BIOLOGICAL REVIEWS
Cambridge Philosophical Society
*Biol. Rev.* (2019), **94**, pp. 1143–1160.
doi: 10.1111/brv.12496


# Effects of developmental stress on animal phenotype and performance: a quantitative review

Harrison J.F. Eyck*, Katherine L. Buchanan, Ondi L. Crino and Tim S. Jessop

*Centre for Integrative Ecology, Deakin University, School of Life and Environmental Sciences, 75 Pigdons rd, Geelong, VIC 3216, Australia*

ABSTRACT

Developmental stressors are increasingly recognised for their pervasive influence on the ecology and evolution of animals. In particular, many studies have focused on how developmental stress can give rise to variation in adult behaviour, physiology, and performance. However, there remains a poor understanding of whether general patterns exist in the effects and magnitude of phenotypic responses across taxonomic groups. Furthermore, given the extensive phenotypic variation that arises from developmental stressors, it remains important to ascertain how multiple processes may explain these responses. We compiled data from 111 studies to examine and quantify the effect of developmental stress on animal phenotype and performance from juveniles to adulthood, including studies from birds, reptiles, fish, mammals, insects, arachnids, and amphibians. Using meta-analytic approaches, we show that across all studies there is, on average, a moderate to large negative effect of developmental stress exposure (posterior mean effect: $|d| = -0.51$) on animal phenotype or performance. Additionally, we demonstrate that interactive effects of timing of stressor onset and the duration of exposure to stressors best explained variation in developmental stress responses. Animals exposed to stressors earlier in development had more-positive responses than those with later onset, whereas longer duration of exposure to a stressor caused responses to be stronger in magnitude. However, the high amount of heterogeneity in our results, and the low degree of variance explained by fixed effects in both the meta-analysis ($R^2 = 0.034$) and top-ranked meta-regression model ($R^2 = 0.02$), indicate that phenotypic responses to developmental stressors are likely highly idiosyncratic in nature and difficult to predict. Despite this, our analyses address a critical knowledge gap in understanding what effect developmental stress has on phenotypic variation in animals. Additionally, our results highlight important environmental and proximate factors that may influence phenotypic responses to developmental stressors.

*Key words*: cross taxa, developmental stress, meta-analysis, meta-regression, phenotypic plasticity, phenotypic variation.

CONTENTS



* Address for correspondence (Tel: +0400194502; E-mail: harrisoneyck@gmail.com)

## I. INTRODUCTION

Conditions encountered during early development can have significant impacts on an animal's existence (Waddington, 1942; Møller & Swaddle, 1997; Francis *et al.*, 1999). Animals can be exposed to developmental stressors through environmental, energetic, physiological, social, behavioural, or pathological processes which by definition alter development. Within this study, 'stressors' are considered any noxious or unpredictable stimuli that elicit a stress response or deviation from homeostasis (Romero, 2004; Koolhaas *et al.*, 2011). Developmental stressors are diverse in their nature, timing and in their persistence, and for this reason they can have complex phenotypic consequences for developing animals, which can affect phenotypic performance across life (Schaefer & Ryan, 2006). Phenotypic consequences of developmental stress are now widely considered integral for how animals respond to many ecological and evolutionary phenomena and will be especially important when considering adaptive outcomes for species exposed to rapid global environmental change (Sultan, 2007; Uller, 2008; Moczek *et al.*, 2011).

A large body of literature suggests that developmental stressors can have sustained effects on morphological, physiological and behavioural traits. Stressors can have significant consequences for organismal fitness (Vallee *et al.*, 1997; Denver, Mirhadi & Phillips, 1998; Varlinskaya & Spear, 2008) as changes to crucial aspects of life history due to a response to a stressor, such as reproductive physiology (Naguib, Nemitz & Gil, 2006) or stress reactivity, can alter an animal's ability to survive or reproduce. A well-documented example of this comes from maternal separation in juvenile rats, which can lead to many significant phenotypic changes, including to stress-regulated protein composition in several brain regions (Liu *et al.*, 1997). As a result, the rats exhibit abnormal responses to stressful stimuli in adult life (Lippmann *et al.*, 2007). Such long-term changes to phenotypic traits in response to developmental conditions are reported across a wide range of species. For example, amphibians can have reduced adult body size due to exposure to habitat desiccation at the tadpole stage, potentially reducing adult survivability (Newman, 1989; Denver, 1997; Crespi & Warne, 2013), whereas avian studies have shown that developmental conditions can reduce song learning and song quality, affecting the ability to attract mates (Spencer *et al.*, 2003; Brumm, Zollinger & Slater, 2009). The wide range of developmental responses across many taxa suggests a general susceptibility of organisms to the consequences of developmental stress exposure (West-Eberhard, 2003). This is not limited to vertebrates; developmental conditions have been shown to have significant impacts on different aspects of invertebrate life history. Fruit flies (*Drosophila melanogaster*) exposed to brief heat shocks at a young age can have increased longevity and thermotolerance as adults (Le Bourg *et al.*, 2001), whilst wolf spiders (*Schizocosa* spp.) nutritionally deprived as juveniles matured slower, had smaller adult body size, and show evidence of altered mate choice among females (Hebets, Wesson & Shamble, 2008). Indeed, developmental stress has been shown to have persistent effects on numerous phenotypic traits across many life-history stages. Some examples of the broad range of phenotypic traits altered by developmental conditions include: animal performance (Chin *et al.*, 2009; Crino *et al.*, 2017), body size (Pavlovska-Teglia *et al.*, 1995), behaviour (Spencer & Verhulst, 2007) and cognitive ability (Nowicki, Searcy & Peters, 2002; Farrell *et al.*, 2015). Furthermore, recent meta-analyses have also shown that specific types of developmental stress (e.g. nutritional stress; developmental thermal environments in reptiles) can directly impact fitness (English & Uller, 2016; Senior *et al.*, 2017; Noble, Stenhouse & Schwanz, 2018).

However, despite being an active research field, there remains a poor understanding of what effect different conditions (e.g. stressor type) have on variation in animal's developmental stressor responses, with considerable variability in the literature. In general, by definition the effects of developmental stress are considered negative (Graham,

Freeman & Emlen, 1993; Gluckman & Hanson, 2004; Gluckman *et al.*, 2009), however, some studies indicate that developmental stress can also result in increased performance through phenotypic changes (Downes & Shine, 1999; Minois, 2000; Relyea, 2001; Hercus, Loeschcke & Rattan, 2003; Crino & Breuner, 2015). For example, Chin *et al.* (2009) found European starlings (*Sturnus vulgaris*) exposed to elevated levels of avian stress hormones in ovo had decreased wing loading and outperformed control birds in flight performance trials at fledging. In addition, Relyea (2001) observed that exposing wood frog (*Rana sylvatica*) tadpoles to predator stress indirectly led to those tadpoles to have longer, wider limbs at metamorphosis, traits which have been associated with increased jump length (Johansson, Lederer & Lind, 2010). Thus, further evaluation is necessary to quantify the basis for the large interspecific and inter-individual variation in phenotypic outcomes arising from developmental stressors (Clarke, 1995; Blas *et al.*, 2007; Van Buskirk & Steiner, 2009). To address this knowledge gap, we undertook a quantitative review to evaluate what factors influence variation in phenotypic responses (herein termed the developmental stress response; DSR) from across taxonomic groups (birds, reptiles, fish, mammals, insects, arachnids, and amphibians) following exposure to a broad range of developmental stressor types and exposure regimes (English & Uller, 2016; Nakagawa *et al.*, 2017).

Our quantitative review had two aims. The first, was to determine the mean (i.e. overall) effect of developmental stress on phenotypic traits. The phenotypic effects of developmental stress have often been considered negative (Micheli-Campbell *et al.*, 2011; Crespi & Warne, 2013), thus, it is predicted that the mean phenotypic response of animals exposed to developmental stressors will be negative. The simple rationale here is that development stressors cause trade-offs during development, whereby energetic or pathological constraints can lead to negative effects on phenotype and performance responses (Barker, 1995; Metcalfe & Monaghan, 2001).

The second objective was to assess quantitatively the basis for the variation in DSR across individuals, species, and stressor types in order to determine if conclusions can be drawn about the additive or interactive effects resulting from the underlying physiological processes. We predict a large heterogeneity in DSR because of the complexity of interactions among developmental stressors, environmental conditions, individuals, species and different phenotypic and performance traits (Mousseau & Fox, 1998; Shine & Downes, 1999; Agrawal, 2001; Storm & Lima, 2010).

To examine the causes of variation in phenotypic responses to developmental stress, we focus on understanding the effects of 11 variables drawn from four key categories of influence that could explain variation in developmental responses (Table 1). The first category of influence (i.e. variation in the stressor regime) we consider is how different attributes of the developmental stressor 'regime' affect variation in later-life phenotypic consequences arising from developmental stress exposure (Johnson *et al.*, 1992). It is well known that phenotypic responses vary in relation to the nature, magnitude, and duration of the developmental stressor (i.e. the stressor regime; citations below). These different attributes are each expected to influence an animal's developmental trajectory and hence the direction and magnitude of later-life phenotypic consequences. Additionally, animals appear differentially susceptible to developmental stressors depending on their developmental stage, especially during the rapid pre-natal/hatch growth phase when rates of cell replication and differentiation are highest (Gonzalez *et al.*, 2014).

The second category of influence (i.e. variation in phenotypic attributes; Table 1) focuses on understanding how inherent differences within and among individuals of the same species could influence phenotypic responses to developmental stressors. Many species show significant differences in phenotypic attributes between males and females (Lande, 1980). To achieve this, males and females undergo highly divergent growth during early life (Badyaev, 2002). As a result of sex-specific early-life developmental trajectories, corresponding differences in response to developmental stressors are also expected. Furthermore, inherent differences in developmental pathways and physiological requirements between traits, within individuals, mean that different phenotypic traits are very likely to differ in their susceptibility to developmental stressors (Møller & Swaddle, 1997; Clarke, 1998). This is predicted to influence how developmental stress affects later-life phenotypic variation.

A third category (i.e. variation in environmental processes; Table 1) considers how broad-scale environmental processes could explain variation in developmental stress responses of animals. Macrophysiological studies strongly demonstrate how large-scale environmental gradients can drive physiological variation amongst organisms (Reich, Walters & Ellsworth, 1997; Gaston, 2000; Chown, Gaston & Robinson, 2004). Global-scale variation in temperature, precipitation, and biogeochemical composition extensively influence various developmental processes, including metabolic rate, growth and reproduction of organisms (Brown *et al.*, 2004; Angilletta, 2009; Schulte, Healy & Fangue, 2011). For example, species that experience greater seasonal-temperature variation are predicted to possess mechanisms to cope better with greater temperature extremes that could lead to increased developmental stability (e.g. canalisation) (Waddington, 1942) or resistance to developmental stressors (Addo-Bediako *et al.*, 2000; Hoffmann & Woods, 2001).

The fourth category (i.e. variation in evolutionary history; Table 1) addresses how evolutionary factors (e.g. intrinsic species traits or shared phylogenetic history), predict variation in developmental stress responses of animals. Many taxa, for example, differ in metabolic organisation (e.g. ectothermy *versus* endothermy) that underpins fundamental differences in how physiological regulation of developmental trajectories proceeds, and this could further influence how developmental stressors manifest in species to produce

Table 1. Description and rationale for predictor variables evaluated in the meta-regression to determine their influences on developmental stress (i.e. phenotypic and fitness) responses of animals

| Predictor | Rationale | Key references |
|---|---|---|
| | I. **Variation in stressor regime** | |
| Developmental stressor source | Different experimental stressors (physiological, environmental, psychological, physical, nutritional) simulate different conditions and affect different physiological systems. As such, organisms are expected to differ in their developmental susceptibility to stressors depending on the type of stressor. | Gibbs (1984); Pacak *et al.* (1998) |
| Exposure stage (pre/post-natal/hatch/both) | Animals are differentially susceptible to developmental stressors depending on developmental stage, especially during the rapid pre-natal/hatch growth phase when rates of cell replication and differentiation are highest. | Gonzalez, Lotto & Hallgrímsson (2014) |
| Relative developmental age at stressor exposure | Phenotypes vary in sensitivity to environmental cues at different points in development due to: the frequency and reliability of cues, the potential fitness benefits of information and/or the constraints on plasticity. Windows of heightened sensitivity during different periods of development mean the age of onset of exposure is likely to determine some of the magnitude in developmental responses. | Fawcett & Frankenhuis (2015); Hoverman & Relyea (2007) |
| Relative exposure duration | Theory predicts that different stressor durations lead to different consequences *via* commensurate alterations to normal developmental processes. Therefore, developmental stress consequences are likely to increase with exposure duration. | Herbert & Cohen (1993); O'Keeffe & Baum (1990) |
| | **2. Variation in phenotypic attributes** | |
| Sex | Offspring developmental trajectories such as growth rates and resource requirements can differ between males and females. In poor conditions, parents invest more resources in the least-resource-intensive sex as they are less negatively affected and show lower mortality rates in response to developmental stressors. Developmental stressors are expected to have different impacts on males and females due to different parental investment in offspring. | Clutton-Brock, Albon & Guinness (1985); Trivers & Willard (1973) |
| Age at phenotype or performance measurement | Some organisms have the capacity to regulate growth when faced with different developmental stressors in order to maintain normal development, leading to an attenuated response to developmental stress. However, the physiological costs of maintaining this are known to manifest themselves in later life, such as reductions in longevity. As a result, we predict there will be differences in how the effects of developmental stressors are manifested across different life-history stages. | Boersma & Wit (1997); English & Uller (2016); Hales & Ozanne (2003) |
| Phenotypic traits | Intrinsic differences in physiological requirements and developmental pathways mean that there will be inherent variation in developmental susceptibility between phenotypic traits, even within individuals. Theory predicts that traits with higher physiological requirements and low functional significance, such as secondary sexual characteristics, will be most susceptible to developmental stressors, whilst those with high functional significance will be the least susceptible due to stabilising selection pressure. | Clarke (1998); Møller & Swaddle (1997) |
| | **3. Variation in environmental processes** | |
| Average temperature/ hot month & cold month | Variation in environmental temperature has a prodigious direct and indirect effect on animals. Physiological tolerances are linked with global clines in thermal variability, with more-variable thermal environments showing selection for higher tolerance. Evidence shows that a similar pattern exists in a species' ability to survive extreme temperatures. We expect that organisms inhabiting areas with extreme temperatures will suffer fewer negative effects of stressors due to higher selection pressure on homeostatic processes, including systems involved in responding to stressors. | Addo-Bediako, Chown & Gaston (2000); Khaliq *et al.* (2014); Stevens (1989); Sunday, Bates & Dulvy (2011) |

