Population and Life-History Consequences of Within-Cohort Individual Variation

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Introduction

Differences in age or stage are important sources of heterogeneity among individuals that are captured by structured population models (Caswell 2001). However, significant demographic variation can also be found within the same stage or age class (Huss et al. 2007). Heterogeneity among individuals may be caused by diverse factors, including differences in size or quality at birth or variation in resource abundance or climatic conditions (McNamara and Houston 1996). At the same time, some of these factors, for example, resource abundance, are affected by population size and structure. For instance, in organisms with food-dependent growth rates, when the population is dense, food levels decrease, thereby reducing individual growth rates. Population dynamics may hence determine which life histories are realized via changes in the environment. In turn, variation in individual life histories influences population dynamics (Benton et al. 2006). Thus, there is a feedback between life histories and population dynamics that is mediated by the environment.

Ecologists have long been interested in understanding how variation among individuals influences population dynamics and community ecology (recently reviewed by Bolnick et al. [2011]). A first set of studies has focused on the one-way influence of individual variation on population dynamics and, in particular, on the questions of whether variation stabilizes or destabilizes dynamics (e.g., Lomnicki 1978; Ebenman and Persson 1988; Grimm and Uchmanski 2002; Filin and Ovadia 2007) and how variation affects the risk of extinction (Kendall and Fox 2002; Vindenes et al. 2008). These questions have been explored with diverse population models (unstructured, structured, and individual based) and assumptions of various forms of variation among individuals, including differences in social rank, body size, or vital rates. A main conclusion of past analyses is that the relationship between individual variation and population stability is complex and usually nonmonotonic. For example, among-cohort variation can
have a stabilizing effect when population dynamics are
inherently unstable, but the effect is destabilizing for in-
herently stable dynamics (Lindström and Kokko 2002).
Likewise, individual variation in body size in grasshoppers
appears to destabilize dynamics under a deterministic,
constant environment, whereas the effect is stabilizing for
a changing, stochastic environment (Filin and Ovadia
2007).

Another important conclusion is that variation in vital
rates can lead to higher or lower extinction risks, de-
pending on the mean-variance relationship in vital rates
(Kendall and Fox 2002). Similarly, Vindenes et al. (2008)
found that demographic stochasticity may increase, de-
crease, or remain unaltered, depending on the specific
vital-rate values explored and how these rates vary over
time among individuals. Altogether, these studies have
shown that there is no unique effect of individual variation
in population dynamics; instead, the predicted effects de-
pend on how variation is modeled. Therefore, models that
generate variation in vital rates or life history without con-
sidering the actual mechanisms generating this variation
likely provide limited insight into how variation affects
natural populations. Instead, biologically relevant conclu-
sions require models that consider and explicitly include
the mechanisms that give rise to individual variation.

Variation among individuals not only affects population
stability but also can influence demographic traits such as
growth, survival, and fecundity rates via feedbacks from
population dynamics to individual life history. A second
set of studies of individual variation, based on physiolog-
ically structured population models (hereafter PSPMs), ac-
knowledges explicitly that the relationship between indivi-
dual life history and population dynamics is intertwined
(Metz and Diekmann 1986). PSPMs show that realized life
histories and population dynamics are emergent properties
of individual-level processes involving energy acquisition
and allocation, social interactions, and so on. The main
implications emerging from this work are that (1) the
feedback between life history and population dynamics is
likely to result in population cycles referred to as genera-
tion or cohort cycles (e.g., de Roos et al. 1992), (2) size-
dependent trophic interactions influence the types of dy-
namics observed (e.g., Claessen et al. 2000), (3) realized
life histories depend strongly on population dynamics
(e.g., Claessen et al. 2000; de Roos et al. 2002), and (4)
food-dependent growth may induce bistability and poten-
tially a catastrophic population collapse (e.g., Persson
et al. 2007). Traditional PSPMs account for variation among
cohorts caused by environmental conditions experienced
during life and plasticity in realized life histories (e.g.,
food-dependent growth) but ignore within-cohort varia-
tion. One exception is a study that demonstrates that sto-
castic within-cohort variability may dampen the ampli-
tude of single generation cycles or lead to small-amplitude,
stochastic fluctuations (van Kooten et al. 2007). Except
for this study, the population-dynamic consequences of
the life-history–population feedback in the context of
within-cohort variation remain largely unexplored.

