographic rates. *Journal of Animal Ecology*, 74, 789–801.

Coulson, T., Kendall, B.E., Barthold, J., Plard, F., Schindler, S., Ozgul, A. et al. (2017) Modeling adaptive and nonadaptive responses of populations to environmental change. *American Naturalist*, 190, 313–336.

Coulson, T., Tuljapurkar, S. & Childs, D.Z. (2010) Using evolutionary demography to link life history theory, quantitative genetics and population ecology. *Journal of Animal Ecology*, 79, 1226–1240.

Cressler, C.E., Bengtson, S. & Nelson, W.A. (2017) Unexpected nongenetic individual heterogeneity and trait covariance in *Daphnia* and its consequences for ecological and evolutionary dynamics. *American Naturalist*, 190, E13–E27.

Dahlgren, J.P., Colchero, F., Jones, O.R., Øien, D.I., Moen, A. & Sletvold, N. (2016) Actuarial senescence in a long-lived orchid challenges our current understanding of ageing. *Proceedings of the Royal Society B*, 283, 20161217.

Deere, J.A., Coulson, T., Cubaynes, S. & Smallegange, I.M. (2017) Unsuccessful dispersal affects life history characteristics of natal populations: the role of dispersal related variation in vital rates. *Ecological Modelling*, 366, 37–47.

Demetrius, L., Gundlach, V.M. & Ochs, G. (2004) Complexity and demographic stability in population models. *Theoretical Population Biology*, 65, 211–225.

Descamps, S., Boutin, S., Berteaux, D., McAdam, A.G. & Gaillard, J.M. (2008) Cohort effects in red squirrels: the influence of density, food abundance and temperature on future survival and reproductive success. *Journal of Animal Ecology*, 77, 305–313.

Dierickx, E.G., Robinson, R.A., de Brooke, M.L. (2019) Survival of a long-lived single island endemic, the Raso lark *Alauda razae*, in relation to age, fluctuating population and rainfall. *Scientific Reports*, 9, 19557.

Doak, D.F., Morris, W.F., Pfister, C., Kendall, B.E. & Bruna, E.M. (2005) Correctly estimating how environmental stochasticity influences fitness and population growth. *American Naturalist*, 166, E14–E21.

Douhard, M., Gaillard, J.-M., Delorme, D., Capron, G., Duncan, P., Klein, F. et al. (2013) Variation in adult body mass of roe deer: early environmental conditions influence early and late body growth of females. *Ecology*, 94, 1805–1814.

Ducros, D., Morellet, N., Patin, R., Atmeh, K., Debeffe, L., Cargnelutti, B. et al. (2020) Beyond dispersal versus philopatry? Alternative behavioural tactics of juvenile roe deer in a heterogeneous landscape. *Oikos*, 129, 81–92.

Ellner, S.P., Geber, M.A. & Hairston, N.G. Jr (2011) Does rapid evolution matter? Measuring the rate of contemporary evolution and its impacts on ecological dynamics. *Ecology Letters*, 14, 603–614.

Engen, S., Lande, R., Sæther, B.E. & Festa-Bianchet, M. (2007) Using reproductive value to estimate key parameters in density-independent age-structured populations. *Journal of Theoretical Biology*, 244, 308–317.

Fay, R., Barbraud, C., Delord, K. & Weimerskirch, H. (2018) From early life to senescence: individual heterogeneity in a long-lived seabird. *Ecological Monographs*, 88, 60–73.

Fox, G.A., Kendall, B.E., Fitzpatrick, J.W. & Woolfenden, G.E. (2006) Consequences of heterogeneity in survival probability in a population of Florida scrub-jays. *Journal of Animal Ecology*, 75, 921–927.

Giaimo, S., Li, X.Y., Traulsen, A. & Baudisch, A. (2018) Evolution of fixed demographic heterogeneity from a game of stable coexistence. *Demographic Research*, 38, 197–226.

Gillespie, D.O.S., Trotter, M.V., Krishna-Kumar, S. & Tuljapurkar, S.D. (2013) Birth-order differences can drive natural selection on aging. *Evolution*, 68, 886–892.

Gimenez, O., Cam, E. & Gaillard, J.M. (2018) Individual heterogeneity and capture-recapture models: what, why and how? *Oikos*, 127, 664–686.