Table 1. Continued

| Predictor | Rationale | Key references |
|---|---|---|
| Seasonality | Theory predicts that increased developmental plasticity is favoured when (*i*) there is environmental heterogeneity (spatial or temporal), (*ii*) there are cues that reliably predict future environmental conditions and, hence, selective regimes and (*iii*) the cost of plasticity is low. Seasonality was calculated by taking the average temperature of the hottest month minus the average temperature of the coldest month. Seasonality reflects temporal heterogeneity in thermal environmental temperature. Therefore, we expect species inhabiting environments with low seasonality to respond negatively to variation in developmental conditions, rather than responding with adaptive plasticity that would be expected if they inhabited very seasonal areas with great variation. | Boyce (1979); Brodie (1975); Uller (2008); Winemiller & Jepsen (1998) |
| Average Precipitation | Annual precipitation often correlates with productivity and ecological variation that could affect environmental quality including nutritional availability/quality, pond depth/consistency and vegetation. Organisms inhabiting areas of higher resource availability and predictability are likely to experience weaker selection on plasticity and homeostatic processes associated with stress responses, and as a result, will exhibit a more negative response to developmental stress. | Alto & Juliano (2001); Love, McGowan & Sheriff (2013); Walls, Barichivich & Brown (2013) |
| | **4. Variation in evolutionary history** | |
| Taxa | This study includes mammals, birds, reptiles, amphibians, fish, insects and arachnids. Differences in reproductive mode (oviparity/viviparity), parental care and developmental environment among species mean they can differ greatly in their modalities for maintaining homeostasis and their capacity to respond to early-life stressors. As a result, we expect to see clear differences in developmental responses among taxa. | Blackburn (1999); Buckley, Hurlbert & Jetz (2012); Royle, Smiseth & Kölliker (2012) |

variation in later-life phenotypic and fitness outcomes (Buckley *et al.*, 2012). Additionally, if related species cannot easily evolve (e.g. phylogenetic constraints), so that their physiological tolerances are limited, it may also predispose certain taxa to exhibit stronger responses to developmental stress (Hoffmann, Chown & Clusella-Trullas, 2013).

Using experimental studies investigating developmental stress up until the age of sexual maturity in multiple animal taxa, we sought to determine the mean effect of developmental stressors on animal phenotype and performance responses and to examine predictors of covariation drawn from the four categories (Table 1). We analysed effect-size data using a multi-level meta-analytic (MLMA) approach to determine the overall magnitude of effect of developmental stressors. Alongside this approach, we used meta-regression (MLMR) models in order to evaluate the relative influence of individual predictor variables and allowed us to test for complex interactions between predictors that may be disproportionately influencing phenotypic variation. With both of these methods, we controlled for sampling error, study and species, and the covariance among effects that are contrasted with the same control-group data.

## II. METHODS

To identify published studies investigating the phenotypic effects of developmental conditions on animals, we conducted a search with *Google Scholar* using one or more of the following key words associated with alterations to developmental environment (developmental, early-life, post-natal/hatch, pre-natal/hatch, stress, plasticity, conditions) and/or combined with a specific type of stressor (temperature, maternal separation, handling, restraint, restriction, desiccation, predator, heat hardening, nutrition, exposure). Taxonomic terms were also included in some searches to increase the number of taxa for statistical comparison (bird, reptile, fish, mammal, insect, arachnid, or amphibian). Studies with human subjects were excluded from this meta-analysis. Only studies that conducted an experimental manipulation of the developmental conditions during pre-natal/hatch and/or post-natal/hatch life up until the age of sexual maturity were included. Studies all included experimental manipulation of an animal's physical, physiological, psychological, environmental or nutritional conditions during these periods.

### (1) Extraction of data from papers

The means, standard deviations and sample sizes, or $F$ or $t$ values and sample sizes from phenotypic and performance-related measures were obtained from 111 studies. Data were obtained from each study through direct reporting in the paper in text or tables or statistical results. Data were taken from figures when explicitly stated; if data were not specifically stated in text, no data were taken (programs designed to extract data from graphs and figures were not used). From the 111 studies, a total

of 866 effect sizes were calculated using the Cohen's *d* estimate (see online Supporting information, Tables S1–S4). The explanatory variables extracted from each study are listed below. When using means and standard deviations, we calculated effect sizes by subtracting the mean of the control group from the mean of the treatment group and dividing the result by the pooled standard deviation of both groups. When using *F* values, we calculated effect sizes by taking the square root of the *F* value multiplied by the pooled sample size, and then divided that figure by the square root of the pooled degrees of freedom multiplied by the square root of the two sample groups multiplied together. When using *t* scores, we calculated effect sizes by multiplying the *t* score by the pooled sample size and dividing it by the square root of the pooled degrees of freedom multiplied by the square root of the two sample group sizes multiplied together. We used a guide of small, medium and large effect sizes as 0.2, 0.5 and 0.8, respectively (Cohen, 1988). We calculated the sampling variance for each study following established methods (Cohen, 1988). Multiple effect sizes were obtained from some individual studies which had multiple stressor treatments compared to a single control, in these cases there was a single effect size calculated per developmental treatment. Where studies measured phenotype or performance traits separately for different sexes and age classes, we calculated independent effect sizes for each of these separate comparisons.

In preliminary diagnostic analyses of effect-size data, a caterpillar plot was used to display variation in effect sizes and their associated sampling variance and to present the mean effect-size estimate. Funnel plots were visually inspected in order to test for publication bias, and by conducting Egger's regression analysis (Egger *et al.*, 1997). A *Q* heterogeneity test was used to measure effect-size heterogeneity ($I^2$) within data. These procedures were completed using the package metafor in R (Viechtbauer, 2010).

## (2) Meta-analysis and meta-regression analyses

Bayesian meta-analysis and meta-regression models were used to evaluate relationships between developmental stress response and putative predictor variables (Table 1; Spiegelhalter *et al.*, 2002). Specifically, Bayesian linear mixed-effect models (BLMMs) were used to estimate effects of predictor variables on effect-size data using Bayesian Markov Chain Monte Carlo (MCMC) methods within the MCMCglmm package (Hadfield, 2015) in R. Effect-size data analysed using BLMMs were fitted with a Gaussian error and a uniform prior distribution (i.e. an inverse Wishart prior with $V = 0.002$ and nu = 1) (Hadfield, 2015). Model parameter estimates are based on 1000 iterations sub-sampled from 650,000 iterations after a 150,000-sample burn-in and a thinning interval of 500 which was more than sufficient for the MCMC chain to reach stationarity. Satisfactory convergence of model parameters (or posterior distributions) were evaluated using the Gelman–Rubin statistic (Gelman & Rubin, 1992). We deemed that model convergence was appropriate if all sets of three chains had a Potential Scale Reduction (PRS) factor < 1.01.

### (*a*) *Meta-analysis*

First, in order to determine how each predictor variable influenced DSR effect sizes, the additive effects of 11 different predictor variables on DSR effect size were evaluated with a single BLMM. These predictor variables consisted of: average temperature (°C) of the hottest month, average temperature (°C) of the coldest month, average precipitation (mm), timing of stressor exposure relative to development period length, duration of exposure relative to development period length, taxa (mammals, fish, amphibians, arachnids, birds, reptiles and insects), class of developmental stressor, age class that performance was measured (juvenile, sub-adult, adult), exposure stage (post-natal/hatch, pre-natal/hatch or both), the class of phenotypic or performance trait measured (reproductive, physiological, morphological, performance, behavioural), sex of the test subjects (male female, both), and two random effects (study ID and species).

### (*b*) *Meta-regression*

Second, in order to assess the basis for variation in DSR effect sizes, and determine the most influential variables, 21 different models from the 11 predictor variables used in the meta-analysis were constructed. These models evaluated the effects of univariate and multivariate combinations of predictor variables alongside null models to explain variation in effect sizes. This included four models that represented the influence of the stressor regime. The first modelled the additive effects of stressor regime duration and timing, the second modelled an interaction between stressor duration and timing, the third modelled the additive effect of the class of stressor, its duration, exposure stage and timing, while the fourth was similar to the third, except timing was evaluated as a cubic term, to determine if DSR effect sizes exhibited a non-linear trend across the range of timing of exposure of developmental stressors. We included a model representing the additive effects of individual variation, which included the age class at which phenotype and performance were measured, the phenotypic and performance trait being measured and the sex of the subjects. Furthermore, there was a model representing environmental variation, which included the additive effects of the average temperature of the hottest month, average temperature of the coldest month and average precipitation. We also included the global model from the meta-analysis to determine how influential it was compared with the other models, as well as a model for each predictor variable independently. Study and species identity were included as random effects in all models. However, to evaluate better the variation due to these random effects, three null models (i.e. intercept-only models that comprised one or both random effects) were considered. A Bayesian information-theoretic approach was then used, *via* the Deviance Information Criterion (DIC) to identify and rank the relative support for each model (Spiegelhalter

*et al.*, 2002). The best-fitting model has the smallest DIC and models were ranked from best to worst, according to the differences between each model's DIC ($\Delta i$) values. Model weights ($w_i$) were computed from the DIC values following Burnham & Anderson (2003). Model weights can be interpreted as the probability of evidence that the model is the 'best' model. The only models considered biologically informative were top-ranked models that exceeded the rank of the null models by ≥2 DIC units (Burnham & Anderson, 2003).

### (c) *Statistical inference for parameter estimates from meta-analysis and meta-regression*

Consistent with use of Bayesian methods, our statistical inference was informed by values of the posterior mean (PM) and 95% higher posterior densities (HPD) or credible intervals (CI) to determine the statistical significance of BLMM parameter effects (Spiegelhalter *et al.*, 2002). Here, if the 95% credible intervals of fixed parameter effects did not overlap zero, these effects were deemed to be statistically influential. Both the marginal $R^2$ to estimate the total variance explained by fixed effects and the conditional $R^2$ to estimate the total variance explained by both fixed and random effects in top-ranked models were also calculated (Nakagawa & Schielzeth, 2013).

## (3) Predictor-variable data collection and estimation

We considered 11 parameters as putative covariates (Table 1) that could be incorporated into models to predict variation in effect sizes. For each effect size, corresponding environmental, stressor regime, phenotypic and taxonomic information were extracted. The parameters considered were collected and estimated as follows.

### (a) *Developmental stressor source*

Each study stated the source of developmental stress that was applied in their methodology. Stressors were grouped into five general stressor classes for comparison: nutritional, environmental, psychological, physical and physiological. Stressors were placed into these classes based on the following criteria: nutritional stressors were specifically any manipulation of an animal's diet or nutrition such as reduction in quality or quantity of food. Environmental stressors included changes to the overall environment in which animals were kept, these include temperature stressors in ectotherms, or rapid reductions in water levels for amphibians. Psychological stressors were comprised of changes in an animal's environment that did not directly impact their survival, but presented a recognisable challenge to the organism; notable examples include visual or chemical exposure to live or simulated predators, or maternal separation in juvenile animals. Physical stressors were stressors which physically impacted the animals, such as physical restraint, or being manually harassed (e.g. chasing). Finally, physiological stressors were those that altered the otherwise regular function of an animal's physiology, including exposure to chemicals or hormones.

### (b) *Exposure stage*

Whether exposure to stressors occurred during pre-natal/hatch, post-natal/hatch or over both stages of an animal's life was stated in each study and this was recorded as a predictor variable. Pre-natal/hatch exposure was recorded as a negative number, whilst post-natal/hatch exposure was recorded as a positive number.

### (c) *Relative age of stressor exposure*

The number of days pre-or post- hatch or birth that an animal was first subjected to treatment within a study was recorded. The average age that the species of study reached sexual maturity was also taken from sites on the internet, mainly http://animaldiversity.org/, or other sites were used when information was unavailable there. To create a standardised variable that represented the age an animal was exposed to the stressor relative to the length of its development, the following calculation was used: $\left(\frac{exp}{mat}\right) * 100$, where $exp$ = number of days pre-or post-hatch or birth that an animal was first subjected to treatment, and $mat$ = average age that the species of study reached sexual maturity.