Our study aims to address this gap in our knowledge
by exploring the question of how explicit consideration of
mechanisms that generate within-cohort variability affects
predicted population dynamics and life history while ac-
counting for feedbacks. We use a detailed, predominantly
mechanistic framework inspired by empirical understand-
ing and knowledge about what causes individual variation
in natural populations. In particular, we develop a PSPM
that accounts for feedback of the population on life history,
keeps tracks of each individual explicitly, and incorporates
stochastic variation in individual fates (see also de Roos
et al. 2009). Introducing within-cohort individual varia-
tion, we relax a common and strong assumption of the
PSPM framework and obtain a model that is basically an
explicit individual-based model (IBM). A common dif-
fERENCE between studies using PSPMs and those using
IBMs is that the former focus on long-term population
dynamics and feedback, whereas the latter usually focus
on a one-way effect of individual variability on population
dynamics. Here, we try to unify these two approaches,
making use of concepts and tools developed in the context
of PSPM theory to analyze the interaction between indi-
vidual variation and population feedback. Our model is
based on the European common lizard Zootoca vivipara,
for which there is strong evidence of phenotypic plasticity
in growth rates, survival, and reproduction (Sorci et al.
1996; Lorenzon et al. 2001; Le Galliard et al. 2010).

We first present a deterministic model without individ-
ual variation and explore general model behavior under
the assumption of a constant environment. Second, we
develop four stochastic model versions aimed at unrav-
eling the effects that two distinct mechanisms generating
variation among individuals have on population dynamics
and realized individual life histories. Important factors
leading to individual differences include heritable life-his-
tory strategies (i.e., foraging or mating strategies), genetic
variation in birth size or body condition, the consequences
of differences in time of birth, and variation in climatic
conditions, food availability, or habitat quality (reviewed
by Le Galliard et al. 2010 for the common lizard). Among
these, we consider stochastic variation in date of birth,
which exposes different individuals to distinct environ-
mental conditions during early life history (potentially a-
fecting development) and generates a range of coexisting
ages in each year class. In addition, we explore stochastic,
among-individual variation of food availability as the most
important and direct determinant of variability in indi-
vidual resource acquisition and thus a potentially critical
factor leading to differences in individual growth and reproduction. The two mechanisms considered here, variation in birth date and variation in food availability, are major sources of individual variation in natural populations of reptiles and other species (Madsen and Shine 2000; Sh t and Olsson 2003; Reznick et al. 2006; Sargeant et al. 2007). We do not explore the role of heritable variation, although it is likely important, because mechanistically including heritable sources of variation requires an understanding of quantitative genetics that is not currently available for our model species. Finally, it is important to emphasize that although our model is species specific, our approach to modeling mechanisms of individual variation is very general. Therefore, our modeling exercise is likely relevant for a wide range of species.

Model Formulation and Parameterization

PSPMs explicitly link individual-level processes (growth, reproduction, mortality) and population dynamics (de Roos 1997). The life history of an individual is represented by a dynamic energy budget model that describes the acquisition of energy and its allocation to maintenance, growth, and reproduction, depending on individual physiological traits, or i-states, and the current state of the environment. Population functions, such as the total population biomass and the population size distribution, are derived by bookkeeping individual contributions, including birth, growth, and death. The mechanistic approach of PSMPs allows a unique understanding of causal relationships between individual-level processes and the resulting population dynamics. In addition, the PSPM framework allows a close connection between model and empirical system: the main model ingredients pertain to the individual level and can be parameterized with experimental data, while outputs, such as population dynamics and realized life history, are observable in the field.

Our PSPM of the common lizard is similar to earlier models of freshwater fish (Persson et al. 1998) and ungulates (de Roos et al. 2009) in its definition of the state of individuals (structural vs. reserves body mass), the use of a simple energy budget model to compute individual growth, and the specification of size-dependent functions for metabolic, food intake, and survival rates. Unique aspects, inspired by the biology of our model species, pertain to assumptions about energy-allocation rules, density dependence, the influence of weather conditions, and the sources of within-cohort variation among individuals (in the stochastic versions). These novelties are presented below, whereas a complete model description can be found in appendix A in the online edition of the American Naturalist.

Model Species

Zootoca vivipara is a small (newborn to adult snout-vent length: 20–70 mm), diurnal, nonterritorial Lacertidae species found in humid habitats across northern Eurasia. This lizard is an active forager that preys on a diversity of invertebrate species (Avery 1966). Populations may be oviparous or ovoviviparous, but we parameterize and validate our model with data from the latter (table 1). Natural populations can be divided into three main age classes: juveniles (<1 year old), yearlings (1–2 years old), and adults (>2 years old). Zootoca vivipara hibernate from September–October to March–April, exhibiting little winter activity and very low winter mortality (Bauwens 1981). Therefore, we explicitly model only the active season, ~200 days per year. Mating occurs soon after emergence from hibernation, but females retain the eggs in the abdominal cavity until embryonic development is completed. Females lay an average of five (range 1–12) noncalcified eggs from early June to early July, and eggs generally hatch within minutes of laying. Additional information on the species’ life history can be found in Massot et al. (1992) and references therein. We model only the female portion of the population and assume that individuals interact only by competition for a food resource.

