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Maternal effects and the stability of population dynamics in noisy environments

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Summary

1. It is widely appreciated that complex population dynamics are more likely in systems where there is a lag in the density dependence. The transmission of maternal environmental conditions to offspring phenotype is a potential cause of such a lag. Maternal effects are increasingly found to be common in a wide range of organisms, and might thus be a frequent cause of nonequilibrium population dynamics.

2. We show that a maternal effects' lag generally increases population variability. This may result from the lag inducing cycles (or more complex dynamics) in a deterministic environment or, in a stochastic environment, from the lag interacting with environmental noise to produce more variable dynamics than would otherwise occur. This may happen whether the underlying dynamics are equilibrium, periodic or more complex.

3. Although maternal effects may generally destabilize dynamics there are a clear set of exceptions to this. For example, including a maternal lag may convert cycles to equilibrium dynamics, which may revert to cycles when external noise is added.

4. The influence of the maternal effect depends importantly on the details of the model, whether it is structured or unstructured, the life-history traits which are maternally affected, and the type of density dependence.

5. Our results indicate that, if maternal effects are possible, failure to include them in models used predictively may result in quantitatively, and perhaps qualitatively, poor predictions.

Key-words: density dependence, environmental stochasticity, life-history traits, structured and unstructured models, time-lags.

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Introduction

An organism's fitness depends on the environment in which it finds itself (Metz, Nisbet & Geritz 1992; Diekmann, Mylius & ten Donkelaar 1999; Benton & Grant 2000). What is increasingly realized is that there is often a time-lag – or memory-effect – of past environments on current fitness (Mousseau & Fox 1998a, 1998b). This may be within a generation, such that an individual's early experiences may alter growth rates and later reproductive capacity, an effect sometimes noticeable for a whole cohort born at the same time (Albon, Clutton-Brock & Guinness 1987). For example, the breeding success of female red deer, *Cervus elaphus* L., is influenced by the environmental conditions in the year of their birth, probably by affecting food availability, including their mothers' lactation (Albon *et al*. 1987; Kruuk *et al*. 1999). Similarly, the conditions experienced by one generation can cause effects across one, or more, generations. Such 'parental effects' have been identified in a wide range of organisms, including red deer where birth weight, which affects aspects of adult reproductive success, is strongly influenced by a mother's nutritional status during gestation which, in turn, is influenced by climatic conditions (Albon *et al*. 1987; Kruuk *et al*. 1999). In the soil mite *Sancassania berleisi* (Michael), the amount of competition experienced during development by one generation causes significant differences in the hatching

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probability, hatching date, juvenile development rate and survival of the next generation, and environmental effects may be detectable up to four generations later (Benton *et al*., unpublished).

Parental effects in general, or maternal effects in particular, have received considerable attention in recent years, particularly from an evolutionary viewpoint, as maternal effects have increasingly been identified as adaptive factors, rather than simply an annoying source of variation masking the underlying genetic effects (Mousseau & Fox 1998a). Maternal condition and subsequent patterns of offspring provisioning has been shown to determine offspring life-history traits, such as growth rate, survival, size and age at reproduction and offspring fecundity, in a wide variety of organisms, e.g. plants (Platenkamp & Shaw 1993; Donohue & Schmitt 1998; Mazer & Wolfe 1998), annelids (Bridges & Heppell 1996), arthropods (Benton *et al*., unpublished; Gliwicz & Guisande 1992; Fox & Mousseau 1998; Fox *et al*. 1999), fish (Lin & Dunson 1995; Heath & Blouw 1998; Heath, Fox & Heath 1999), amphibians (Semlitsch & Gibbons 1990), lizards (Sinervo 1991), birds (Price 1998), and mammals (Boonstra & Hochachka 1997; Inchausti & Ginzburg 1998; Dodenhoff, Van Vleck & Gregory 1999; Saatci, Dewi & Ulutas 1999). Maternal choice of mate, nest site selection or oviposition site selection have also been investigated as having important consequences for offspring fitness, although the consequences of mate choice have rarely been viewed from a 'maternal effects' angle (Mousseau & Fox 1998a; Moore, Wolf & Brodie 1998).

In biology, the focus of studies on maternal effects has thus been on their evolutionary implications, although there is also a large applied literature on estimating their influence in studies of selective breeding for improved agricultural yields, e.g. Dodenhoff *et al*. (1999); Saatci *et al*. (1999); Tosh, Kemp & Ward (1999). The ecological consequences of maternal effects, particularly in population dynamic terms, has received relatively little attention, though it was first considered by P.H. Leslie in 1959 (Leslie 1959). It has long been recognized that delayed-density dependence can destabilize population dynamics and may, in particular, promote cyclic dynamics (Schaffer & Kot 1986; Turchin 1990; Berryman 1992). One obvious cause of a delay is through maternal effects (Rossiter 1991, 1994; Ginzburg & Taneyhill 1994; Crone & Taylor 1996; Ginzburg 1998). Using simple, unstructured, deterministic models Ginzburg & Taneyhill (1994) showed that multigenerational cycling was indeed promoted by maternal effects, as is found in a range of forest Lepidoptera. Furthermore, Inchausti & Ginzburg (1998) show that maternal effects are a plausible cause of microtine rodent cycles.

Although extremely valuable, Ginzburg and collaborators' models do not address three important issues:

1. Most organisms do not show cyclical population dynamics; so, do maternal effects have detectable effects for these noncyclic populations?

2. Most – if not all – organisms live in worlds where environmental stochasticity is ubiquitous; so, is there an interaction between maternal effects and environmental variance? A priori we might think that the combination of noise and maternal effects may be important in some situations. For example, if a maternal effect slightly destabilizes equilibrium population dynamics to the extent of generating decaying oscillations, then the long-term deterministic behaviour of the maternal and non-maternal effects will be the same: equilibrium dynamics. However, in noisy environments the dynamics could be different as the maternally generated decaying oscillations can be perturbed into sustained oscillations (Kaitala, Ranta & Lindstrom 1996; Greenman & Benton 2001).

3. For many biological questions, age, or stage, structured models are useful as they 'include more biologically significant mechanisms, more experimentally measurable parameters, more environmental variability and more important nonlinear processes' than unstructured models, and 'the resulting dynamics can be profoundly different' from those of unstructured models (Caswell *et al*. 1997: 10). Although the unstructured models of Ginzburg & Taneyhill (1994) and Inchausti & Ginzburg (1998) provide a good fit to a number of extant forest Lepidoptera and microtine rodent time series, there has been no investigation of the impacts of maternal effects on complex, biologically structured, population models (although Inchausti & Ginzburg (1998; Appendix 1) shows that maternal-effects cycles are also plausible within a simple age-structured model). With structured models, it might be possible for the different age/stage classes to fluctuate out of synchrony, such that the total population size remains unaffected by the destabilizing maternal effects which are shown in unstructured models.