### (d) *Relative stressor exposure duration*

The amount of time (days) that an animal was exposed to a stressor was recorded from each study. Once again this was standardised by making it relative to each species age until sexual maturity. This was calculated by: $\left(\frac{dur}{mat}\right) * 100$, where $dur$ = amount of time in days that an animal was exposed to a stressor, and $mat$ = average age that the species of study reached sexual maturity.

### (e) *Sex of subject*

For each study it was recorded whether the subjects involved in a study were male, female or both.

### (f) *Age at phenotype or performance testing*

We recorded the age at which species phenotype and performance were measured and grouped them into three different age classes: juvenile, sub-adult and adult. These were added as a predictor variable to assess what effect aging has on developmental stress responses.

### (g) *Phenotypic trait class*

The specific phenotypic trait that a study measured the performance of was recorded. For use as a predictor variable, these were grouped into five different classes: physiological, morphological, behavioural, reproductive or performance. Physiological traits were those which measured

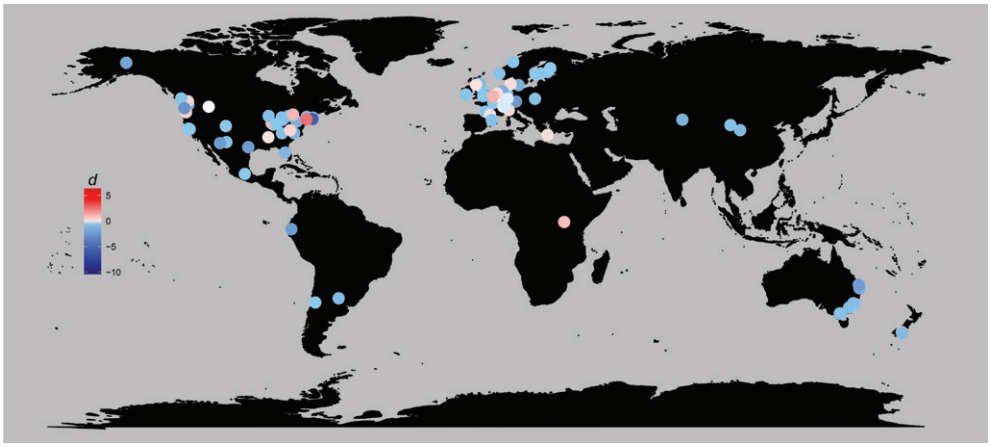


**Fig. 1.** (A) Global map of published study localities ($N = 111$) and associated effect sizes (Cohen's $d$; $N = 866$) of developmental stressor effects on phenotypic variation and performance in animals.

the functional aspect of an animal's body. Morphological traits were measurements of the structure of an organism's body. Behavioural traits related to measurements of activity or decision making of animals, reproductive traits were measurements of reproductive capacity, while performance traits were measurements of how well an animal performed at a task.

### (*h*) *Variation in environmental processes*

To determine temperature and climatic variables of studies, latitude and longitude were collected. Latitude and longitude generally were indicated in the text of the study; however, some gave place names instead of coordinates. In this case, *Google Earth* version 6 (Google Inc., Mountain View, CA, USA. http://www.google.com/earth) was used to search for the place names and obtain the coordinates.

### (*i*) *Temperature and climatic variables*

Climate data were recorded, but as climate data specific to each of the studied locations were not reported, an approximation of the temperature around the location sites was given. This was achieved by identification of the closest weather station to the study. Data regarding average temperature, hot month, cold month and average precipitation were gathered from the website weatherbase (http://www.weatherbase.com/). Seasonality for each location was calculated by taking the average temperature of the hottest month, minus the average temperature of the coldest month.

### (*j*) *Variation due to phylogenetic affiliations*

Taxonomic class was recorded for each species in the database. There were seven different taxonomic classes comprising: Aves, Mammalia, Insecta, Arachnida, Actinopterygii, Amphibia and Reptilia.

## III. RESULTS

### (1) Meta-analysis diagnostics

One hundred and eleven studies were examined comprising 64 species culminating in 866 Cohen's $d$ effect-size measurements of developmental stressors across phenotypic responses of animals (Fig. 1). The posterior mean (PM) value of Cohen's $d$ DSR effect size was −0.510 (LCI −0.667, UCI −0.370) (Fig. 2A). This estimate indicated a moderately strong negative effect that was significant as the range of the upper (UCI) and lower (LCI) credible intervals did not overlap zero. This result clearly indicated that animals exposed to developmental stressors had on average decreased phenotypic and performance responses relative to animals in control treatments. DSR effect sizes exhibited highly significant heterogeneity among studies ($I^2 = 96.18\%$; $Q$ heterogeneity test = 21164.151, $P < 0.0001$), with no evidence for publication bias (Egger's test for funnel plot asymmetry: $z = 0.778$, $P = 0.436$; Fig. 2B).

### (2) Meta-analysis

Results of the meta-analysis indicated that multiple predictor variables had significant effects on DSR effect sizes of animals (Fig. 3). Upper environmental temperature limit, represented by the mean temperature of the hottest month in summer, had a significant negative impact on DSR effect sizes (PM hottest month: −0.206, LCI −0.357, HCI −0.056, $P = 0.012$), indicating that species exposed to higher temperatures were more likely to exhibit adverse long-term effects of developmental stressor exposure. Additionally, increased duration of developmental stressor exposure (relative to an animal's time until sexual maturity) was found to significantly reduce DSR measures (PM relative stressor duration: −0.201, LCI −0.339, HCI −0.069, $P = 0.002$). Effect sizes were also found to be significantly more negative in juveniles compared with adult animals (PM juveniles: −0.447, LCI −0.754, UCI −0.140, $P = 0.008$), whilst

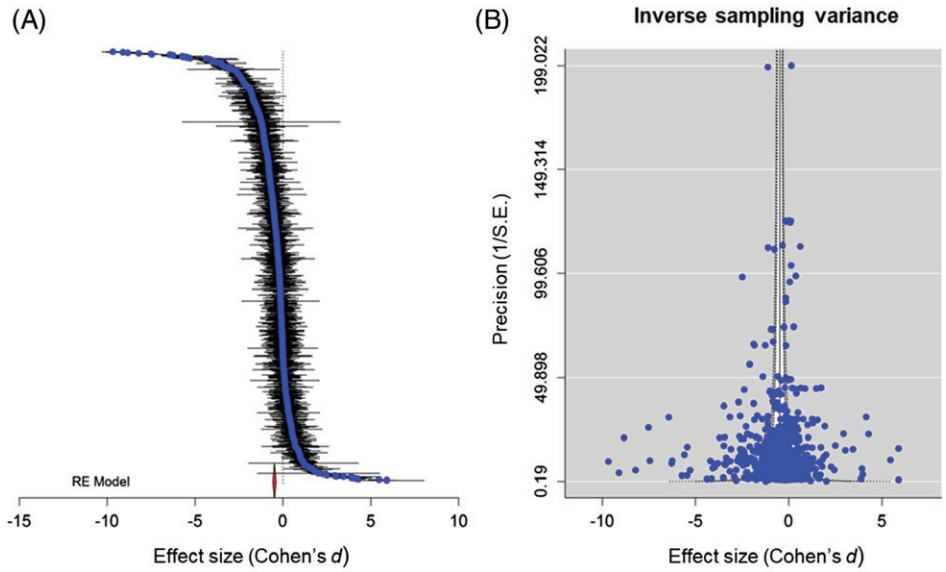


**Fig. 2.** (A) Caterpillar plot of ordered mean Cohen's *d* effect size (blue filled circles) and study sampling variance (black line) and the mean effect size (red diamond) of all studies used in the meta-regression analysis. (B) Funnel plot presenting distribution of Cohen's *d* effect size (blue filled circles) against study precision estimates, with counter-shaded confidence intervals (90% and 95% CI).

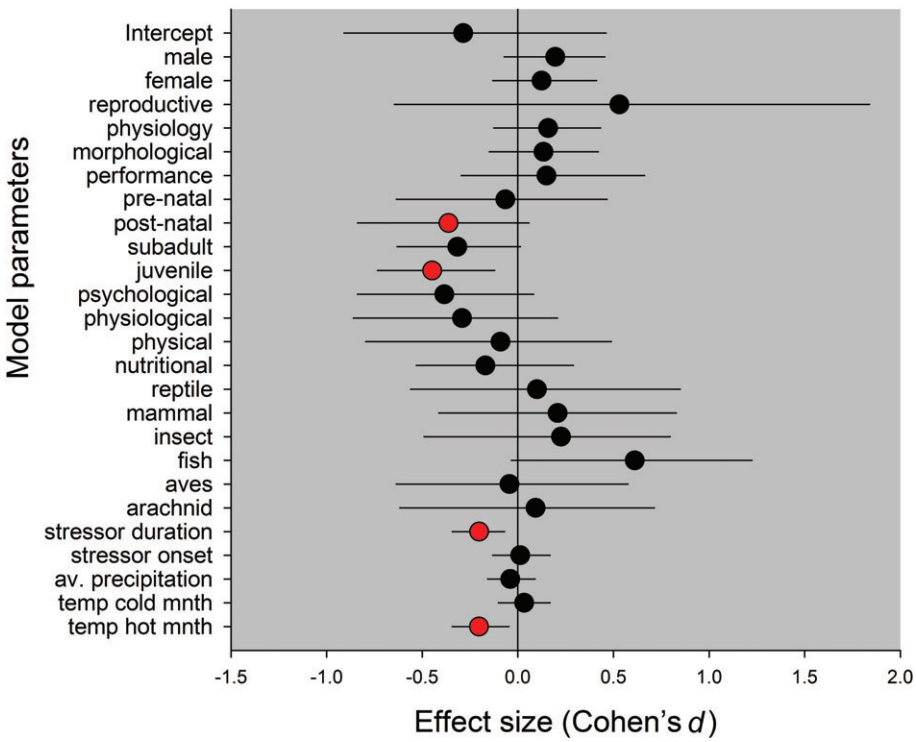


**Fig. 3.** Meta-analysis coefficient plot depicting parameter effects of all putative covariates on the posterior mean and 95% CI of effect sizes. Parameters with red filled posterior means are considered to have significant influence (i.e. CI do not overlap zero) on the magnitude of effect sizes estimated from studies reporting effects of developmental stressors on phenotype and performance response measures. Reference groups for comparison of categorical variables were: male/female compared to 'both'; reproductive/physiology/morphological/performance compared to 'behaviour'; pre-natal/post-natal/hatch compared to 'both'; juvenile/subadult compared to 'adult'; psychological/physiological/physical/nutritional compared to 'environmental'; reptiles/mammals/insect/arachnids/birds/fish compared to 'amphibians'.

Table 2. Summary table of Bayesian linear mixed meta-analysis regression models ranked by Deviance Information Criterion (DIC), Delta Deviance Information Criterion (ΔDIC) indicating parameter-fit differences among models, the model weight ($\omega_i$), the number of parameters ($K$) within each model and the model likelihood (logLik) deviance for each model.

| Model | DIC | ΔDIC | $\omega_i$ | $K$ | logLik |
|---|---|---|---|---|---|
| Duration × timing | 2526.6 | 0 | 0.629 | 6 | −1177.62 |
| Duration + timing | 2529.2 | 2.54 | 0.177 | 8 | −1178.04 |
| Duration | 2530.2 | 3.57 | 0.106 | 6 | −1178.9 |
| Null(species) | 2534.2 | 7.58 | 0.014 | 4 | −1183.78 |
| Hotmnth (°C) | 2534.5 | 7.86 | 0.012 | 6 | −1181.53 |
| Taxa | 2534.8 | 8.2 | 0.01 | 11 | −1182.26 |
| Age | 2535.2 | 8.56 | 0.009 | 7 | −1182.4 |
| Null(studyID+species) | 2535.2 | 8.62 | 0.008 | 5 | −1181.92 |
| Coldmnth (°C) | 2535.6 | 9.02 | 0.007 | 6 | −1181.98 |
| Exposure | 2535.8 | 9.2 | 0.006 | 7 | −1181.07 |
| Timing | 2536.2 | 9.54 | 0.005 | 6 | −1182.69 |
| Stressor + duration + exposure + timing | 2536.3 | 9.68 | 0.005 | 6 | −1181.54 |
| Precipitation (mm) | 2536.9 | 10.31 | 0.004 | 13 | −1179.28 |
| Hotmnth (°C) + coldmnth (°C) + precipitation (mm) | 2537.1 | 10.53 | 0.003 | 7 | −1182.71 |
| Sex | 2537.8 | 11.22 | 0.002 | 8 | −1182.81 |
| Stressor | 2541.1 | 14.47 | 0 | 9 | −1184.04 |
| Trait | 2541.7 | 15.13 | 0 | 9 | −1185.92 |
| Age + trait + sex | 2545.1 | 18.52 | 0 | 13 | −1184.63 |
| Hotmnth (°C) + coldmnth (°C) + precipitation (mm) + timing + duration + taxa + stressor + age + exposure + trait + sex | 2547.8 | 21.15 | 0 | 30 | −1183.8 |
| Null(studyID) | 2550.9 | 24.25 | 0 | 4 | −1188.17 |
| Stressor + duration + exposure + timing$^2$ | 2551.8 | 25.15 | 0 | 31 | −1182.83 |

Model parameter definitions include: duration = the relative developmental stressor duration; exposure = whether stressor occurred during pre- or postnatal/hatch development; timing = the relative onset of the stressor during development; stressor = the class of developmental stressor; hotmnth = the average temperature (°C) of the warmest month of the year in sampling/study location; coldmnth = the average temperature (°C) of the coldest month of the year in sampling/study location; precipitation = yearly average precipitation (mm) of the sampling/study location; taxa = the class of species in the study; age = the age of the animals in the study; traits = the class of trait affected; sex = the sex of the animal studied; studyID = individual study number from database; species = random term representing individual study number from database.

subadult animals had a marginally non-significant negative trend compared to adults (PM subadults: −0.322, LCI −0.632, HCI 0.032, $P = 0.070$). There were no differences between the effect sizes of any of the different taxa included in the meta-analysis. The marginal $R^2$ (i.e. proportion of variance explained by fixed factors) and conditional $R^2$ (i.e. proportion of variance explained by both the fixed and random factors) within the meta-analysis model explained 3.4% (3–3.7%) and 17% (9–25.2%) of variation in effect sizes, respectively.