Model Outline

The state of individuals is defined by three i-state variables: age, structural mass (i.e., bone, organs), and reserves mass (i.e., adipose and reproductive tissues). We assume that energy acquisition, growth, survival, and reproduction are functions of body mass defined by an energy budget model (app. A). Food intake and metabolism also depend on environmental conditions, that is, sunshine duration, to reflect the importance of weather on lizard life history (Adolph and Porter 1993). Food intake is also a function of a density-dependent scaling function D(B) that provides feedback from population dynamics to the individual process of food consumption. Whereas PSPMs usually model the dynamics of the resource population explicitly, we cannot accurately model prey dynamics because the common lizard feeds on a large variety of prey and its functional response is not well understood (Avery 1966; González-Suárez et al. 2011). In the absence of enough empirical data to adequately define the consumer-resource interaction in this species, we model density dependence in a phenomenological way, using a simple function D(B) that reflects our general knowledge of the species’ feeding biology. An individual’s feeding rate is obtained by multiplying its empirical, size-dependent feeding rate under standard conditions (see González-Suárez et al. 2011) by the function D(B), which is a decreasing function of the
Table 1: Parameters of a physiologically structured population model of the lizard *Zootoca vivipara*

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Value</th>
<th>Units</th>
<th>Definition</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>(\lambda_1)</td>
<td>61.693</td>
<td>mm ((g^{\lambda_1})^{-1})</td>
<td>Allometric scalar</td>
<td>Unpublished data 1</td>
</tr>
<tr>
<td>(\lambda_2)</td>
<td>0.303</td>
<td>...</td>
<td>Allometric exponent</td>
<td>Unpublished data 1</td>
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<tr>
<td>(W)</td>
<td>2.941</td>
<td>...</td>
<td>Dry to wet mass conversion</td>
<td>Avery 1971</td>
</tr>
<tr>
<td>Food intake:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(\gamma_i)</td>
<td>34.449</td>
<td>mg (g^{-3}) (day^{-1})</td>
<td>Food intake scalar</td>
<td>Unpublished data 2</td>
</tr>
<tr>
<td>(\gamma_s)</td>
<td>0.69</td>
<td>...</td>
<td>Food intake exponent</td>
<td>Unpublished data 2</td>
</tr>
<tr>
<td>(\text{sun})</td>
<td>4(^a)</td>
<td>h (day^{-1})</td>
<td>Hours of sunshine per day</td>
<td>Standard conditions</td>
</tr>
<tr>
<td>Density dependence:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(B_0)</td>
<td>1,500(^b)</td>
<td>g</td>
<td>Population biomass resulting in observed food intake rates</td>
<td>Free parameter</td>
</tr>
<tr>
<td>(\delta)</td>
<td>1(^b)</td>
<td>...</td>
<td>Density-dependent function coefficient</td>
<td>Free parameter</td>
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<tr>
<td>Ontogeny:</td>
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<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(x_{mat})</td>
<td>0.569</td>
<td>g</td>
<td>Structural dry mass at maturation</td>
<td>Unpublished data 1</td>
</tr>
<tr>
<td>(M_0)</td>
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<td>g</td>
<td>Body mass at birth</td>
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<td>(\Omega)</td>
<td>0.4</td>
<td>...</td>
<td>Reproduction costs</td>
<td>Avery 1975; Massot et al. 1992</td>
</tr>
<tr>
<td>(q_{ni})</td>
<td>0.197</td>
<td>...</td>
<td>Neonate body condition</td>
<td>Nagy 1983</td>
</tr>
<tr>
<td>(q_{na})</td>
<td>0.205</td>
<td>...</td>
<td>Body condition after reproduction</td>
<td>Nagy 1983</td>
</tr>
<tr>
<td>(q_s)</td>
<td>0.084</td>
<td>...</td>
<td>Starvation body condition</td>
<td>Nagy 1983</td>
</tr>
<tr>
<td>Metabolism and growth:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(\tau)</td>
<td>0.0226</td>
<td>kJ (mg^{-1})</td>
<td>Prey mass conversion factor</td>
<td>Avery 1971</td>
</tr>
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<td>(\nu)</td>
<td>0.82</td>
<td>...</td>
<td>Assimilation efficiency coefficient</td>
<td>Avery 1975</td>
</tr>
<tr>
<td>(\sigma_a)</td>
<td>1.0</td>
<td>kJ (day^{-1})</td>
<td>Basal activity scope</td>
<td>Grant and Porter 1992</td>
</tr>
<tr>
<td>(M_{sun})</td>
<td>8(^b)</td>
<td>h (day^{-2})</td>
<td>Maximum number of hours of sunshine per day</td>
<td>Standard conditions</td>
</tr>
<tr>
<td>(\rho_i)</td>
<td>0.23</td>
<td>kJ (day^{-1} g^{-2})</td>
<td>Maintenance scalar</td>
<td>Cragg 1978</td>
</tr>
<tr>
<td>(\rho_j)</td>
<td>1.02</td>
<td>...</td>
<td>Maintenance exponent</td>
<td>Cragg 1978</td>
</tr>
<tr>
<td>(k_i)</td>
<td>0.42(^b)</td>
<td>...</td>
<td>Energy allocation rule for immature animals (before first reproduction)</td>
<td>Free parameter</td>
</tr>
<tr>
<td>(k_a)</td>
<td>0.74(^b)</td>
<td>...</td>
<td>Energy allocation rule for adults</td>
<td>Free parameter</td>
</tr>
<tr>
<td>(\Phi)</td>
<td>0.0286</td>
<td>g kJ(^{-1})</td>
<td>Energy-to-structural-mass conversion coefficient (with costs of synthesis)</td>
<td>Avery 1971; Peterson et al. 1999</td>
</tr>
<tr>
<td>(\Psi)</td>
<td>0.0210</td>
<td>g kJ(^{-1})</td>
<td>Energy-to-reserves-mass conversion coefficient (with cost of synthesis)</td>
<td>Peterson et al. 1999; Kooijman 2000</td>
</tr>
<tr>
<td>Mortality:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>(\mu_s)</td>
<td>0.0025</td>
<td>day(^{-1})</td>
<td>Background mortality rate</td>
<td>Unpublished data 3</td>
</tr>
<tr>
<td>(\alpha)</td>
<td>6.2</td>
<td>day(^{-1})</td>
<td>Length-dependent mortality scalar</td>
<td>Unpublished data 3</td>
</tr>
<tr>
<td>(\beta)</td>
<td>0.51</td>
<td>mm(^{-1})</td>
<td>Length-dependent mortality exponent</td>
<td>Unpublished data 3</td>
</tr>
<tr>
<td>(L_{min})</td>
<td>15</td>
<td>mm</td>
<td>Minimum body length size for survival</td>
<td>Unpublished data 3</td>
</tr>
</tbody>
</table>

