# Heritability and developmental plasticity of growth trajectories in an oviparous lizard

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# Abstract

Selective processes act on phenotypic variation however the evolutionary potential of any given trait relies on the underlying heritable variation. Developmental plasticity is an important driver of phenotypic variation and can also promote changes in genetic variation, yet we have a limited understanding on how they are both impacted. Here, we quantified the influence of developmental temperature on the growth trajectory in delicate skinks (*Lampropholis delicata*) and partitioned the total variance using an animal model fitted with a genomic relatedness matrix. We measured mass for 262 individuals (nhot = 126, ncold = 136) over 16 months (nobservations = 3002) and estimated heritability and maternal effects over time. Our results show that developmental temperature did not impact the shape of growth trajectories but rather mass at hatching. Lizards born in cold developmental temperatures had a higher hatching mass compared to lizards from the hot developmental temperatures. On average, additive genetic variance, maternal effects and heritability were higher in hot developmental temperature treatment, however these differences were not statistically significant. All variance components changed dynamically over time. Heritability increased with age, whereas maternal effects decreased upon hatching but increased again at a later age. Our work suggests that evolutionary potential of growth is complex, age dependent and not overtly affected by natural developmental temperatures.

# Keywords

Body mass, growth rate, additive genetic variance, incubation temperature, maternal effects

# Introduction

Developmental plasticity plays a key role in generating phenotypic variation. Environmentally-induced changes may result in a better match between adult phenotype and the selective environment if developmental cues predict later-life conditions (Beldade et al., 2011), however in some cases maladaptive phenotypes can arise (Ghalambor et al., 2007). As such, developmental plasticity is considered as a promising immediate solution for populations to adjust to environmental fluctuations (West-Eberhard, 2003). Over longer terms, populations can either migrate to more favourable habitats or evolve genetic adaptations in response to environmental change (Sgrò & Hoffmann, 2004). However, the capacity to evolve may be dependent on developmental environments because environmental cues experienced during development can also affect the underlying standing genetic variation which select acts on. Understanding how phenotypic and genotypic variation respond to developmental environments is necessary in order to predict population persistence.

Environmental dependence of genetic variation implies that evolutionary change may speed up or slow down under certain contexts. Such implications has sparked researchers to describe the conditions under which genetic variation may change (Charmantier & Garant, 2005; Hoffmann & Merilä, 1999; Rowiński & Rogell, 2017). Several hypotheses have been proposed to explain environmentally-induced changes in genetic variance (Charmantier & Garant, 2005; Hoffmann & Merilä, 1999; Rowiński & Rogell, 2017). Notably, higher mutation rates (Hoffman & Parsons, 1991); reduced selection pressures which allows for the accumulation of genetic variation (Hoffmann & Merilä, 1999) and the breakdown of buffering mechanisms that releases of cryptic genetic variation are expected to increase genetic variation under novel, stressful environments (Paaby & Rockman, 2014). On the other hand, genetic variation can also decrease under stressful environments if gene expression is condition-dependent (Coltman et al., 2001) or under strong selection (Hunt et al., 2007).

Comparative studies have shown that the influence of developmental stress on genetic variance is not straightforward (Charmantier & Garant, 2005; Hoffmann & Merilä, 1999; Rowiński & Rogell, 2017). In lab studies, high developmental stress have been shown to increase the heritability of morphological traits (Hoffmann & Merilä, 1999), whereas wild, non-domestic populations tend to have higher heritability in favourable environments (Charmantier & Garant, 2005). Lack of consensus may be related to lab and wild conditions, with the latter being more variable and therefore more difficult to detect environmental patterns. The nature of environmental stressor under investigation (e.g. heat shock vs. habitat quality) could also yield variable patterns of gene expression if they are not strongly genetically correlated , making comparisons more heterogenous (Charmantier & Garant, 2005; Dahlgaard & Hoffmann, 2000). Environmental comparisons of heritability have been criticised as estimates may show no changes, but the relative contributions of non-genetic variance actually differ. Indeed, additive genetic and environmental variance of life history traits which is supposedly more important to fitness components were found to increase under high stress conditions. The opposite holds true for morphological traits, where the same variance components decreased in high stress environments. Interestingly, h2 showed no differences among environments which suggests that the capacity to evolve is contingent on non-genetic sources of variance as well as the trait of interest (Rowiński & Rogell, 2017).

