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

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abstract: The consequences of within-cohort (i.e., among-individual) variation for population dynamics are poorly understood, in particular for the case where life history is density dependent. We develop a physiologically structured population model that incorporates individual variation among and within cohorts and allows us to explore the intertwined relationship between individual life history and population dynamics. Our model is parameterized for the lizard *Zootoca vivipara* and reproduces well the species' dynamics and life history. We explore two common mechanisms that generate within-cohort variation: variability in food intake and variability in birth date. Predicted population dynamics are inherently very stable and do not qualitatively change when either of these sources of individual variation is introduced. However, increased within-cohort variation in food intake leads to changes in morphology, with longer but skinnier individuals, even though mean food intake does not change. Morphological changes result from a seemingly universal nonlinear relationship between growth and resource availability but may become apparent only in environments with strongly fluctuating resources. Overall, our results highlight the importance of using a mechanistic framework to gain insights into how different sources of intraspecific variability translate into life-history and population-dynamic changes.

Keywords: demography, Jensen's inequality, phenotypic plasticity, physiologically structured population models, stochasticity, *Zootoca vivipara.*

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

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have a stabilizing effect when population dynamics are inherently unstable, but the effect is destabilizing for inherently 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, depending on the mean-variance relationship in vital rates (Kendall and Fox 2002). Similarly, Vindenes et al. (2008) found that demographic stochasticity may increase, decrease, 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 depend on how variation is modeled. Therefore, models that generate variation in vital rates or life history without considering the actual mechanisms generating this variation likely provide limited insight into how variation affects natural populations. Instead, biologically relevant conclusions 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 physiologically structured population models (hereafter PSPMs), acknowledges explicitly that the relationship between individual 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 generation or cohort cycles (e.g., de Roos et al. 1992), (2) size-dependent trophic interactions influence the types of dynamics 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 potentially 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 variation. One exception is a study that demonstrates that stochastic within-cohort variability may dampen the amplitude 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 accounting for feedbacks. We use a detailed, predominantly mechanistic framework inspired by empirical understanding 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 variation, 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 difference 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 individual 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 individual variation and explore general model behavior under the assumption of a constant environment. Second, we develop four stochastic model versions aimed at unraveling 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-history 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 environmental conditions during early life history (potentially affecting 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 individual 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; Shine 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*

Symbol	Value	Units	Definition	Reference[a]
Length-mass relationship:				
λ_1	61.693	mm $(g^{\lambda_2})^{-1}$	Allometric scalar	Unpublished data 1
λ_2	.303	...	Allometric exponent	Unpublished data 1
W	2.941	...	Dry to wet mass conversion	Avery 1971
Food intake:				
γ_1	34.449	mg $g^{-\gamma_2}$ day^{-1}	Food intake scalar	Unpublished data 2
γ_2	.69	...	Food intake exponent	Unpublished data 2
sun	4[b]	h day^{-1}	Hours of sunshine per day	Standard conditions
Density dependence:				
B_0	1,500[b]	g	Population biomass resulting in observed food intake rates	Free parameter
δ	1[b]	...	Density-dependent function coefficient	Free parameter
Ontogeny:				
x_{mat}	.569	g	Structural dry mass at maturation	Unpublished data 1
M_0	.0557	g	Body mass at birth	Unpublished data 1
Ω	.4	...	Reproduction costs	Avery 1975; Massot et al. 1992
q_N	.197	...	Neonate body condition	Nagy 1983
q_R	.205	...	Body condition after reproduction	Nagy 1983
q_S	.084	...	Starvation body condition	Nagy 1983
Metabolism and growth:				
τ	.0226	kJ mg^{-1}	Prey mass conversion factor	Avery 1971
ε	.82	...	Assimilation efficiency coefficient	Avery 1975
σ_0	1.0	kJ day^{-1}	Basal activity scope	Grant and Porter 1992
Msun	8[b]	h day^{-1}	Maximum number of hours of sunshine per day	Standard conditions
ρ_1	.23	kJ day^{-1} $g^{-\rho_2}$	Maintenance scalar	Cragg 1978
ρ_2	1.02	...	Maintenance exponent	Cragg 1978
k_P	.42[b]	...	Energy allocation rule for immature animals (before first reproduction)	Free parameter
k_A	.74[b]	...	Energy allocation rule for adults	Free parameter
Φ	.0286	g kJ^{-1}	Energy-to-structural-mass conversion coefficient (with costs of synthesis)	Avery 1971; Peterson et al. 1999
Ψ	.0210	g kJ^{-1}	Energy-to-reserves-mass conversion coefficient (with cost of synthesis)	Peterson et al. 1999; Kooijman 2000
Mortality:				
μ_0	.0025	day^{-1}	Background mortality rate	Unpublished data 3
α	6.2	day^{-1}	Length-dependent mortality scalar	Unpublished data 3
β	.51	mm^{-1}	Length-dependent mortality exponent	Unpublished data 3
L_{min}	15	mm	Minimum body length size for survival	Unpublished data 3

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.