Gimenez, O., Lebreton, J.D., Gaillard, J.M., Choquet, R. & Pradel, R. (2012) Estimating demographic parameters using hidden process dynamic models. *Theoretical Population Biology*, 82, 307–316.

Gomes, M.G.M. (2019) On the mathematics of populations. *bioRxiv*, 612366. https://doi.org/10.1101/612366.

Gould, A.L., Zhang, V., Lamberti, L., Jones, E.W., Obadia, B., Korasidis, N. et al. (2018) Microbiome interactions shape host fitness. *Proceedings of the National Academy of Sciences of the United States of America*, 115, E11951–E11960.

Guillemain, M., Pradel, R., Devineau, O., Simon, G. & Gauthier-Clerc, M. (2014) Demographic heterogeneity among individuals can explain the discrepancy between capture-mark-recapture and waterfowl count results. *Condor*, 116, 293–302.

Hairston, N.G. Jr, Ellner, S.P., Geber, M.A., Yoshida, T. & Fox, J.A. (2005) Rapid evolution and the convergence of ecological and evolutionary time. *Ecology Letters*, 8, 1114–1127.

Hamel, S., Côté, S.D., Gaillard, J.M. & Festa-Bianchet, M. (2009a) Individual variation in reproductive costs of reproduction: high-quality females always do better. *Journal of Animal Ecology*, 78, 143–151.

Hamel, S., Gaillard, J.M., Festa-Bianchet, M. & Côté, S.D. (2009b) Individual quality, early-life conditions, and reproductive success in contrasted populations of large herbivores. *Ecology*, 90, 1981–1995.

Hamel, S., Gaillard, J.M. & Yoccoz, N.G. (2018) Introduction to: individual heterogeneity – the causes and consequences of a fundamental biological process. *Oikos*, 127, 643–647.

Hamel, S., Yoccoz, N.G. & Gaillard, J.M. (2017) Assessing variation in life-history tactics within a population using mixture regression

models: a practical guide for evolutionary ecologists. *Biological Reviews*, 92, 754–775.

Hart, S.P., Schreiber, S.J. & Levine, J.M. (2016) How variation between individuals affects species coexistence. *Ecology Letters*, 19, 825–838.

Hartemink, N. & Caswell, H. (2018) Variance in animal longevity: contributions of heterogeneity and stochasticity. *Population Ecology*, 60, 89–99.

Heino, J., Melo, A.S. & Bini, L.M. (2015) Reconceptualising the beta diversity-environmental heterogeneity relationship in running water systems. *Freshwater Biology*, 60, 223–235.

Hesse, E., Rees, M. & Müller-Schärer, H. (2008) Life-history variation in contrasting habitats: flowering decisions in a clonal perennial herb (*Veratrum album*). *American Naturalist*, 172, E196–E213.

Hill, G.E. (2011) Condition-dependent traits as signals of the functionality of vital cellular processes. *Ecology Letters*, 14, 625–634.

Janeiro, M.J., Coltman, D.W., Festa-Bianchet, M., Pelletier, F. & Morrissey, M.B. (2017) Towards robust evolutionary inference with integral projection models. *Journal of Evolutionary Biology*, 30, 270–288.

Jansen, M., Zuidema, P.A., Anten, N.P.R. & Martínez-Ramos, M. (2012) Strong persistent growth differences govern individual performance and population dynamics in a tropical forest understorey palm. *Journal of Ecology*, 100, 1224–1232.

Jenouvrier, S., Aubry, L.M., Barbraud, C., Weimerskirch, H. & Caswell, H. (2018) Interacting effects of unobserved heterogeneity and individual stochasticity in the life history of the southern fulmar. *Journal of Animal Ecology*, 87, 212–222.

Jenouvrier, S., Aubry, L., van Daalen, S., Barbraud, C., Weimerskirch, H. & Caswell, H. (2019) When the going gets tough, the tough get going: effect of extreme climate on an Antarctic seabird's life history. *bioRxiv*, 791855. https://doi.org/10.1101/791855.

Jolles, J.W., King, A.J. & Killen, S.S. (2020) The role of individual heterogeneity in collective animal behaviour. *Trends in Ecology & Evolution*, 35, 278–291.

Jouvet, L., Rodríguez-Rojas, A. & Steiner, U.K. (2018) Demographic variability and heterogeneity among individuals within and among clonal bacteria strains. *Oikos*, 127, 728–737.