Body size is a heritable, fitness related trait and is strongly affected by a number of factors. Developmental environments such as temperature and nutrition play pivotal roles on variation in body size, largely through shifts in how organisms grow and develop (Eyck et al., 2019; Noble et al., 2017). Naturally, maternal contributions on offspring development environments such as in egg size, nest site selection or timing of birth can also promote variation in body size and growth. Selection on body size is therefore likely to vary across ontogeny. For example, high juvenile mortality has favoured high maternal investment to larger birth weights in Soay sheep (Wilson, Coltman, et al., 2005). Accordingly, maternal contributions are expected to dissipate with age presumably because mothers cannot continue to influence her offspring’s phenotype post-weaning (Krist, 2010; Wilson, Kruuk, et al., 2005). Moreover, body size at sexual maturity is likely a major target of selection as it strongly predicts reproductive success and survival (Bartheld et al., 2015; Calsbeek & Sinervo, 2002) and may have favoured the evolution of compensatory growth strategies which offsets body size variance at later stages (Hector & Nakagawa, 2012). Given that selection operates on body size at certain life stages, it is reasonable to expect that its genetic and non-genetic variance to change across ontogeny. A more temporal approach is needed in order to evaluate when evolutionary potential of body size is great.

Here we investigated the impact of developmental temperature on growth and mass in an oviparous skink (*Lampropholis delicata*) and how developmental environments affect evolutionary potential in these traits. We quantified growth trajectories (nobservations = 3002) for lizards that hatched from two incubation treatments (nhot = 126, ncold = 136), Using 8,433 SNP markers, we derived a genomic relatedness matrix to estimate quantitative genetics parameters to address the following key questions: 1) How does developmental temperature affect the shape of growth trajectories (initial mass, growth rate and curvature of growth trajectory)?; 2) Do differences in growth result in variation in age at maximum mass?; 3) How does developmental temperature affect genetic and non-genetic variance components as well as heritability overall and across age? Based on the temperature-size rule, we expect lizards that experienced cold developmental temperatures to have larger initial masses, slower growth rates and would reach maximum size at a later age compared to lizards that experienced hot developmental temperatures (Angilletta Jr et al., 2017). We expect genetic and non-genetic to differ among treatments, and predict that additive genetic to increase under higher thermal stress. We expect maternal effects and permanent environmental effects to decline as lizards mature.

# Materials and Methods

## Lizard collection and husbandry

From 2015 – 2017, we established a breeding colony of adult *L. delicata* (nfemales = 144, nmales = 50) using wild individuals collected across five sites throughout the Sydney region between 28 August and 8 September 2015. Using a half-sib breeding design we paired three females with a single male in opaque plastic enclosures measuring 35cm 25cm 15cm (L W H). Enclosures were kept under UV lights (12L:12D) in a temperature controlled room set to 24ºC. Lizards were given access to a heat lamp that elevated temperatures to between 28-32 ºC. Each enclosure was lined with newspaper and lizards had constant access to water and tree bark as refuge. Adult lizards were fed medium sized crickets *ad libitum* (*Acheta domestica*) dusted with calcium powder and multi-vitamin every two days. From the beginning of egg laying seasons (October of each year), we replaced newpaper lining with garden potting mix and placed an opaque plastic box (12 cm 17.5 cm 4.3 cm) containing moistened vermiculite in each enclosure for females to oviposit their eggs. During this time, enclosures were sprayed gently with water every second day to maintain a relatively humid environment. From October to November, egg boxes were checked every day. Tail tissue samples (~1 mm) were taken from adults that were from enclosures producing eggs for DNA extraction (see below). All tissues were stored in 70% ethanol. Animal collection was approved by the New South Wales National Parks and Wildlife Service (SL101549) and all procedures were approved by the Macquarie University Ethics committee (ARA 2015/015) and University of New South Wales Animal Care and Ethics committee (ACEC 15/51A).

