Evolutionary Ecology Research, 2016, **17**: 1–19

Environmental integration: patterns of correlation between environmental factors, early life decisions, and their long-term consequences

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ABSTRACT

Questions: How does the temporal correlation of key environmental variables (here temperature and precipitation) affect life-history decisions in early life, and what are the long-term consequences (accumulated damage, survival, and expected reproductive success)?

Hypothesis: Strong environmental correlation (environmental integration) is an important signal for the development of life-history traits, such as growth rate, phenotype, and reproduction, leading to higher fitness; plastic and fixed developmental strategies have different fitness depending upon the level of environmental integration.

Mathematical methods: A dynamic state-dependent model in which the state of the organism is characterized by mass, reproductive investment, and accumulated damage, all of which are affected by feeding activity and developmental costs mediated by the environment. Fitness is measured as expected lifetime reproduction.

Key assumptions: We assume that at each time step the resources gained by an individual through foraging activity are determined by developmental phenotype, which itself is the result of a decision process, and are then allocated to somatic growth, repair of cellular damage (e.g. oxidative stress) or gonadal tissue.

Results/Conclusions: (1) The differences in growth rate and reproductive investment between the plastic and fixed phenotypic strategies are greater at low levels (both positive and negative directions) of environmental integration. (2) Optimal resource allocation changes as a function of environmental gradient only for the plastic phenotypic strategy, and the difference in the onset of resource allocation between the plastic and fixed strategies is greater at low levels of environmental integration (i.e. the correlation between environmental factors and their fluctuation affects the reproductive timing decision though modification of resource allocation). (3) There is a marked difference in fitness when there is a low correlation and high fluctuation – conversely, the correlation has little effect when environmental fluctuation is low. (4) Even with costs, the investment in phenotype–environment matching has greater payoffs for individuals who are better able to track changes in their environment. Our results highlight that

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to understand the interactions between developmental decisions, we need to take into account not only the average environmental conditions but also their dynamics through time (variance and covariance).

Keywords: climate change, developmental phenotype, dynamic state model, growth, life history, reproduction.

INTRODUCTION

Understanding the pattern of spatial and temporal environmental fluctuation is important for ecological processes at all scales (Guadayol *et al.*, 2014). The effects of fluctuations, expressed in terms of means, trends, seasonality, and variances, have been studied at larger scales, whereas individual responses (e.g. behavioural and physiological variability) to environmental fluctuations occur mainly at small scales (e.g. Falciatore *et al.*, 2000; Benedetti-Cecchi, 2003). Therefore, it is important to characterize the whole spectrum of environmental mean and variance in order to understand how the responses of individuals to environmental perturbations scale up to an ecosystem response. In particular, the life-history decisions (e.g. growth rate, morphology) of organisms inhabiting environments with the same mean values for important environmental factors may depend not only on fluctuations through time, but also on how these factors co-vary in time.

The effect of multiple environmental factors is often assumed to be an additive accumulation of impacts associated with single factors (e.g. Halpern *et al.*, 2007; Ban and Alder, 2008). Some studies of multiple factors (Myers, 1995; Breitburg *et al.*, 1998) have suggested that the cumulative effects of environmental factors are greater than the effect of a single factor, whereas others have not (e.g. Crain *et al.*, 2008). We lack a conceptual framework to assess the effects of temporal changes of multiple environmental factors, and at least two major issues that can potentially impact fitness have to date been completely neglected. The first relates to the scale (frequency) of changes, in that two geographic regions could have similar means but different temporal patterns of fluctuation among multiple factors. The second relates to how environmental factors are correlated. Here, we introduce the concept of 'environmental integration' as the pattern of correlation between environmental factors in which two regions can have the same mean environmental factors and the same temporal scales, but different strengths of correlation (i.e. different levels of environmental integration) (Fig. 1). As such, we considered both a variety of patterns (levels) of environmental factors (Fig. 2a) and their correlations through time (Fig. 2b). Recent studies of several taxa have revealed that multiple environmental factors showing different patterns (high or low) have an effect on fitness (Table 1). Therefore, we hypothesize that high levels of environmental integration (positive or negative) may have important fitness consequences in the sense that such high levels may provide stronger signals to important life-history traits, such as growth, phenotype, and reproduction.

For a particular phenotype to be adaptive in response to environmental change, the phenotypic-induced fitness benefits in a given environmental condition must exceed the costs of the ability to be plastic [i.e. the 'cost of phenotypic development' (see Callahan *et al.*, 2008; Auld *et al.*, 2010)]. The cost of phenotypic development has been investigated with respect to development to some stage in life, such as the costs involved in the production of inducible phenotypes. Driessen *et al.* (2011) argued that phenotypic change in repeatedly fluctuating

Table 1. Experimental examples of two environmental factors that show different patterns

Species	Environmental factor #A	Pattern #A	Environmental factor #B	Pattern #B	Reference
Hyla eximia	Temperature	Low	Precipitation	High	Cruz-Ruiz *et al.* (2015)
Oenanthe oenanthe	Temperature	Low	Precipitation	Low	Oberg *et al.* (2015)
Mycteria leucocephala	Temperature	Low	Precipitation	High	Ismail and Rahman (2013)
Athene cunicularia	Temperature	High	Precipitation	High	Fisher *et al.* (2015)
Ambystoma maculatum	Temperature	High	Precipitation	Low	Sexton *et al.* (1990)
Somateria mollissima	Temperature	Low	North Atlantic Oscillation	High	Descamps *et al.* (2010)
Chen caerulescens	Temperature	High	Snow cover	Low	Dickey *et al.* (2008)
Rissa tridactyla	Sea surface temperature	High	Sea ice concentration	High	Moe *et al.* (2009)

Note: We searched for studies in which multiple environmental factors affected fitness. Pattern (level) of environmental factor (low or high) is represented in Fig. 2.