Kendall, B.E. & Fox, G.A. (2003) Unstructured individual variation and demographic stochasticity. *Conservation Biology*, 17, 1170–1172.

Kendall, B.E., Fox, G.A., Fujiwara, M. & Nogeire, T.M. (2011) Demographic heterogeneity, cohort selection, and population growth. *Ecology*, 92, 1985–1993.

Kendall, B.E., Fujiwara, M., Diaz-Lopez, J., Schneider, S., Voigt, J. & Wiesner, S. (2019) Persistent problems in the construction of matrix population models. *Ecological Modelling*, 406, 33–43.

Knight, T.M., Barfield, M. & Holt, R.D. (2008) Evolutionary dynamics as a component of stage-structured matrix models: an example using *Trillium grandiflorum*. *American Naturalist*, 172, 375–392.

Kohyama, T.S., Kohyama, T.I. & Sheil, D. (2018) Definition and estimation of vital rates from repeated censuses: choices, comparisons and bias corrections focusing on trees. *Methods in Ecology and Evolution*, 9, 809–821.

Krieger, M.S., Denison, C.E., Anderson, T.L., Nowak, M.A. & Hill, A.L. (2020) Population structure across scales facilitates coexistence and spatial heterogeneity of antibiotic-resistant infections. *PLoS Computational Biology*, 16, e1008010.

Kruuk, L.E.B. (2004) Estimating genetic parameters in natural populations using the 'animal model'. *Philosophical Transactions of the Royal Society of London. Series B: Biological Sciences*, 359, 873–890.

Lande, R. (1982) A quantitative genetic theory of life history evolution. *Ecology*, 63, 607–615.

Lefkovitch, L.P. (1965) The study of population growth in organisms grouped by stages. *Biometrics*, 21, 1–18.

Lemaître, J.-F., Berger, V., Bonenfant, C., Douhard, M., Gamelon, M., Plard, F. et al. (2015) Early-late life trade-offs and the evolution of ageing in the wild. *Proceedings of the Royal Society B*, 282, 20150209.

Lemaître, J.-F., Cheynel, L., Douhard, F., Bourgoin, G., Débias, F., Ferté, H. et al. (2018) The influence of early-life allocation to antlers on male performance during adulthood: Evidence from contrasted populations of a large herbivore. *Journal of Animal Ecology*, 87, 921–932.

Leslie, P.H. (1945) On the use of matrices in certain population mathematics. *Biometrika*, 33, 183–212.

Letcher, B.H., Coombs, J.A. & Nislow, K.H. (2011) Maintenance of phenotypic variation: repeatability, heritability and size-dependent processes in a wild brook trout population. *Evolutionary Applications*, 4, 602–615.

Lewis, E.G. (1942) On the generation and growth of a population. *Sankhya*, 6, 93–96.

Lloyd, K.J., Oosthuizen, W.C., Fay, R., Bester, M.N. & de Bruyn, P.J.N. (2020) Selective disappearance of frail juveniles: consequences for understanding social dominance in adult male elephant seals. *Oikos*, 129, 1566–1578.

McGuigan, K. & Blows, M.W. (2009) Asymmetry of genetic variation in fitness-related traits: apparent stabilizing selection on g_{max}. *Evolution*, 63, 2838–2847.

McLean, E.M., Archie, E.A. & Alberts, S.C. (2019) Lifetime fitness in wild female baboons: trade-offs and individual heterogeneity in quality. *American Naturalist*, 194, 745–759.

McNamara, J.M. & Houston, A.I. (1996) State-dependent life histories. *Nature*, 380, 215–221.

Melbourne, B.A. & Hastings, A. (2008) Extinction risk depends strongly on factors contributing to stochasticity. *Nature*, 454, 100–103.

Moyes, K., Morgan, B., Morris, A., Morris, S., Clutton-Brock, T. & Coulson, T. (2011) Individual differences in reproductive costs examined using multi-state methods. *Journal of Animal Ecology*, 80, 456–465.

Nepoux, V., Babin, A., Haag, C., Kawecki, T.J. & Le Rouzic, A. (2015) Quantitative genetics of learning ability and resistance to stress in *Drosophila melanogaster*. *Ecology and Evolution*, 5, 543–556.