## Developmental Temperature Manipulations

Eggs were collected over two years from 2016–2017 and were typically laid between October to March. As soon as eggs were found, they were weighed using a digital scale to the nearest 0.01g (Ohaus Scout SKX123). We also measured egg length (distance between the furthest points along the longest axis of the egg) and egg width (distance between the widest points along the axis perpendicular to the longest axis of the egg) using digital callipers to the nearest 0.01mm. Following measurements, each egg was placed in a plastic cup (80ml) containing three grams of vermiculite and four grams of water. Each cup was then covered using cling wrap and secured using an elastic band. Each clutch was pseudo-randomly assigned to one of two developmental temperature treatments. We used two incubators to precisely control the temperature of eggs (LabWit, ZXSD-R1090). The ‘hot’ treatment was exposed to a mean temperature of 29ºC whereas the ‘cold’ treatment was exposed to a mean temperature of 23ºC. However, both incubators fluctuated +/- 3ºC over a 24 hour period. These treatments represent the temperature extremes of natural nest sites of *L. delicata* (Cheetham et al., 2011). Egg cups were rotated within each incubator weekly to avoid uneven heat circulation within incubators. Incubators were also checked daily for hatchlings. On average, the incubation period for the ‘hot’ treatment was 29.36 days (SD = 2.17, range = 15 - 49) days and 48.48 days (SD = 4.18, range = 25 - 56) for the ‘cold’ treatment.

## Quantifying Growth Rate

Newly emerged hatchlings were weighed to the nearest 0.01g and a small tail tip clipping (~2mm) was taken for genetic analyses (see below). Ventral photographs were taken for digital measurement (Nikon Coolpix A900). For the first two months, photographs of hatchlings were taken approximately every 14 days. After which, hatchlings were photographed at approximately a 35-day interval. From approximately six months onwards, we manually measured hatchling SVL using a clear ruler. Growth measurements continued until we had approximately 16 measures per individual (mean = 11.5 , range = 1 - 18). From the photographs, we extracted snout-vent-length (SVL; from tip of snout to the beginning of the cloaca opening) using ImageJ software (Rueden et al., 2017). For the first initial nine months, hatchlings were housed individually in opaque plastic enclosures (32.3cm x 18.5cm x 6cm) lined with newspaper. Hatchlings were fed the same number of crickets every second day and had constant access to a tree bark refuge and water. Hatchling enclosures were placed in a temperature control room under the same conditions as described above for the adult colony. For logistical reasons, at approximately nine months, hatchlings were housed in groups of five in opaque bins with the same measurements as the adult enclosures. We pseudo-randomised individuals to each shared enclosure while maintaining a similar number of individuals from each treatment.

Genomic Relatedness Matrix

We derived a genomic relatedness matrix (GRM) using single nucleotide polymorphism (SNP) genotypes for all 262 offspring with growth data (132 putative parents; nfemales = 69, nmales = 63). While our half-sib breeding design allowed us to assign parentage to derive a pedigree, high levels of sperm storage and low levels of multiple paternity (94% of females had been sired by a single male) meant our pedigree had low resolution to effectively estimate additive genetic variation. Recent studies have shown that GRM derived from SNPs have low error rates (<0.3%) and are able to reconstruct pedigree relationships when at least 200 SNP loci are used (Bérénos et al., 2014; Huisman, 2017). Moreover, both relatedness and heritability values estimated from a GRM are strongly correlated to those inferred using a pedigree (Bérénos et al., 2014; Huisman, 2017). Single nucleotide polymorphism libraries were designed and animals genotyped using DArTseq™ ( Diversity Arrays Technology) methods. For more details on DNA extraction and SNP genotyping see ESM.