Our aim is to investigate a range of models to explore whether the known destabilizing properties of maternal effects are indeed general. We inspect both structured and unstructured population models with a range of deterministic behaviours and with noise added to them. For the first part of the investigation, we use a simple, unstructured model with and without maternal effects which cause the fecundity and/or competitive ability of individuals at time t to be a noisy function of the population size at time t–1. For the second part of the investigation, we take the validated, structured, Larvae, Pupae, Adult (LPA) model of *Tribolium castaneum* (Herbst) population dynamics (Dennis *et al*. 1995), and make larval recruitment (a measure of fecundity) a function of the population size during their mother's generation, in both deterministic and noisy environments. For both model types, varying control parameters allows the impact of noise and maternal effects to be identified when the underlying dynamics vary from equilibrium to quasi-periodicity. This investigation should be regarded as a 'what if … ?' study rather than one aiming to generate particular system-specific and testable hypotheses. If maternal effects are found to have important dynamical

consequences in a range of plausible scenarios then this would indicate that they should be considered in any modelling scenario, especially if modelling is undertaken in order to produce qualitative predictions of future population size.

Methods

UNSTRUCTURED MODEL

We shall adopt two population renewal processes differing in details of the density-dependent structure:

$$N_{t+1} = \left\{ N_t \exp\left[r\left(1 - \frac{N_t}{K}\right)\right]\right\}\mu_t$$

and eqn 1

$$N_{t+1} = \left(\frac{\lambda N_t}{1 + aN_t^b}\right)\mu_t,$$

the Ricker dynamics and the Maynard Smith–Slatkin dynamics, respectively (e.g. May & Oster 1976; Royama 1992). The population size at time t and subsequent time units is N_t. For the Ricker dynamics r is the population growth rate while K is the carrying capacity (here r ranges from 1 to 3·5 and $K = 1$). For the Maynard Smith–Slatkin dynamics λ is the number of offspring produced, and the carrying capacity is given with the parameter a (here $\lambda = 5$ and $a = 1$), while the value of the exponent b indicates whether the competition of the resources is contest, when $b = 1$, or scramble, when $b > 1$ (here b ranges from 1 to 5). The decision to work with the two models is because the Ricker model has an exponential growth function while the Maynard Smith–Slatkin model has a hyperbolic growth function (Royama 1992). These growth functions create a notable difference in the form of the density dependent feed back.

The term μ_t in equation 1 is an external disturbance forcing the population dynamics (the Moran effect: Moran 1953; Royama 1992). It is implemented here by drawing random numbers μ_t from a uniform distribution between 0·7 and 1·3 (e.g. Ranta, Kaitala & Lundberg 1997). We decided to let the Moran effect influence population dynamics after equation 1 as all natural populations are affected by noise of some sort (Kaitala *et al*. 1997). Thus, it is of particular interest to see to what extent the maternal effect becomes visible through the noisy dynamics. The behaviour of the two models without the Moran noise is indicated in panels (a) and (b) in Fig. 1.

There is ample evidence that the past population size affects the realized fecundity, r or λ (it was first demonstrated by Park 1935; for a recent review, see Wade 1998). However, maternal effects can also affect the realized competitive ability (Wulff 1986), due to the effects of differential provisioning of propagules leading to differing growth rates and final adult sizes. Thus, the number of organisms that an environment can support, the carrying capacity, K, may vary over time. This suggests that maternal effects can operate via the r–K dichotomy (Pianka 1970; Roff 1984; Stearns 1992). Thus, there is a choice of where to impose the maternal effect in the equation 1, so the following three scenarios were adopted: (i) it affects the fecundity (r or λ) (ii) it comes via the population carrying capacity (K or a), or (iii) via both ways simultaneously. When it affects simultaneously both r and K, we assumed the effect to be negatively correlated (according to the r–K selection dichotomy).

As with previous authors, we assume that the maternal effect acts via population density lagged one time step. In particular, if $N_{t-1} > N_t$ then $r_t < r$, and $K_t > K$, while if $N_{t-1} < N_t$ then $r_t > r$, and $K_t < K$. Of course when $N_{t-1} = N_t$ then $r_t = r$, and $K_t = K$. We assume no memory of the maternal effect between subsequent time steps. The effect on r and λ is implemented as follows. With $N_{t-1} > N_t$ we draw a random number w_t from a triangular distribution from the range of 0·6–1·2 with a mode at 1, with $N_{t-1} < N_t$ the triangular distribution has a range of 0·8–1·4, and a mode again at 1. The maternal effect, w_t, affects population growth rate by: $r_t = w_t r$ or $\lambda_t = w_t\lambda$. The long-term average of w_t is 1·0, but if $N_{t-1} > N_t$ the expectation of w_t is less than 1·0, so population growth is generally, but not always, reduced, and vice versa for $N_{t-1} < N_t$. This rationale for this implementation is that most of the time there is an effect in the expected direction, but on occasion the effect is opposite to the expected, and that larger effects are more uncommon than smaller effects. The maternal effect on K or a is implemented in a similar way: when $N_{t-1} < N_t$, w_t is drawn from the distribution with the range of 0·6–1·2, with $N_{t-1} > N_t$ the distribution has the range of 0·8–1·4 and $K_t = w_t K$ or $a_t = w_t a$. When the maternal effect is implemented on r and K (or λ and a) simultaneously we assume there is a negative correlation between the two effects because when the environment is crowded r_t is low, and due to the competition individuals have access to fewer resources and so are smaller, so the same unit of resource makes more individuals and K increases. To incorporate the maternal effect into the carrying capacity we select $r_t = w_t r$ or $\lambda_t = \omega_t\lambda$ as described above and make $K_t = (2 - r_t) + \varepsilon_t$ and $a_t = (2 - \lambda_t) + \varepsilon_t$, where ε_t comes from uniform random numbers between –0·1 and 0·1. This results in r and K (or λ and a) being correlated with an average of –0·2 in our simulations.