Noonburg, E.G., Chen, A., Shima, J.S. & Swearer, S.E. (2015) Demographic heterogeneity and the dynamics of open populations. *Ecology*, 96, 1159–1165.

Ofstad, E.G., Markussen, S.S., Sæther, B.-E., Solberg, E.J., Heim, M., Haanes, H. et al. (2020) Opposing fitness consequences of habitat use in a harvested moose population. *Journal of Animal Ecology*, 89, 1701–1710.

Okuyama, T. (2019) Census timing alters stage duration distributions in matrix population models. *Ecology and Evolution*, 9, 8500–8508.

Olijnyk, A.M. & Nelson, W.A. (2013) Positive phenotypic correlations among life-history traits remain in the absence of differential resource ingestion. *Functional Ecology*, 27, 165–172.

Oosthuizen, W.C., Postma, M., Altwegg, R., Nevoux, M., Pradel, R., Bester, M.N. et al. (2019) Individual heterogeneity in life-history trade-offs with age at first reproduction in capital breeding elephant seals. *Population Ecology*, 61, 421–435.

Orzack, S.H., Steiner, U.K., Tuljapurkar, S. & Thompson, P. (2011) Static and dynamic expression of life history traits in the northern fulmar *Fulmarus glacialis*. *Oikos*, 120, 369–380.

Paoli, A., Weladji, R.B., Holand, Ø. & Kumpula, J. (2020) Early-life conditions determine the between-individual heterogeneity in plasticity of calving date in reindeer. *Journal of Animal Ecology*, 89, 370–383.

Paterson, J.T., Rotella, J.J., Link, W.A. & Garrott, R. (2018) Variation in the vital rates of an Antarctic marine predator: the role of individual heterogeneity. *Ecology*, 99, 2385–2396.