Prior to deriving our GRM, we filtered our SNPs using the R package *dartR* (Gruber et al., 2018). We filtered loci based on various metrics in the following order: 1) read depth (8 – 40); reproducibility (> 0.996); call rate by loci (> 0.97) and then by individual (> 0.80); monomorphic loci; minor allele frequencies (> 0.02); Hamming Distance among loci (> 0.25) and Hardy Weinberg Equilibrium. This clean-up process resulted in a dataset of 8,438 loci with an average call rate of 98.5% (see ESM and provided code). Using these 8,438 loci we derived a GRM, which describes the proportion of the genome that is identical by descent (VanRaden, 2008). We calculated a GRM for all hatchlings using the *snpReady* R package (Granato et al., 2018) following methods described by VanRaden, 2008:

where *Z* is the centered matrix of SNP genotypes of all individuals. This is calculated from a matrix of SNP genotypes coded as -1, 0, 1 for homozygote for the reference allele, heterozygote and homozygote for the SNP allele. *pi*is the frequency of the second locus at position locus *i.* The denominator scales the GRM matrix so that the values approximates to a relatedness matrix derived from a pedigree. The GRM was then inverted for modelling fitting (see ESM and provided code).

# Statistical Analyses

All analyses were performed using *R* (Core Team, 2013). We checked the data for potential input errors using histograms, scatterplots and Cleveland plots. We fitted Bayesian linear mixed effects models (LMM) in *brms* (Bürkner, 2017). Mass was log-transformed, and age was z-transformed. For all models we used noninformative priors with 4000 iterations with a burn in of 1500, sampling from the posterior distribution every fifth iteration. We ensured proper mixing by inspecting trace plots and checked that scale reduction factors were less than 1.01. We report posterior means and 95% credible intervals for all parameters throughout.

Impact of Developmental Temperature on Additive Genetic Variance and Maternal Effects Across Age

First, we tested whether developmental temperature influenced the overall heritability of mass and the relative contributions of variance irrespective of age. For each treatment group, we fitted intercepts only in the fixed effects with random intercepts for additive genetic variance (*G*), maternal effects (*M*) and permanent environmental effects (*PE*) as we had repeated measures of the same individuals (Wilson et al., 2010). The model also estimated residual variance (*R*). We included our GRM to estimate additive genetic variation. Overall. Heritability (*h2*) of mass using this intercept (*I*) model was calculated as:

To then test how *G*, *M* and *h2* change across age, we used model selection to determine the most appropriate random effects structure for our data as we had no *a priori* knowledge of how the variance components change with age. We fitted seven models with varying complexity in their random effects and compared their WAIC values (Table S1). We fitted random intercepts and random slopes by including either a linear age term or both linear and quadratic age terms to partition variance across age. Two models were equally supported, the first included a random linear and quadratic slope for *G* and *M* and *PE*. (Model 3 - Table S1) and the second included a random linear and quadratic slope for *G* and *M* and a random intercept for *PE* (Model 7 – Table S1)*.* To avoid overfitting, we selected the more parsimonious model and used this random effect structure for the remaining analysis unless stated otherwise.

Residual variance may conflate with estimates of other variance components if it changes over time (heterogenous variance) and is not properly accounted for. We therefore explicitly modelled residual variance to verify if this was the case using WAIC values. We fitted two models, both of which had the same fixed and random effects structure as Model 7 described above. The first model had homogenous residual variance whereas in the second model we modelled residual variance with a linear slope thereby allowing it to vary with age. The model with heterogenous variance was best supported (Table S2), we therefore modelled heterogenous variance in all subsequent models unless stated otherwise

For each treatment group, we fitted an intercept in our fixed effects and used the best supported random effect structure (Model 7) with heterogenous residual variance. We estimated a genetic variance-covariance matrix for each treatment (), where the diagonal elements represent the additive genetic variances for the intercept (), slope () and the quadratic () across age. The off-diagonal elements are the additive genetic covariances between the growth curve parameters, for example, is the additive genetic variance between the intercept and the quadratic slope.

Similarly, the variance-covariance matrix for dams () can be decomposed in the same manner as .