### (3) Meta-regression

Further to evaluate processes influencing variation in the DSR of animals we estimated and ranked the effects of 21 candidate BLMM models that comprised a combination of one of more predictor variables (Table 2). Here, a single model resulted in highly supported (i.e. ΔDIC < 2) *a priori* parameter effects on DSR effect sizes. The top-ranked model indicated that two aspects of the stressor regime, onset timing and duration, were simultaneously influencing two factors of DSR effect-size responses: both the direction of a response and its magnitude. Timing of stressor onset being early or late during development led to opposing responses (positive/negative) in DSR effect size. Earlier exposure to a stressor led to more-positive DSR effect sizes, whilst exposure later during development led to more-negative DSR effect sizes. Simultaneously, longer exposure to a stressor caused DSR effect-size responses to be stronger in magnitude, regardless of whether it was a positive or negative response (HPD stressor duration × stressor onset timing: PM −0.48, LCI −0.63, UCI −0.34; Fig. 4). This model of the stressor regime greatly outperformed the three null models (ΔDIC = 7.6–24.25) parameterised with one or both random effects (i.e. study or species identity). For the top-ranked model, the marginal $R^2$ (i.e. proportion of variance explained by fixed factors) and conditional $R^2$ (i.e. proportion of variance explained by both the fixed and random factors) explained 2 and 20% of variation in effect sizes, respectively.

## IV. DISCUSSION

Developmental stressors are increasingly recognised for their pervasive influence on the ecology and evolution of animals (Pankhurst & Munday, 2011; Baumann, Talmage &

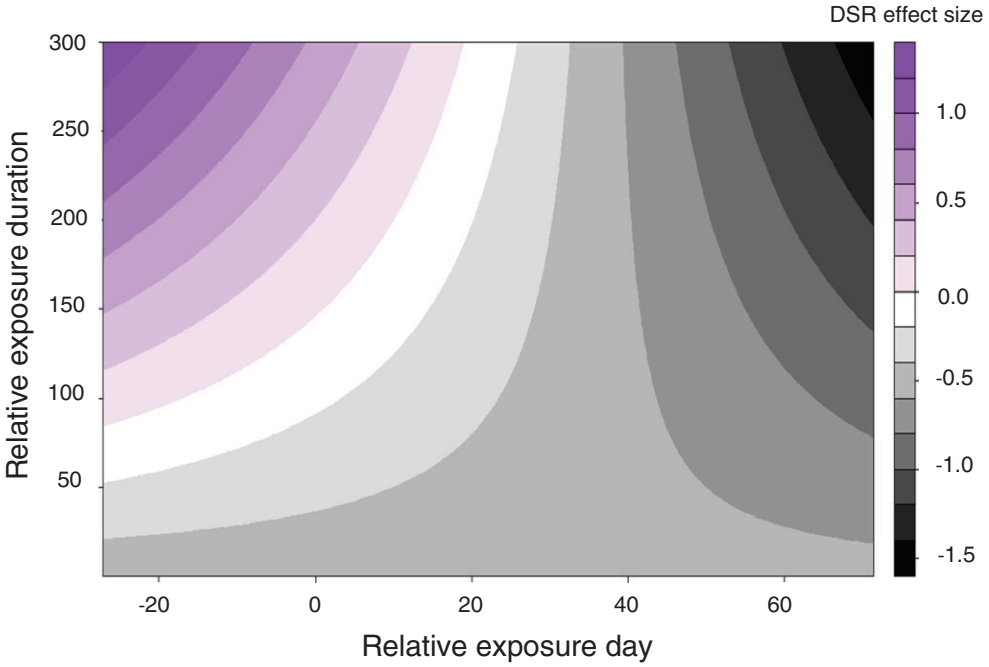


**Fig. 4.** Contour plot depicting parameter effects of the top-ranked meta-regression model on effect sizes of animals. DSR, developmental stressor effect.

Gobler, 2012; Buchanan, Grindstaff & Pravosudov, 2013). However, key questions remain, such as: what is the general effect of developmental stress on phenotypic trait variation and performance? Our quantitative review results confirm that developmental stressors produce predominantly negative phenotypic effects in animals. However, it is important to note that we observed considerable heterogenity in developmental stress responses, including neutral and positive responses. Neutral effects may indicate that, at least for some species, there may be potential for developmental stability, or that these species were exposed to relatively benign developmental stressors (Siegal & Bergman, 2002). Similarly, evidence of positive effects are consistent with some theoretical expectations and evidence that developmental stressors arising from maternal or environmental influences can invoke improved phenotype and performance responses (Mousseau & Fox, 1998; Shine & Downes, 1999; Agrawal, 2001; Storm & Lima, 2010; Crino *et al.*, 2014*b*). Indeed, it is well known that in plant and invertebrate species low to mild sub-lethal exposure to developmental stressors can confer positive consequences such as improved lifespan (Le Bourg *et al.*, 2001; Hercus *et al.*, 2003), or increased tolerances to subsequent stressor exposure (Chen, Shen & Li, 1982; Xu *et al.*, 2006; Wiegant *et al.*, 2009).

### (1) Meta-analysis

Our meta-analysis evaluated 11 putatively important predictor variables, of which three parameters significantly influenced variation in the developmental stress responses of animals: environmental variation, attributes of the developmental stressor regime, and the age-class of animals. With respect to environmental influences, we showed that temperature had a significant influence on the DSR of animals. Specifically, effect sizes were more negative for animals inhabiting areas with higher average temperatures during the warmest month of the year. This finding, contrary to our predictions (Table 1), indicates that adaptation to extreme temperatures does not necessarily lead to greater stressor resistance, this is despite stress-response systems such as the hypothalamo–pituitary–adrenocortical (HPA) axis (Michel *et al.*, 2007) and heat shock proteins (Feder & Hofmann, 1999) playing a major role in responding to thermal stressors. Developing animals may utilise other life-history strategies to counteract the effects of thermal stress that do not require a physiological response to stressors. For example, our study does not account for how species might adapt behaviourally to use microhabitats to escape extreme temperatures (Baudier *et al.*, 2015), or whether animals time their breeding and development periods to be when the costs of thermoregulation are low (Piersma, Cadée & Daan, 1995), or even migrating before extreme temperatures occur. Species adapted to avoid extreme temperature exposure rather than tolerate such conditions through homeostatic processes may explain why species inhabiting areas with higher average temperatures during the warmest month of the year responded negatively to stressors. More broadly, these results suggest, similar to other macro-physiology studies, that attributes of environmental temperature can be an important determinant of global patterns of phenotypic variation of animals (Chown *et al.*, 2004; Clusella-Trullas, Blackburn & Chown, 2011). Thus, large-scale temperature-induced patterns in DSR variation

could underlie developmental effects on life-history traits or trade-offs that influence phenotypic variation (Crespi *et al.*, 2013). Plausibly, environmental variation in temperature, either through direct or indirect effects, could influence developmental processes such as growth rate, particularly in ectotherms (Van der Have & De Jong, 1996; Laugen, Laurila & Merilä, 2003).

Secondly, as predicted, increased stressor duration led to more-negative DSR effect sizes. This is perhaps intuitive because increased duration of developmental stressor exposure would elevate physiological trade-offs in which resources nominally allocated to normal development and growth would instead be lost to costs or defensive measures (Stevens *et al.*, 1999; Amat, Aguilera & Visser, 2007; Walters & Heil, 2007). Furthermore, this trend makes clear that persistent exposure to developmental stressors does not necessarily promote resistance or tolerance to stressors.

Although there is abundant literature describing how effects of developmental stress can persist across life, our analyses indicate that animals exposed to developmental stressors can at least partially compensate through subsequent phenotypic development (e.g. compensatory growth). This is demonstrated in our results, with juveniles having significantly lower DSR effect sizes, and sub-adult age classes having marginally non-significant DSR effect sizes as a result of developmental stress compared with adults, which were the age class comparison group (Fig. 3). This attenuation (i.e. 'wash-out') of the early-life effects of stressful conditions across ontogeny (Boersma & Wit, 1997) would likely mean adult animals have less-compromised phenotype and performance responses than juveniles and sub-adults. However, this ability to compensate comes with several significant disadvantages not quantified by phenotype and performance response variables. Not only would survivability potentially be reduced during vulnerable juvenile stages, but the costs of harsh developmental conditions could ultimately have direct and indirect fitness costs to adults (Simmons, Templeton & Gertz, 2001), such as through reduced longevity (Metcalfe & Monaghan, 2001; Hales & Ozanne, 2003) or sexual attractiveness (Spencer *et al.*, 2005).

### (2) Meta-regression

To evaluate further the relative importance of variation on DSR effect sizes we used a meta-regression approach to identify the most influential predictor variables and to address the potential for multiple mechanisms to drive phenotypic variation. The best model indicated that the interaction between the duration of the developmental stress exposure and the timing the stressor commenced during development explained the most variation in the DSR of animals. The interaction between these two attributes influenced DSR effect sizes in two respects. Clearly, increases in stressor duration led to increases in response. It is likely that intense stressors trigger greater physiological responses, thus leading to a greater differentiation from the default developmental pathway (Natelson *et al.*, 1988). Intriguingly, the developmental timing (i.e. early or late) of stressor onset led to opposing responses in DSR. Contact with stressors later in development led to more-negative DSR effect sizes than those encountered in phases of early development (e.g. particularly pre-natal/hatch exposure), which is consistent with some experimental studies of developmental stressors (Shanks *et al.*, 2000; Martin, 2009) showing improved performance from very early stressor exposure. Importantly, this result helps describe a basis to how timing of developmental stressors could explain the difference between a predictive adaptive response (Gluckman, Hanson & Spencer, 2005; Saastamoinen *et al.*, 2010) and the more commonly associated negative DSR. Previous work has found that exposure to stressors during developmental 'critical windows' (i.e. periods of trait senstivity) (Burggren & Mueller, 2015; Fawcett & Frankenhuis, 2015) leads to the most dramatic changes in phenotype across life history (Hoverman & Relyea, 2007; Carvajal-Salamanca *et al.*, 2008; Kotrschal, Szidat & Taborsky, 2014). Our results indicate these periods are primarily very early during development, however, these positive phenotypic responses and their critical windows are related to species- and stressor-specific cues about the environment (Fawcett & Frankenhuis, 2015) and, therefore, are likely inherently varied and incredibly hard to predict. Furthermore, based on evidence in the literature (Graham *et al.*, 1993; Gluckman & Hanson, 2004; Gluckman *et al.*, 2009), and the overall negative trend in DSR found throughout the meta-analysis, clearly positive phenotypic responses are not common. However, the more common negative phenotypic responses to stress are not necessarily maladaptive, and could constitute an adaptive response, particularly if the developmental stressor predicts conditions that persist throughout an organism's life (Bateson, Gluckman & Hanson, 2014). Studies from humans (Bateson, 2007; Obradović *et al.*, 2010) as well as mice (*Mus musculus*) and rats (*Rattus norvegicus*) (Oomen *et al.*, 2010; Santarelli *et al.*, 2014) demonstrate that the adaptive value of a stress response is determined primarily by environmental context (Ellis & Boyce, 2008). The studies included herein (see Table S4) were exclusively experimental and lacked the context of a natural environment, therefore, we cannot determine whether changes in phenotype and performance responses under laboratory conditons would constitute adaptive or maladaptive stress responses.

### (3) Strength of evidence

Although our meta-analysis and meta-regression models produced significant effects and outperformed null models, the relative extent of their capacity to explain variation in DSR effect sizes was small, accounting for only 3.4 and 2.0% of variation, respectively. Although low, these results are consistent with other meta-analyses observing developmental stress effects, such as that by English & Uller (2016), which saw nutritional deprivation during development explain 8% of variation in mean longevity. Within our study, the combination of fixed and random

effects extended the explained variance to 17 and 20% for the global and top-ranked models. Clearly, idiosyncratic qualities relating to differences among individual species and studies were responsible for a relatively larger proportion of the variance observed within DSR effect sizes. Rather than following any systematic patterns that can easily be predicted, the low level of variance explained by variables within both the top ranked and global models suggests that developmental stress responses are highly heterogenous in nature. This is not entirely unexpected, as there are a multitude of different and complex mechanisms by which stress can affect phenotype (Johnson *et al.*, 1992; Herman *et al.*, 2003). In this study, we considered many processes that are intuitive or have been recognised to be important determinants of variation in stress responses of organisms. Although the interaction between stressor duration and the onset of the stressor performed the best of all the predictors we evaluated, it is likely that developmental responses often remain inherently variable in nature.