[a] Unpublished data 1 = J.-F. Le Galliard, M. Massot, and J. Clobert, unpublished data; unpublished data 2 = M. González-Suárez, unpublished data; unpublished data 3 = J.-F. Le Galliard, unpublished data.

[b] Default values defined for free parameters or standard conditions.

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*).

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,

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 ($\mu_0 + \mu_L + \mu_S$; 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. González-Suárez, unpublished data). Results do not generally change when the stochastic food consumption factor is drawn at longer intervals (2–15 days). A final model (*F&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 EBTtool 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 EBTtool 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 $x > x_{mat}$ (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 Prédictive (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_N, q_R, and q_S), 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_0 and δ 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_A 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 (*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 $\pm$ 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. 1*B*–1*D*). However, changes in k_P affect population dynamics noticeably (fig. 1*A*). 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. 1*A*, 2). For example, for $k_P = 0.42$ (the default value), all individuals mature during their second year of life (fig. 2*A*), 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. 2*B*). 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-

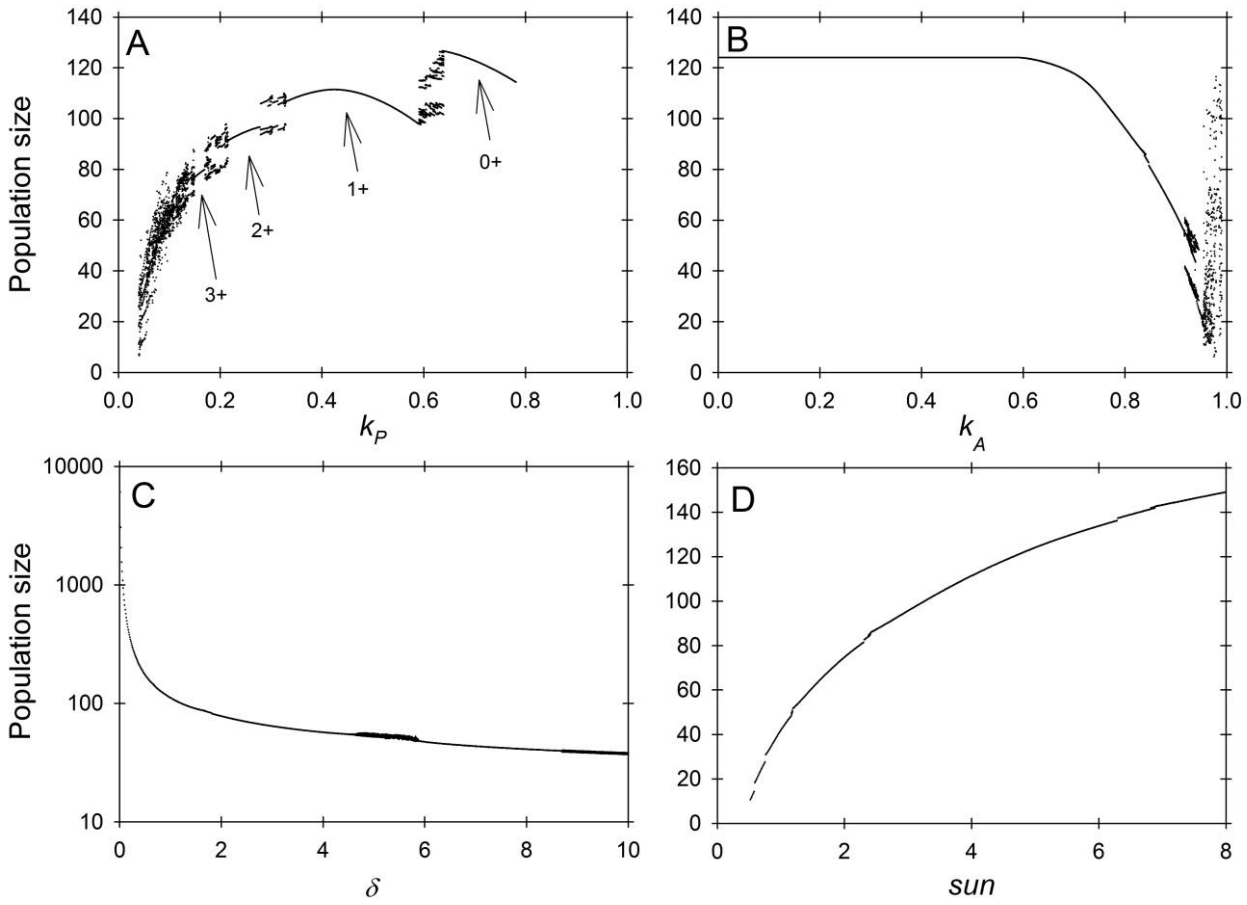

Figure 1: Bifurcation diagrams of the free parameters and *sun* in the deterministic model. Each panel illustrates the results for a different parameter: *A*, energy allocation to structural growth in nonreproducing individuals k_P; *B*, energy allocation to structural growth in adults k_A; *C*, strength of density dependence δ; 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.