STRUCTURED (LPA) MODEL

The structured model we used was based on the LPA model of larvae (L), pupae (P) and adults (A) of the flour beetle *Tribolium* (Dennis *et al*. 1995). Dennis *et al*. (1995) express the LPA model as a set of three difference equation:

$$\begin{aligned} L_{t+1} &= bA_t \exp(-c_{ea}A_t - c_{el}L_t) \\ P_{t+1} &= L_t(1 - \mu_l) \\ A_{t+1} &= P_t \exp(-c_{pa}A_t) + A_t(1 - \mu_a) \end{aligned}$$ eqn 2

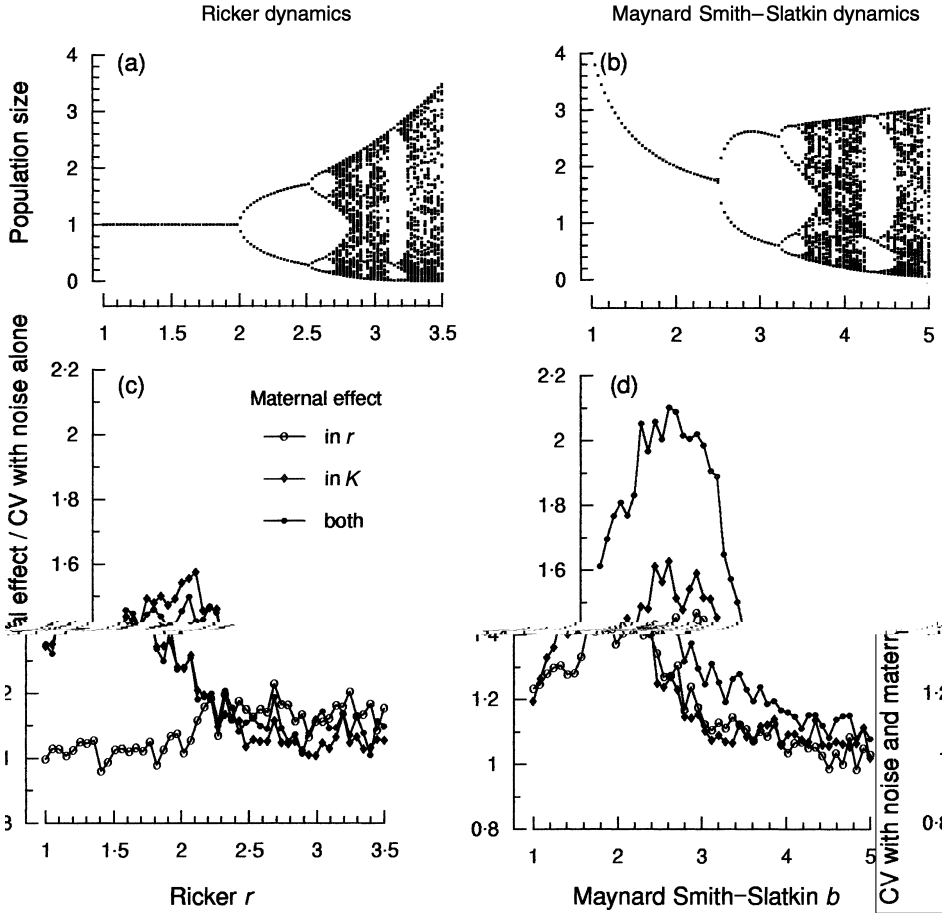

Fig. 1. (a) Bifurcation diagram for the Ricker dynamics against growth rate, r. (b) Bifurcation diagram for the Maynard Smith–Slatkin dynamics against the exponent b in equation 1. Panels (c) and (d) give the measure of standardized (see text for details) population stability against the bifurcation parameters in the Ricker equation and in the Maynard Smith–Slatkin equation. If the coefficient of variation (CV) in the four different treatments (see text) is averaged by splitting the data into two after the bifurcation parameter we get the following averages for the CVs:

Treatment	$r \leq 2$	$r > 2$	Treatment	$\lambda \leq 2{\cdot}6$	$\lambda > 2{\cdot}6$
Moran noise alone	22%	77%	Moran noise alone	22%	67%
Maternal effect in r	22%	88%	Maternal effect in λ	30%	73%
Maternal effect in K	31%	83%	Maternal effect in a	31%	73%
Maternal effect in r and K	30%	85%	Maternal effect in λ and a	41%	80%.

The parameters of the model are explained in Table 1. Table 1 also includes the numerical estimates of the parameters obtained by Dennis *et al.* (1995) from data on the corn oil sensitive strain of *Tribolium castaneum* (Desharnais & Costantino 1985; Desharnais & Liu 1987).

In equation 2 larval recruitment (which is a measure of fecundity) is a constant. We modify (2) by making fecundity density-dependent, as a product of b and an exponential function of the numbers of competitors of the adults:

$$b_{dd} = b\exp\left[R\left(1 - \frac{L_t + A_t}{K}\right)\right] \qquad \text{eqn 3}$$

Where b takes the value from Table 1, R is a constant which acts as a 'control parameter' and changes the slope of the relationship between b_{dd} and density, and K is a scaling parameter and was arbitrarily set to 100.

In many arthropods, the amount of food mothers can invest in their eggs has a strong influence on the life-history of the subsequent animal (Benton *et al.*, unpublished; Gliwicz & Guisande 1992; Fox & Mousseau 1998; Fox *et al.* 1999). Competition, if it acts equally on members in a population, might constrain parental provisioning of offspring to create a 'cohort effect', such as has been hypothesized as the cause of forest Lepidoptera outbreaks (Rossiter 1994). Such a 'cohort effect' is also known for vertebrates (Leslie 1959; Albon *et al.* 1987; Albon & Clutton-Brock 1988; Kruuk *et al.* 1999). We therefore model maternal effects by making b_{dd} a function of the number of competitors at the time that their mothers were laying eggs, rather than the number of current competitors. The lag between the current time, t, and the time at which the median mother was laying an egg which develops into an adult at time t was estimated as:

Table 1. Parameters in the LPA model, biological meaning and numerical estimates obtained for all four replicate cultures analysed by Dennis *et al.* (1995)

Parameter	Biological interpretation	Numerical value
b	Average number of larvae recruited per time step in the absence of cannibalism	11·6772
μ_a	Probability of adults dying from causes other than cannibalism	0·1108
μ_L	Probability of larvae dying from causes other than cannibalism	0·5129
c_{ea}	Rate of cannibalism of eggs by adults	0·0110
c_{el}	Rate of cannibalism of eggs by larvae	0·0093
c_{pa}	Rate of cannibalism of pupae by adults	0·0178

$$lag = \left(3 + \frac{\log(0{\cdot}5)}{\log(1-\mu_a)}\right)_{nint} \qquad \text{eqn 4}$$

where *n*int refers to rounding to the nearest integer. The 3 refers to an average development time of 3 time steps, and the $\log(0{\cdot}5)/\log\mu_a$ is an estimate of the age of the median female alive, given the extrinsic mortality. Therefore the maternal effect on fecundity becomes:

$$b_{dd} = b\exp\left[R\left(1 - \frac{L_{t-lag} + A_{t-lag}}{K}\right)\right] \qquad \text{eqn 5}$$

Following (Dennis *et al.* 1995), we use μ_a as a control parameter, and vary it from 0·01 to 0·99. This changes the population dynamics, as shown in Fig. 2a.