environments is a recurrent process and the costs of producing an environment-induced phenotype (i.e. cost of phenotypic development) will have to be paid multiple times. The total cost involved in developing a particular phenotype will then depend on the response to the environment and the magnitude of fitness reduction due to the cost of making environment-induced phenotypes. We hypothesized that phenotypic changes (or development) due to environmental fluctuations could alter life-history strategies as evolutionary trajectories of adaptive plasticity. First, developmental plasticity as an adaptive mechanism may buffer a developmental trajectory from deviations induced by environmental fluctuations in early life. Empirical evidence shows that the growth trajectory of juvenile organisms, consistent with the idea of life-history strategies in resource allocation or feeding activity, determined to maximize expected reproductive success, is predicted to respond to environmental change (e.g. Lee *et al.*, 2012, 2015). Second, environmental change may produce new phenotypes through gene regulatory circuits (Espinosa-Soto *et al.*, 2011). In other words, differences in adult phenotype among individuals can originate through differences in developmental condition early in life induced by environmental change.

Here, we construct a novel life-history model to support the concept of environmental integration. We assume that the model organism can evolve strategic decisions (e.g. phenotype and resource allocation) based on its physiological state and the environment (McNamara and Houston, 1996), and that individuals respond dynamically to condition, providing a basis to predict and adapt to a future environment. We focus on

enumerating all the life-history shapes (i.e. growth trajectory shapes) possible within a fairly broad range of the parameter space. Therefore, the model is general and flexible. We begin by introducing a state-dependent life-history model to explore (1) how the strength of correlation between important environmental features (e.g. temperature and precipitation) affects life-history traits in early life such as somatic growth, damage accumulation, and growth for gonadal tissue (reproductive investment); and (2) how the costs associated with different phenotypic strategies vary as a function of their response to environmental change in early life and the expected fitness in later life. We then discuss how the correlation between environmental features provides ecological signals for important life-history decisions.

THE MODEL

We characterize an individual by a set of states that describe variables such as somatic growth, development of reproductive tissue, and damage accumulation, which are related both to resource availability (i.e. food) and environmental conditions (temperature and precipitation).

Our model links stochastic environmental change (e.g. environmental fluctuation, seasonality) with individual life-history traits (survival, growth, and reproduction) for (i) patterns of fluctuation (different patterns of gradation in Fig. 1) and (ii) the correlation between environmental factors (i.e. temperature and precipitation in this study). Environmental change directly affects phenotypic variation and physiological processes (arrows in Fig. 1). Therefore, we consider both phenotypic and physiological decisions in the model. We assume that the costs of developing a particular phenotype to match a given environmental condition may affect resource allocation strategies involved in the developmental processes, which in turn affect the balance of anabolic and catabolic factors that determine growth.

It is well documented that a single genotype likely gives rise to distinct phenotypes when exposed to different environmental conditions (i.e. reaction norms), but age and size are clearly not independent traits, since change in one generally results in a correlated change in the other (Roff, 1992; Steams, 1992). In line with studies across a wide variety of species, we also consider the costs associated with developing a particular phenotype as the accumulated level of investment over the entire developmental period. For example, one may assume that up to a certain point, the greater the amount of environmental fluctuation, the higher the investment in phenotype–environment matching. We then predict that the optimal pattern of resource allocation and related fitness consequences may be ultimately related to optimal phenotypic decisions to match patterns of environmental fluctuation.

State-dependent life-history model

We build on Lee *et al.* (2011) and consider a state-dependent life-history theory governed by three state variables: mass $W(s)$, reproductive tissue $O(s)$, and the accumulation of oxidative or cellular damage $D(s)$ at the beginning of a time step (week) s. We assume that the resources gained by an individual are related to the level of activity a for a given developmental phenotype i (see Frankenhuis and Panchanathan, 2011). These are affected by key environmental factors (here we consider temperature and precipitation as the relevant environmental variables).