Thus the robustness of this study was tested using appropriate methods (Møller & Jennions, 2001) and it still gives strong insights into what influences phenotypic responses in regards to developmental stress. Therefore, the results can be considered to be an accurate representation of the effects of developmental stress (Duval & Tweedie, 2000).

## V. CONCLUSIONS

(1) Utilising both meta-regression and meta-analysis techniques to analyse the phenotypic effects of developmental stress has allowed several important questions to be answered.

(2) Phenotypic and performance-based responses to developmental stressors are typically moderately strong and negative, however studies document the capacity for neutral and positive responses.

(3) Meta-regression model ranking shows that, regardless of species or environmental conditions, the interaction between stressor onset timing and duration has the most influence over the way in which animals respond to developmental stressors. Longer exposure to a stressor, relative to the duration of an animal's development, results in greater phenotypic or performance responses, however, the outcome, whether the response is adverse or beneficial, varies dependent upon the age stressor exposure began.

(4) Higher environmental temperature led to more-negative responses to developmental stressors. Furthermore, the phenotypic effects of developmental stress are more negative during juvenile life stages compared to adult life stages.

(5) Consistent with the results from other meta-analyses, there was evidence of only a small degree of phenotypic variation explained by environmental and physiological factors. Evidently, animal responses to development stressors are highly variable in nature, and inherently difficult to predict.

(6) Future directions should focus on determining whether the effects of developmental stress could translate into adaptive or maladaptive changes within the context of an animal's natural environment.

## VI. ACKNOWLEDGEMENTS

We are grateful to all authors who contributed data (listed in the electronic supplementary material).

Author contributions: T.S.J. and H.E. conceived the study with input from K.L.B and O.L.C.. H.E. compiled the metadata. T.S.J. and H.E. conducted analyses and produced results and figures. H.E. wrote the manuscript with significant input from T.S.J., K.L.B and O.L.C.

## VII. REFERENCES

*References marked with asterisk have been cited within the supporting information.*

Addo-Bediako, A., Chown, S. L. & Gaston, K. J. (2000). Thermal tolerance, climatic variability and latitude. *Proceedings of the Royal Society of London B: Biological Sciences* **267**, 739–745.

Agrawal, A. A. (2001). Phenotypic plasticity in the interactions and evolution of species. *Science* **294**, 321–326.

*Almasi, B., Roulin, A., Korner-Nievergelt, F., Jenni-Eiermann, S. & Jenni, L. (2012). Coloration signals the ability to cope with elevated stress hormones: effects of corticosterone on growth of barn owls are associated with melanism. *Journal of Evolutionary Biology* **25**, 1189–1199.

*Alonso-Alvarez, C., Bertrand, S., Devevey, G., Prost, J., Faivre, B., Chastel, O., Sorci, G. & Promislow, D. (2006). An experimental manipulation of life-history trajectories and resistance to oxidative stress. *Evolution* **60**, 1913–1924.

Alto, B. W. & Juliano, S. A. (2001). Precipitation and temperature effects on populations of *Aedes albopictus* (Diptera: Culicidae): implications for range expansion. *Journal of Medical Entomology* **38**, 646–656.

Amat, J. A., Aguilera, E. & Visser, G. H. (2007). Energetic and developmental costs of mounting an immune response in greenfinches (*Carduelis chloris*). *Ecological Research* **22**, 282.

*Amiel, J. J. & Shine, R. (2012). Hotter nests produce smarter young lizards. *Biology Letters* **8**, 372–374.

*Andersen, S. L. & Teicher, M. H. (2004). Delayed effects of early stress on hippocampal development. *Neuropsychopharmacology* **29**, 1988.

Angilletta, M. J. (2009). *Thermal Adaptation: A Theoretical and Empirical Synthesis*. Oxford University Press, Oxford, U.K.

Badyaev, A. V. (2002). Growing apart: an ontogenetic perspective on the evolution of sexual size dimorphism. *Trends in Ecology & Evolution* **17**, 369–378.

Barker, D. J. (1995). Fetal origins of coronary heart disease. *British Medical Journal* **311**, 171.

*Barton, B. A., Schreck, C. B. & Barton, L. D. (1987). Effects of chronic cortisol administration and daily acute stress on growth, physiological conditions, and stress responses in juvenile rainbow trout. *Diseases of Aquatic Organisms* **2**, 173–185.

Bateson, P. (2007). Developmental plasticity and evolutionary biology. *The Journal of Nutrition* **137**, 1060–1062.

Bateson, P., Gluckman, P. & Hanson, M. (2014). The biology of developmental plasticity and the Predictive Adaptive Response hypothesis. *The Journal of Physiology* **592**, 2357–2368.

Baudier, K. M., Mudd, A. E., Erickson, S. C. & O'donnell, S. (2015). Microhabitat and body size effects on heat tolerance: implications for responses to climate change (army ants: Formicidae, Ecitoninae). *Journal of Animal Ecology* **84**, 1322–1330.

Baumann, H., Talmage, S. C. & Gobler, C. J. (2012). Reduced early life growth and survival in a fish in direct response to increased carbon dioxide. *Nature Climate Change* **2**, 38–41.

*Bengston, S. E., Pruitt, J. N. & Riechert, S. E. (2014). Differences in environmental enrichment generate contrasting behavioural syndromes in a basal spider lineage. *Animal Behaviour* **93**, 105–110.

*Benson-Amram, S., Weldele, M. L. & Holekamp, K. E. (2013). A comparison of innovative problem-solving abilities between wild and captive spotted hyaenas, *Crocuta crocuta*. *Animal Behaviour* **85**, 349–356.

*Birkhead, T., Fletcher, F. & Pellatt, E. J. (1999). Nestling diet, secondary sexual traits and fitness in the zebra finch. *Proceedings of the Royal Society of London B: Biological Sciences* **266**, 385–390.

Blackburn, D. G. (1999). Viviparity and oviparity: evolution and reproductive strategies. In *Encyclopedia of Reproduction* (ed. E. Knobil and J. D. Neill), pp. 994–1003. Academic Press, New York

Blas, J., Bortolotti, G., Tella, J., Baos, R. & Marchant, T. (2007). Stress response during development predicts fitness in a wild, long lived vertebrate. *Proceedings of the National Academy of Sciences of the United States of America* **104**, 8880–8884.

*Blount, J. D., Metcalfe, N. B., Arnold, K. E., Surai, P. F., Devevey, G. L. & Monaghan, P. (2003). Neonatal nutrition, adult antioxidant defences and sexual attractiveness in the zebra finch. *Proceedings of the Royal Society of London B: Biological Sciences* **270**, 1691–1696.

Boersma, B. & Wit, J. M. (1997). Catch-up growth. *Endocrine Reviews* **18**, 646–661.

*Boggs, C. L. & Niitepõld, K. (2016). Effects of larval dietary restriction on adult morphology, with implications for flight and life history. *Entomologia Experimentalis et Applicata* **159**, 189–196.

*Bonisoli-Alquati, A., Matteo, A., Ambrosini, R., Rubolini, D., Romano, M., Caprioli, M., Dessì-Fulgheri, F., Baratti, M. & Saino, N. (2011*a*). Effects of egg testosterone on female mate choice and male sexual behavior in the pheasant. *Hormones and Behavior* **59**, 75–82.

*Bonisoli-Alquati, A., Rubolini, D., Caprioli, M., Ambrosini, R., Romano, M. & Saino, N. (2011*b*). Egg testosterone affects wattle color and trait covariation in the ring-necked pheasant. *Behavioral Ecology and Sociobiology* **65**, 1779.

Boyce, M. S. (1979). Seasonality and patterns of natural selection for life histories. *The American Naturalist* **114**, 569–583.

Brodie, P. (1975). Cetacean energetics, an overview of intraspecific size variation. *Ecology* **56**, 152–161.

Brown, J. H., Gillooly, J. F., Allen, A. P., Savage, V. M. & West, G. B. (2004). Toward a metabolic theory of ecology. *Ecology* **85**, 1771–1789.

Brumm, H., Zollinger, S. A. & Slater, P. J. (2009). Developmental stress affects song learning but not song complexity and vocal amplitude in zebra finches. *Behavioral Ecology and Sociobiology* **63**, 1387–1395.

Buchanan, K. L., Grindstaff, J. L. & Pravosudov, V. V. (2013). Condition dependence, developmental plasticity, and cognition: implications for ecology and evolution. *Trends in Ecology & Evolution* **28**, 290–296.

Buckley, L. B., Hurlbert, A. H. & Jetz, W. (2012). Broad-scale ecological implications of ectothermy and endothermy in changing environments. *Global Ecology and Biogeography* **21**, 873–885.

*Burger, J. (1991). Effects of incubation temperature on behavior of hatchling pine snakes: implications for reptilian distribution. *Behavioral Ecology and Sociobiology* **28**, 297–303.

Burggren, W. W. & Mueller, C. A. (2015). Developmental critical windows and sensitive periods as three-dimensional constructs in time and space. *Physiological and Biochemical Zoology* **88**, 91–102.

Burnham, K. P. & Anderson, D. R. (2003). *Model Selection and Multimodel Inference: A Practical Information-Theoretic Approach*. Springer Science & Business Media, New York, U.S.A.

*Capelle, P. M., Semeniuk, C. A., Sopinka, N. M., Heath, J. W. & Love, O. P. (2016). Prenatal stress exposure generates higher early survival and smaller size without impacting developmental rate in a Pacific salmon. *Journal of Experimental Zoology Part A: Ecological Genetics and Physiology* **325**, 641–650.

*Carducci, J. P. & Jakob, E. M. (2000). Rearing environment affects behaviour of jumping spiders. *Animal Behaviour* **59**, 39–46.

Carvajal-Salamanca, J., Aránguiz-Acuña, A., Ramos-Jiliberto, R. & Zúñiga, L. R. (2008). Immediate and delayed life-history responses of *Daphnia ambigua* to conspecific cues. *Journal of Plankton Research* **30**, 1117–1122.

*Chaby, L. E., Cavigelli, S. A., White, A., Wang, K. & Braithwaite, V. A. (2013). Long-term changes in cognitive bias and coping response as a result of chronic unpredictable stress during adolescence. *Frontiers in Human Neuroscience* **7**, 107–116.

*Chaby, L. E., Sheriff, M. J., Hirrlinger, A. M. & Braithwaite, V. A. (2015). Does early stress prepare individuals for a stressful future? Stress during adolescence improves foraging under threat. *Animal Behaviour* **105**, 37–45.

*Chapman, L., Albert, J. & Galis, F. (2008). Developmental plasticity, genetic differentiation, and hypoxia-induced trade-offs in an African cichlid fish. *Open Evolution Journal* **2**, 75–88.

Chen, H.-H., Shen, Z.-Y. & Li, P. (1982). Adaptability of crop plants to high temperature stress 1. *Crop Science* **22**, 719–725.

Chin, E. H., Love, O. P., Verspoor, J. J., Williams, T. D., Rowley, K. & Burness, G. (2009). Juveniles exposed to embryonic corticosterone have enhanced flight performance. *Proceedings of the Royal Society of London B: Biological Sciences* **276**, 499–505.

*Chinathamby, K., Reina, R. D., Bailey, P. C. & Lees, B. K. (2006). Effects of salinity on the survival, growth and development of tadpoles of the brown tree frog, *Litoria ewingii*. *Australian Journal of Zoology* **54**, 97–105.

Chown, S., Gaston, K. & Robinson, D. (2004). Macrophysiology: large-scale patterns in physiological traits and their ecological implications. *Functional Ecology* **18**, 159–167.

*Christy, M. T. & Dickman, C. R. (2002). Effects of salinity on tadpoles of the green and golden bell frog (*Litoria aurea*). *Amphibia-Reptilia* **23**, 1–11.

Clarke, G. M. (1995). Relationships between developmental stability and fitness: application for conservation biology. *Conservation Biology* **9**, 18–24.

Clarke, G. M. (1998). The genetic basis of developmental stability. V. Inter-and intra-individual character variation. *Heredity* **80**, 562.

*Clarke, A. S., Wittwer, D., Abbott, D. & Schneider, M. (1994). Long-term effects of prenatal stress on HPA axis activity in juvenile rhesus monkeys. *Developmental Psychobiology* **27**, 257–269.

Clusella-Trullas, S., Blackburn, T. M. & Chown, S. L. (2011). Climatic predictors of temperature performance curve parameters in ectotherms imply complex responses to climate change. *The American Naturalist* **177**, 738–751.

Clutton-Brock, T. H., Albon, S. D. & Guinness, F. E. (1985). Parental investment and sex differences in juvenile mortality in birds and mammals. *Nature* **313**, 131.

Cohen, J. (1988). *Statistical Power Analysis for the Behavioral Sciences*, Second Edition (). Erlbaum Associates, Hillsdale.

*Connolly, J. D. & Cree, A. (2008). Risks of a late start to captive management for conservation: phenotypic differences between wild and captive individuals of a viviparous endangered skink (*Oligosoma otagense*). *Biological Conservation* **141**, 1283–1292.

*Coomber, P., Crews, D. & Gonzalez-Lima, F. (1997). Independent effects of incubation temperature and gonadal sex on the volume and metabolic capacity of brain nuclei in the leopard gecko (*Eublepharis macularius*), a lizard with temperature-dependent sex determination. *Journal of Comparative Neurology* **380**, 409–421.