Note: Values derive from a literature survey or unpublished data sets or cannot be parameterized a priori ("free parameter"). See "Model Parameterization." All mass is given as dry mass.


\(^b\) Default values defined for free parameters or standard conditions.

Understanding how assimilated energy is actually channeled in an organism is complicated, and numerous energy allocation rules have been proposed (Kooijman 2000; Claessen et al. 2009). We assume that individuals follow a “net allocation model” (Kooijman 2000) before first reproduction and a “gross-production allocation model” (Kooijman 2000) after the first reproduction event (see app. A for details). These two allocation models reflect observed differences in prioritization between reproductive lizards, population’s weighted abundance \(B\) (app. A). Although the exact mechanisms by which increased abundance affects individual food intake, and hence body growth, remain unknown, \(D(B)\) appears to be a reasonable representation that largely captures the observed density-dependent response in body growth of *Z. vivipara* (app. B in the online edition of the *American Naturalist*).
which prioritize reproduction, and nonreproductive individuals, which prioritize structural growth (Andrews 1982).

**Model Implementation**

First, we describe a deterministic PSPM (Det) that follows cohorts of identical individuals over their life span. Cohorts may differ because of differences in the environmental conditions they encounter, but individuals within a cohort remain identical. We also present four individual-based stochastic versions of Det in which individuals, instead of cohorts, are followed. The first stochastic version (referred to as the Dis model) is a simple discretization of the Det model: each individual is represented explicitly, and its death and reproduction are modeled as discrete, stochastic events. Unless otherwise stated, all model assumptions and functions for Dis (and the other stochastic model versions) are as described in appendix A. For the Dis model, the time of death is determined through the expected-survival curve, computed for each individual with its time-varying total mortality rate (see app. A). The expected number of offspring depends on reserves mass, according to equation (A13) in the online edition of the American Naturalist. However, equation (A13) calculates real numbers that must be rounded into integers for use in the individual-based model versions. The simplest approach would be to round to the nearest integer, but this could introduce a bias. Therefore, in order to make sure that the expected fecundity exactly equals the fecundity based on the bioenergetic allocation rules (eq. [A13]; app. A), we round fecundity by using a simple probabilistic rule. If a female has, for example, an expected fecundity of 4.3, she produces either 5 offspring with probability 0.3 or 4 offspring with probability 0.7. The energetic investment of the mother is still defined as 4.3 in order to maintain a fully equivalent energy budget model, as in the Det model. As an artifact, this approach introduces limited stochasticity in the number of offspring per female. We have verified, however, that the overall distribution of offspring numbers per female predicted by the model generally captures the empirically observed distribution (app. C in the online edition of the American Naturalist). Note that in our models, the distribution of the number of offspring per female is a model prediction, not an a priori model assumption. Variability in the number of offspring, at a given time, results from the within-population variability in terms of age, size, and body condition of females and hence, indirectly, from their feeding history as well as from the (past) dynamics of the population through density-dependent feeding.