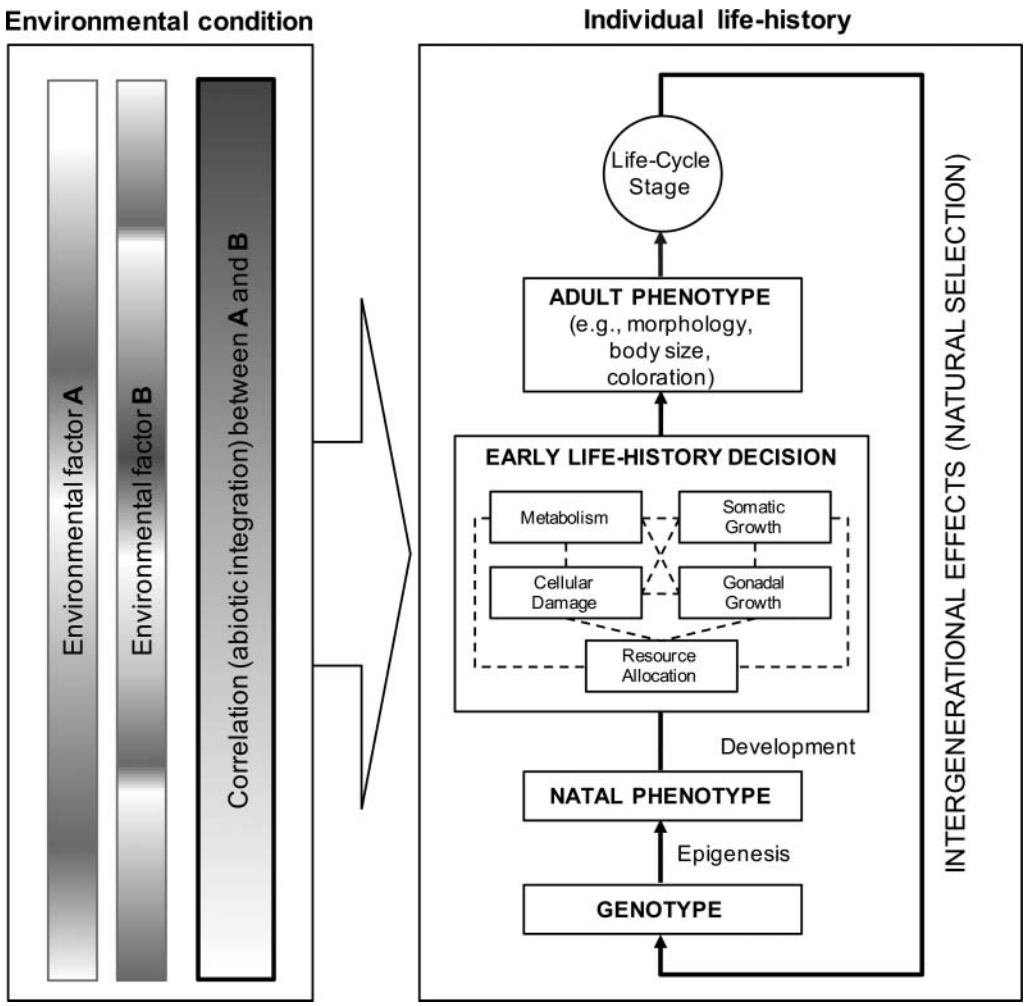

Fig. 1. Schematic representation of the model linking environmental change (left) to individual life history (right). Environmental conditions affecting individual life history are based on two environmental factors (A and B) differing in their temporal dynamics (represented by different patterns of gradation between two factors) and environmental integration (the correlation between two factors). In the individual life compartment (right), adult phenotype is determined by early life-history decisions (e.g. growth rate, resource allocation) that are a consequence of resource allocation due to the temporal dynamics of the two environmental factors as well as their correlations.

To explore how the fitness advantage of being able to switch phenotypes according to environmental changes is traded against the energetic cost of being able to maintain this ability, we consider two phenotypic strategies: (1) a fixed (but optimal) strategy that does not invest in adapting to changing conditions, and (2) a strategy capable of changing phenotypic trajectories (plastic strategy) as a way to track environmental changes. To do so, we consider two variants of the model. The first variant assumes that an individual has

a fixed phenotype – chosen to optimize fitness – throughout its entire life, averaged over environmental variation, whereas the second variant assumes that an individual may switch phenotypes within its lifespan.

For either strategy, the net gain of resource $N(a,i)$ depends on the level of activity a for a given developmental phenotype i and the balance of anabolic G and catabolic C factors:

$$N(a,i) = G(a,i) - C(a,i), \tag{1}$$

where the amount of food gained $G(a,i)$ is

$$G(a,i) = \eta A(a) I(i) W(s)^{0.75}. \tag{2}$$

In equation (2), η is the weight coefficient related to food consumption, and $A(a)$ and $I(i)$ denote the influence of activity and developmental phenotype on anabolism, respectively. We assume that no consumption occurs in the absence of activity and that consumption saturates with activity (Mangel and Munch, 2005):

$$A(a) = \frac{\xi a}{a + \kappa}, \tag{3}$$

where ξ is the maximum rate of resource gain related to activity and κ is the level of activity at which resource gain is half its maximum. We define the influence of developmental phenotype as the multiples of the anabolism as

$$I(i) = 1 + v_G i, \tag{4}$$

where v_G is the phenotypic gain coefficient for food consumption; i varies between 0 and 1 (Table 2) and controls the level of phenotypic flexibility (0 = most canalized phenotype and 1 = most flexible phenotype). Higher values of i lead to higher rates of resource gain during

Table 2. Summary of the parameter space searched

Parameter	Description	Range searched
ζ	Maximum rate of energy gain	1.5–1.75
κ	Half-saturation of food consumption	0.31–0.41
ε_T	Temperature coefficients for metabolic costs	0.01–0.1
ε_P	Precipitation coefficients for metabolic costs	0.001–0.5
m_r	Multiplier for time spent resting	0.005–0.1
ρ_D	Damage reinforcement rate	1.001–1.1
ρ_R	Energy to damage conversion	0.01–0.03
ϕ_a	Parameter for damage accumulation of activity level	0.201–0.321
ϕ_i	Parameter for damage accumulation of developmental phenotype	7.01–8.01
a_D	Activity level at zero damage	0.001–0.15
i_D	Phenotypic cost at zero damage	0.001–0.02
μ	Mortality rate while active	0.005–0.02
μ_r	Mortality rate while resting	0.005–0.05
μ_d	Mortality rate due to damage	0.002–0.02
μ_s	Mortality rate due to developmental phenotype	0.001–0.99
μ_b	Mortality rate during breeding season	0.01–0.1

growth, allowing the match between phenotype and environmental condition to control for consumption (e.g. Frédérich *et al.*, 2012).

Physiological costs $C(a,i)$ depend on a level of activity a for a given developmental phenotype i and temperature $T(s)$ and precipitation $P(s)$. For computation, we use an individual with a fixed phenotypic strategy (canalized) during its lifespan and the cost, C_{canal}, is

$$C_{canal}(a,i) = \alpha_s(a,i)[e^{\varepsilon_T T(s)} + \varepsilon_P P(s)]W(s), \tag{5a}$$

where $\alpha_S(a,i)$ is the specific metabolic cost at a level of activity a for a given developmental phenotype i, $T(s)$ is temperature (°C) at time s, $P(s)$ is precipitation at time s, and ε_T and ε_P are the temperature and precipitation coefficients for the metabolic costs of growth, respectively. Hence, $e^{\varepsilon_T T(s)} + \varepsilon_P P(s)$ characterizes the environmental costs of growth (Brett and Groves, 1979). The exponential cost of temperature on growth is well established (Brett and Groves, 1979). In the case of precipitation, however, indirect effects on development have been reported (Roitberg *et al.*, 1993; Voigt *et al.*, 2011), thus we assumed a linear effect.