Crespi, E. J. & Warne, R. W. (2013). Environmental conditions experienced during the tadpole stage alter post-metamorphic glucocorticoid response to stress in an amphibian. *Integrative and Comparative Biology* **53**, 989–1001.

Crespi, E. J., Williams, T. D., Jessop, T. S. & Delehanty, B. (2013). Life history and the ecology of stress: how do glucocorticoid hormones influence life-history variation in animals? *Functional Ecology* **27**, 93–106.

Crino, O. L. & Breuner, C. W. (2015). Developmental stress: evidence for positive phenotypic and fitness effects in birds. *Journal of Ornithology* **156**, 389–398.

*Crino, O., Driscoll, S. C., Ton, R. & Breuner, C. W. (2014*a*). Corticosterone exposure during development improves performance on a novel foraging task in zebra finches. *Animal Behaviour* **91**, 27–32.

Crino, O. L., Prather, C. T., Driscoll, S. C., Good, J. M. & Breuner, C. W. (2014*b*). Developmental stress increases reproductive success in male zebra finches. *Proceedings of the Royal Society of London B: Biological Sciences* **281**, 20141266.

Crino, O. L., Klaassen van Oorschot, B., Crandell, K. E., Breuner, C. W. & Tobalske, B. W. (2017). Flight performance in the altricial zebra finch: developmental effects and reproductive consequences. *Ecology and Evolution* **7**, 2316–2326.

*Criscuolo, F., Monaghan, P., Nasir, L. & Metcalfe, N. B. (2008). Early nutrition and phenotypic development: 'catch-up' growth leads to elevated metabolic rate in adulthood. *Proceedings of the Royal Society of London B: Biological Sciences* **275**, 1565–1570.

Denver, R. J. (1997). Environmental stress as a developmental cue: corticotropin-releasing hormone is a proximate mediator of adaptive phenotypic plasticity in amphibian metamorphosis. *Hormones and Behavior* **31**, 169–179.

Denver, R. J., Mirhadi, N. & Phillips, M. (1998). Adaptive plasticity in amphibian metamorphosis: response of *Scaphiopus Hammondiitadpoles* to habitat desiccation. *Ecology* **79**, 1859–1872.

*Dias, B. G., Ataya, R. S., Rushworth, D., Zhao, J. & Crews, D. (2007). Effect of incubation temperature and androgens on dopaminergic activity in the leopard gecko, *Eublepharis macularius*. *Developmental Neurobiology* **67**, 630–636.

Downes, S. J. & Shine, R. (1999). Do incubation-induced changes in a lizard's phenotype influence its vulnerability to predators? *Oecologia* **120**, 9–18.

*Duplouy, A., Woestmann, L., Gallego Zamorano, J. & Saastamoinen, M. (2017). Impact of male condition on his spermatophore and consequences for female reproductive performance in the Glanville fritillary butterfly. *Insect Science* **25**, 284–296.

Duval, S. & Tweedie, R. (2000). Trim and fill: a simple funnel-plot–based method of testing and adjusting for publication bias in meta-analysis. *Biometrics* **56**, 455–463.

Egger, M., Smith, G. D., Schneider, M. & Minder, C. (1997). Bias in meta-analysis detected by a simple, graphical test. *British Medical Journal* **315**, 629–634.

Ellis, B. J. & Boyce, W. T. (2008). Biological sensitivity to context. *Current Directions in Psychological Science* **17**, 183–187.

*Elphick, M. J. & Shine, R. (1998). Longterm effects of incubation temperatures on the morphology and locomotor performance of hatchling lizards (*Bassiana duperreyi*, *Scincidae*). *Biological Journal of the Linnean Society* **63**, 429–447.

English, S. & Uller, T. (2016). Does early-life diet affect longevity? A meta-analysis across experimental studies. *Biology Letters* **12**, 20160291.

Farrell, T. M., Neuert, M. A., Cui, A. & MacDougall-Shackleton, S. A. (2015). Developmental stress impairs a female songbird's behavioural and neural response to a sexually selected signal. *Animal Behaviour* **102**, 157–167.

Fawcett, T. W. & Frankenhuis, W. E. (2015). Adaptive explanations for sensitive windows in development. *Frontiers in Zoology* **12**, S3.

Feder, M. E. & Hofmann, G. E. (1999). Heat-shock proteins, molecular chaperones, and the stress response: evolutionary and ecological physiology. *Annual Review of Physiology* **61**, 243–282.

Francis, D., Diorio, J., Liu, D. & Meaney, M. J. (1999). Nongenomic transmission across generations of maternal behavior and stress responses in the rat. *Science* **286**, 1155–1158.

*Fraser, T., Hansen, T., Fleming, M. & Fjelldal, P. (2015). The prevalence of vertebral deformities is increased with higher egg incubation temperatures and triploidy in Atlantic salmon *Salmo salar L. Journal of Fish Diseases* **38**, 75–89.

Gaston, K. J. (2000). Global patterns in biodiversity. *Nature* **405**, 220–227.

Gelman, A. & Rubin, D. B. (1992). Inference from iterative simulation using multiple sequences. *Statistical Science* **7**, 457–472.

*Georgakopoulou, E., Sfakianakis, D., Kouttouki, S., Divanach, P., Kentouri, M. & Koumoundouros, G. (2007). The influence of temperature during early life on phenotypic expression at later ontogenetic stages in sea bass. *Journal of Fish Biology* **70**, 278–291.

Gibbs, D. M. (1984). Dissociation of oxytocin, vasopressin and corticotropin secretion during different types of stress. *Life Sciences* **35**, 487–491.

*Gil, D., Heim, C., Bulmer, E., Rocha, M., Puerta, M. & Naguib, M. (2004). Negative effects of early developmental stress on yolk testosterone levels in a passerine bird. *Journal of Experimental Biology* **207**, 2215–2220.

Gluckman, P. D. & Hanson, M. A. (2004). Living with the past: evolution, development, and patterns of disease. *Science* **305**, 1733–1736.

Gluckman, P. D., Hanson, M. A. & Spencer, H. G. (2005). Predictive adaptive responses and human evolution. *Trends in Ecology & Evolution* **20**, 527–533.

Gluckman, P. D., Hanson, M. A., Bateson, P., Beedle, A. S., Law, C. M., Bhutta, Z. A., Anokhin, K. V., Bougnères, P., Chandak, G. R. & Dasgupta, P. (2009). Towards a new developmental synthesis: adaptive developmental plasticity and human disease. *The Lancet* **373**, 1654–1657.

Gonzalez, P. N., Lotto, F. P. & Hallgrímsson, B. (2014). Canalization and developmental instability of the fetal skull in a mouse model of maternal nutritional stress. *American Journal of Physical Anthropology* **154**, 544–553.

Graham, J. H., Freeman, D. C. & Emlen, J. M. (1993). Developmental stability: a sensitive indicator of populations under stress. In *Environmental Toxicology and Risk Assessment* (ed. W. G. Landis, J. S. Hughes and M. A. Lewis), pp. 136–158. ASTM International, Philadelphia, U.S.A.

*Guibert, F., Lumineau, S., Kotrschal, K., Möstl, E., Richard-Yris, M.-A. & Houdelier, C. (2013). Trans-generational effects of prenatal stress in quail. *Proceedings of the Royal Society of London B: Biological Sciences* **280**, 20122368.

*Gutzke, W. & Crews, D. (1988). Embryonic temperature determines adult sexuality in a reptile. *Nature* **332**, 832–834.

*Guzman, C., Cabrera, R., Cardenas, M., Larrea, F., Nathanielsz, P. & Zambrano, E. (2006). Protein restriction during fetal and neonatal development in the rat alters reproductive function and accelerates reproductive ageing in female progeny. *The Journal of Physiology* **572**, 97–108.

Hadfield, J. (2015). MCMCglmm: MCMC generalised linear mixed models. *Royal Society Open Science* **3**, 160087.

*Hagman, M., Hayes, R. A., Capon, R. & Shine, R. (2009). Alarm cues experienced by cane toad tadpoles affect post-metamorphic morphology and chemical defences. *Functional Ecology* **23**, 126–132.

Hales, C. & Ozanne, S. (2003). The dangerous road of catch-up growth. *The Journal of Physiology* **547**, 5–10.

Hebets, E. A., Wesson, J. & Shamble, P. S. (2008). Diet influences mate choice selectivity in adult female wolf spiders. *Animal Behaviour* **76**, 355–363.

*Hector, K., Bishop, P. & Nakagawa, S. (2012). Consequences of compensatory growth in an amphibian. *Journal of Zoology* **286**, 93–101.

*Helle, H., Koskela, E. & Mappes, T. (2012). Life in varying environments: experimental evidence for delayed effects of juvenile environment on adult life history. *Journal of Animal Ecology* **81**, 573–582.

*Hellstedt, P. & Kallio, E. R. (2005). Survival and behaviour of captive-born weasels (*Mustela nivalis nivalis*) released in nature. *Journal of Zoology* **266**, 37–44.

Herbert, T. B. & Cohen, S. (1993). Stress and immunity in humans: a meta-analytic review. *Psychosomatic Medicine* **55**, 364–379.

Hercus, M. J., Loeschcke, V. & Rattan, S. I. (2003). Lifespan extension of *Drosophila melanogaster* through hormesis by repeated mild heat stress. *Biogerontology* **4**, 149–156.

Herman, J. P., Figueiredo, H., Mueller, N. K., Ulrich-Lai, Y., Ostrander, M. M., Choi, D. C. & Cullinan, W. E. (2003). Central mechanisms of stress integration: hierarchical circuitry controlling hypothalamo–pituitary–adrenocortical responsiveness. *Frontiers in Neuroendocrinology* **24**, 151–180.

Hoffmann, A. & Woods, R. (2001). Trait variability and stress: canalization, developmental stability and the need for a broad approach. *Ecology Letters* **4**, 97–101.

Hoffmann, A. A., Chown, S. L. & Clusella-Trullas, S. (2013). Upper thermal limits in terrestrial ectotherms: how constrained are they? *Functional Ecology* **27**, 934–949.

*Holveck, M.-J. & Riebel, K. (2010). Low-quality females prefer low-quality males when choosing a mate. *Proceedings of the Royal Society of London B: Biological Sciences* **277**, 153–160.

*Houslay, T. M., Hunt, J., Tinsley, M. C. & Bussiere, L. F. (2015). Sex differences in the effects of juvenile and adult diet on age-dependent reproductive effort. *Journal of Evolutionary Biology* **28**, 1067–1079.

Hoverman, J. T. & Relyea, R. A. (2007). How flexible is phenotypic plasticity? Developmental windows for trait induction and reversal. *Ecology* **88**, 693–705.

*Jespersen, L. & Toft, S. (2003). Compensatory growth following early nutritional stress in the wolf spider *Pardosa prativaga*. *Functional Ecology* **17**, 737–746.

Johansson, F., Lederer, B. & Lind, M. I. (2010). Trait performance correlations across life stages under environmental stress conditions in the common frog, *Rana temporaria*. *PLoS One* **5**, e11680.

Johnson, E. O., Kamilaris, T. C., Chrousos, G. P. & Gold, P. W. (1992). Mechanisms of stress: a dynamic overview of hormonal and behavioral homeostasis. *Neuroscience & Biobehavioral Reviews* **16**, 115–130.

*Jourdan, J., Baier, J., Riesch, R., Klimpel, S., Streit, B., Müller, R. & Plath, M. (2016). Adaptive growth reduction in response to fish kairomones allows mosquito larvae (*Culex pipiens)* to reduce predation risk. *Aquatic Sciences* **78**, 303.

*Kapoor, A. & Matthews, S. G. (2005). Short periods of prenatal stress affect growth, behaviour and hypothalamo–pituitary–adrenal axis activity in male guinea pig offspring. *The Journal of Physiology* **566**, 967–977.

Khaliq, I., Hof, C., Prinzinger, R., Böhning-Gaese, K. & Pfenninger, M. (2014). Global variation in thermal tolerances and vulnerability of endotherms to climate change. *Proceedings of the Royal Society of London B: Biological Sciences* **281**, 20141097.

*Kitaysky, A., Kitaiskaia, E., Piatt, J. & Wingfield, J. (2003). Benefits and costs of increased levels of corticosterone in seabird chicks. *Hormones and Behavior* **43**, 140–149.

Koolhaas, J. M., Bartolomucci, A., Buwalda, B. d., De Boer, S., Flügge, G., Korte, S., Meerlo, P., Murison, R., Olivier, B. & Palanza, P. (2011). Stress revisited: a critical evaluation of the stress concept. *Neuroscience & Biobehavioral Reviews* **35**, 1291–1301.

Kotrschal, A., Szidat, S. & Taborsky, B. (2014). Developmental plasticity of growth and digestive efficiency in dependence of early-life food availability. *Functional Ecology* **28**, 878–885.

Lande, R. (1980). Sexual dimorphism, sexual selection, and adaptation in polygenic characters. *Evolution* **34**, 292–305.

Laugen, A. T., Laurila, A. & Merilä, J. (2003). Latitudinal and temperature-dependent variation in embryonic development and growth in *Rana temporaria*. *Oecologia* **135**, 548–554.

Le Bourg, E., Valenti, P., Lucchetta, P. & Payre, F. (2001). Effects of mild heat shocks at young age on aging and longevity in *Drosophila melanogaster*. *Biogerontology* **2**, 155–164.