After discretization of the model, we introduce further modifications to explore two sources of within-cohort individual variation, first separately and then in combination. These additional stochastic model versions follow the same discretization approach as Dis. The Birth model modifies the Dis model to include a birthing period of 30 days instead of a single, population-wide birth pulse. The expected birth date for each female is drawn from a normal distribution with a mean equal to half the birthing-period length and a variance of half the mean, but truncated to span only the birthing period. The birthing day for each mother is assigned randomly each year to reflect variation in time of parturition of natural populations (J.-F. Le Galliard, unpublished data). Mature individuals with insufficient reserves to breed on their assigned date are given a second opportunity to reproduce on the last day of the breeding season. However, for the parameter values explored, more than 99.9% of the females that reproduce give birth on the originally assigned date. Results do not qualitatively change when different birthing-period lengths are considered (10–90 days).

The Food model modifies the Dis model by introducing temporal variation in prey availability for each lizard. For each individual separately, a stochastic food consumption factor is drawn daily from a normal distribution with a mean of 1 and a standard deviation reflecting observed variability in consumption rates of a lizard population (SD = 0.195; M. Gonzalez-Suarez, unpublished data). Results do not generally change when the stochastic food consumption factor is drawn at longer intervals (2–15 days). A final model (Fe-B) combines both sources of individual variability.

We use a numerical integration method known as the Escalator Boxcar Train (EBT; de Roos et al. 1992), implemented in the EBT software, to explore all these model versions. A general description of the EBT approach and the complete code files necessary to run the deterministic and stochastic model versions with the EBT software are available in appendix D in the online edition of the American Naturalist. We simulate population dynamics under varying initial conditions that have no effect on the stable dynamics. The code files in appendix D include the set of initial conditions used in most analyses. For each stochastic model version, we report results from 10 different simulations of population dynamics over a 300-year period. To discard transient dynamics, population statistics are calculated over the last 200 years. Additional details of the simulation methods are provided in appendix D.

Below, age is expressed in years such that 0+ represents the first year of life, 1+ the second year, and so on. For comparison with empirical estimates based on three age classes, we also present results grouped by ages 0+ (juveniles), 1+ (yearlings), and >1+ (adults). The term “mature individuals” refers to those with structural mass $x > x_{\text{mat}}$ (table 1), while “reproductive individuals” are those that have reproduced at least once.
Model Parameterization

Most parameters are defined on the basis of data for *Z. vivipara* from published sources (table 1) or a large unpublished data set (J.-F. Le Galliard, M. Massot, and J. Clobert, unpublished data) based on >1,200 *Z. vivipara* individuals from an experimental population at Centre de Recherche en Écologie Expérimentale et Prédective (CEREEP), France. We use the unpublished data set to estimate the length-to-mass relationship, fecundity rates, and body size at birth and at maturation and, in conjunction with published estimates of annual survival rates (Massot et al. 1992; Le Galliard et al. 2010), to define size-dependent daily mortality rates. The feeding rate is defined with data from a seminatural experiment at CEREEP based on >100 *Z. vivipara* (González-Suárez et al. 2011; M. González-Suárez, unpublished data). Standard conditions of sunshine duration at CEREEP during the lizard active season are used to define the climate-related parameters sun and Msun. When data from the common lizard are not available, we use information from related species (table 1). We use observations from the garter snake *Thamnophis sirtalis fitchi* to estimate the energetic costs of reserves- and structural-mass growth (Peterson et al. 1999). Data from the lizard *Uta stansburiana* is used to estimate condition thresholds (\(q_0\), \(q_1\), and \(q_2\)), defined as ratios of dry lipid and reproductive body mass over remaining dry mass (bones, organs, etc.) for different age classes and body conditions (Nagy 1983, pp. 32–33). These body condition thresholds are consistent with rough estimates calculated for *Z. vivipara* on the basis of dry lipid contents for adults (Avery 1970).

Data are not available to define some parameters (“free parameters” in table 1), and so we define their default values as follows. At equilibrium, the density-dependence parameters \(B\) and \(\delta\) determine population size. Their default values are chosen so that size at equilibrium is approximately 100–150 individuals. Growth allocation rule parameters \(k_p\) and \(k_s\) are defined to broadly fit empirical patterns of individual growth, fecundity, and age of maturation from CEREEP (J.-F. Le Galliard, unpublished data). We explore the sensitivity of model predictions to changes in the default values of these free parameters. In addition, because observation error is possible, we also explore the sensitivity of the deterministic model predictions to changes in empirically determined parameter values.

Model Validation

There are difficulties in validating stochastic-model predictions (Waller et al. 2003; Grimm and Railsback 2005), and Grimm and Railsback (2005) suggest that simple comparisons are often best. We obtain empirical estimates independent of those used for model parameterization to calculate fecundity, survival, and body size. Empirical estimates (\(\text{Emp}\)) are calculated from a long-term data set (\(>11\) years) of detailed individual measures of body size, clutch size, and a recapture series from a natural population in the Cévennes, France (Le Galliard et al. 2010; M. Massot and J.-F. Le Galliard, unpublished data). We estimate fecundity (number of female offspring per female) from total clutch size, assuming a 1:1 sex ratio. Survival estimates are calculated from mark-recapture histories, allowing for interannual variation and heterogeneous capture probabilities. Body size at fixed age is estimated from individuals of known age. These empirical estimates are compared with model predictions to address the question of whether observed data appear consistent with the model. In particular, we assess differences in central tendencies by determining whether empirical mean estimates fall within a narrow confidence interval (mean ± 1 SD) of model predictions. We explore data dispersion, comparing empirical and predicted variances with tests of homogeneity in variances. All unpublished data sets used to parameterize and validate the model are deposited at the Dryad Digital Repository (http://dx.doi.org/10.5061/dryad.jh87h).