The model is deterministic but we added stochasticity to its elements to ensure the results were robust. For the stochastic model, at each time step each of the six parameters was multiplied by random variables which

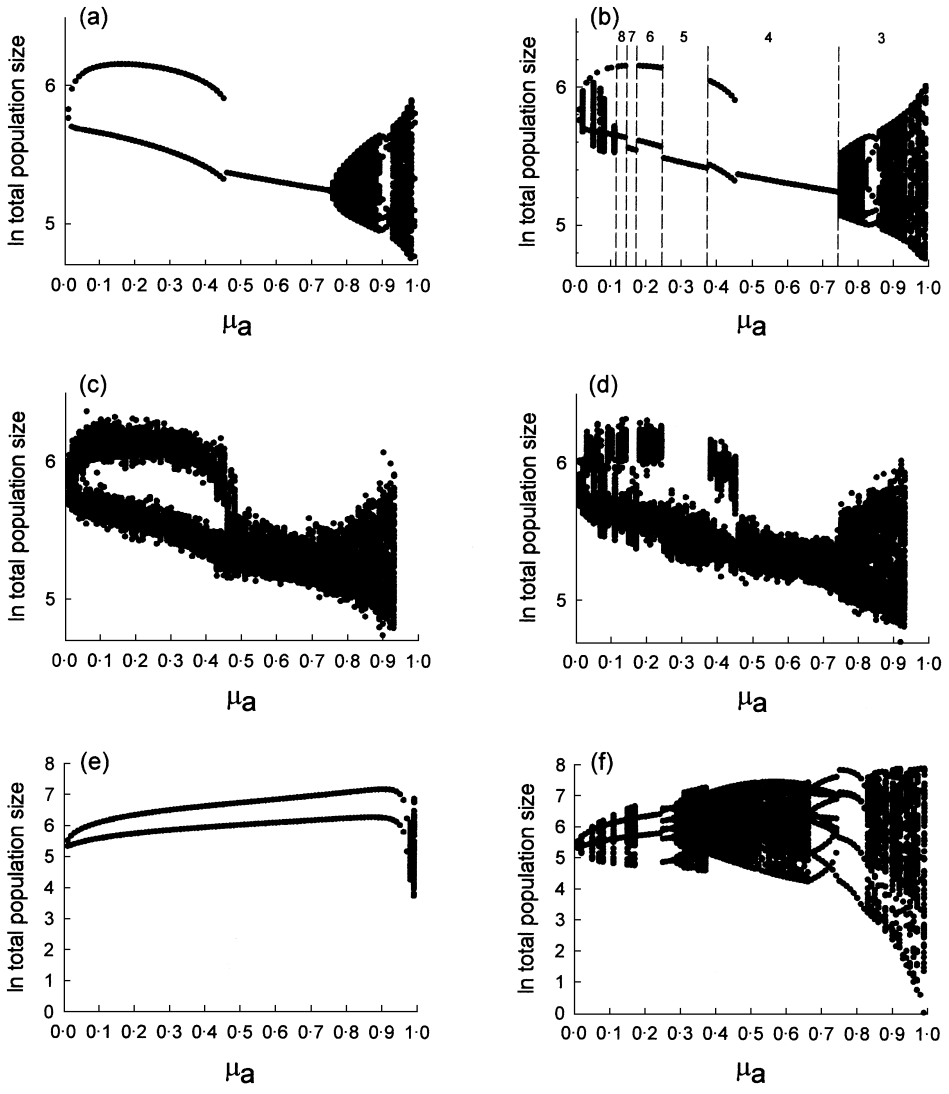

Fig. 2. Bifurcation diagrams for modified LPA model given by equations 2–5 and varying the extrinsic adult mortality (μ_a). (a) bifurcation diagram for deterministic model without maternal effects (eqns 2, 3), $R = 0{\cdot}1$. (b) with maternal effects (eqns 2, 5), $R = 0{\cdot}1$. (c) as (a) but with added stochasticity on each parameter (Table 1) in the model, $R = 0{\cdot}1$. (d) as (b) but with added stochasticity, $R = 0{\cdot}1$. (e) as (a), but $R = 3{\cdot}0$. (f) as (b), but $R = 3{\cdot}0$. The dashed lines and numbers in (b) indicate the length of the lag modelling maternal effects, longer lags (9–72) occur for $\mu_a < 0{\cdot}12$. Addition of maternal effects may stabilise (b, lag 5), destabilise (f vs. e) or not change (b, lag 4) the dynamics. Addition of noise makes generally blurs the dynamics, though there are examples where qualitative changes occur.

Table 2. Output from a GLM model, with response variable the CV of the population over 100 time steps, and the independent variables maternal effects ('ME', presence or absence), stochasticity ('stoch', presence or absence), adult mortality rate (μ_a) and strength of density dependence in fecundity (R).

Source	d.f.	Seq SS	Adj SS	Adj MS	*F*	*P*
μ_a	1	0·98596	0·01708	0·01708	15·29	0·000
ME	1	0·31515	0·01653	0·01653	14·79	0·000
R	1	7·76719	0·51080	0·51080	457·13	0·000
stoch	1	0·00186	0·00738	0·00738	6·61	0·010
ME*μ_a	1	0·22731	0·04410	0·04410	39·47	0·000
mu*R	1	0·58657	0·58846	0·58846	526·63	0·000
stoch*μ_a	1	0·00042	0·00579	0·00579	5·18	0·023
ME*R	1	0·13620	0·00397	0·00397	3·55	0·060
ME*stoch	1	0·00542	0·00003	0·00003	0·03	0·864
stoch*R	1	0·00001	0·00542	0·00542	4·85	0·028
ME*mu*R	1	0·02423	0·01627	0·01627	14·56	0·000
ME*stoch*μ_a	1	0·01947	0·00006	0·00006	0·06	0·810
stoch*μ_a*R	1	0·00699	0·00685	0·00685	6·13	0·013
ME*stoch*R	1	0·00380	0·00496	0·00496	4·44	0·035
ME*stoch*μ_a*R	1	0·01361	0·01361	0·01361	12·18	0·000
Error	2248	2·51191	2·51191	0·00112		
Total	2263	12·60611				

were independent and identically distributed (taken from a normal distribution, with a mean of 1·0 and a standard deviation of 0·05). The coefficient of variation (CV: ratio of standard deviation to mean) of 5% is small, but none the less causes population extinction for about 11% of the parameter space explored. Larger CVs cause more frequent extinctions but do not alter conclusions qualitatively.

Results

UNSTRUCTURED MODEL

In our simulations, with the two differing population renewal models, we had the following treatments: (1) dynamics with the Moran noise alone, (2) dynamics with the Moran noise and a maternal effect in r or λ, (3) dynamics with the Moran noise and maternal effect in the carrying capacity, K or a, and finally (4) dynamics with the Moran noise and maternal effect via fecundity and carrying capacity, assuming a weak negative correlation between the two maternal effect components. In the four combinations for the Ricker dynamics and for the Maynard Smith–Slatkin dynamics we scored the coefficient of variation in population size as a measure of stability of the realized dynamics. The bifurcation parameter in the Ricker dynamics was r and in the Maynard Smith–Slatkin dynamics it was the exponent b.

