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Testing the role of phenotypic plasticity for local adaptation: growth and development in time-constrained *Rana temporaria* populations

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time constraints;
trade-off.

Abstract

Phenotypic plasticity can be important for local adaptation, because it enables individuals to survive in a novel environment until genetic changes have been accumulated by genetic accommodation. By analysing the relationship between development rate and growth rate, it can be determined whether plasticity in life-history traits is caused by changed physiology or behaviour. We extended this to examine whether plasticity had been aiding local adaptation, by investigating whether the plastic response had been fixed in locally adapted populations. Tadpoles from island populations of *Rana temporaria*, locally adapted to different pool-drying regimes, were monitored in a common garden. Individual differences in development rate were caused by different foraging efficiency. However, developmental plasticity was physiologically mediated by trading off growth against development rate. Surprisingly, plasticity has not aided local adaptation to time-stressed environments, because local adaptation was not caused by genetic assimilation but on selection on the standing genetic variation in development time.

Introduction

Phenotypic plasticity is the ability of one genotype to express more than one phenotype depending upon environmental conditions (DeWitt & Scheiner, 2004). Theoretical models (e.g. Via & Lande, 1985; Sultan & Spencer, 2002) as well as studies in natural systems (Richter-Boix *et al.*, 2006; Lind & Johansson, 2007; Hollander, 2008; Lind *et al.*, 2011) have shown that phenotypic plasticity is a beneficial strategy in a heterogeneous environment. In addition to this well-known benefit of adaptive phenotypic plasticity, it has also been suggested that ancestral plasticity can be beneficial when adapting to novel environments, by a mechanism known as genetic accommodation (West-Eberhard, 2003; Lande, 2009). According to the genetic accommodation model, the presence of plasticity in a trait can enable survival in a novel environment long enough for a change in the trait mean or in the degree of plasticity to evolve (as originally suggested by Baldwin, 1896). Genetic accommodation is proposed to be a process involving three steps; first, plasticity enables survival, which allows the mean trait expression to change (Baldwin effect), which may be followed by canalization and reduction in plasticity in the new environment (genetic assimilation) (Waddington, 1952, 1961).

Plasticity has only recently been invoked as an important mechanism for local adaptation (Price *et al.*, 2003; Crispo, 2007; Lande, 2009) and speciation (West-Eberhard, 2003; Crispo, 2007). In nature, the role of plasticity in local adaptation and speciation has been investigated by measuring the plasticity of adaptive traits in ancestral lines, and investigating whether this ancestral plasticity mirrors the specialization in the more recent lines that have evolved a change in the mean trait value. Indeed, there are now documented cases where plasticity seems to have aided adaptation to new environments (Gomez-Mestre & Buchholz, 2006; Kamimura, 2006; Wund *et al.*, 2008; Scoville & Pfrender, 2010).

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However, determining the role of plasticity in local adaptation is not easy, because plasticity in a trait may be present in the founder population yet be of no importance for the local adaptation of the derived populations. Moreover, local adaptation may be aided by the presence of plasticity, which enables survival in the novel environment, but the mechanism of adaptation can still be different from the mechanism behind the plastic response. For example, plasticity in development time among spadefoot toads and parsley frogs is mediated physiologically by adjusting the levels of thyroid hormone, whereas the local adaptation of the different species is governed by their sensitivity to the hormone (Gomez-Mestre & Buchholz, 2006). If the mechanism of plasticity is known on the hormonal or gene expression level, its contribution to local adaptation can be investigated (Gomez-Mestre & Buchholz, 2006; Scoville & Pfrender, 2010). However, if the mechanistic basis of plasticity is not known, there is no simple way to test whether the mechanism behind the initial plasticity has been important for local specialization.