For each treatment group, we then calculated additive genetic variance at a given age using the random slope terms and their covariances as follows,

where is a specific age. Age-specific maternal effect was calculated using the same formula structure but with the relevant variance components from . Age-specific heritability is thus a ratio of all variance components at a given age . The proportion of variance explained by maternal effects (*m2*) is calculated in the same manner.

As the mean body mass increases over time, the variance may also increase concurrently due to scale effects and potentially bias estimates of quantitative genetics parameters (Wilson, Kruuk, et al., 2005). We therefore calculated coefficients of variation (CV) across age for each variance component by dividing variance by the predicted mean mass at a given age. Interpretations using CV estimates did not change our overall conclusions for additive genetic variance or maternal effects, we therefore present the raw estimates of each variance component below (See ESM).

The Influence of Developmental Temperature on Growth Trajectories

We fitted three models that varied in their fixed effect structure to determine how developmental temperatures affect 1) initial mass (intercept of curve), 2) linear rate of growth (linear slope) and 3) curvature of the growth trajectory (quadratic term). We also wanted to test for treatment differences in age at which lizards reach their maximum mass by solving for the maxima of quadratic regression equation. We fitted mass as the response and the most supported random effect structure we identified above. The first model included the main effect of developmental temperature and the linear and quadratic term for age (Table S2). The other two models differed in their interaction terms between developmental temperature with age and age2 (Table 2, S3). We then compared DIC values to select the best model for our data that explained changes in mass across age between the two developmental temperature treatments (Table 1).

# Results

Over two years, we collected 3002 observations of mass data for a total of 261 individuals ( = 125, = 136). The average age for hot incubated lizards was 335.82 (range: 0 – 711) and for cold incubated lizards it was 384.8 (range: 0 – 707). On average, a lizard had 11.5 measurements (SD = 4.71).

## The influence of developmental temperature on additive genetic variance and maternal effects across age

**Figure 1** Pie charts depicting the overall relative contributions of mass variance for the hot (n = 126) and cold (n =136) developmental treatment group irrespective of age. Point estimates and 95% credible intervals are presented in Table S3. There were no significant differences in variance components between developmental temperature treatments.

Overall, additive genetic variance, permanent environmental variance and heritability of growth appears to be higher in the hot developmental temperature treatment (Fig. 1). However, there were no significant differences among treatment groups (Table S3). Treatment groups did not differ in how the relative contributions of and changed with age as their 95% credible intervals overlapped (Fig. S1). Additive genetic variance remained relatively low and constant upon emergence until approximately nine months of age, after which it increased rapidly (Fig. S1). Maternal effects decreasing sharply upon hatching and dropped to the minimum at approximately six months before it increased (Fig. S1). There were some differences among developmental treatments in how residual variance changed with age (Fig. S1). Residual variance in cold incubated lizards showed a reasonably shallow increase whereas it increased more steeply in hot incubated lizard (Fig. S1). We investigated whether increases in mass mean over time would result in scale effects that can bias variance estimates. We found that CV of *G* and *M* followed the same pattern was the raw variance estimates and conclude that scale effects did not influence our results (Fig. S2). After accounting for heterogenous residual variance, we found no treatment differences in or the proportion of variance explained by maternal effects () (Fig. 2). Heritability was very low for the first year of growth in *L. delicata* and only began increasing at one year of age (Fig. 2). As predicted decreased since hatching, however it increased slightly from six months of age (Fig. 2). The and matrices for each treatment group are presented in Table S4-S5.



**Figure 2** Graph showing the relationship of between heritability (*h2,* **A**) and the proportion of total variance explained by maternal effects (M2, **B**) change with age for the hot developmental treatment (n = 125, red) and the cold developmental treatment (n = 136, blue). Points represent estimates generated from the posterior distribution of the variance-covariance matrix, thin lines represent the 95% credible intervals, thick lines represent the mean for each treatment group.