For each bifurcation parameter value we replicated the simulations 100 times with random initial values (between 0 and 1). For illustrative purposes we standardized the treatment effects by averaging the CVs from the 100 simulations in treatments 2–4, and dividing them by the CV of treatment 1, the dynamics with Moran noise alone (see Fig. 1). Inspection of the non-standardized CVs indicated a quadratic relationship between the CV and the bifurcation parameter. To remove the effect of the bifurcation parameter from the CV we fitted a second-order regression, and took the residuals for an analysis of variance (ANOVA). The ANOVA indicates unambiguously that the four treatment groups differ in the resulting variability in population size: $F_{3,196} = 45{\cdot}4$, $P < 0{\cdot}001$, $F_{3,196} = 220{\cdot}91$, $P < 0{\cdot}001$, Ricker dynamics and Maynard Smith–Slatkin dynamics, respectively. Tukey's *post hoc* HSD test indicated that with the Ricker dynamics there were two clusters of treatments (Fig. 1c), comprising the 'control group' of the dynamics with the Moran noise only (residual mean –5·5) against the three other treatments with both Moran noise and maternal effect (residual means: 2: 1·09, 3: 1·48, 4: 2·96). The Tukey HSD test clustered the four treatments in the Maynard Smith–Slatkin dynamics into three groups: treatment 1 (residual mean –7·29), treatments 2 and 3 (means of –0·89 and 0·23) and treatment 4 (7·95, Fig. 1d).

STRUCTURED (LPA) MODEL RESULTS

The amount that the population varies over time is influenced significantly by the presence or absence of maternal effects, i.e. the dependence of current fecundity on past population density, as well as stochastic variation in parameters (Table 2). Variability is also dependent on the values of μ_a and R (Table 2). There are significant interactions between all four dependent variables in this analysis. The interaction terms indicate that the presence of maternal effects has different influences on population variability depending on the values of the other variables.

The presence of maternally effected fecundity may destabilize (Fig. 2f vs. Fig. 2e), stabilize (Fig. 2b vs. Fig. 2a, $\mu_a = 0{\cdot}25 - 0{\cdot}37$) or leave unchanged the population dynamics (Fig. 2b vs. Fig. 2a, $\mu_a = 0{\cdot}38 - 0{\cdot}75$). There were 594 different combinations of parameters in this investigation: $R = 0{\cdot}1$, 1, 2, 3, 4, and for each value μ_a varies from 0·01 to 0·99. In 62·0% of cases ($n = 594$), addition of a maternal-effects lag caused the population to fluctuate more, as measured by the CV (Fig. 3). When stochasticity was added to the parameters in the model a similar proportion, 62·1%, lead to an increase

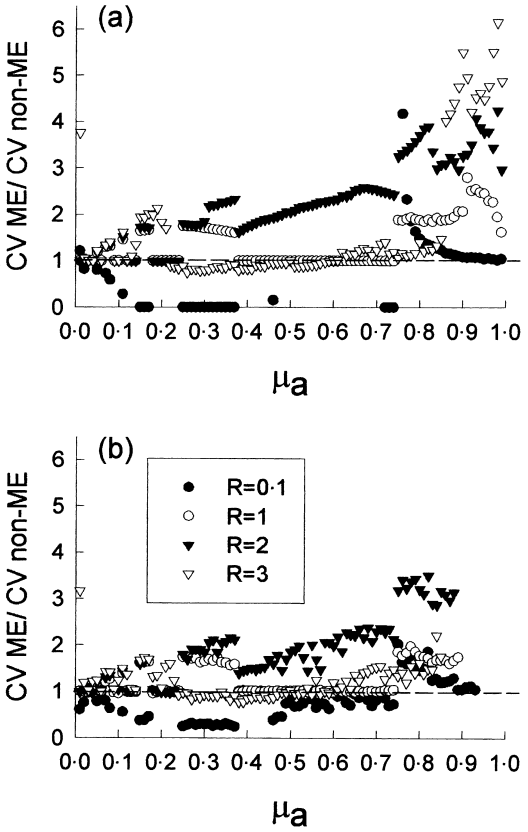

Fig. 3. Ratio of CV (standard deviation divided by mean population size, for 100 time steps of the model starting at $t = 900$) for maternal effects model to standard model, plotted for μ_a and R 0·1–3. CVs > 1 (shown by dotted line) indicate that the maternal effects destabilize the population dynamics. (a) deterministic model, (b) stochastic model. ●, R = 0·1; ○, R = 1; ▼, R = 2; ▽, R = 3.

in variability. However, with some parameter values, maternal effects may reduce the variability (20·2% of cases for deterministic models, 37·5% of cases for stochastic models) or make no difference (17·7% of cases in deterministic models). The latter occurs when the deterministic dynamics cycles with a periodicity which is the same as the lag due to maternal effect.

The instability caused by the maternal-effects' lag can be marked. The median change in CV for the deterministic models was 1·41 times (IQ range 1·00–1·837, $n = 537$), with a maximum of 67·9 times (in a further 57 cases the addition of maternal effects created variability when there was none, giving a CV of infinity). There is no simple relationship between the strength of the density dependence in fecundity, b, as measured by R, and the dynamical effect of maternal effects. The mean ratio of CV in the maternal effects model to CV in the non-maternal effects model was 0·8 when $R = 0·1$, 1·3 when $R = 1$, 2·2 when $R = 2$, 1·5 when $R = 3$ and 1·4 when $R = 4$.

Adding stochasticity does not change these figures qualitatively: 37·5% of the time the CV diminishes when a maternal-effect lag is introduced, 9·8% of the time the population goes extinct. Of the non-extinct populations ($\mu_a < \sim 0·9$), the median change in CV caused by adding maternal effects was 1·19 times (IQ range 0·963–1·464, $n = 536$). With some parameter values, noise interacts with the density dependence to destabilize the model. For example, a deterministic equilibrium can be converted into noisy two-point cycle with the addition of stochasticity (Fig. 2).