Lippmann, M., Bress, A., Nemeroff, C. B., Plotsky, P. M. & Monteggia, L. M. (2007). Long-term behavioural and molecular alterations associated with maternal separation in rats. *European Journal of Neuroscience* **25**, 3091–3098.

Liu, D., Diorio, J., Tannenbaum, B., Caldji, C., Francis, D., Freedman, A., Sharma, S., Pearson, D., Plotsky, P. M. & Meaney, M. J. (1997). Maternal care, hippocampal glucocorticoid receptors, and hypothalamic-pituitary-adrenal responses to stress. *Science* **277**, 1659–1662.

*Livingston, J. D., Kahn, A. T. & Jennions, M. D. (2014). Sex differences in compensatory and catch-up growth in the mosquitofish *Gambusia holbrooki*. *Evolutionary Ecology* **28**, 687–706.

*Loman, J. & Claesson, D. (2003). Plastic response to pond drying in tadpoles *Rana temporaria*: tests of cost models. *Evolutionary Ecology Research* **5**, 179–194.

Love, O. P., McGowan, P. O. & Sheriff, M. J. (2013). Maternal adversity and ecological stressors in natural populations: the role of stress axis programming in individuals, with implications for populations and communities. *Functional Ecology* **27**, 81–92.

*Lyons, D. E. & Roby, D. D. (2011). Validating growth and development of a seabird as an indicator of food availability: captive-reared Caspian Tern chicks fed ad libitum and restricted diets. *Journal of Field Ornithology* **82**, 88–100.

*MacDonald, I. F., Kempster, B., Zanette, L. & MacDougall-Shackleton, S. A. (2006). Early nutritional stress impairs development of a song-control brain region in both male and female juvenile song sparrows (*Melospiza melodia*) at the onset of song learning. *Proceedings of the Royal Society of London B: Biological Sciences* **273**, 2559–2564.

*Marasco, V., Spencer, K. A., Robinson, J., Herzyk, P. & Costantini, D. (2013). Developmental post-natal stress can alter the effects of pre-natal stress on the adult redox balance. *General and Comparative Endocrinology* **191**, 239–246.

*Marcó, M. V. P., Piña, C. I., Simoncini, M. & Alejandro, L. (2010). Effects of incubation and rearing temperatures on *Caiman latirostris* growth. *Zoological Studies* **49**, 367–373.

*Márquez-García, M., Correa-Solís, M. & Méndez, M. (2010). Life-history trait variation in tadpoles of the warty toad in response to pond drying. *Journal of Zoology* **281**, 105–111.
Martin, L. B. (2009). Stress and immunity in wild vertebrates: timing is everything. *General and Comparative Endocrinology* **163**, 70–76.
*McGhee, K. E., Pintor, L. M., Suhr, E. L. & Bell, A. M. (2012). Maternal exposure to predation risk decreases offspring antipredator behaviour and survival in threespined stickleback. *Functional Ecology* **26**, 932–940.
*Mestre, L. & Bonte, D. (2012). Food stress during juvenile and maternal development shapes natal and breeding dispersal in a spider. *Behavioral Ecology* **23**, 759–764.
Metcalfe, N. B. & Monaghan, P. (2001). Compensation for a bad start: grow now, pay later? *Trends in Ecology & Evolution* **16**, 254–260.
Michel, V., Peinnequin, A., Alonso, A., Buguet, A., Cespuglio, R. & Canini, F. (2007). Decreased heat tolerance is associated with hypothalamo–pituitary–adrenocortical axis impairment. *Neuroscience* **147**, 522–531.
Micheli-Campbell, M., Campbell, H., Cramp, R., Booth, D. & Franklin, C. (2011). Staying cool, keeping strong: incubation temperature affects performance in a freshwater turtle. *Journal of Zoology* **285**, 266–273.
*Milston, R. H., Fitzpatrick, M. S., Vella, A. T., Clements, S., Gundersen, D., Feist, G., Crippen, T. L., Leong, J. & Schreck, C. B. (2003). Short-term exposure of Chinook salmon (*Oncoryhnchus tshawytscha*) to o, p-DDE or DMSO during early life-history stages causes long-term humoral immunosuppression. *Environmental Health Perspectives* **111**, 1601.
Minois, N. (2000). Longevity and aging: beneficial effects of exposure to mild stress. *Biogerontology* **1**, 15–29.
Moczek, A. P., Sultan, S., Foster, S., Ledón-Rettig, C., Dworkin, I., Nijhout, H. F., Abouheif, E. & Pfennig, D. W. (2011). The role of developmental plasticity in evolutionary innovation. *Proceedings of the Royal Society of London B: Biological Sciences* **278**, 2705–2713.
*Moe, B., Brunvoll, S., Mork, D., Brobakk, T. E. & Bech, C. (2004). Developmental plasticity of physiology and morphology in diet-restricted European shag nestlings (*Phalacrocorax aristotelis*). *Journal of Experimental Biology* **207**, 4067–4076.
Møller, A. P. & Jennions, M. D. (2001). Testing and adjusting for publication bias. *Trends in Ecology & Evolution* **16**, 580–586.
Møller, A. P. & Swaddle, J. P. (1997). *Asymmetry, Developmental Stability and Evolution*. Oxford University Press, Oxford, U.K.
*Monaghan, P., Heidinger, B. J., D'Alba, L., Evans, N. P. & Spencer, K. A. (2012). For better or worse: reduced adult lifespan following early-life stress is transmitted to breeding partners. *Proceedings of the Royal Society of London B: Biological Sciences* **279**, 709–714.
*Moretz, J. A., Martins, E. P. & Robison, B. D. (2007). The effects of early and adult social environment on zebrafish (*Danio rerio*) behavior. *Environmental Biology of Fishes* **80**, 91–101.
Mousseau, T. A. & Fox, C. W. (1998). The adaptive significance of maternal effects. *Trends in Ecology & Evolution* **13**, 403–407.
*Müller, W. & Eens, M. (2009). Elevated yolk androgen levels and the expression of multiple sexually selected male characters. *Hormones and Behavior* **55**, 175–181.
*Naguib, M., Riebel, K., Marzal, A. & Gil, D. (2004). Nestling immunocompetence and testosterone covary with brood size in a songbird. *Proceedings of the Royal Society of London, Series B: Biological Sciences* **271**, 833–838.
Naguib, M., Nemitz, A. & Gil, D. (2006). Maternal developmental stress reduces reproductive success of female offspring in zebra finches. *Proceedings of the Royal Society of London B: Biological Sciences* **273**, 1901–1905.
Nakagawa, S. & Schielzeth, H. (2013). A general and simple method for obtaining R2 from generalized linear mixed-effects models. *Methods in Ecology and Evolution* **4**, 133–142.
Nakagawa, S., Noble, D. W., Senior, A. M. & Lagisz, M. (2017). Meta-evaluation of meta-analysis: ten appraisal questions for biologists. *BMC Biology* **15**, 18.
Natelson, B. H., Ottenweller, J. E., Cook, J. A., Pitman, D., McCarty, R. & Tapp, W. N. (1988). Effect of stressor intensity on habituation of the adrenocortical stress response. *Physiology & Behavior* **43**, 41–46.
Newman, R. A. (1989). Developmental plasticity of *Scaphiopus couchii* tadpoles in an unpredictable environment. *Ecology* **70**, 1775–1787.
Noble, D. W., Stenhouse, V. & Schwanz, L. E. (2018). Developmental temperatures and phenotypic plasticity in reptiles: a systematic review and meta-analysis. *Biological Reviews* **93**, 72–97.
Nowicki, S., Searcy, W. & Peters, S. (2002). Brain development, song learning and mate choice in birds: a review and experimental test of the" nutritional stress hypothesis". *Journal of Comparative Physiology A: Neuroethology, Sensory, Neural, and Behavioral Physiology* **188**, 1003–1014.
Obradović, J., Bush, N. R., Stamperdahl, J., Adler, N. E. & Boyce, W. T. (2010). Biological sensitivity to context: the interactive effects of stress reactivity and family adversity on socioemotional behavior and school readiness. *Child Development* **81**, 270–289.
O'Keeffe, M. K. & Baum, A. (1990). Conceptual and methodological issues in the study of chronic stress. *Stress and Health* **6**, 105–115.
*O'Mahony, S. M., Marchesi, J. R., Scully, P., Codling, C., Ceolho, A.-M., Quigley, E. M., Cryan, J. F. & Dinan, T. G. (2009). Early life stress alters behavior, immunity, and microbiota in rats: implications for irritable bowel syndrome and psychiatric illnesses. *Biological Psychiatry* **65**, 263–267.
Oomen, C. A., Soeters, H., Audureau, N., Vermunt, L., van Hasselt, F. N., Manders, E. M., Joëls, M., Lucassen, P. J. & Krugers, H. (2010). Severe early life stress hampers spatial learning and neurogenesis, but improves hippocampal synaptic plasticity and emotional learning under high-stress conditions in adulthood. *Journal of Neuroscience* **30**, 6635–6645.
Pacak, K., Palkovits, M., Yadid, G., Kvetnansky, R., Kopin, I. J. & Goldstein, D. S. (1998). Heterogeneous neurochemical responses to different stressors: a test of Selye's doctrine of nonspecificity. *American Journal of Physiology* **275**, R1247–R1255.
Pankhurst, N. W. & Munday, P. L. (2011). Effects of climate change on fish reproduction and early life history stages. *Marine and Freshwater Research* **62**, 1015–1026.
*Paris, J. J., Brunton, P. J., Russell, J. A. & Frye, C. A. (2011). Immune stress in late pregnant rats decreases length of gestation and fecundity, and alters later cognitive and affective behaviour of surviving pre-adolescent offspring. *Stress* **14**, 652–664.
*Partadiredja, G. & Bedi, K. (2010*a*). Undernutrition during either the pre-or immediate post-weaning period does not affect longevity in Quackenbush mice. *Nutritional Neuroscience* **13**, 33–42.
*Partadiredja, G. & Bedi, K. (2010*b*). Undernutrition during the gestation and suckling periods does not cause any loss of pyramidal neurons in the CA2–CA3 region of the rat hippocampus. *Nutritional Neuroscience* **13**, 102–108.
Pavlovska-Teglia, G., Stodulski, G., Svendsen, L., Dalton, K. & Hau, J. (1995). Effect of oral corticosterone administration on locomotor development of neonatal and juvenile rats. *Experimental Physiology* **80**, 469–476.
Piersma, T., Cadée, N. & Daan, S. (1995). Seasonality in basal metabolic rate and thermal conductance in a long-distance migrant shorebird, the knot (*Calidris canutus*). *Journal of Comparative Physiology B* **165**, 37–45.
*Piña, C. I., Larriera, A., Medina, M. & Webb, G. J. (2007). Effects of incubation temperature on the size of *Caiman latirostris* (Crocodylia: *Alligatoridae*) at hatching and after one year. *Journal of Herpetology* **41**, 205–210.
*Plesnar-Bielak, A., Woch, K., Małszycki, M., Alkhawlany, A., Hołysz, A., Assis Correia, J., Turk, N., Ugrin, M., Kramarz, P. & Prokop, Z. (2017). Larval and adult nutrition effects on reproductive traits in the red flour beetle. *Journal of Zoology* **302**, 79–87.
*Pravosudov, V. V. & Kitaysky, A. S. (2006). Effects of nutritional restrictions during post-hatching development on adrenocortical function in western scrub-jays (*Aphelocoma californica*). *General and Comparative Endocrinology* **145**, 25–31.
*Prijs, H. (1996). Effects of brood size manipulations on sexual attractiveness of offspring in the zebra finch. *Animal Behaviour* **51**, 699–708.
*Punzo, F. & Ludwig, L. (2002). Contact with maternal parent and siblings affects hunting behavior, learning, and central nervous system development in spiderlings of *Hogna carolinensis* (Araeneae: *Lycosidae*). *Animal Cognition* **5**, 63–70.
*Qualls, C. P. & Andrews, R. M. (1999). Cold climates and the evolution of viviparity in reptiles: cold incubation temperatures produce poor-quality offspring in the lizard, *Sceloporus virgatus*. *Biological Journal of the Linnean Society* **67**, 353–376.
Reich, P. B., Walters, M. B. & Ellsworth, D. S. (1997). From tropics to tundra: global convergence in plant functioning. *Proceedings of the National Academy of Sciences of the United States of America* **94**, 13730–13734.
Relyea, R. A. (2001). The lasting effects of adaptive plasticity: predator-induced tadpoles become long-legged frogs. *Ecology* **82**, 1947–1955.
*Richter-Boix, A., Llorente, G. A. & Montori, A. (2006). Effects of phenotypic plasticity on post-metamorphic traits during pre-metamorphic stages in the anuran *Pelodytes punctatus*. *Evolutionary Ecology Research* **8**, 309–320.
*Riedstra, B., Pfannkuche, K. & Groothuis, T. (2013). Increased exposure to yolk testosterone has feminizing effects in chickens, *Gallus gallus domesticus*. *Animal Behaviour* **85**, 701–708.
*Roche, D. P., McGhee, K. E. & Bell, A. M. (2012). Maternal predator-exposure has lifelong consequences for offspring learning in threespined sticklebacks. *Biology Letters* **8**, 932–935.
Romero, L. M. (2004). Physiological stress in ecology: lessons from biomedical research. *Trends in Ecology & Evolution* **19**, 249–255.
*Rosa, E. & Saastamoinen, M. (2017). Sex-dependent effects of larval food stress on adult performance under semi-natural conditions: only a matter of size? *Oecologia* **184**, 633–642.
Royle, N. J., Smiseth, P. T. & Kölliker, M. (2012). *The Evolution of Parental Care*. Oxford University Press, Oxford, U.K.
*Ruuskanen, S., Lehikoinen, E., Nikinmaa, M., Siitari, H., Waser, W. & Laaksonen, T. (2013). Long-lasting effects of yolk androgens on phenotype in the pied flycatcher (*Ficedula hypoleuca*). *Behavioral Ecology and Sociobiology* **67**, 361–372.