Developmental plasticity in growth trajectories in response to temperature

While the model containing an full interaction between treatment and linear and quadratic age was best supported, the improvement was marginal (Table 1). Moreover, the linear growth rate (Age) and curvature of the growth trajectory (Age2) did not differ significantly between the two developmental temperature treatments in any of the models containing interactions (Table S5, Table S7-9). Irrespective of treatment, a lizard mass increased by 1.65g for every 1 SD unit increase in age. Developmental temperature did however impact initial mass (Table 1, Fig. 3). Lizards from the ‘cold’ treatment were on average 0.030 g (0.018g – 0.041g) heavier compared to lizards from the ‘hot’ treatment (Table. S5). Larger initial masses meant that lizards from the ‘cold’ treatment reached their maximum mass slightly earlier (382.97 days, 95% CI: 358.84– 409.78) compared to lizards from ‘hot’ treatment (413.04 days, 95% CI: 379.70 – 452.34). The population *G* and *M* matrices and other variance components are presented in Table S6.

**Table 1** Comparisons of WAIC values of four models with different combinations of treatment interactions with age parameters. = 3982. represents the difference in expected log predicted density.

|  |  |  |  |
| --- | --- | --- | --- |
| Formula of Fixed Effects | WAIC |  | Std. Error |
| Treatment + Age + Treatment Age + Treatment Age2 | -3301 | 0 | 0 |
| Treatment + Age + Age2+ Treatment Age | -3295 | -0.62 | 1.182 |
| Treatment + Age + Treatment Age2 | -3300 | -2.798 | 1.375 |
| Treatment + Age + Age2 | -3292 | -4.452 | 1.563 |



**Figure 3** Model predictions of log-transformed mass over age for a random subset of 40 lizards from the two developmental temperatures. Points represent mean estimates for each lizard from the hot developmental treatment (hot) and the cold developmental treatment (blue). Thick lines represent average growth curve for each treatment. Faint grey lines are each individual’s growth curve.

# Discussion

Lizards from hot early developmental environments weighed less than lizards from cold temperatures. However, growth trajectories were not significantly impacted by thermal environment. In addition, developmental temperature did not impact the expression of additive genetic and maternal effect variance. Heritability of mass varied over ontogeny, peaking at six months before gradually increasing again due to a rise in additive genetic variance around nine months of age. As we predicted, maternal effects on offspring mass declined in the first month presumably because maternal non-genetic contributions were less influential on mass over time. Unexpectedly, maternal effect variance increased again at approximately six months and continued to remain high, possibly resulting from maternal genetic factors impacting mass. Our study suggests that environmental sources contributed the most mass variation and both genetic and non-genetic sources of maternal effects could influence the evolutionary potential of body mass over time.

In ectotherms, temperature plays a pervasive role in phenotypic development. Lizards from the cold incubation treatment had higher mass upon hatching compared to lizards from the hot incubation treatment, however growth rate did not differ between our treatment groups. These results partially support the temperature-size-rule whereby individuals reared in cold temperatures tend to be larger compared to their counterparts reared in hot temperatures (Angilletta Jr et al., 2017). Larger sizes are usually achieved by compensatory growth strategies (prolonging growth or increasing growth rates), however our results does not support either of this hypothesis (Hector & Nakagawa, 2012). Instead, temperature variation during embryonic growth may have resulted in differences in hatching weight (Storm & Angilletta, 2007). For example, turtle embryos exposed to high temperatures had enhanced mitochondrial metabolism and metabolic enzymic activity which shortened developmental time but reduce overall hatching size (Ji et al., 2003; Sun et al., 2015). We found some evidence that cold incubated lizards reached their maximum weight i.e. sexual maturity more quickly compared to hot incubated lizards. Plasticity in embryonic development in response to temperature may confer a selective advantage for lizards born late in the season when nest temperatures are generally colder (Warner & Shine, 2008; While et al., 2015). Heavier weight at emergence could mean that hatchlings are in better condition to compete with lizards that hatched earlier and evade predators (Downes & Shine, 1999; Gifford et al., 2017; Qualls & Shine, 2000).