One situation where it is possible to bypass this need for knowledge of such physiological detail is in the case of life-history plasticity. Here, we suggest, following Ball & Baker (1996) and Beckerman *et al.* (2007), the connection between growth rate and development rate is a fruitful avenue for a better understanding of the mechanism of phenotypic plasticity. By linking plasticity and local adaptation in a variable environment, it can be understood whether variation in life history is governed either via behaviour or by a facultative life-history shift mediated by a change in physiology (Ball & Baker, 1996).

Conceptual model

It is well known that organisms, experiencing a time-stressed environment, might increase both development rate and growth rate (Fig. 1a), for example by increased food intake (Abrams *et al.*, 1996; Johansson & Rowe, 1999; Stoks *et al.*, 2005). This is known as a behavioural regulation of the life history (Stoks *et al.*, 2005; Beckerman *et al.*, 2007) and implies that the organisms will develop faster without a substantial reduction in final mass. However, organisms may instead trade off final mass for an increase in development rate, if less investment in mass gives the possibility to develop faster. The second mechanism is not obtained by a changed behaviour, but is regarded as a facultative life-history shift (Fig. 1b), mediated by physiological mechanisms (Ball & Baker, 1996; Beckerman *et al.*, 2007). Because the organisms increase the development rate without increasing growth rate, the shorter time available for growth will give the organisms a smaller final mass.

The model of genetic accommodation and assimilation by Lande (2009) is useful when analysing the consequence of phenotypic plasticity for local adaptation to a time-constrained environment. In the absence of plasticity, selection will favour individuals with high development rate, which is also individuals with high growth rate, because they typically are correlated within a population and determined by foraging activity (Abrams *et al.*, 1996). Thus, populations with high mean development rate will also have high growth, and the slope of the population means in development and growth rates will follow the slope of the individuals within each population (Fig. 1c). Therefore, the local adaptation of populations will be based upon the standing genetic variation in growth and development rate. The same pattern will be present if the individuals express plasticity to time constraints by increasing foraging activity.

However, if plasticity is present as a facultative life-history shift (Ball & Baker, 1996), the predictions can change. By a plastic change in their physiology, independent of their foraging activity, individuals experiencing time constraints increase their development rate without a corresponding increase in their growth rate (Beckerman *et al.*, 2007, 2010). If genetic accommodation takes place, it is predicted that this physiological shift in the life history, caused by plasticity, will be genetically accommodated in populations constantly experiencing time constraints and expressed regardless of the environment (Lande, 2009). Thus, we can predict that individuals that are locally adapted to time-constrained environments will have considerably higher development rate than individuals from populations adapted to less time-constrained environments, without an equally large change in their growth rate. As a consequence, a regression of the population means of development rate on growth rate (which is a result of genetic accommodation of the physiological life-history shift) will have a steeper slope than regressions of the within-population development rates on growth rate (which are determined by different foraging efficiency among the individuals within each population); see Fig. 1d. If local adaptation is caused by genetic accommodation of a physiological life-history shift originally only expressed by plasticity, this will leave a clear signal in the growth and development rates of individuals of different populations. The approach of comparing within and among population patterns is powerful, and a similar approach has recently been used to investigate the role of local adaptation and plasticity for egg-laying date in the common frog *Rana temporaria* across Britain (Phillimore *et al.*, 2010).

Testing the conceptual model

An excellent system to test whether local adaptation in life-history traits was caused by the genetic accommodation of a plastic response, as outlined in the conceptual model above, is the island system of the common frog (*R. temporaria*) off the Baltic coast in northern Sweden. The island populations were founded during the last 70–800 years (Johansson *et al.*, 2005) by individuals migrating from mainland populations (Lind *et al.*, 2011); these