*Saastamoinen, M. & Rantala, M. J. (2013). Influence of developmental conditions on immune function and dispersal-related traits in the Glanville fritillary (*Melitaea cinxia*) butterfly. *PLoS One* **8**, e81289.

Saastamoinen, M., Van der Sterren, D., Vastenhout, N., Zwaan, B. J. & Brakefield, P. M. (2010). Predictive adaptive responses: condition-dependent impact of adult nutrition and flight in the tropical butterfly *Bicyclus anynana*. *The American Naturalist* **176**, 686–698.

*Saastamoinen, M., Hirai, N. & Van Nouhuys, S. (2013). Direct and trans-generational responses to food deprivation during development in the Glanville fritillary butterfly. *Oecologia* **171**, 93–104.

*Sakata, J. T., Coomber, P., Gonzalez-Lima, F. & Crews, D. (2000). Functional connectivity among limbic brain areas: differential effects of incubation temperature and gonadal sex in the leopard gecko, *Eublepharis macularius*. *Brain, Behavior and Evolution* **55**, 139–151.

*Salzberg, M., Kumar, G., Supit, L., Jones, N. C., Morris, M. J., Rees, S. & O'brien, T. J. (2007). Early postnatal stress confers enduring vulnerability to limbic epileptogenesis. *Epilepsia* **48**, 2079–2085.

Santarelli, S., Lesuis, S. L., Wang, X.-D., Wagner, K. V., Hartmann, J., Labermaier, C., Scharf, S. H., Müller, M. B., Holsboer, F. & Schmidt, M. V. (2014). Evidence supporting the match/mismatch hypothesis of psychiatric disorders. *European Neuropsychopharmacology* **24**, 907–918.

Schaefer, J. & Ryan, A. (2006). Developmental plasticity in the thermal tolerance of zebrafish *Danio rerio*. *Journal of Fish Biology* **69**, 722–734.

*Schneider, M. L. (1992). Prenatal stress exposure alters postnatal behavioral expression under conditions of novelty challenge in rhesus monkey infants. *Developmental Psychobiology* **25**, 529–540.

Schulte, P. M., Healy, T. M. & Fangue, N. A. (2011). Thermal performance curves, phenotypic plasticity, and the time scales of temperature exposure. *Integrative and Comparative Biology* **51**, 691–702.

*Searcy, W. A., Peters, S. & Nowicki, S. (2004). Effects of early nutrition on growth rate and adult size in song sparrows *Melospiza melodia*. *Journal of Avian Biology* **35**, 269–279.

Senior, A., Nakagawa, S., Raubenheimer, D., Simpson, S. & Noble, D. (2017). Dietary restriction increases variability in longevity. *Biology Letters* **13**, 20170057.

Shanks, N., Windle, R. J., Perks, P. A., Harbuz, M. S., Jessop, D. S., Ingram, C. D. & Lightman, S. L. (2000). Early-life exposure to endotoxin alters hypothalamic–pituitary–adrenal function and predisposition to inflammation. *Proceedings of the National Academy of Sciences of the United States of America* **97**, 5645–5650.

Shine, R. & Downes, S. J. (1999). Can pregnant lizards adjust their offspring phenotypes to environmental conditions? *Oecologia* **119**, 1–8.

Siegal, M. L. & Bergman, A. (2002). Waddington's canalization revisited: developmental stability and evolution. *Proceedings of the National Academy of Sciences of the United States of America* **99**, 10528–10532.

Simmons, R. A., Templeton, L. J. & Gertz, S. J. (2001). Intrauterine growth retardation leads to the development of type 2 diabetes in the rat. *Diabetes* **50**, 2279–2286.

*Sjöåsen, T. (1996). Survivorship of captive-bred and wild-caught reintroduced European otters *Lutra lutra* in Sweden. *Biological Conservation* **76**, 161–165.

*Sørensen, J., Kristensen, T. N., Kristensen, K. & Loeschcke, V. (2007). Sex specific effects of heat induced hormesis in Hsf-deficient *Drosophila melanogaster*. *Experimental Gerontology* **42**, 1123–1129.

Spencer, K. & Verhulst, S. (2007). Delayed behavioral effects of postnatal exposure to corticosterone in the zebra finch (*Taeniopygia guttata*). *Hormones and Behavior* **51**, 273–280.

Spencer, K., Buchanan, K., Goldsmith, A. & Catchpole, C. (2003). Song as an honest signal of developmental stress in the zebra finch (*Taeniopygia guttata*). *Hormones and Behavior* **44**, 132–139.

Spencer, K., Wimpenny, J., Buchanan, K., Lovell, P., Goldsmith, A. & Catchpole, C. (2005). Developmental stress affects the attractiveness of male song and female choice in the zebra finch (*Taeniopygia guttata*). *Behavioral Ecology and Sociobiology* **58**, 423–428.

Spiegelhalter, D. J., Best, N. G., Carlin, B. P. & Van Der Linde, A. (2002). Bayesian measures of model complexity and fit. *Journal of the Royal Statistical Society: Series B (Statistical Methodology)* **64**, 583–639.

*Squires, Z. E., Bailey, P. C., Reina, R. D. & Wong, B. B. (2010). Compensatory growth in tadpoles after transient salinity stress. *Marine and Freshwater Research* **61**, 219–222.

Stevens, G. C. (1989). The latitudinal gradient in geographical range: how so many species coexist in the tropics. *The American Naturalist* **133**, 240–256.

Stevens, D. J., Hansell, M. H., Freel, J. A. & Monaghan, P. (1999). Developmental trade–offs in caddis flies: increased investment in larval defence alters adult resource allocation. *Proceedings of the Royal Society of London B: Biological Sciences* **266**, 1049–1054.

Storm, J. J. & Lima, S. L. (2010). Mothers forewarn offspring about predators: a transgenerational maternal effect on behavior. *The American Naturalist* **175**, 382–390.

Sultan, S. E. (2007). Development in context: the timely emergence of eco-devo. *Trends in Ecology & Evolution* **22**, 575–582.

Sunday, J. M., Bates, A. E. & Dulvy, N. K. (2011). Global analysis of thermal tolerance and latitude in ectotherms. *Proceedings of the Royal Society of London B: Biological Sciences* **278**, 1823–1830.

*Székely, D., Denoël, M., Székely, P. & Cogălniceanu, D. (2017). Pond drying cues and their effects on growth and metamorphosis in a fast developing amphibian. *Journal of Zoology* **303**, 129–135.

*Taborsky, B. (2006). The influence of juvenile and adult environments on life-history trajectories. *Proceedings of the Royal Society of London B: Biological Sciences* **273**, 741–750.

*Tobler, M., Hasselquist, D., Smith, H. G. & Sandell, M. I. (2010). Short-and long-term consequences of prenatal testosterone for immune function: an experimental study in the zebra finch. *Behavioral Ecology and Sociobiology* **64**, 717–727.

Trivers, R. L. & Willard, D. E. (1973). Natural selection of parental ability to vary the sex ratio of offspring. *Science* **179**, 90–92.

*Tu, M. P. & Tatar, M. (2003). Juvenile diet restriction and the aging and reproduction of adult *Drosophila melanogaster*. *Aging Cell* **2**, 327–333.

*van Uitregt, V. O., Hurst, T. P. & Wilson, R. S. (2012). Reduced size and starvation resistance in adult mosquitoes, *Aedes notoscriptus*, exposed to predation cues as larvae. *Journal of Animal Ecology* **81**, 108–115.

Uller, T. (2008). Developmental plasticity and the evolution of parental effects. *Trends in Ecology & Evolution* **23**, 432–438.

Vallee, M., Mayo, W., Dellu, F., Le Moal, M., Simon, H. & Maccari, S. (1997). Prenatal stress induces high anxiety and postnatal handling induces low anxiety in adult offspring: correlation with stress-induced corticosterone secretion. *Journal of Neuroscience* **17**, 2626–2636.

*Valtonen, T. M., Kangassalo, K., Pölkki, M. & Rantala, M. J. (2012). Transgenerational effects of parental larval diet on offspring development time, adult body size and pathogen resistance in *Drosophila melanogaster*. *PLoS One* **7**, e31611.

Van Buskirk, J. & Steiner, U. K. (2009). The fitness costs of developmental canalization and plasticity. *Journal of Evolutionary Biology* **22**, 852–860.

Van der Have, T. & De Jong, G. (1996). Adult size in ectotherms: temperature effects on growth and differentiation. *Journal of Theoretical Biology* **183**, 329–340.

Varlinskaya, E. I. & Spear, L. P. (2008). Social interactions in adolescent and adult Sprague–Dawley rats: impact of social deprivation and test context familiarity. *Behavioural Brain Research* **188**, 398–405.

*Veenema, A. H., Blume, A., Niederle, D., Buwalda, B. & Neumann, I. D. (2006). Effects of early life stress on adult male aggression and hypothalamic vasopressin and serotonin. *European Journal of Neuroscience* **24**, 1711–1720.

*Veenema, A. H., Bredewold, R. & Neumann, I. D. (2007). Opposite effects of maternal separation on intermale and maternal aggression in C57BL/6 mice: link to hypothalamic vasopressin and oxytocin immunoreactivity. *Psychoneuroendocrinology* **32**, 437–450.

*Vega-Trejo, R., Jennions, M. D. & Head, M. L. (2016). Are sexually selected traits affected by a poor environment early in life? *BMC Evolutionary Biology* **16**, 263.

*Verhulst, S., Holveck, M.-J. & Riebel, K. (2006). Long-term effects of manipulated natal brood size on metabolic rate in zebra finches. *Biology Letters* **2**, 478–480.

Viechtbauer, W. (2010). Conducting meta-analyses in R with the metafor package. *Journal of Statistical Software* **36**, 1–48.

Waddington, C. H. (1942). Canalization of development and the inheritance of acquired characters. *Nature* **150**, 563–565.

Walls, S. C., Barichivich, W. J. & Brown, M. E. (2013). Drought, deluge and declines: the impact of precipitation extremes on amphibians in a changing climate. *Biology* **2**, 399–418.

Walters, D. & Heil, M. (2007). Costs and trade-offs associated with induced resistance. *Physiological and Molecular Plant Pathology* **71**, 3–17.

*Wapstra, E. (2000). Maternal basking opportunity affects juvenile phenotype in a viviparous lizard. *Functional Ecology* **14**, 345–352.

*Warner, D. A. & Andrews, R. M. (2002). Laboratory and field experiments identify sources of variation in phenotypes and survival of hatchling lizards. *Biological Journal of the Linnean Society* **76**, 105–124.

West-Eberhard, M. J. (2003). *Developmental Plasticity and Evolution*. Oxford University Press, Oxford, U.K.

*Whiteside, M. A., Sage, R. & Madden, J. R. (2016). Multiple behavioural, morphological and cognitive developmental changes arise from a single alteration to early life spatial environment, resulting in fitness consequences for released pheasants. *Royal Society Open Science* **3**, 160008.

Wiegant, F., Surinova, S., Ytsma, E., Langelaar-Makkinje, M., Wikman, G. & Post, J. (2009). Plant adaptogens increase lifespan and stress resistance in *C. elegans*. *Biogerontology* **10**, 27–42.

Winemiller, K. O. & Jepsen, D. B. (1998). Effects of seasonality and fish movement on tropical river food webs. *Journal of Fish Biology* **53**, 267–296.

*Wisely, S., Ososky, J. & Buskirk, S. (2002). Morphological changes to black-footed ferrets (*Mustela nigripes*) resulting from captivity. *Canadian Journal of Zoology* **80**, 1562–1568.

*Xiao-long, T., Feng, Y., Ming, M., Ningbo, W., Jianzheng, H. & Qiang, C. (2012). Effects of thermal and hydric conditions on egg incubation and hatchling phenotypes in two *Phrynocephalus* lizards. *Asian Herpetological Research* **3**, 184–191.

Xu, S., Li, J., Zhang, X., Wei, H. & Cui, L. (2006). Effects of heat acclimation pretreatment on changes of membrane lipid peroxidation, antioxidant metabolites, and ultrastructure of chloroplasts in two cool-season turfgrass species under heat stress. *Environmental and Experimental Botany* **56**, 274–285.

*Zimmer, C., Boogert, N. J. & Spencer, K. A. (2013). Developmental programming: cumulative effects of increased pre-hatching corticosterone levels and post-hatching unpredictable food availability on physiology and behaviour in adulthood. *Hormones and Behavior* **64**, 494–500.

## VIII. SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

**Table S1.** Legend for final analysis data.

**Table S2.** Final analysis data.

**Table S3.** Legend for meta-data.

**Table S4.** Meta-data.

(*Received 4 April 2018; revised 8 December 2018; accepted 13 December 2018; published online 4 January 2019*)