Discussion

It is well appreciated that time delays in density dependence can be sufficient to cause complex population dynamics (Leslie 1959; Schaffer & Kot 1986; Turchin 1990; Berryman 1992), and that for a given set of parameters, delayed density dependence makes complex dynamics more likely (Guckenheimer, Oster & Ipatchki 1977; Ginzburg & Taneyhill 1994). Plausible causes of delay in density dependence include the actions of parasites and pathogens, predators or prey (or food, more generally), and *trans*-generational effects such as the maternal effects discussed here. Turchin & Taylor (1990) and Witteman, Redfearn & Pimm (1990) found that delayed density dependence was not uncommon for a range of invertebrate and vertebrate time series. There are some good examples where the delay has been shown to be due to extrinsic factors (e.g. parasites in red grouse: Hudson *et al.* 1998; trophic interactions between Canada lynx and snowshoe hare: Krebs *et al.* 1995; Stenseth *et al.* 1998), but it has generally been found that strongly density-dependent trophic interactions (whether with food plants or natural enemies) have been difficult to establish (Ginzburg & Taneyhill 1994). Although trophic interactions, specifically consumer–resource interactions, are implicit in maternal effects, the destabilization is not caused by dynamic changes in an extrinsic resource, food, which a population tracks. Instead, the destabilization is intrinsic to the system: for a set amount of resources, the population density determines aspects of individual quality which are carried over to future generations through, for example, changing offspring provisioning.

Given the paucity of evidence for extrinsic factors causing delayed density dependence, and given the 'ubiquity of maternal effects in plants and animals' (Mousseau & Fox 1998b, p. 135) we might *a priori* often expect maternal effects to be important in generating delays in density dependence. In a series of modelling papers tackling this subject, Ginzburg and colleagues (Ginzburg & Taneyhill 1994; Ginzburg 1998; Inchausti & Ginzburg 1998) formulate models where individual quality depends on the quality of the parents, where individual quality is a function of density and where quality determines fecundity. The output from such models provided time-series which closely resembled those of the outbreaks in forest Lepidoptera (Ginzburg & Taneyhill 1994) and cycles in microtine rodents (Inchausti & Ginzburg 1998). These models indicate that, indeed, intrinsic maternal effects can destabilize population dynamics and generate cycles. Our research does not focus on the generation of population cycles, but extends the former work to ask how frequently maternal effects destabilize dynamics

(i.e. over what range of parameter types and values do maternal effects increase population variability) and whether the impact of maternal effects is sensitive to environmental variation and/or biological structure.

DO MATERNAL EFFECTS ALWAYS DESTABILIZE THE DYNAMICS?

Although the presence of a lag in density dependence due to a maternal effect generally caused populations to fluctuate more than they would do otherwise in our simulations, this is not a necessary effect of the lag. In both the structured and unstructured models, there are areas of parameter space where the maternal effects model is more stable than the one without the delay. The structured model, in particular, displayed examples of major *stabilization* over a significant portion of parameter space caused by the lag. For example, the unlagged LPA model shows 2-cycles for $\mu_a < 0{\cdot}44$. However, addition of a maternal effect changes the dynamics to equilibrium for $0{\cdot}25 < \mu_a < 0{\cdot}37$ (Fig. 2a,b). This is because the maternal effect effectively smoothes out the highs and lows in population size: when density is low, animals cannot reproduce at the maximum rate because they are low quality, as they are the product of the previous high-density generation, and vice versa. As well as occasional examples of stabilizing the dynamics, the presence of a lag may have no measurable effect (in deterministic models: see Fig. 2a vs. 2b for $0{\cdot}45 > \mu_a > 0{\cdot}74$) or very little effect (in stochastic models: see Fig. 1c, with maternal effect acting on r and with $r < 2$).

In the unstructured model, the influence of the maternal effect depends on the parameters affected by the lag. In the Ricker model, the lag affecting population growth rate (r) has little impact on the population variability, whereas the lag affecting carrying capacity (K) has a marked effect, especially at low r. Contrariwise, in the Maynard Smith–Slatkin model, the presence of a maternal effect on growth rate (λ) has a similar magnitude of effect as a maternal effect on carrying capacity (a) and interestingly, the presence of both types of maternal effect has a greater impact than the sum of the individual effects. Such multiple maternal effects are likely in the real world because maternal provisioning of propagules often leads to differentially sized offspring which may differ both in their competitive ability and their eventual fecundity (owing to the marked size dependence of fecundity across many taxa). For example, in the mite *Sancassania berlesei* offspring of mothers from high density cultures lay smaller eggs which hatch into smaller juveniles, which develop into smaller adults which then have lower fecundities (Benton *et al*., unpublished). Similarly, cohorts of female red deer on Rum, born after cold springs, tend to have low birth weights, slower development rates and shorter lifespans and, in turn, give birth to smaller offspring with lower viability than average (Albon *et al*. 1987; Albon & Clutton-Brock 1988). Simulation of the red deer population dynamics, using randomly assigned cold, average and warm springs, indicates t
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also change. Therefore, if maternal effects are strong in an organism, ESS (or optimality) approaches may lose predictive value if the maternal effects are not incorporated into the analysis.

WHAT IS THE RESULT OF THE INTERACTION OF NOISE, DYNAMICS AND DELAY?

Our simulations indicate that noise, maternal effects and dynamics may interact. At some parameter values the deterministic dynamics are oscillations decaying to equilibrium, which can be sustained by noise (Fig. 2a,c $\mu_a = 0{\cdot}46–0{\cdot}47$; Kaitala *et al*. 1996); such noise-induced changes in dynamics are common close to bifurcations in the underlying dynamics (Greenman & Benton, unpublished). Addition of a maternal-effects' lag can cause a change in the dynamics, and if the lag causes the dynamics to move close to, or away from, a bifurcation then noise may change the dynamics again (e.g. at $R = 0{\cdot}01$ and $\mu_a = 0{\cdot}35$, the deterministic behaviour is 2-cycles, becoming equilibrium on addition of a lag, but revert to noisy 2-cycles with random variation). We conclude that, as with any population model, noise can sometime be an important determinant of dynamical behaviour, but adding noise does not alter the conclusion that, in general, dynamics are destabilised by maternal effects. The effects of noise will, however, depend on the details: in very noisy environments the influence of a maternal effect will be swamped, and the distribution from which the noise is drawn may make a qualitative difference to the dynamics (Greenman & Benton 2001).

WHAT IS THE IMPACT OF BIOLOGICAL STRUCTURE?

In the unstructured model, the addition of a maternal effect generally destabilizes the dynamics. In the structured model, the range of effects is more varied. Adding biological structure seems to generate a richer range of dynamical responses to the maternal effects. In the models described above, however, the structured

model differs from the unstructured in another important regard. The maternal effect in the structured model is a response to the density at which the mother developed which, depending on mortality, may have been last time step (for high μ_a) or several time steps previously (for low μ_a). We investigated the impact of this and used a maternal effect with a constant lag of a single time-step; biologically the egg size depends on the density when mothers laid the eggs, rather than the density when they themselves developed. A constant lag does not substantially alter the conclusions: maternal effects in structured models lead to more varied consequences than in the unstructured models. In soil mites (Benton *et al.*, unpublished), we find offspring life histories are influenced strongly both by the density at which their mothers were reared and the density experienced by their mothers when the eggs were laid.