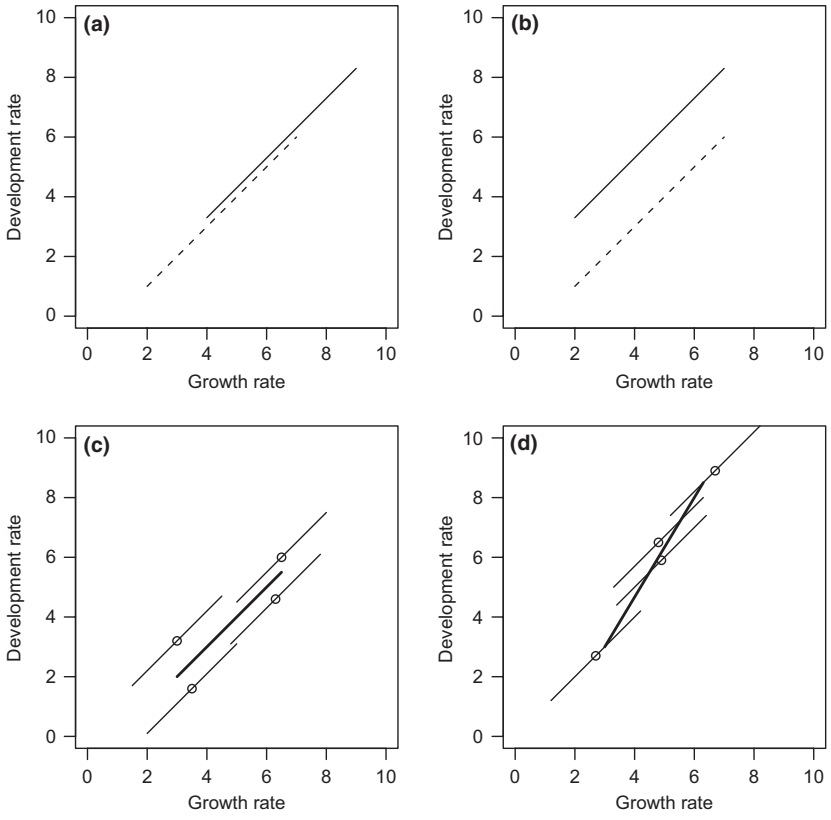

Fig. 1 Predicted relationship between development rate and growth rate among genotypes and populations in response to pool drying. (a) and (b) represents the slope of population means in two treatments, whereas (c) and (d) illustrates the slope of individual populations and the overall population means under the same treatment. A linear relationship between development rate and growth rate of genotypes is predicted if the individual differences are caused by different behaviours (a). Note that individuals under drying water conditions (*solid line*) increase their development and growth rates compared to individuals under constant water conditions (*hatched line*). The increase in development and growth rates is achieved with an increased foraging effort. However, if the phenotypic plasticity in development rate is caused by a facultative life-history shift, the model predicts a shift in the relationship between development and growth rates when comparing individuals in the artificial drying treatment (*solid line*) and the constant water level treatment (*hatched line*) (b). This model can be extended to investigate the basis of local adaptation in populations that live in different environments that select for different developmental rates. If plasticity in development time is not present, or caused by increased foraging activity, genotypes with high foraging efficiency will be selected for in populations experiencing pool drying. Thus, the local adaptation is caused by the same mechanisms that are responsible for the differences in development rate among genotypes (e.g. foraging effort), and we predict that the slope of the growth rate–development rate regression should be equal whether you compare genotypes within a population (*open circles, dotted lines*) or among populations (*bold line*) (c). Points lying off the bold line indicated physiological differences among the populations. However, if plasticity is present and caused by a facultative life-history shift where development rate is traded off against growth rate, adaptation by genetic accommodation will result in a steeper slope when comparing populations than when comparing genotypes, because the individual differences in development rate among genotypes from the same population still is caused by different foraging efficiency (d).