Results

Deterministic PSPM

Model dynamics are insensitive to moderate changes in the empirically estimated parameters, and we find no evidence of alternative stable states. Bifurcation analyses reveal that long-term dynamics of our model remain relatively unaffected by changes in most free parameters or those defined by standard conditions (fig. 1B–1D). However, changes in \(k_p\) affect population dynamics noticeably (fig. 1A). The parameter \(k_p\) defines energy allocation to growth before first reproduction and thus determines the age at maturation. Stable fixed-point dynamics occur when all cohorts mature at the same time, while cycles or irregular dynamics are observed when cohorts mature at different ages (figs. 1A, 2). For example, for \(k_p = 0.42\) (the default value), all individuals mature during their second year of life (fig. 2A), but for \(k_p = 0.32\), some cohorts mature at age 1+ and others at age 2+, generating cyclic dynamics with years of higher and lower population sizes (fig. 2B). These 3-year cycles are akin to juvenile-driven cohort cycles (Persson et al. 1998), in that every three years a dense year class of newborns is born that reduces growth and reproduction of the older year classes. For \(k_p > 0.79\), individuals reach the maturation size quickly but body condition remains below the reproductive threshold because growth of reserves mass is very limited. As a result, no reproduction occurs and popula-
Demographic Consequences of Variability

Figure 1: Bifurcation diagrams of the free parameters and sun in the deterministic model. Each panel illustrates the results for a different parameter: A, energy allocation to structural growth in nonreproducing individuals $k_f$; B, energy allocation to structural growth in adults $k_A$; C, strength of density dependence $d$; and D, daily sunshine duration $sun$. Other parameters are set to their default values (see table 1). In A, the arrows indicate stable fixed-point regions in which all cohorts mature at the age indicated. The free parameter $B_0$ scales the population size linearly at equilibrium, and its bifurcation diagram is not shown.

In A, the arrows indicate stable fixed-point regions in which all cohorts mature at the age indicated. The free parameter $B_0$ scales the population size linearly at equilibrium, and its bifurcation diagram is not shown. All mature individuals reproduce every year.

Stochastic Individual-Based PSPM

The dynamic behavior of the stochastic versions of our model does not differ qualitatively from that of the deterministic version. Model runs starting with as few as 10 individuals show similar long-term behavior over 300 years. Results from the discretized model ($Dis$) and the deterministic model ($Det$) are also quantitatively very similar, although population size and mean fecundity are slightly lower in $Dis$ (figs. 3A, 4A). On the other hand, introducing variation in birth date and food intake influences quantitative predictions. Scenarios including individual variation in birth date predict slightly larger median population sizes, while scenarios considering food intake variability predict a median population size lower than that from the $Det$ or $Dis$ model (fig. 3A). Combining both sources of variation ($F&B$), we find a median population size slightly higher than that from the $Food$ model but still lower than that from the $Det$ or $Dis$ model. The age structure of the population on the last day of the season, defined as the relative proportion of individuals in each of the three age classes (0+, 1+, and >1+), is, however, nearly identical in all versions.

Mean fecundity is slightly reduced in all the stochastic model versions, with the lowest mean fecundity for $F&B$ (fig. 4A). Reduced fecundity is accompanied by slightly higher survival rates of individuals of age 0+ (fig. 3B). Because there is no starvation mortality, older individuals
are subject only to background mortality, and their survival rates do not differ among model versions. Growth curves (snout-vent length, SVL) have a similar pattern in all model versions, with rapid growth in early stages of life and decreasing rates for older animals (fig. 4B). However, the mean SVL at fixed ages varies among versions. Older individuals are larger in the Food and F&B models. Variability in SVL among individuals within each age class is small in all versions (SD < 0.54 mm), but differences are more noticeable in models with food variation (Food and F&B) and among older age classes. The increase in mean SVL in older individuals in the Food and F&B models is accompanied by a decrease in body condition, so that these adults are characterized by being longer and skinnier. Variation in birth date alone (Birth) results in differences in SVL between individuals born early in the birthing period (first 10 days) and those born late (last 10 days), with the former being significantly larger at all ages and reaching larger maximum sizes (Student t-tests: $P < .031$). Although significant, the actual differences in size are generally small (<1 mm). Interestingly, variability in food consumption eliminates these differences. There are no differences in SVL between earlier- and later-born individuals in the F&B model (Student t-test: $P > .10$).