Conclusions

In conclusion, we show that the addition of a lagged density-dependent effect whose mechanism is a maternal effect generally causes populations to fluctuate more than otherwise. This is often the case even if the underlying dynamics are equilibrium: the lag destabilizes the dynamics. Given the ubiquity of environmental variation and maternal effects (Mousseau & Fox 1998b), a full understanding of the impact of environmental variation on population dynamics may need to consider maternal effects especially, perhaps, if models are constructed to predict future population sizes accurately. Similarly, if one is interested in the relationship between population size and environmental noise, the presence of maternal effects would reduce the likely correspondence between environmental signal and population response, which is already expected to be weak (Ranta *et al*. 2000; Laakso, Kaitala & Ranta 2001; Greenman & Benton 2001). Thus, maternal effects may be one of the reasons why forcing environmental noise and population variation may fail to be well correlated.

Maternal effects, however, can have some unexpected consequences, especially in interaction with noise and biological structure: we present one example where the non-maternal effects model shows two cycles, which are converted to equilibrium dynamics by the addition of a lag, and then revert to two cycles when environmental noise is added to the maternal effects. Such results, as well as the general destabilization that occurs suggests that, whatever the underlying dynamics, maternal effects may make a significant contribution to patterns in population dynamics. Our simulations suggest that this will be true whether the organism exhibits equilibrium dynamics or periodicity, whether the environmental variation is small or large and whether the organisms is modelled using structured or unstructured models.

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References

Albon, S.D. & Clutton-Brock, T.H. (1988) Climate and the population dynamics of red deer in Scotland. *Ecological Change in the Uplands* (eds M.B. Usher & D.B.A. Thompson), pp. 93–107. Blackwell Science, Oxford.

Albon, S.D., Clutton-Brock, T.H. & Guinness, F. (1987) Early development and population dynamics in red deer. II. Density-independent effects and cohort variation. *Journal of Animal Ecology*, **56**, 69–81.

Benton, T.G. & Grant, A. (2000) Evolutionary fitness in ecology: comparing measures of fitness in stochastic, density dependent environments. *Evolutionary Ecology Research*, **6**, 769–789.

Berryman, A.A. (1992) On choosing models for describing and analysing ecological time series. *Ecology*, **73**, 694–698.

Boonstra, R. & Hochachka, W.M. (1997) Maternal effects and additive genetic inheritance in the collared lemming (*Dicrostonyx groenlandicus*). *Evolutionary Ecology*, **11**, 169–182.

Bridges, T.S. & Heppell, S. (1996) Fitness consequences of maternal effects in *Streblospio benedicti* (Annelida, Polychaeta). *American Zoologist*, **36**, 132–146.

Caswell, H., Nisbet, R.M., de Roos, A.M. & Tuljapurkar, S. (1997) Structured population models: many methods, a few basic concepts. *Structured Population Models in Marine, Terrestrial and Freshwater Ecosystems* (eds S. Tuljapurkar & H. Caswell), pp. 3–17. Chapman & Hall, New York.

Crone, E.E. & Taylor, D.R. (1996) Complex dynamics in experimental populations of an annual plant, *Cardamine pensylvanica*. *Ecology*, **77**, 289–299.

Dennis, B., Desharnais, R.A., Cushing, J.M. & Costantino, R.F. (1995) Nonlinear demographic dynamics – mathematical models, statistical methods, and biological experiments. *Ecological Monographs*, **65**, 261–281.

Desharnais, R.A. & Costantino, R.F. (1985) Genetic-analysis of a population of Tribolium 8. The stationary stochastic dynamics of adult numbers. *Canadian Journal of Genetics and Cytology*, **27**, 341–350.

Desharnais, R.A. & Liu, L. (1987) Stable demographic limit-cycles in laboratory populations of *Tribolium castaneum*. *Journal of Animal Ecology*, **56**, 885–906.

Diekmann, O., Mylius, S.D. & ten Donkelaar, J.R. (1999) Saumon a la Kaitala et Getz, sauce Hollandaise. *Evolutionary Ecology Research*, **1**, 261–275.

Dodenhoff, J., Van Vleck, L.D. & Gregory, K.E. (1999) Estimation of direct, maternal and grandmaternal genetic effects for weaning weight in several breeds of beef cattle. *Journal of Animal Science*, **77**, 840–845.

Donohue, K. & Schmitt, J. (1998) Maternal environmental effects in plants: adaptive plasticity? *Maternal effects as adaptations* (eds T.A. Mousseau & C.W. Fox), pp. 137–158. Oxford University Press, Oxford.

Fox, C.W., Czesak, M.E., Mousseau, T.A. & Roff, D.A. (1999) The evolutionary genetics of an adaptive maternal effect: egg size plasticity in a seed beetle. *Evolution*, **53**, 552–560.

Fox, C.W. & Mousseau, T.A. (1998) Maternal effects as adaptations for transgenerational phenotypic plasticity in insects. *Maternal Effects as Adaptations* (eds T.A. Mousseau & C.W. Fox), pp. 159–177. Oxford University Press, Oxford.

Ginzburg, L.R. (1998) Inertial growth: population dynamics based on maternal effects. *Maternal Effects as Adaptations* (eds T.A. Mousseau & C.W. Fox), pp. 42–53. Oxford University Press, Oxford.

Ginzburg, L.R. & Taneyhill, D. (1994) Population cycles of forest Lepidoptera: a maternal effects hypothesis. *Journal of Animal Ecology*, **63**, 79–92.

Gliwicz, Z.M. & Guisande, C. (1992) Family planning in *Daphnia*: resistance to starvation in offspring born to mothers grown at different food levels. *Oecologia*, **91**, 463–467.

Greenman, J.V. & Benton, T.G. (2001) The impact of stochasticity on nonlinear population models: synchrony and the Moran effect. *Oikos*, **93**, 343–351.

Guckenheimer, J., Oster, G.F. & Ipatchki, A. (1977) The dynamics of density dependent population models. *Journal of Mathematical Biology*, **4**, 101–107.

Heath, D.D. & Blouw, D.M. (1998) Are maternal effects in fish adaptive or merely physiological side effects? *Maternal Effects as Adaptations* (eds T.A. Mousseau & C.W. Fox), pp. 178–201. Oxford University Press, Oxford.

Heath, D.D., Fox, C.W. & Heath, J.W. (1999) Maternal effects on offspring size: variation through early development of chinook salmon. *Evolution*, **53**, 1605–1611.

Hudson, P.J., Dobson, A.P. & Newborn, D. (1998) Prevention of population cycles by parasite removal. *Science*, **282**, 2256–2258.

Inchausti, P. & Ginzburg, L.R. (1998) Small mammal cycles in Northern Europe: patterns and evidence for a maternal effects hypothesis. *Journal of Animal Ecology*, **67**, 180–194.