An individual that maintains the ability to switch phenotypes (a plastic or flexible phenotypic strategy) pays an additional energetic cost for this ability, so we replace equation (5a) by

$$C_{flex}(a,i) = \alpha_S(a,i)[e^{\varepsilon_T T(s)} + \varepsilon_P P(s) + \omega]W(s), \tag{5b}$$

where ω is the additional cost for maintaining the ability to switch phenotypes. Clearly, equation (5b) reduces to equation (5a) when $\omega = 0$ but we separate them to help emphasize the difference.

The specific metabolic cost for a given level of activity a for a given developmental phenotype i is

$$\alpha_S(a,i) = a\alpha + (1-a)\alpha m_r + v_C i, \tag{6}$$

and depends upon the weight-specific catabolic rate α, the multiplier for time spent resting m_r, and developmental phenotype where v_C is the coefficient for phenotype related to metabolic costs. With this framework there is an intermediate optimal level of activity (Mangel and Munch, 2005).

In each time step, we assume that individuals allocate net resource gains to growth of somatic or gonadal tissue and repair of damaged cells. Resource allocation is also related to the level of activity for a given developmental phenotype to be adaptive, in that individuals having different costs to develop a particular phenotype may have different resource investment in life-history traits. If f_W is the fraction ($0 \le f_W \ge 1$) of resource allocated to mass gain, then body mass $W(s)$ follows the dynamics

$$W(s+1) = W(s) + f_W N(a,i). \tag{7}$$

Reproductive tissue (e.g. oocyte) $O(s)$ follows the dynamics

$$O(s+1) = O(s) + f_O N(a,i), \tag{8}$$

where f_O is the fraction ($0 \le f_O \le 1 - f_W$) of resource allocated to reproductive tissues. Allocation to the repair of damage is then $(1 - f_W - f_O)$. Finally, the level of damaged tissue follows the dynamics

$$D(s+1) = \rho_D D(s) - \rho_R(1 - f_W - f_O)N(a,i) + \phi_a(a - a_D) + \phi_i(i - i_D), \tag{9}$$

where ρ_D is the rate at which damage is reinforced, ρ_R is the efficiency of repair that links repairing investment to actual reduction in damage, ϕ_a connects activity to damage, a_D is the level of activity that results in zero net production of damage (i.e. repair = production), i_D is the phenotypic level resulting in zero damage, and ϕ_i connects phenotype to damage (for further details, see Lee *et al.*, 2011).

We assume that the probability of survival depends upon the level of activity, the accumulated damage (cf. Mangel and Munch, 2005; Lee *et al.*, 2011), and a given developmental phenotype according to

$$\beta(a,i) = e^{-\mu a - \mu_r(1-a) - \mu_d d - \mu_s i}, \tag{10}$$

where the mortality rate while active (e.g. as a result of predation) is μ, the mortality rate while resting is μ_r, the mortality rate due to damage is μ_d, the level of damage is d, and the mortality rate due to developmental phenotype is μ_s (Table 2).

To determine the optimal time- and state-dependent pattern of activity for a given developmental phenotype, which in turn determines the pattern of growth and survival (i.e. resource allocation), we assumed that the expected reproductive success at the end of the fixed growth interval, when mass is $W(s)$, is

$$F(w,o,d,S) = (W(S) - w_C)^{\phi}, \tag{11}$$

where w_C is the critical mass required for reproduction and ϕ is an allometric parameter relating reproductive output to mass (Table 2).

We require two fitness functions (Mangel and Clark, 1988; Mangel and Ludwig, 1992; Houston and McNamara, 1999; Clark and Mangel, 2000). For the fixed phenotypic strategy, we let $\bar{F}(w,o,d,s|i)$ denote the maximum expected terminal fitness for an individual with fixed phenotype i given that mass at time s is w, accumulated oocyte mass is o, and the level of damage is d. The optimal choice for a fixed phenotypic strategy is then found by choosing the value of i that maximizes $\bar{F}(w_0,0,d_0,1|i)$, where w_0 and d_0 are the initial mass and level of damage, respectively (we assume no gonads at the start). We let $\bar{F}^*(w_0,0,d,1)$ denote the maximum fitness for an individual with a fixed phenotype strategy.

Each of the $\bar{F}(w,o,d,s|i)$ satisfy the end condition

$$\bar{F}(w,o,d,S|i) = oe^{-\mu_b d}, \tag{12}$$

where μ_b is the mortality rate during the breeding season. For previous time steps, we have the equation of stochastic dynamic programming (Mangel and Clark, 1988; Mangel and Ludwig, 1992; Houston and McNamara, 1999; Clark and Mangel, 2000):

$$F_{canal}(w,o,d,s|i) = \max_a \max_{f_W f_O} \left[\beta(a,i) F_{canal} \begin{pmatrix} w + f_W N(a,i), \\ o + f_O N(a,i), \\ \rho_D d - \rho_R(1 - f_W - f_O)N(a,i) + \phi_a(a - a_D) + \\ \quad \phi_i(i - i_D), \\ s + 1 | i \end{pmatrix} \right], \tag{12a}$$

where $N(a,i)$ is computed using equation (5a).

Our second fitness function $F_{flex}(w,o,d,s)$ is the maximum expected terminal fitness for an individual with a plastic phenotypic strategy. This function satisfies the same end condition as for equation (11) but a different equation of dynamic programming, in which maximization is taken over phenotype as well as activity and allocation:

$$F_{flex}(w,o,d,s) = \max_{i} \max_{a} \max_{f_W f_O} \left[\beta(a,i) F_{flex} \begin{pmatrix} w + f_W N(a,i), \\ o + f_O N(a,i), \\ \rho_D d - \rho_R (1 - f_W - f_O) N(a,i) + \phi_a (a - a_D) + \\ \quad \phi_i (i - i_D), \\ s + 1 \mid i \end{pmatrix} \right], \tag{12b}$$

where $N(a,i)$ is computed using equation (5b).