tions rapidly collapse. Under the default values, starvation mortality is absent, the maximum age is 8 years, and maturation occurs at age 319 days (~1.6 years, where a year refers to a ~200-day active season). 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. 3*A*, 4*A*). 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. 3*A*). 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. 4*A*). Reduced fecundity is accompanied by slightly higher survival rates of individuals of age 0+ (fig. 3*B*). Because there is no starvation mortality, older individuals

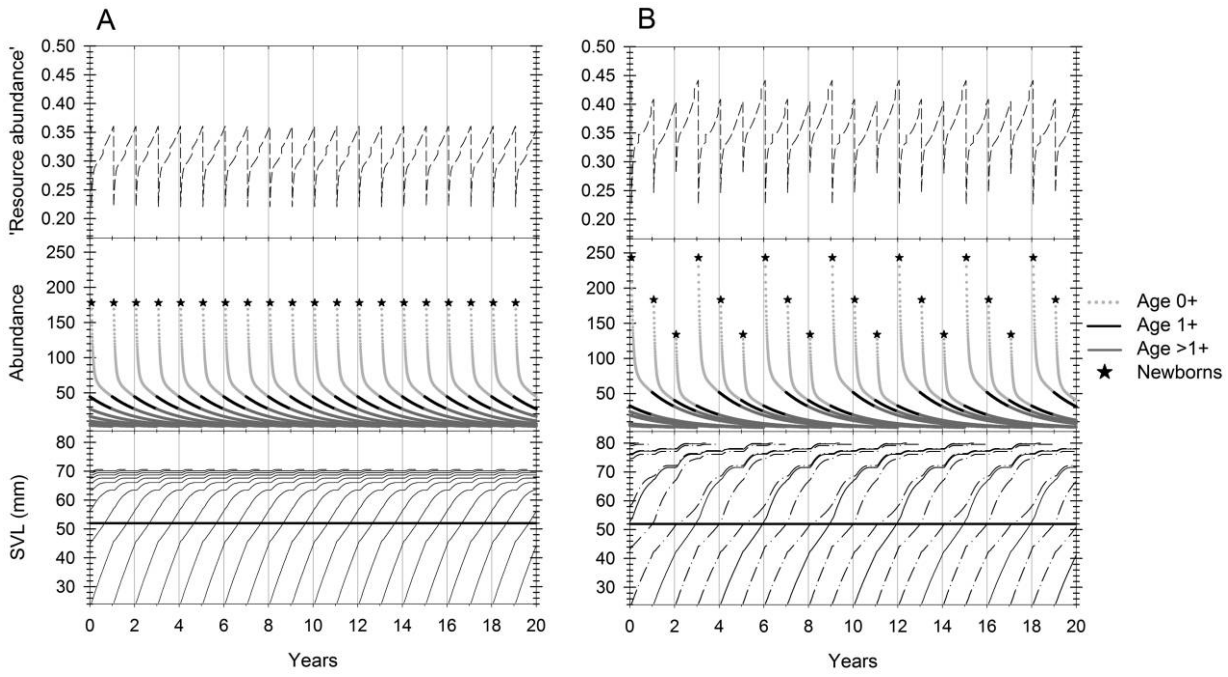

Figure 2: Time series of long-term dynamics predicted by the deterministic model for fixed-point dynamics (*A*; $k_p = 0.42$) and cyclic dynamics (*B*; $k_p = 0.32$). The top panels present the values of the density-dependence factor $D(B)$ (see app. A in the online edition of the *American Naturalist* 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).

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. 4*B*). 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