Maturation always occurs during the second year of life, but individuals mature slightly earlier in the Food model because of faster growth rates (mean ± SD: Dis, 324 ± 23.5 days; Food, 313 ± 29.3 days; Birth, 331 ± 24.1 days; and F&B, 321 ± 28.8 days; ANOVA: $F = 136.4, P < .001$). All mature individuals reproduce every year in all versions of our model. The Food and F&B models assume that consumption varies stochastically for each individual independently. Interestingly, an alternative model version assuming that food intake varies stochastically but simultaneously for all individuals (i.e., shared environmental stochasticity) predicts the same population-dynamic and life-history patterns, including the long-and-skinny syndrome, but eliminates variability among individuals.

**Model Validation**

Independent empirical observations are generally consistent with model predictions. All model versions predict multiple coexisting cohorts (age classes), as observed in

![Figure 2: Time series of long-term dynamics predicted by the deterministic model for fixed-point dynamics (A; $k_0 = 0.42$) and cyclic dynamics (B; $k_0 = 0.32$). The top panels present the values of the density-dependence factor $D(B)$ (see app. A in the online edition of the *American Naturalist* for detailed definition) that can be interpreted as "resource abundance." The middle panels illustrate cohort abundance per age class, including newborns (<1 day old). The bottom panels present cohort growth curves (solid lines represent cohorts maturing at age 1+ and dot-dashed lines cohorts maturing at age 2+). The reference line in the bottom panel indicates body size at maturation. Other parameters are set to their default values (see table 1).](image)
natural populations (Massot et al. 1992). The predicted age structure (fig. 3A) agrees well with empirical estimates (Massot et al. 1992). Mean empirical estimates of growth curves, fecundity, and survival rates fall within the range (mean ± 1 SD) of predicted estimates, except for a few estimates of SVL (figs. 3, 4). However, interannual variability in survival observed in natural populations is higher than predicted (F-test for homogeneity of variances: \( P < .01 \); fig. 3B). Similarly, observed variation in SVL for younger age classes (newborns and 200-day-old individuals) is greater than that predicted by the model (F-test for homogeneity of variances: \( P < .001 \); fig. 4B). Interestingly, observed variation in SVL for the older adult class is also greater than that predicted for models without variation in food intake (Dis and Birth; F-test for homogeneity of variances: \( P < .02 \)) but not greater than that for models in which food varies stochastically (Food and Food-B; F-test for homogeneity of variances: \( P > .12 \)). Empirical and predicted variances in fecundity rates are not significantly different (F-test for homogeneity of variances: \( P > .05 \)), although the extreme values are farther from the mean in the empirical data set (fig. 4A).

**Discussion**

We follow a largely mechanistic framework to construct a physiologically structured population model for the common lizard *Zootoca vivipara*. This model allows us to evaluate the demographic consequences of considering variation in birth date and variation in food availability as mechanisms responsible for generating within-cohort individual differences. Parameterized with realistic values and ranges for the stochastic processes, our model predicts differences in life history among individuals that are generally smaller than the observed levels of within-cohort variability. This result is not surprising, because mechanisms other than the ones considered here can generate individual variation in natural populations (Tuljapurkar et al. 2009). For example, heritable traits, maternal effects, and climate conditions are potentially important sources of variation among common lizards (Massot et al. 2002; Marquis et al. 2008; Le Galliard et al. 2010). Yet our results indicate that variation in food availability and birth date can generate individual differences in life history and influence population dynamics.

**Population-Dynamic Consequences of Within-Cohort Variability**

The median population abundance depends on the model version, but we find no qualitative effects on model dynamics of incorporating within-cohort individual variation. Qualitative effects have been reported by previous studies, although the direction of the effects seems to vary. For example, Fox (2005) introduced heritable individual variation and concluded that increased individual variation reduces the risk of extinction by reducing population-level variance. However, other authors have reported more complex, nonmonotonic patterns (Grimm and Uchmanski 2002; Vindenes et al. 2008) and have shown that there is no unique, simple effect of intraspecific variability on population dynamics, as reviewed in the “Introduction.” Our
results show that qualitative changes may not always occur in response to individual variation (see also Vindenes et al. 2008). Instead, population-dynamic effects depend on diverse factors, including the intrinsic population dynamics, the source of individual variation, and how this source is incorporated into the model. The fact that population-dynamic effects of individual variation are context and approach dependent highlights the importance of using a mechanistic framework. Although models will always include a degree of structural uncertainty, as our knowledge of dynamics and mechanisms is never complete, a mechanistic framework can provide greater insights because the processes responsible for generating observed patterns are explicitly investigated. Mechanistic models are key to understanding the importance of the diverse sources of intraspecific variability and to gaining insight into how particular factors and mechanisms translate into life-history and population-dynamic changes. An example is our prediction of long, skinny individuals in response to variability in food intake rate (see below).