Kaitala, V., Ranta, E. & Lindström, J. (1996) Cyclic population dynamics and random perturbations. *Journal of Animal Ecology*, **69**, 245–251.

Kaitala, V., Ylikarjula, J., Ranta, E. & Lundberg, P. (1997) Population dynamics and the colour of environmental noise. *Proceedings of the Royal Society of London B*, **264**, 943–948.

Krebs, C.J., Boutin, S., Boonstra, R., Sinclair, A.R.E., Smith, J.N.M., Dale, M.R.T., Martin, K. & Turkington, R. (1995) Impact of food and predation on the snowshoe hare cycle. *Science*, **269**, 1112–1115.

Kruuk, L.E.B., Clutton-Brock, T.H., Rose, K.E. & Guinness, F.E. (1999) Early determinants of lifetime reproductive success differ between the sexes in red deer. *Proceedings of the Royal Society London Series B*, **266**, 1655–1661.

Laakso, J., Kaitala, V. & Ranta, E. (2001) How does environmental variation translate into biological processes? *Oikos*, **92**, 119–122.

Leslie, P.H. (1959) The properties of a certain lag type of population growth and the influence of an external random factor on a number of such populations. *Physiological Zoology*, **32**, 151–159.

Lin, H.-C. & Dunson, W.A. (1995) An explanation of the high strain diversity of a self-fertilizing hermaphroditic fish. *Ecology*, **76**, 593–605.

May, R.M. & Oster, G. (1976) Bifurcation and dynamic complexity in simple ecological models. *American Naturalist*, **110**, 573–5499.

Mazer, S.J. & Wolfe. L.M. (1998) Density-mediated maternal effects on seed size in wild radish: genetic variation and its evolutionary implications. *Maternal Effects as Adaptations* (eds T.A. Mousseau & C.W. Fox), pp. 323–344. Oxford University Press, Oxford.

Metz, J.A.J., Nisbet, R.M. & Geritz, S.A.H. (1992) How should we define fitness for general ecological scenarios? *Trends in Ecology and Evolution*, **7**, 198–202.

Moore, A.J., Wolf, J.B. & Brodie, III, ed. (1998) The influence of direct and indirect genetic effects on the evolution of behaviour: social and sexual selection meets maternal effects. *Maternal Effects as Adaptations* (eds T.A. Mousseau & C.W. Fox), pp. 22–41. Oxford University Press, Oxford.

Moran, P.A.P. (1953) The statistical analysis of the Canadian lynx cycle. II. Synchronization and meteorology. *Australian Journal of Zoology*, **1**, 291–298.

Mousseau, T.A. & Fox, C.W. (1998a) The adaptive significance of maternal effects. *Trends in Ecology and Evolution*, **13**, 403–407.

Mousseau, T.A. & Fox, C.W. eds (1998b) *Maternal Effects as Adaptations*. Oxford University Press, Oxford.

Park, T. (1935) Studies in population physiology IV. Some physiological effects of conditioned flour upon *Tribolium confusum* Duval and its populations. *Physiological Zoology*, **8**, 91–115.

Pianka, E.R. (1970) On r and K selection. *American Naturalist*, **104**, 592–597.

Platenkamp, G.A.J. & Shaw, R.G. (1993) Environmental and genetic maternal effects on seed characters in *Neomophila menziessii*. *Evolution*, **47**, 540–555.

Price, T. (1998) Maternal and paternal effects in birds: effects on optimum fitness. *Maternal Effects as Adaptations* (eds T.A. Mousseau & C.W. Fox), pp. 202–226. Oxford University Press, Oxford.

Ranta, E., Kaitala, V. & Lundberg, P. (1997) The spatial dimension in population fluctuations. *Science*, **278**, 1621–1623.

Ranta, E., Lunderg, P., Kaitala, V. & Laakso, J. (2000) Visibility of the environmental noise modulating population dynamics. *Proceedings of the Royal Society, London, B*, **267**, 1851–1856.

Roff, D.A. (1984) The evolution of life history parameters in teleosts. *Canadian Journal of Fisheries and Acquatic Science*, **41**, 989–1000.

Rossiter, M.C. (1991) Environmentally based maternal effects: a hidden force in insect population dynamics? *Oecologia*, **87**, 288–294.

Rossiter, M.C. (1994) Maternal effects hypothesis of herbivore outbreak. *Bioscience*, **44**, 752–762.

Royama, T. (1992) *Analytical Population Dynamics*. Chapman & Hall, London.

Saatci, M., Dewi, I.A. & Ulutas, Z. (1999) Variance components due to direct and maternal effects and estimation of breeding values for 12-week weight of Welsh Mountain lambs. *Animal Science*, **69**, 345–352.

Schaffer, W.M. & Kot, M. (1986) Chaos in ecological systems: the coals that Newcastle forgot. *Trends in Ecology and Evolution*, **1**, 58–63.

Semlitsch, R.D. & Gibbons, J.W. (1990) Effects of egg size on success of larval salamanders in complex aquatic environments. *Ecology*, **71**, 1789–1795.

Sinervo, B. (1991) Experimental and comparative analyses of egg size in lizards: constraints on the adaptive evolution of maternal investment per offspring. *The Unity of Evolutionary Biology* (ed. E.C. Dudley), pp. 794–799. Dioscorides Press. Portland, Oregon.

Stearns, S.C. (1992) *The Evolution of Life Histories*. Oxford University Press, Oxford.

Stenseth, N.C., Falck, W., Chan, K.S., Bjornstad, O.N., O'Donoghue, M., Tong, H., Boonstra, R., Boutin, S., Krebs, C.J. & Yoccoz, N.G. (1998) From patterns to processes: phase and density dependencies in the Canadian lynx cycle. *Proceedings of the National Academy of Sciences of the USA*, **95**, 15430–15435.

Tosh, J.J., Kemp, R.A. & Ward, D.R. (1999) Estimates of direct and maternal genetic parameters for weight traits and backfat thickness in a multibreed population of beef cattle. *Canadian Journal of Animal Science*, **79**, 433–439.

Turchin, P. (1990) Rarity of density dependence or population regulation with lags? *Nature*, **344**, 660–663.

Turchin, P. & Taylor, A. (1990) Complex dynamics in ecological time series. *Ecology*, **73**, 289–305.

Wade, M.J. (1998) The evolutionary genetics of maternal effects. *Maternal Effects as Adaptations* (eds T.A. Mousseau & C.W. Fox), pp. 5–22. Oxford University Press, Oxford.

Witteman, G.J., Redfearn, A. & Pimm, S.L. (1990) The extent of complex population changes in nature. *Evolutionary Ecology*, **4**, 173–183.

Wulff, R.D. (1986) Seed size variation in *Desmodium paniculatum*. III. Effects on reproductive yields and competitive ability. *Journal of Ecology*, **74**, 99–114.

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