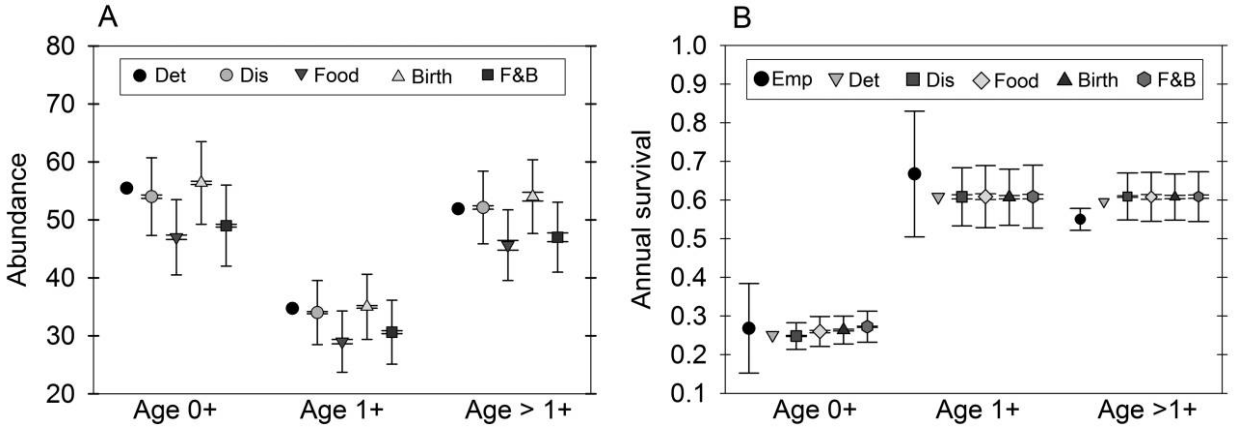

Figure 3: Model predictions per age class of median abundance (*A*) and annual mean survival rates (*B*). Model versions include a deterministic physiologically structured population model (*Det*), a discretized stochastic model (*Dis*), and stochastic versions with individual variation in food intake (*Food*), birth date (*Birth*), or both (*F&B*). *B* also presents empirical estimates (Emp) of annual survival rates. In both panels, longer error bars represent standard deviation (SD) among years, except for the Emp age 1+ estimate, which represents estimates of SD among cohorts because annual estimates are not available. Shorter error bars illustrate the SD among 10 stochastic replicates.

natural populations (Massot et al. 1992). The predicted age structure (fig. 3*A*) 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. 3*B*). 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. 4*B*). 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 *F&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. 4*A*).

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

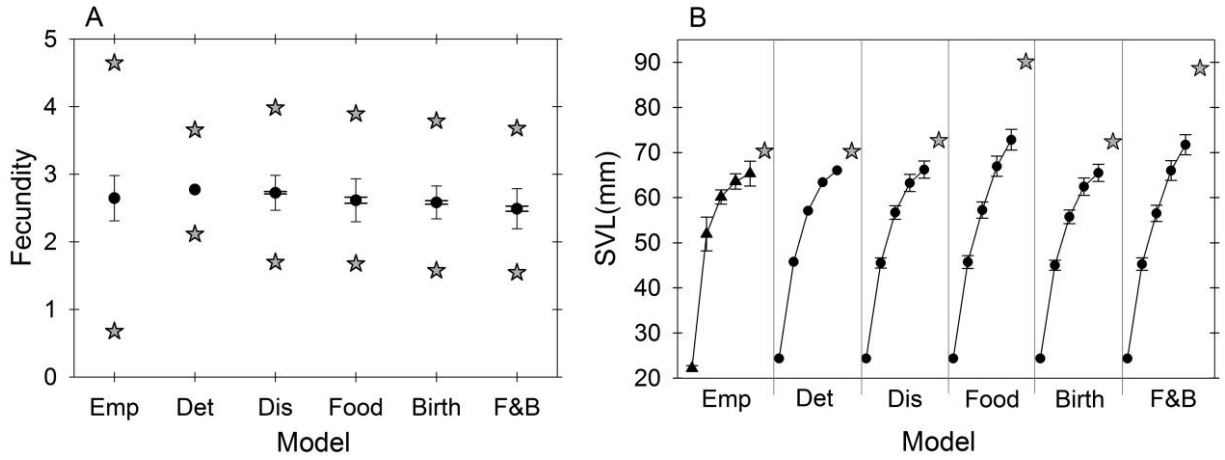

Figure 4: Empirical (Emp) and predicted estimates of female fecundity (*A*) and growth curves (*B*). Predictions are obtained from the models *Det*, *Dis*, *Food*, *Birth*, and *F&B* (see fig. 3 for definitions of the models). In *A*, circles represent mean fecundity (number of female offspring per adult female), longer error bars are for the SD among years, and shorter error bars are for the SD among 10 stochastic replicates. Triangles and stars represent extreme values, estimated as the mean of yearly maxima and minima. In *B*, circles represent mean snout-vent length (SVL) at fixed ages (0, 200, 400, 600, and 800 days), error bars are for the SD among years, and stars represent maximum SVL estimates as the means of yearly maxima.