populations express phenotypic plasticity in development rate (Almfelt, 2005), which likely represents the ancestral state. The frogs breed in rocky pools that vary in their water permanence (from temporary to permanent pools). Individual survival depends on completion of larval development and metamorphosis before the end of season or before the pool dries up. However, a short development time bears the consequence of decreased metamorphic weight (Lind & Johansson, 2007), an important fitness trait (Smith, 1987; Altwegg & Reyer, 2003). As pool permanence can vary among pools in the same area, or even between years, adjustment of the development rate and growth rate to the local conditions is selected for (Laugen *et al.*, 2003; Lind *et al.*, 2011). Many amphibians develop in pools of varying duration, and local specialization to the mean pool-drying regime (temporary or permanent pools) is present in these systems (Lind & Johansson, 2007; Lind *et al.*, 2008). Thus, populations originating from islands with mainly permanent pools are experiencing a different environment than populations from islands with mainly temporary pools, selecting for higher development rate in

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temporary pools (Lind *et al.*, 2011). It is, however, not known whether this local specialization could be explained by local, adaptive increases in both development and growth rates from standing genetic variation, or whether these populations have incorporated part of a historical plastic response to time-constrained environments by genetic accommodation.

We investigate the mechanisms behind plasticity and local specialization in development rate in island populations of *R. temporaria*, originating from islands with different pool-drying regimes. We employed a common garden approach with two water level treatments, simulating permanent and temporary pools (time constraints) and evaluated development rate and growth rate of 81 families from 10 populations. First, we compared the growth rate and development rate patterns among families within populations, and under both water level treatments, to determine whether the plastic response to pool drying is governed by a changed behaviour or by a facultative life-history shift. Second, we investigated the development rate/growth rate relationship on population means. If the differences in development time of the locally adapted populations are caused by selection on standing genetic variation in development time, we expect the relationship between development rate and growth rate to be the same among populations as among families within a population. However, if the present local adaptation to environments with temporary pools (Lind & Johansson, 2007; Lind *et al.*, 2011) is enabled by genetic accommodation of a plastic facultative life-history shift, we expect a significantly steeper slope of the growth rate–development relationship among populations than among families (see Fig. 1c,d).

Materials and methods

Population sampling

To sample populations from a range of possible pool-drying regimes, eggs of *R. temporaria* were collected from 10 islands in the archipelago of Umeå, northern Sweden. The sampling procedure and common garden experiment are described in detail elsewhere (Lind & Johansson, 2007), and a map showing the location of the 10 populations is to be found in Lind *et al.* (2011). Briefly, up to 10 (on average eight) egg clumps (each egg clump corresponding to the offspring of one female) from each of 10 islands were collected on the 2nd and 6th of May, 2005, and brought to the laboratory. To assess the pool-drying regime present on an island, the decrease in water level of the pools was estimated as follows. Maximum pool depth was measured at egg collection and at June 26. The percentage decrease in pool depth between the two sampling dates was then used as a proxy for the hydroperiod, following Lind & Johansson (2007). If egg clumps were found in multiple pools on an island, egg clumps were collected from all pools and the drying regime on that island was calculated as the average drying regime of the breeding pools, weighted by the density of egg clumps.

Experimental procedure

After hatching, at Gosner stage 23 (Gosner, 1960), the tadpoles were individually placed in plastic containers (9.5 × 9.5 cm, height 10 cm), filled with 750 mL of aged and aerated tap water. The water was replaced every fourth day, before feeding. In the common garden experiment, the temperature was set to 22 °C and the tadpoles were fed *ad libitum* every fourth day on a mixture (1 : 2) of finely ground fish flakes and rabbit chow. To estimate the degree of phenotypic plasticity in tadpole development time as a response to the drying out of the pool, the tadpoles were subjected to one of two treatments: either a constant water volume (C) or a simulated pool drying (D). In the pool-drying treatment, the initial water volume of 750 mL was lowered by 33% every fourth day. Two siblings from each female clutch were individually raised under each water level treatment, giving 325 experimental units in total, which was the maximum number that could fit into the climate-controlled laboratory. The experiment was terminated at Gosner stage 42 (front legs visible), and the time to reach this stage was recorded as development time. Tadpoles were also weighed to obtain an estimate of their weight at metamorphosis. Plasticity in development time was calculated as the mean development time for the offspring of a female under constant water level, minus the development time under the artificial pool-drying treatment. Because of random mortality of some replicate siblings, we calculated the mean sibling trait values and used this family mean in all analyses.