The fitness advantage of plasticity is then measured by

$$\Delta F(\omega) = F(w_0,0,d,1) - \bar{F}^*(w_0,0,d,1), \tag{13}$$

which will be a declining function of ω and will depend upon the environment (average values and their correlations through time).

At each time step and state in the solution of equations (12a) and (12b), we determined the optimal levels of activity $a^*(w,s)$ and developmental phenotype $i^*(w,s)$ that maximize the fitness function. Given these values, we then simulate the trajectory of life history by a forward Monte Carlo simulation (Mangel and Clark, 1988; Clark and Mangel, 2000), feeding the calculated values for optimal activity and developmental phenotype (i.e. reflecting a change in the amount of mass gained during early growth) at each time step into equations (1–9).

The virtual life-history experiment

Since the space of possible parameter combinations is too broad to investigate completely, we used Monte Carlo simulations to sweep over the parameter space (see Mangel and Munch, 2005; Munch and Mangel, 2006; Mangel, 2008). To maximize variation within our parameter space, we divided the range of each parameter into 30 (or 20 in the case of two parameters; see Table 2) uniformly distributed values and sampled from these values instead of any possible value within the parameter range, which in many instances could have generated individuals very similar to each other for any given parameter. An individual was thus characterized by a random selection of values within intervals across all parameters. In total, the same 10,000 individuals (parameter combinations) were used across all simulations (i.e. fixed and plastic strategies and across different patterns and correlations of environmental fluctuation; see next section).

Simulation and analysis

To consider both a variety of environmental factors (fluctuations in temperature and precipitation in this study; Fig. 2a) and their correlations through time (Fig. 2b) in a systematic way, we combined three levels of fluctuation (θ_T and $\theta_P = 0.1$, 0.5, and 1.0: low, intermediate, and high, respectively; see www.evolutionary-ecology.com/data/3021Appendix.pdf) across six levels of correlation between temperature and precipitation [high ($r = 0.9$ or -0.9), intermediate ($r = 0.5$ or -0.5), and low ($r = 0.1$ or -0.1) degree of correlation respectively, in both positive and negative directions]. Thus, overall within the combinations of fluctuation and correlation there were 18 manipulation groups (three levels of fluctuation × six levels of correlation), which enabled us to examine the effects of environmental integration (correlation) on life-history traits and to determine whether the costs associated with a

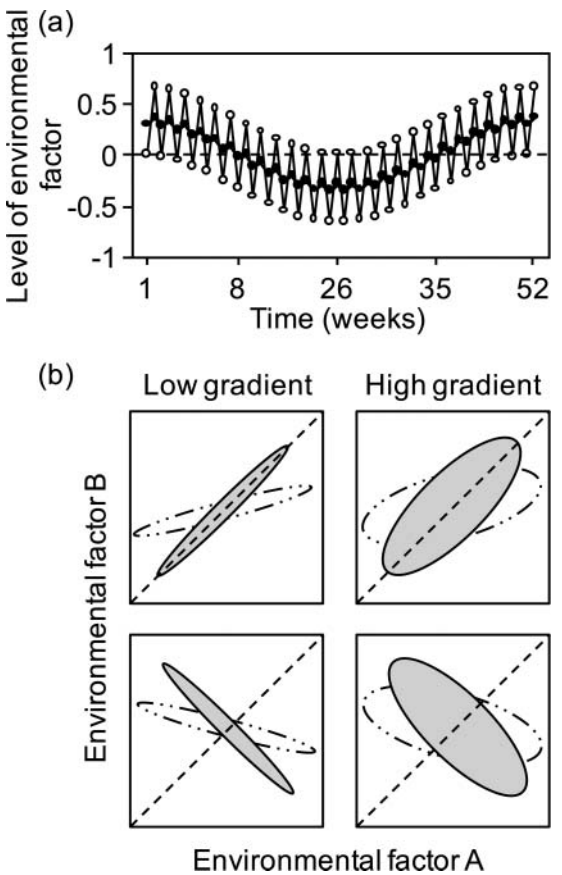

Fig. 2. (a) Simulated annual pattern (level) of an environmental factor (temperature or precipitation in this study; see 3021Appendix.pdf): solid circle = the shallowest gradient, open circle = the steepest gradient. (b) Patterns of correlation between environmental factor A and environmental factor B (environmental integration) vary according to the level (gradient) of environmental fluctuation [either positive (above) or negative (below)]: low gradient (left) and high gradient (right). Large ellipses (right) represent high gradients (high environmental fluctuation), while narrow ellipses (left) represent low gradients (low environmental fluctuation). There are different coefficients of correlation (environmental integration): high correlation (ellipses with solid lines) and low correlation (ellipses with dashed lines).

different phenotype or strategy vary as a function of their response to environmental fluctuation.

To investigate how decisions in early life and the associated costs of phenotypic development in response to environmental integration affect long-term consequences, we computed growth rate, reproductive investment (mass of reproductive tissue at the onset of the breeding season), and accumulated damage (total amount of damaged tissue). In particular, we compared the differences in all life-history traits and fitness between plastic and fixed phenotypic strategies to examine how the costs and benefits associated with different phenotypic strategies vary as a function of their response to environmental change (environmental integration).

To examine how the variation in the additional cost associated with maintaining the ability to switch phenotypes affects fitness, we simulated four different cases (increase or decrease of 60% or 30% of the additional cost, respectively) and calculated their fitness difference between the plastic and fixed phenotypic strategies.