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

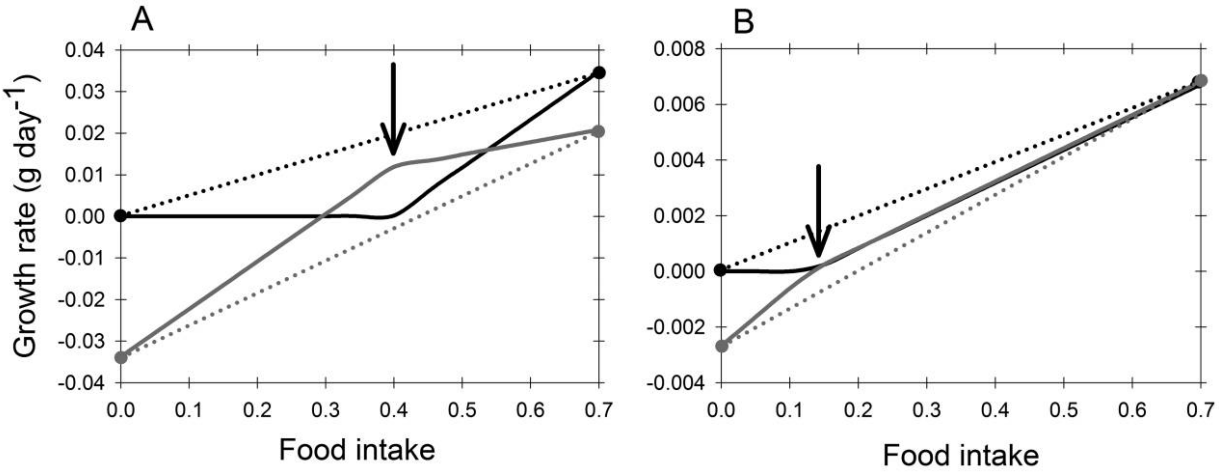

Figure 5: How Jensen's inequality influences the energy budget for adults (body mass 4.5 g; *A*) and juveniles (body mass 0.4 g; *B*). Solid lines show that, assuming a constant food intake rate, the relationship between the growth rates of reserves mass (gray) or structural mass (black) and food intake rate is piecewise linear. The arrows indicate the transition in growth rates that occurs when the assimilated energy is insufficient to cover metabolic costs. Dotted lines show the average growth rates with daily stochastic variation in food intake, assuming that the daily food intake rate takes the extreme values only (i.e., either 0 or 0.7), with a certain probability. Along the dotted line, the probability of "lucky days" changes from 0 to 1. See appendix A in the online edition of the *American Naturalist* for food intake rates and mass growth functions (parameters as in table 1; $D(B) = 0.31$).

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, nonmechanistic approaches to models that allow exploration of how individual differences are generated and the effects of particular sources of variation. In agreement with Vindenes 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.

Acknowledgments

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Appendix A from M. González-Suárez et al., "Population and Life-History Consequences of Within-Cohort Individual Variation"

(Am. Nat., vol. 178, no. 4, p. 525)

Description of the Deterministic Model (*Det*)

The model includes three *i*-state variables: age, structural mass, and reserves mass (see table 1 for descriptions and values of all model parameters described below). Body mass is separated into two components to account for mass that can be converted into energy for reproduction and during periods of starvation (y, reserves mass in g) and structural body mass that cannot be reconverted to energy (x, in g). In standard PSPM terminology, these two types of mass are also referred to as irreversible (structural) and reversible (reserves) mass.

The individual food intake rate (in kJ day^{-1}) is assumed to be the product of a size-dependent, empirically estimated typical food intake rate ($\gamma_1 x^{\gamma_2}$), a density-dependent factor $D(B)$, sunshine duration (*sun*), and a prey mass-to-energy conversion factor τ, and it is given by

$$C(x) = D(B) \cdot \tau \cdot sun \cdot \gamma_1 x^{\gamma_2}. \tag{A1}$$

We use a daily food intake rate because lizards digest their prey within 24 h (Avery 1973). A positive effect of sunshine duration on food intake is suggested by the literature (Avery 1971; González-Suárez et al. 2011). The density-dependent factor $D(B)$ is given by

$$D(B) = \exp\left(\delta\left(1 - \frac{B}{B_0}\right)\right), \tag{A2}$$

where B_0 is a scaling factor such that when $B = B_0$, $D(B)$ is 1 (i.e., the feeding rate equals the empirical estimate), and δ is the sensitivity to density changes. The quantity $B = \sum_{j=1}^{H} \sum_{i=1}^{N_j} \gamma_1 x_i^{\gamma_2}$ measures the population-level consumption potential, with H being the number of cohorts and N_j the number of individuals in cohort j. Equation (A2) assumes that prey availability depends on the consumption potential of the entire lizard population.