Development and growth rates

Development rate was recorded as the number of developmental stages from the start of the experiment (Gosner stage 23) until the metamorphosis (Gosner stage 42) that the tadpoles passed per day (19 Gosner stages divided by the development time). This is essentially the same as using 1 divided by development time (the more common method of calculating development rate), because in both cases we divide a constant with development time. Growth rate was recorded as the difference in weight (log transformed) between Gosner stage 42 (metamorphosis) and 23 (start of experiment), divided by the development time.

Maternal effects

Maternal effects can potentially influence the life history of the offspring (Mousseau & Fox, 1998) and need to be accounted for. Ideally, maternal and other nonadditive

genetic effects are best estimated using a North Carolina II half-sib breeding design (Lynch & Walsh, 1998). However, due to the large number of populations used (with synchronous breeding) and the small population sizes on the islands, collection of breeding frogs was not possible. However, a half-sib breeding design has been performed using one of the island populations, and it concluded that maternal effects were of minor importance, explaining only 5% of the phenotypic variance in this system (Lind & Johansson, 2007). As maternal effects in *R. temporaria* are mostly transmitted through differential investment in the size of the eggs (Laugen *et al.*, 2002), we measured the mean egg size of every clutch. This was performed by placing 10 eggs from the clutches collected in nature in a petri-dish, covering them with water and photographing them together with a scale. The egg sizes were then measured from the photographs using the software IMAGEJ (http://rsbweb.nih.gov/ij/), and the mean egg size for each female egg clutch was used as a covariate in all analyses.

Statistical analyses

To test the Ball–Baker model of the relationship between development rate and growth rate (Beckerman *et al.*, 2007), as well as testing for differences in slopes among families and populations, we investigated their relationship in the two water level treatments. To avoid bias in slope estimates, and to enable comparison of slope coefficients of different hierarchical levels (family and population level), we used the technique of within-subject mean centring (van de Pol & Wright, 2009). This technique involves subtracting the subject mean from each observational value, i.e. to subtract the mean population growth rate from every growth rate measure on the family level. The within-subject centring thus gives us a new predictor variable that only expresses the within-population component of growth rate, whereas the mean growth rate of each population expresses the among population differences in growth rate, and both variables can be included in a mixed model of the form:

$$z_{ij} = \mu + A_i + \beta_W(x_{ij} - \bar{x}_j) + \beta_B\bar{x}_j + C_{ij} + \alpha_j + \varepsilon_{ij} \qquad (1)$$

where z_{ij} is the mean development rate of the ith family from the jth population. In the equation, μ is the grand mean, A_i denotes the two water level treatments, α_j is the random population effect, β_W is the within-population slope of the family-level growth rate x_{ij}, standardized by the mean growth rate $\bar{x}_j$ of the population it belongs to. β_B is the slope coefficient of the mean growth rate of the populations, C_{ij} is the effect of the covariate egg size and ϵ_{ij} is the residual error term. This allows us to test whether there is a trade-off between growth and development rate by assessing the effect of the water level treatment, and also to test whether the slope of the relationship between growth and development rate differs among the different hierarchical levels. Equation (1) was implemented in a Bayesian MCMC framework using the package MCMCglmm (Hadfield, 2009) in the statistical software R 2.11.1 (http://www.r-project.org/). Treatment, the within-subject-centred family-growth rates and the mean population growth rate were fitted as fixed factors, egg size as a covariate and population origin as a random effect. The model was run for 200 000 iterations with a burn-in of 2000 iterations and a thinning interval of 10. We used a proper but weak prior, partitioning the observed variance equally between the random effects and the residuals. The estimates of the fixed effects (which were the only effects of interest) were not affected by the choice of prior. Parameter values were estimated as the mode of their highest posterior distribution, and their difference from the null hypothesis (0) was estimated using the 95% highest posterior density (HPD). When two parameter estimates were compared, we used the 84% HPD interval, because this corresponds to $\alpha = 0.05$ when confidence interval overlap of parameters estimated from data are used for hypothesis testing (Schenker & Gentleman, 2001; Payton *et al.*, 2003). Note that when investigating whether a parameter is significantly different from an entity that is measured without uncertainty (such as zero, which is the most common null hypothesis in statistical testing), a confidence interval of 95% should obviously be used.