For any given combination of fluctuation and correlation, we followed 10,000 individuals (parameter combinations; see previous section on 'The virtual life-history experiment'). We implemented the simulation model in Microsoft Visual Basic 2010 (Microsoft, 2010) and code can be obtained from the corresponding author.

RESULTS

In Figure 3, we show model predictions between life-history traits and the strength of correlation between temperature and precipitation across different levels of environmental fluctuation through time (i.e. gradient). Differences in growth rate (Fig. 3a) and reproductive investment (Fig. 3c) between the plastic and fixed phenotypic strategies were greater

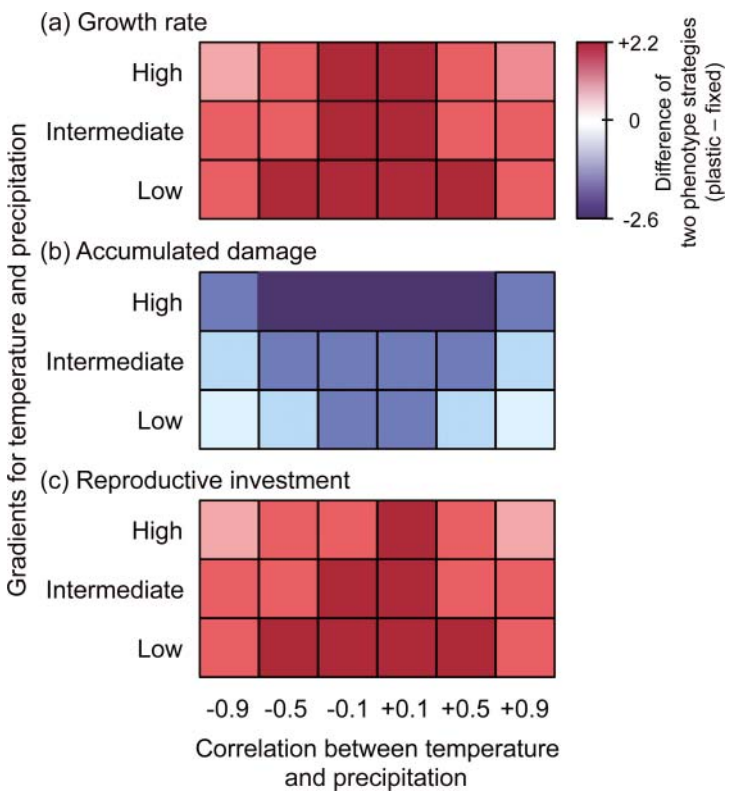

Fig. 3. Difference (normalized mean) in (a) growth rate, (b) accumulated damage, and (c) reproductive investment between the plastic and fixed phenotypic strategies in relation to the correlation between temperature and precipitation (environmental integration) for different levels of environmental fluctuation (low, intermediate, and high). The deeper the red, the higher the value of the plastic phenotypic strategy, whereas the deeper the blue, the higher the value of the fixed phenotypic strategy.

when the correlation between environmental factors was low (both positive and negative directions), while the difference in accumulated damage was more negative the lower the correlation (Fig. 3b). Similarly, differences in growth rate (Fig. 3a) and reproductive investment (Fig. 3c) differed with environmental fluctuation, with more marked differences at a low environmental gradient, while differences in accumulated damage were greater at a high environmental gradient (Fig. 3b).

In Figure 4, we show how the strength of correlation between temperature and precipitation across different levels of environmental fluctuation through time are related to optimal allocation of resources to reproduction. Optimal allocation of resources changes as a function of environmental gradient for the plastic phenotypic strategy only, not the fixed strategy (Fig. 4): the onset of resource allocation to reproductive tissue for the plastic

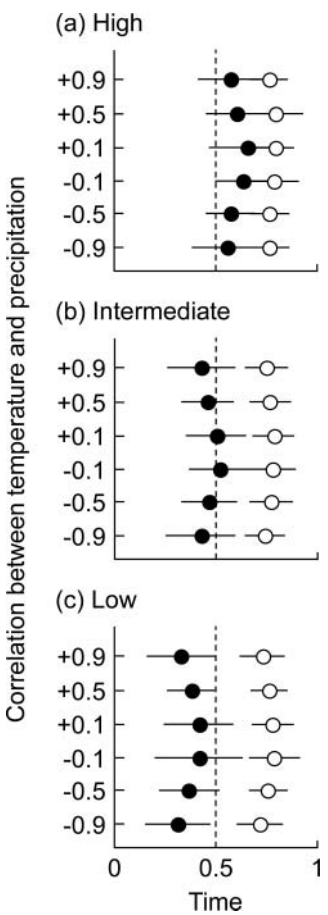

Fig. 4. Time (normalized mean ± S.D.) when resource is first allocated to reproductive tissue in relation to the correlation between temperature and precipitation (environmental integration) for the plastic (solid circle) and fixed (open circle) phenotypic strategies. The strength of the correlation varies according to three levels (gradients) of environmental fluctuation: (a) high gradient, (b) intermediate gradient, and (c) low gradient. Normalized time is from 0 (the initiation of life) to 1 (the onset of the breeding season). The dashed line represents the middle of life during the developmental period.

strategy was faster at a low environmental gradient. Furthermore, differences in the onset of resource allocation between the plastic and fixed strategies were greater when the correlation was low (Fig. 4). In other words, the correlation between environmental factors and their fluctuation affects the reproductive timing decision by modifying allocation of resources, so that the growth of reproductive tissues (reproductive investment) is different.

In Figure 5, we compare differences in fitness of the two phenotypic strategies (i.e. plastic and fixed) with strength of correlation between temperature and precipitation and level of fluctuation over time. There was a marked difference in fitness with a low correlation and high fluctuation; in contrast, the correlation between environmental factors had little effect when the environment fluctuated. The strength of correlation has a clear effect on fitness, and this effect is particularly strong when the fluctuation in environmental factors is relatively higher.