After ingestion, a fixed portion ε of the consumed food is assimilated, while $(1 - \varepsilon)$ is lost as waste and digestion costs. Assimilated energy, $E(x)$, depends on structural mass only and is allocated to cover metabolic costs and growth of structural and reserves mass (in kJ d^{-1}). Growth is assumed to be indeterminate (i.e., no maximum structural or reserves mass is defined a priori). Before first reproduction, individuals follow a "net-production-allocation" model in which active metabolic costs $M(x, y)$ are covered first and a portion k_P of the remaining energy, if any, is allocated to growth of structural mass:

$$E_x(x, y) = \begin{cases} k_P \cdot (E(x) - M(x, y)) & E(x) \geq M(x, y) \\ 0 & \text{otherwise} \end{cases}. \tag{A3}$$

The portion $1 - k_P$ of net production is allocated to reserves mass:

$$E_y(x, y) = \begin{cases} (1 - k_P) \cdot (E(x) - M(x, y)) & E(x) \geq M(x, y) \\ E(x) - M(x, y) & \text{otherwise} \end{cases}. \tag{A4}$$

After first reproduction, individuals follow a "gross-production-allocation" model in which a fixed portion $(1 - k_A)$ of the assimilated energy (i.e., gross production) goes to reserves-mass growth,

$$E_y(x, y) = \begin{cases} (1 - k_A)E(x) & k_A E(x) \geq M(x, y) \\ E(x) - M(x, y) & \text{otherwise} \end{cases}, \tag{A5}$$

while the remainder (k_A) is used to cover first active metabolic costs and then structural-mass growth,

$$E_x(x, y) = \begin{cases} k_A E(x) - M(x, y) & k_A E(x) \geq M(x, y) \\ 0 & \text{otherwise} \end{cases}. \quad \text{(A6)}$$

Allocated energy is directly converted into tissue, accounting for synthesis costs and energy-to-tissue conversion rates for structural mass (table 1),

$$\frac{dx}{dt} = \Phi E_x(x, y), \quad \text{(A7)}$$

and for reserves mass,

$$\frac{dy}{dt} = \Psi E_y(x, y). \quad \text{(A8)}$$

Metabolic costs are defined in light of an empirically estimated basal metabolic rate with parameters ρ_1 and ρ_2, such that

$$M(x, y) = \sigma \rho_1 (x + y)^{\rho_2}, \quad \text{(A9)}$$

where the activity scope σ depends on sunshine duration (*sun*) and expected maximum sunshine (*Msun*):

$$\sigma = \sigma_0 + \frac{sun}{Msun}. \quad \text{(A10)}$$

We assume that as observed in natural populations (Avery 1971), activity is 0 in the absence of sunshine (metabolic rate = basal rate), while maximum activity occurs at maximum sunshine duration. Lizards generally have an activity scope of ~1.5 (Grant and Porter 1992; Buckley 2008); thus, by default we set *Msun* to twice the sunshine duration (*sun*). Changing the default values of *sun* or *Msun* had no qualitative effect on the results.

In addition to a background mortality rate μ_0, our model includes two size-dependent sources of mortality (Le Galliard et al. 2004; J.-F. Le Galliard, unpublished data). The size-dependent mortality rate μ_L represents processes such as competitive social interactions, susceptibility to predation and environmental changes, and aging, and it is modeled as

$$\mu_L(x) = \alpha e^{-\beta \cdot (L(x) - L_{\min})}, \quad \text{(A11)}$$

where $L(x)$ is snout-vent length and scales allometrically with structural mass $L(x) = \lambda_1 x^{\lambda_2}$ and $L_{\min}$ defines a threshold length for survival. Starvation mortality,

$$\mu_S(x, y) = \begin{cases} q_S \frac{x}{y} - 1 & \frac{y}{x} < q_S \\ 0 & \text{otherwise} \end{cases} \quad \text{(A12)}$$

(following Persson et al. 1998), occurs when body condition (defined as the ratio of reserves to structural mass) falls below the starvation threshold q_S (table 1).

In the deterministic model version, reproduction is modeled as a single event occurring once a year, on the mean day of the birthing season (see stochastic versions in "Model Implementation"). In *Zootoca vivipara*, reproduction investment in litter mass increases with body size mainly because of an increase in the number of eggs rather than an increase in egg size (Bauwens and Verheyen 1987; Massot et al. 1992). Therefore, we assume that all individuals are born with identical body mass m_0 and body condition q_N. Reproduction is modeled in two separate steps. Step 1 determines whether a female will attempt breeding, with the following rule: after reaching maturation size x_{mat}, individuals may reproduce if their body condition is greater than the reproductive threshold q_R. If a female attempts to breed, then the number of female offspring produced per female at each birth is determined by a function of reserves mass, assuming a constant and balanced sex ratio at birth,

$$F(x, y) = \begin{cases} \omega \frac{y - q_R x}{2m_0} & \frac{y}{x} \geq q_R \\ 0 & \text{otherwise} \end{cases}, \quad \text{(A13)}$$

where ω reflects reproduction efficiency, accounting for a 50% loss of invested mass during embryo and egg development (Avery 1975) and a 10% egg failure (Massot et al. 1992).