The evolutionary potential of any trait is represented by additive genetic variance and this is known to vary across environments. Overall, we found no differences in additive genetic variance among treatments groups. Our incubation temperatures were selected based on temperature extremes of naturally occurring nests of *L.delicata* and may not be stressful and different enough to elicit any changes in gene expression . Furthermore, treatment differences may be harder to detect as we used a fluctuating temperature regime which meant that temperatures of both treatment groups overlapped at certain times of the day. Many researchers have hypothesised that genetic variance should increase under stressful and novel conditions and may be crucial for adaptive evolution, however generality has been difficult to establish (Charmantier & Garant, 2005; Hoffmann & Merilä, 1999; Rowiński & Rogell, 2017). Defining an environment as stressful or novel requires detail knowledge of a given species’ past environmental exposure which could influence their stress tolerance and therefore gene expression (Roelofs et al., 2010). Integrating species’ stress physiology and thermal biology such as corticosterone levels, critical thermal limits may benefit future experimental designs as stress-induced manipulations can be more objective and species specific (Hoffmann & Sgrò, 2018). Moreover, this method also allow better species comparisons as researchers can report the relative change from each specie’s limit rather than absolute temperature values (i.e. 20% change increase relative to upper thermal limits). Our results suggest that our incubation treatments did not modify the evolutionary potential of mass, however this should be interpreted with caution as estimates of quantitative parameters from laboratory studies can differ from wild populations (Sgrò & Hoffmann, 2004; Weigensberg & Roff, 1996).

The genetic and non-genetic components of body size variance are expected to change throughout ontogeny. Selection pressures on body size are likely to increase at critical life stages such as at birth or at sexual maturation thereby reducing genetic variance (Rollinson & Rowe, 2015). Moreover, maternal contributions to offspring body size is expected to highest during early life stages and decline as offspring mature (Cheverud, 1984; Wilson, Kruuk, et al., 2005). We observed heritability of mass changed with age with periods of high heritability associated with a decrease of maternal effects, rather than an increase in additive genetic variance. This suggests that maternal effects play an important role in evolutionary dynamics of body size (Wolf & Wade, 2016). In line with our prediction and other studies, maternal effects declined upon hatching (Pick et al., 2016; Wilson, Coltman, et al., 2005; Wilson, Kruuk, et al., 2005). Non-genetic maternal investment such as clutch size or egg quality has been shown to influence hatching size (Brown & Shine, 2009; Warner & Lovern, 2014), however these effects dissipated post-hatching as mothers can no longer alter the phenotype of her precocial offspring (Pick et al., 2016; Réale et al., 1999). Interestingly, maternal contributions increased at a later age and remained relatively high for the remainder of the study. The cause of resurgence in maternal effects is unclear however, this pattern may indicate other maternally inherited components such as mitochondria that promotes variation in body size (Pick et al., 2016). Indeed, variation in mitochondria have been linked to an individual’s metabolic rate and growth and is thus an important driver of body size variance (Salin et al., 2016, 2019). Greater environmental variation later in life (increased density due to housing conditions) coincided with the resurgence of maternal effects, suggesting that maternal effects on offspring fitness may be context dependent.

# Conclusion

Our results show that developmental temperatures affected phenotypic variation of growth but did not the impact its genetic and maternal components of variance. Seasonal variation in temperatures and its effects on growth may drive life history differences within populations which can alter population dynamics and structure. We show that heritability and maternal effects is not static over time and suggests that potential for evolutionary change is age dependent. This has important implications for selection acting on body mass at certain life stages and warrants long term studies to investigate these age-related patterns. Maternal contributions on offspring phenotype are prevalent and are known to weaken over time. However, we provide evidence of maternal inheritance later in life that may promote variance in body mas a and thus influence its evolution.

# Author contributions

FK, DN, SN conceived the study, FK and DN collected and analysed the data, FK wrote the first draft, FK, DN and SN edited the manuscript.

# Data accessibility

Datasets and code used to generate results of this study is accessible via Open Science Framework (DOI: XXXXXXXXXXX)

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