Life-History Consequences of Food Variability

Although our stochastic models predict population dynamics qualitatively similar to those of the deterministic model, we report important consequences of introducing individual variation for the predicted life histories and population densities. In particular, stochastic variation in the food intake rate results in long and skinny individuals, even though the mean food intake remains constant. Skinny individuals are also less fecund, and thus the number of offspring per female decreases, which reduces competition among the newborn class and leads to higher survival of young individuals. The predicted change in individual morphology can be explained by Jensen’s inequality and the nonlinear relationship between body growth and daily food intake (fig. 5). Jensen’s inequality states that for a set of values \( x_i \) with mean \( E(x_i) \), the average result of the nonlinear function \( f(x_i) \) (denoted \( E(f(x_i)) \)) need not equal the function of the average \( f(E(x_i)) \) (Ruel and Ayres 1999). Here, the nonlinearity in body growth is caused by the transition that occurs when the assimilated energy is not sufficient to cover metabolic costs. This transition leads to a concave-up relationship between structural-mass growth and resource availability, because growth is halted when energy intake is below maintenance costs (fig. 5). As a result, when food intake varies daily, the structural-mass mean growth rate is higher than the growth rate predicted for the mean food intake. Conversely, reserves-mass growth has a concave-down relationship because reserves are converted back to energy used to cover maintenance costs when food intake is insufficient. As a result, the mean growth rate of reserves mass in a stochastic environment is lower.

Transitions in growth are expected whenever individuals are able to survive for some time by using energy reserves and body growth is reduced or stopped at the time when food intake is not sufficient to cover maintenance costs. These simple requirements are met by a wide variety of taxa (Kooijman 2000); thus, the nonlinear relationship
between body growth and food availability should be very widespread. However, morphological changes may not be apparent if food availability always remains above or below the transition point. Changes in morphology will become apparent only when food intake falls below maintenance costs for some individuals at some point in time. This is likely to occur in food-regulated populations when population size is near carrying capacity or in habitats with high intrinsic stochasticity in food availability. Confirming our predictions, laboratory studies have shown that changing the temporal variance in food availability while keeping the mean constant results in morphological changes in sticklebacks and sea urchin larvae (Ali and Wootton 1999; Miner and Vonesh 2004). Whether the observed morphological changes have demographic consequences in natural populations remains to be clarified. However, our results suggest that population sizes may change, thereby affecting overall resource levels.

**Life-History Consequences of Birth Date Variability**

Variation in birth date has some permanent effects on individual body size (with earlier-born individuals staying larger), but even large variation in birth date has few consequences on the predicted life histories and population dynamics. This suggests that birth date variation may be relatively unimportant in generating demographic differences in *Z. vivipara*, in contrast with empirical results from other lizards (Olsson and Shine 1997; Warner and Shine 2007). On the other hand, one interesting effect of assuming an extended birth period is the attenuated pressure on the resource by the newborns, such that resource levels do not drop sharply, as compared to the case of simultaneous reproduction. Previous PSPMs considering a single reproduction event have shown how newborn cohorts can quickly overexploit resources and starve older cohorts, generating single-cohort cycles (Persson et al. 1998; Claessen et al. 2000). Our model suggests that resources are not as quickly reduced by the newborns when reproduction is extended over a period of time, which may facilitate the coexistence of multiple cohorts. To test this idea, it would be interesting to explore whether including realistic variation in birth date is sufficient to eliminate the single-cohort cycles predicted by some PSPMs (e.g., Persson et al. 1998). The study by van Kooten et al. (2007) hints at this mechanism. Although reproduction in their model remains pulsed on a single day, the within-cohort spreading of life histories over groups and over a spatial gradient indeed reduces the strength of the birth pulse. This also results in dynamics other than cohort cycles that are instead characterized as stochastic fluctuations with relatively small amplitude.

**Implications and Perspectives**

Models that link individual physiological processes, environmental conditions, and population dynamics are use-
ful tools that have become increasingly popular (Kearney and Porter 2009). This study shows that these models may be used to explore the relative importance of diverse factors capable of generating individual variation but that no single, unique response of population dynamics to all sources of individual variation can be expected. This is an important message, and it suggests that the goal of modeling individual variation should shift from general, non-mechanistic approaches to models that allow exploration of how individual differences are generated and the effects of particular sources of variation. In agreement with Vin denes et al. (2008), a first important finding is that qualitative changes in population dynamics may not always occur in response to increased variation among individuals. Although unspectacular, this result is also reassuring and lends support to the deterministic approach to modeling populations. A second important finding is the fact that individual variation may lead to morphological changes, which in turn can affect population processes. In particular, we find that stochastic variation in food availability can affect individual morphology because of a general, widespread nonlinearity in the response of growth to resource availability. Interestingly, whether a morphological response would be observed depends on environmental and population conditions, illustrating the intertwined relationship between individual processes and population dynamics and once again highlighting the importance of using models that account for this feedback in a mechanistic framework. Future work based on this general mechanistic approach is necessary to better understand the consequences of other potentially important sources of variation, such as heritability, maternal effects, and climate conditions.

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Literature Cited