Plard, F., Bonenfant, C., Delorme, D. & Gaillard, J.M. (2012) Modeling reproductive trajectories of roe deer females: fixed or dynamic heterogeneity? *Theoretical Population Biology*, 82, 317–328.
Plard, F., Fay, R., Kéry, M., Cohas, A. & Schaub, M. (2019) Integrated population models: powerful methods to embed individual processes in population dynamics models. *Ecology*, 100, e02715.
Plard, F., Gaillard, J.M., Coulson, T. & Tuljapurkar, S. (2016) Des differences, pourquoi? Transmission, maintenance and effects of phenotypic variance. *Journal of Animal Ecology*, 85, 356–370.
Plard, F., Schindler, S., Arlettaz, R. & Schaub, M. (2018) Sex-specific heterogeneity in fixed morphological traits influences individual fitness in a monogamous bird population. *American Naturalist*, 191, 106–119.
Rees, M. & Ellner, S.P. (2016) Evolving integral projection models: evolutionary demography meets eco-evolutionary dynamics. *Methods in Ecology and Evolution*, 7, 157–170.
Rees, M. & Ellner, S.P. (2019) Why so variable: can genetic variance in flowering thresholds be maintained by fluctuating selection? *American Naturalist*, 194, E13–E29.
Ronget, V., Garratt, M., Lemaître, J.F. & Gaillard, J.M. (2017) The 'evo-demo' implications of condition-dependent mortality. *Trends in Ecology & Evolution*, 32, 909–921.
Rowe, L. & Houle, D. (1996) The lek paradox and the capture of genetic variance by condition dependent traits. *Proceedings of the Royal Society of London, Series B: Biological Sciences*, 263, 1415–1421.
Russell, T.L., Lwetoijera, D.W., Knols, B.G.J., Takken, W., Killeen, G.F. & Ferguson, H.M. (2011) Linking individual phenotype to density-dependent population growth: the influence of body size on the population dynamics of malaria vectors. *Proceedings of the Royal Society B*, 278, 3142–3151.
Sæther, B.E. & Engen, S. (2015) The concept of fitness in fluctuating environments. *Trends in Ecology & Evolution*, 30, 273–281.
Saito, N., Ishihara, S. & Kaneko, K. (2013) Baldwin effect under multipeaked fitness landscapes: phenotypic fluctuation accelerates evolutionary rate. *Physical Review E*, 87, 052701.
Schindler, D.E., Hilborn, R., Chasco, B., Boatright, C.P., Quinn, T.P., Rogers, L.A. et al. (2010) Population diversity and the portfolio effect in an exploited species. *Nature*, 465, 609–612.
Singh, B.K., Rao, J.S., Ramaswamy, R. & Sinha, S. (2004) The role of heterogeneity on the spatiotemporal dynamics of host-parasite metapopulation. *Ecological Modelling*, 180, 435–443.
Snyder, R.E. & Ellner, S.P. (2018) Pluck or luck: does trait variation or chance drive variation in lifetime reproductive success? *American Naturalist*, 191, E90–E107.
Stein, A., Gerstner, K. & Kreft, H. (2014) Environmental heterogeneity as a universal driver of species richness across taxa, biomes and spatial scales. *Ecology Letters*, 17, 866–880.
Steiner, U.K. & Tuljapurkar, S. (2012) Neutral theory for life histories and individual variability in fitness components. *Proceedings of the National Academy of Sciences of the United States of America*, 109, 4684–4689.
Steiner, U.K., Tuljapurkar, S. & Orzack, S.H. (2010) Dynamic heterogeneity and life history variability in the kittiwake. *Journal of Animal Ecology*, 79, 436–444.
Stover, J.P., Kendall, B.E. & Fox, G.A. (2012) Demographic heterogeneity impacts density-dependent population dynamics. *Theoretical Ecology*, 5, 297–309.
Tompkins, E.M. & Anderson, D.J. (2019) Sex-specific patterns of senescence in Nazca boobies linked to mating system. *Journal of Animal Ecology*, 88, 986–1000.
Tuljapurkar, S. & Steiner, U.K. (2010) Dynamic heterogeneity and life histories. *Annals of the New York Academy of Sciences*, 1204, 65–72.
Tuljapurkar, S., Steiner, U.K. & Orzack, S.H. (2009) Dynamic heterogeneity in life histories. *Ecology Letters*, 12, 93–106.
Tuljapurkar, S., Zuo, W., Coulson, T., Horvitz, C. & Gaillard, J.M. (2020) Skewed distributions of lifetime reproductive success: beyond mean and variance. *Ecology Letters*, 23, 748–756.
van Daalen, S. & Caswell, H. (2020) Variance as a life history outcome: sensitivity analysis of the contributions of stochasticity and heterogeneity. *Ecological Modelling*, 417, 108856.
van de Pol, M., Bruinzeel, L.W., Heg, D., van der Jeugd, H. & Verhulst, S. (2006) A silver spoon for a golden future: long-term effects of natal origin on fitness prospects of oystercatchers (*Haematopus ostralegus*). *Journal of Animal Ecology*, 75, 616–626.
van de Pol, M. & Verhulst, S. (2006) Age-dependent traits: a new statistical model to separate within- and between-individual effects. *American Naturalist*, 167, 766–773.
Vedder, O. & Bouwhuis, S. (2018) Heterogeneity in individual quality in birds: overall patterns and insights from a study on common terns. *Oikos*, 127, 719–727.
Vindenes, Y., Engen, S. & Sæther, B.E. (2008) Individual heterogeneity in vital parameters and demographic stochasticity. *American Naturalist*, 171, 455–467.
Vindenes, Y. & Langangen, Ø. (2015) Individual heterogeneity in life histories and eco-evolutionary dynamics. *Ecology Letters*, 18, 417–432.
Vindenes, Y., Sæther, B.E. & Engen, S. (2012) Effects of demographic structure on key properties of stochastic density-independent population dynamics. *Theoretical Population Biology*, 82, 253–263.
Waddle, E., Piedrahita, L.R., Hall, E.S., Kendziorski, G., Morris, W.F., Peterson, M.L. et al. (2019) Asynchrony in individual and subpopulation fecundity stabilizes reproductive output of an alpine plant population. *Ecology*, 100, e02639.
Wilson, A.J. & Nussey, D.H. (2010) What is individual quality? An evolutionary perspective. *Trends in Ecology & Evolution*, 25, 207–214.
Wilson, A.J., Réale, D., Clements, M.N., Morrissey, M.M., Postma, E., Walling, C.A. et al. (2010) An ecologist's guide to the animal model. *Journal of Animal Ecology*, 79, 13–26.
Zuidema, P.A., Brienen, R.J.W., During, H.J. & Güneralp, B. (2009) Do persistently fast-growing juveniles contribute disproportionately to population growth? A new analysis tool for matrix models and its application to rainforest trees. *American Naturalist*, 174, 709–719.

SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

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