An important consequence of annual, pulsed reproductive events is that the population consists of cohorts of identical individuals. The abundance of the newborn cohort equals the sum of the fecundities of all reproducing individuals,

$$N_{\text{newb}} = \sum_{j=1}^{H} \sum_{i=1}^{N_j} F(x_i, y_i). \tag{A14}$$

During the rest of the year, cohort abundance changes over time as a result of mortality,

$$\frac{dN_j}{dt} = -\mu(x, y)N_j, \tag{A15}$$

with cohorts considered extinct when formed by less than 1 individual. The total number of cohorts present in the population (H) is unlimited and may vary over time. However, in our case H stayed generally less than 10.

Literature Cited Only in Appendix A

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Appendix B from M. González-Suárez et al., "Population and Life-History Consequences of Within-Cohort Individual Variation"

(Am. Nat., vol. 178, no. 4, p. 525)

Verification of the Density-Dependent Function

The function $D(B)$ is defined phenomenologically on the basis of our general knowledge of the species. However, we explore whether the proposed relationship is realistic using empirical data. In particular, we compare model predictions that assume different population densities with data from an experimental manipulation of population density (M. Mugabo and J.-F. Le Galliard, unpublished data). The general experimental setup is described by González-Suárez et al. (2011). In short, five different density treatments, with several replicates per treatment, were established in seminatural enclosures during the summer of 2008, after reproduction had occurred. All treatments were started with comparable age-class and sex ratios and with individuals of equivalent body size in each age class. After approximately 10 months, and before reproduction, all surviving lizards were recaptured and their body mass was measured. These data allow us to estimate the effect of population density on body growth (measured as final body mass) and thus, arguably, on food intake rates.

To simulate the experimental treatments, we used our deterministic model (*Det*) with five different initial conditions, replicating those in the experiment. In particular, we used the same initial number of individuals per treatment and the same average body mass for each age class measured at the beginning of the experiment. Population dynamics were then simulated for the equivalent of the 10 months of the experiment (155 active days in our model). As in the experiment, there was no reproduction during these simulations. Model parameter values were those described in table 1, except for changes necessary to reflect the distinct experimental conditions. In particular, we set $B_0 = 180$, $sun = 1.32$, and $Msun = 2.64$; *Msun* was set to twice *sun*, as in the other simulations. Figure B1 shows that the empirically observed relationship between final body mass and population density is generally captured by the density function $D(B)$.

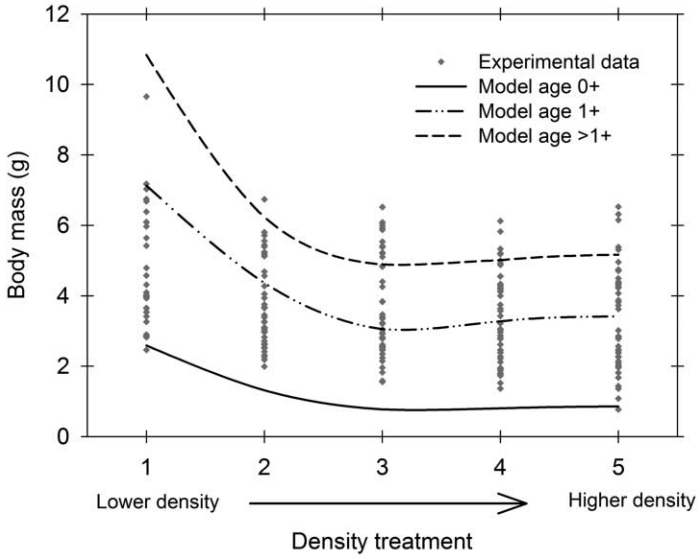

Figure B1: Observed wet body mass of all surviving *Zootoca vivipara* individuals under five density treatments and the average wet body mass per age class predicted by the deterministic model in a simulation of the experiment.

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Appendix C from M. González-Suárez et al., "Population and Life-History Consequences of Within-Cohort Individual Variation"

(Am. Nat., vol. 178, no. 4, p. 525)

Distribution of the Number of Female Offspring per Female

We use a probabilistic approach to discretize the expected number of female offspring per female (eq. [A13]). To explore whether our approach generates a realistic distribution of the number of offspring per female, we compared values predicted by all the model versions with values observed in a natural population in the Cévennes, France (Le Galliard et al. 2010; M. Massot and J.-F. Le Galliard, unpublished data). The empirical number of offspring was calculated as described in the "Model Validation" subsection of "Model Formulation and Parameterization," assuming an equal sex ratio (e.g., 50% of females that produce 3 offspring were assumed to produce 2 female offspring, and the remaining 50% were assumed to produce 1 female offspring, for an average of 1.5 female offspring per female). Fecundity estimates from the deterministic models (real numbers) were rounded to the nearest integer for illustration purposes. In the individual-based stochastic model versions, the number of offspring was calculated as an integer, as described in "Model Implementation." Figure C1 shows that the model-predicted and observed distributions of female offspring per female are very similar, although extreme values, observed with a small frequency in the empirical data set (0 and 6), are not predicted by any model version.