In Figure 6, we show how differences in fitness between the plastic and the fixed phenotypic strategies are related to the additional cost of maintaining the ability to switch phenotypes (ω). Here, for any strength of correlation, we averaged across all 30,000 individuals for all three levels of environmental fluctuation. Our model shows that the difference in fitness is a negative function of the absolute strength of correlation between temperature and precipitation (Fig. 6a) and is negatively related to the additional cost of maintaining the ability to switch phenotypes (Fig. 6b). This result holds true even for

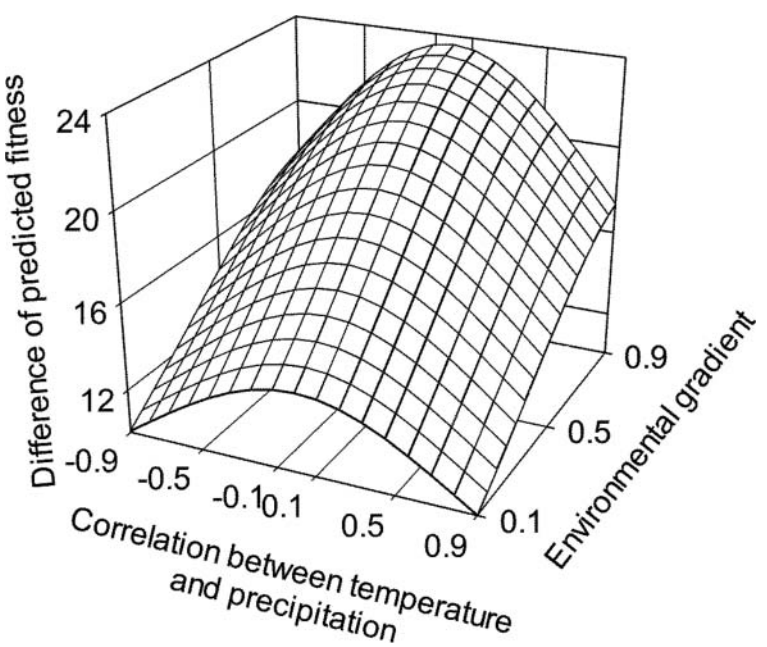

Fig. 5. Differences in fitness between the two phenotypic strategies (plastic and fixed) and the correlation of temperature and precipitation (environmental integration) as well as environmental fluctuation over time. Values are based on the mean of three different environmental gradients (low = 0.1, intermediate = 0.5, and high = 0.9) for different strengths of correlation between temperature and precipitation.

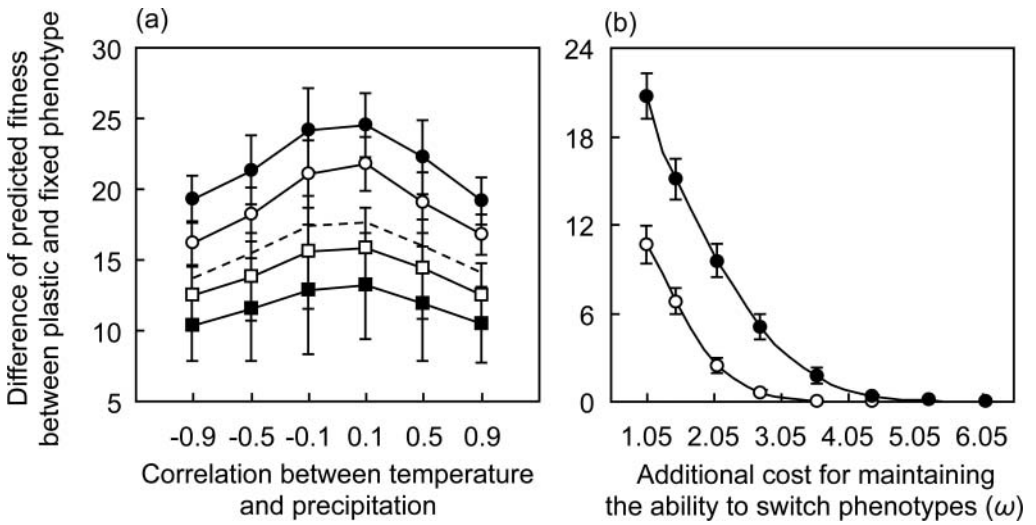

Fig. 6. (a) Differences in fitness between the plastic and fixed phenotypic strategies in relation to the correlation between temperature and precipitation for different additional energetic costs of maintaining the ability to switch phenotypes (ω) versus the original costs used in all simulations: solid squares represent an increase of 60%, open squares an increase of 30%, open circles a decline of 30%, and solid circles a decline of 60%. Values are based on the mean of three different environmental gradients (low, intermediate, and high) for the strength of correlation between temperature and precipitation. (b) Differences in fitness between the plastic and fixed phenotypic strategies in relation to the additional costs of maintaining the ability to switch phenotypes (ω): open circle = the fitness differences are small, solid circle = the fitness differences are large.

increasingly relative amounts of energy invested in phenotype–environment matching. Therefore, in our model, investment in phenotype–environment matching is beneficial, as individuals are better able to track changes in their environment, thus increasing their relative fitness compared with the fixed phenotype.

DISCUSSION

We have explored how environmental integration (patterns of correlation between environmental factors) is related to early life-history decisions and fitness consequences, ultimately demonstrating how these interactions affect the tempo of adaptive phenotype–environment matching. By combining a dynamic state-dependent model and virtual experiments (utilizing a broad parameter space), we conclude that temporal dynamics in important environmental features critically affects life-history traits, including optimal choice of resource allocation, growth rate, reproductive investment, and stress accumulation. To our knowledge, this is the first study to focus on fitness consequences and environmental integration (temporal dynamics and patterns of correlation in environmental features). Our model shows that higher environmental integration (i.e. a high correlation between temperature and precipitation) led to greater reproductive investment and fitness consequences, while the environmental gradient is a negative function of the link between environmental integration and life-history traits.