Results

We found a positive relationship between growth rate and development rate at the family level, indicating that the differences in development rate and growth rate among families within the populations were caused by different foraging efforts (Ball & Baker, 1996). In addition, the significant effect of the artificial drying treatment shows that individuals in the drying treatment had a significantly higher development rate for a given growth rate than in the constant water level treatment, a pattern that strongly resembles the prediction under a facultative life-history shift (Beckerman *et al.*, 2007; see Figs 1b and 2a, Table 1). Maternal effects mediated through egg size also had no significant effect on the development rate.

As for families, there was a significant positive relationship between development and growth rates among populations. However, the slope coefficient for population means (84% HPD: 5.65–7.90) was not significantly different from the slope coefficient estimated among families (84% HPD: 7.17–7.79), which is shown by the substantial overlap of their 84% HPD intervals. Therefore, the results (Fig. 2b) follow the prediction in Fig. 1c and suggest that the local adaptation is caused by selection on standing genetic variation and not by genetic accommodation of a plastic trait.

Table 1 The effect of family-level growth rate (centred within population), mean population growth rate, water level treatment and maternal effects (through egg size) on development. The relationship was analysed as a mixed model in a Bayesian MCMC framework with population incorporated as a random factor. Parameter estimates are presented with their posterior density modes and 95% highest posterior density (HPD) intervals.

Coefficient	Posterior mode (95% HPD interval)
Intercept	0.022 (–0.134–0.202) n.s.
Treatment	0.031 (0.027–0.035) *
Family growth rate	7.450 (7.046–7.909) *
Population mean growth rate	6.751 (5.181–8.319) *
Egg size	0.004 (–0.154–0.192) n.s.

Significant coefficients (not overlapping zero) are indicated by asterisks (*).

Discussion

We found that life-history plasticity in tadpoles of *R. temporaria* as a response to time constraints was caused by a physiological life-history shift, in which final mass was traded off against development rate. Moreover, we extended the use of Ball & Baker's (1996) model to investigate the role of life-history plasticity in local adaptation. By examining the relationship between development rate and growth rate among multiple populations, we suggest that the local adaptation to temporary island pools was not aided by the presence of ancestral plasticity in development rate. Although the individuals show adaptive plasticity in development time by inducing a facultative life-history shift in response to time constraints, this plastic response does not seem to have become incorporated in the development of populations adapted to time-constrained environments. The study highlights the importance of measuring two life-history traits to fully understand the role of phenotypic plasticity for local adaptation.

The mechanism of phenotypic plasticity

By analysing the relationship between development rate and growth rate for families of tadpoles from 10 island populations of *R. temporaria*, we found that, within each treatment, the families with the highest growth rate also had the highest development rate. This indicates that the individual growth and development rates are regulated by the behaviour or energy-acquisition ability of the individual (Ball & Baker, 1996; Beckerman *et al.*, 2007, 2010), because an increase in both development rate and growth rate is a common response to increased energy acquisition mediated through increased foraging (Abrams *et al.*, 1996) and a synchronous decrease in development rate and growth rate is a commonly found result of reduced foraging in the presence of predators (Skelly & Werner, 1990; Ball & Baker, 1996; Benard, 2004). In contrast, when subjected to a time constraint (an artificial pool-drying treatment), the individuals increased their development rate for a given growth rate (Fig. 2a). This pattern indicates that the individuals express life-history plasticity by trading off final mass