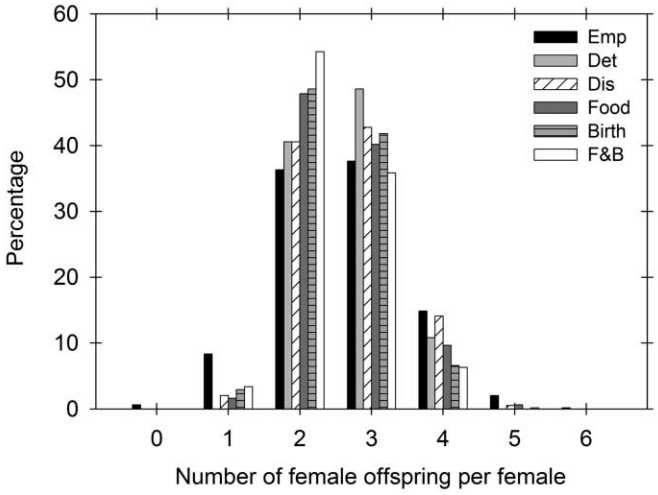

Figure C1: Distribution of the number of female offspring per female, as predicted by all models—*Det*, *Dis*, *Food*, *Birth*, and *F&B*—and as observed in a natural population of *Zootoca vivipara* (Emp). See "Model Implementation" for definitions of the models.

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Appendix D from M. González-Suárez et al., "Population and Life-History Consequences of Within-Cohort Individual Variation"

(Am. Nat., vol. 178, no. 4, p. 525)

Model Simulation and the EBT Tool

All model versions were simulated with the software package called the Escalator Boxcar Train (EBT), developed by André M. de Roos. The software is available from http://staff.science.uva.nl/~aroos/EBTsoftware.htm. A detailed description of the simulation method can be found in De Roos (1997) and in the accompanying online documentation (http://staff.science.uva.nl/~aroos/Ebt.htm).

The EBT method is designed to numerically integrate physiologically structured population models (PSPMs). A basic step in the formulation of PSPMs is to distinguish individual-level and population-level state variables, referred to as *i*-state and *p*-state variables, respectively. The *i*-state variables in our model are age (a), structural body mass (x), and reserves body mass (y). All other individual-level processes (e.g., survival and fecundity) are explicit functions of these *i*-state variables, as defined in appendix A. The *p* state is the distribution of individuals over the *i*-state space (e.g., the population age/size distribution). The EBT method computes the dynamics of the *i*- and *p*-state variables by integrating differential equations. For populations with pulsed reproduction, as is the case in our *Det* model, the population distribution (over the *i*-state space) is naturally discretized into year classes, which we refer to as cohorts. In the EBT method, all individuals within a cohort are assumed to be identical, and their dynamics are described by the same differential equations. The EBT method keeps track of the number of cohorts, the state of the individuals in each cohort, and the abundance of each cohort over time. Reproduction events lead to the creation of new cohorts, whereas cohorts can become extinct and are removed from the population when the cohort abundance drops below a certain threshold value because of mortality (in our model, <1 individual).

In the stochastic versions of the model, individuals are no longer lumped into year classes but are accounted for individually. Technically, this is done by defining a new cohort for each individual that is born. Death is now modeled as a discrete event. Otherwise, the *i*-state dynamics are equivalent to those of the *Det* model. See "Model Implementation" for how the different stochastic versions are defined.

The EBT is coded in the C language. The four files needed to run the deterministic model version and the four files needed to run any of the stochastic models are available in a zip file. The user-defined model functions are coded in two files (extensions .c and .h). Two further files contain the model parameter values and initial conditions (extensions .cvf and .isf, respectively). The different stochastic model versions are obtained by changing two parameter values in the .cvf file, as described in table D1. The parameter REPRO_DUR is for the duration of the breeding period in days, and NORM_SD defines the SD for the normal distribution with mean of 1 from which the stochastic food consumption factor is drawn daily in model versions with variability in food intake.

Model output: time series and bifurcation plots. Two kinds of model output are presented in the main text: time series and bifurcation plots. Time series are obtained by plotting a selection of representative variables against time with a short output time interval. Bifurcation plots are obtained by plotting the output of a whole set of time series, each run with a slightly different value of one particular parameter, against the value of this parameter. For this kind of plot, output is generated only once per year. Bifurcation plots allow us to depict the asymptotic model behavior for a whole range of parameter values.

Table D1. Parameter values for stochastic models

	Value of parameter in .cvf file	
Model version	REPRO_DUR	NORM_SD
Dis	1	.000
Food	1	.195
Birth	30	.000
F&B	30	.195