The costs of producing an environment-induced phenotype (developing a particular phenotype to match a given environmental condition) could be changed through a direct relationship between environmental fluctuation and an adaptive strategy. For example, more variable environments (i.e. temperature or precipitation) could negatively affect growth and potential reproduction if they decrease the efficiency by which an individual allocates resources between growth and the ability to switch phenotypes (i.e. allocation strategies). There is some evidence to suggest that rapid fluctuations in temperature can interfere with the optimal strategy for resource allocation (Kozłowski *et al.*, 2004) and cause adjustments to juvenile phenotypes (Shine and Harlow, 1996), but the impacts of rapid fluctuations on phenotypic adaptation remain unclear (but see Martin and Leberg, 2011). Extreme environmental variation could also negatively affect phenotypic adaptation if it increases cellular damage (e.g. metabolic or oxidative stress) and thereby decreases the amount of energy available for growth and reproduction. Recently, Telemeco *et al.* (2013) found that extreme nest temperature in painted turtles (*Chrysemys picta*) increased the frequency of abnormal shell morphologies, which in turn are negatively correlated with survival and fitness. However, the effects of accumulated stress on the costs of phenotypic plasticity remain equivocal (Storz *et al.*, 2010; Kelly *et al.*, 2012).

The results of our model suggest that patterns of temporal variation in temperature and precipitation (environmental integration) may affect life-history decisions profoundly, even if the means are the same across different environments. In this case, two species living in different habitats with the same annual mean temperature and precipitation may have very different life-history decisions to make (involving, for example, foraging behaviour, reproduction, and phenotypic costs) if the correlation between temperature and precipitation is different across habitats. Moreover, the timing of key events in an organism's life history, such as breeding season, may also be different across habitats based on different decisions in resource allocation induced by different environmental integration. Robin *et al.* (2009) showed that food availability in the Australian avifauna living in different locations with similar temperature depends very strongly on rain. Increased metabolic rates due to high temperatures can be attenuated during periods of higher precipitation (e.g. Voigt *et al.*, 2011). Taken together, both negative and positive effects on life-history traits may depend on the correlation between temperature and precipitation, or other important environmental factors.

Adaptation (via phenotype–environment matching) can speed up the evolutionary process if phenotypic modification is well synchronized with environmental variation. For instance, changes in a particular phenotype (e.g. size or morphology) vary across environmental conditions because impacts of environmental fluctuation on the phenotype can act directly on the developmental processes through changing cell size and number (cellular growth) as well as the trade-off between growth and reproduction. Márquez-García *et al.* (2009) showed that environmental heterogeneity (different correlations between temperature and desiccation) promotes phenotypic variation in both morphology and life-history traits in an anuran (*Rhinella spinulosa*). In particular, Tejedo *et al.* (2010) found that higher temperatures induced smaller heads in frogs (16 species) without a shift in hind-leg length. Although phenotype–environment matching can be initiated by current environmental stimuli or cues, organisms can also evolve mechanisms to sense and adaptively respond to certain cues that predict environmental change (Nijhout, 2002). For instance, the change of hydraulic condition that may predict weather (rain or temperature) affects development rate in stonefly (*Nemoura cinerea*) nymphs (Franken *et al.*, 2008) and penis length in barnacles (*Balanus glandula*) (Neufeld and Palmer, 2008).

While adaptive changes through phenotype–environment matching may increase fitness if the changes are rapid enough to affect the ecological dynamics (Hairston *et al.*, 2005; Miner *et al.*, 2005), there may also be an overwhelmingly detrimental fitness cost in other circumstances. Roff (1996) suggested that the plastic production of large spines or heavy armour in an organism (e.g. a prey) in response to the environmental conditions or presence of predators may enhance the mechanism for adaptation or defence, but may also have negative effects on feeding, migration, mating or reproduction – an illustration of the trade-offs involved in different phenotypic strategies. Hence, the advantage of any phenotype is relative to specific spatial and temporal conditions. The benefits and costs associated with different phenotypes vary across different environments as a function not only of mean conditions but also patterns of correlations across relevant environmental factors, which is important for understanding adaptive phenotype–environment matching. This finding is particularly relevant because species distribution and climate biodiversity models use average values of climatic variables through time (e.g. Araújo *et al.*, 2009). Whether patterns of local extinction and colonization are also affected by how life-history decisions are made in relation to patterns of covariation among relevant environmental factors ('environmental integration') remains to be investigated, but our model indicates that these should also be relevant when coupling population models with bioclimatic habitat models (e.g. Keith *et al.*, 2008).

In conclusion, we have shown that life-history optimization leading to different phenotypic strategies has consequences early in life (growth, long-term damage accumulation and reproductive investment) together with fitness in response to environmental integration due to a trade-off between the costs of developmental phenotype and resource allocation. Moreover, our results show that the degree and direction of environmental integration (the correlation between important environmental variables; here, temperature and precipitation) affect the timing of life-history events through phenotypic development and resource allocation. Our results also emphasize that an understanding of the interactions between developmental decisions needs to take into account not only the environmental conditions but also their dynamics through time.

ACKNOWLEDGEMENTS

W.S.L. was funded by an FQRNT (Québec) postdoctoral fellowship. P.R.P.N. was supported by the Canada Research Chair program (Chair in Spatial Modelling and Biodiversity) and NSERC Discovery grants. P.R.P.N. was funded by the 'Fonds vert' of the Government of Québec via the Ouranos Consortium on Regional Climatology and Adaptation to Climate Change (project 554010-107).M.M. was partially funded by NSF grants EF-0924195, OCE-1130483, and DEB 14-51931

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