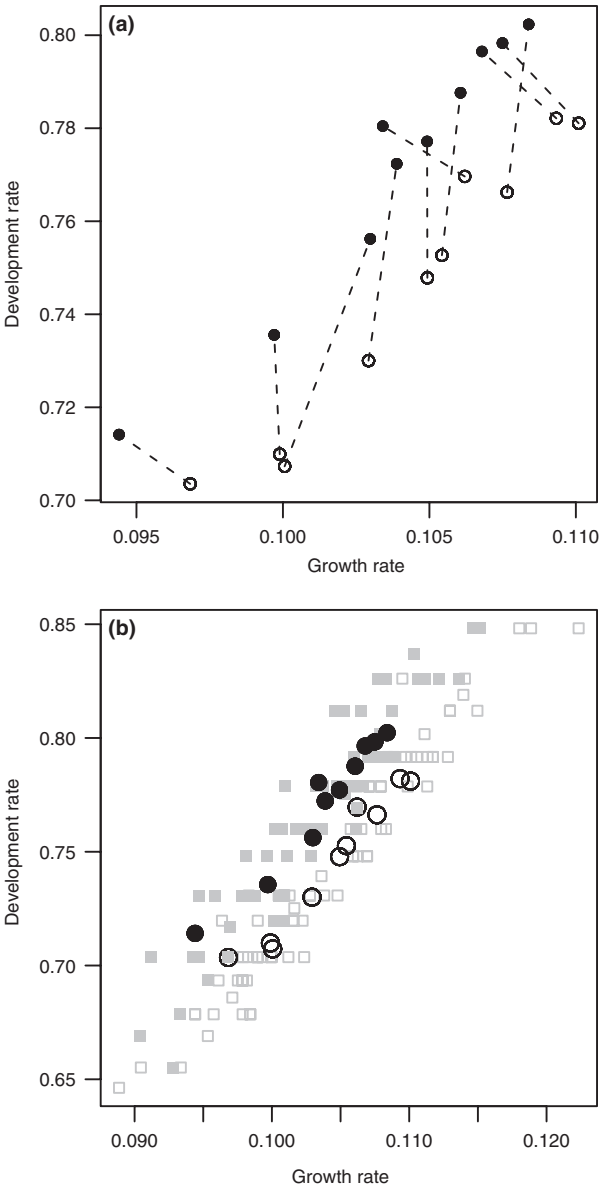

Fig. 2 (a) The population-level expression of development rate and growth rate under constant (*open circles*) and artificial drying treatment (*filled circles*). A hatched line connects the two treatments for each population. For all populations, the plastic response in the artificial drying treatment is to increase development rate, in some populations by trading it off against growth rate. (b) The relationship between growth rates and development rates for families (*grey squares*) and population means (*black circles*) for the two water level treatments (constant water level treatment: open circles/squares, artificial drying treatment: *filled circles/squares*).

against development rate via a physiologically mediated facultative life-history shift (as suggested by Ball & Baker, 1996) and not by increasing both development rate and growth rate, as predicted if plasticity were behaviourally mediated. It should be noted that physiology and behaviour are both affected by pool drying in amphibians (Denver, 1997). Pond drying induces production of stress hormone, which alters both behaviour (up- or down-regulation of appetite and foraging behaviour; Crespi & Denver, 2004) and physiology (through increasing development rate; Denver, 1997). However, as stress hormone increases development rate and allows for less time for growth, the tadpoles will reach a lower final mass despite increased foraging, i.e. a facultative life-history shift will take place.

The role of phenotypic plasticity for local adaptation

In this system, we have shown that there is local adaptation to the degree of pool drying present on the islands, caused by divergent natural selection (Lind *et al.*, 2011), so that the populations with the highest development rates inhabit the islands with the most temporary pools (Johansson *et al.*, 2005; Lind & Johansson, 2007; Lind *et al.*, 2008). Local adaptation to these time-constrained environments can theoretically occur via two routes: selection on standing genetic variation and by genetic accommodation of a plastic response. In the absence of plasticity, the individuals expressing the highest development rate will be favoured by selection. These individuals have both high development rate and growth rate, because of selection on efficient food acquisition. Thus, when plotting the slope of the development rate and growth rate among populations, the slope should be the same as the slope among individuals within the populations, because selection has worked on the within-population variation in food acquisition (Fig. 1c).

However, substantial plasticity in development rate is present in this system (Lind *et al.*, 2011) as well as in the mainland populations (Almfelt, 2005), which are the ancestral populations (Lind *et al.*, 2011). Therefore, the individuals experiencing a time-constrained environment will respond by a physiologically mediated life-history shift by which they will increase their development rate (see previous paragraph). The theory of genetic accommodation predicts that phenotypic plasticity in a trait can be genetically accommodated and expressed independent of the environmental cue that initially was needed (West-Eberhard, 2003; Lande, 2009). As a consequence of genetic accommodation of the plastic trait, the individuals in these populations are predicted to have a higher development rate than the individuals originating from islands with more permanent pools, without a corresponding increase in growth rate (Fig. 1d).

However, when analysing the relationship between growth rate and development rate, we found that the slope coefficients did not differ whether they were estimated from families or from population means (Fig. 2b). Therefore, the plastic life-history shift that is induced by pool drying has not been incorporated in the development path of individuals inhabiting islands with high risk of pool drying. Instead, our analysis suggests that local adaptation has been caused by selection on standing genetic variation in development rate. However, plasticity is retained in the system, even in populations with high mean development rate, because plasticity is needed to express the most extreme development rates (Lind & Johansson, 2009).

In contrast to the findings in a number of studies on amphibians, insects and fish, where local adaptation aided by the presence of ancestral plasticity have been found (commonly by using a phylogenetic approach, Gomez-Mestre & Buchholz, 2006; Kamimura, 2006; Wund *et al.*, 2008), we find no evidence that the plastic response to pool drying has been incorporated in the normal development of populations locally adapted to pool-drying conditions. However, our result does not conclude that plasticity has been unimportant during local adaptation. Plasticity in development rate may well have enabled survival on islands with high risk of pool drying. All we can conclude is that the local adaptation has not involved genetic accommodation of the plastic facultative life-history shift.

Until now, it has not been possible to construct a falsifiable hypothesis regarding the role of phenotypic plasticity for local adaptation without knowledge on the molecular or hormonal basis of plasticity. With our extension of the Ball & Baker (1996) model, we have suggested a framework to investigate whether local adaptation in development rate (or growth rate) can be a result of the genetic accommodation of a plastic physiological life-history shift. From this framework, it is possible to produce testable hypothesis that is possible to falsify. Our framework is restricted to the analysis of two interacting life-history traits, and in cases where plasticity is not expressed by a facultative life-history shift but by behavioural regulation of the life history, we cannot construct a falsifiable null hypothesis, because the slope of the within and among population regressions are predicted to be the same regardless of whether plasticity has aided local adaptation. Nevertheless, plasticity in development rate and growth rate is a common response to time-stressed environments (Merilä *et al.*, 2000; Altwegg, 2002), predators (Altwegg, 2002; Mikolajewski *et al.*, 2005; Van Buskirk & Arioli, 2005), seasonality (Johansson & Rowe, 1999) and temperature (Yamahira *et al.*, 2007). This plasticity seems to induce facultative life-history shift as a response both to time-stressed environments (this study) and to predators (Beckerman *et al.*, 2007, 2010). Therefore, the suggested framework will have important implications for our study of life-history evolution and to test for the role of phenotypic plasticity for local adaptation.

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