BIOLOGICAL REVIEWS
Cambridge Philosophical Society
Biol. Rev. (2016), **91**, pp. 1134–1148.
doi: 10.1111/brv.12214

The positive correlation between maternal size and offspring size: fitting pieces of a life-history puzzle

Njal Rollinson* and Locke Rowe

Department of Ecology and Evolutionary Biology, University of Toronto, 25 Willcocks St., Toronto, Ontario M5S 3B2, Canada

ABSTRACT

The evolution of investment per offspring (I) is often viewed through the lens of the classic theory, in which variation among individuals in a population is not expected. A substantial departure from this prediction arises in the form of correlations between maternal body size and I, which are observed within populations in virtually all taxonomic groups. Based on the generality of this observation, we suggest it is caused by a common underlying mechanism. We pursue a unifying explanation for this pattern by reviewing all theoretical models that attempt to explain it. We assess the generality of the mechanism upon which each model is based, and the extent to which data support its predictions. Two classes of adaptive models are identified: models that assume that the correlation arises from maternal influences on the relationship between I and offspring fitness [$w(I)$], and those that assume that maternal size influences the relationship between I and maternal fitness [$W(I)$]. The weight of evidence suggests that maternal influences on $w(I)$ are probably not very general, and even for taxa where maternal influences on $w(I)$ are likely, experiments fail to support model predictions. Models that assume that $W(I)$ varies with maternal size appear to offer more generality, but the current challenge is to identify a specific and general mechanism upon which $W(I)$ varies predictably with maternal size. Recent theory suggests the exciting possibility that a yet unknown mechanism modifies the offspring size–number trade-off function in a manner that is predictable with respect to maternal size, such that $W(I)$ varies with size. We identify two promising avenues of inquiry. First, the trade-off might be modified by energetic costs that are associated with the initiation of reproduction ('overhead costs') and that scale with I, and future work could investigate what specific overhead costs are generally associated with reproduction and whether these costs scale with I. Second, the trade-off might be modified by virtue of condition-dependent offspring provisioning coupled with metabolic factors, and future work could investigate the proximate cause of, and generality of, condition-dependent offspring provisioning. Finally, drawing on the existing literature, we suggest that maternal size *per se* is not causatively related to variation in I, and the mechanism involved in the correlation is instead linked to maternal nutritional status or maternal condition, which is usually correlated with maternal size. Using manipulative experiments to elucidate why females with high nutritional status typically produce large offspring might help explain what specific mechanism underlies the maternal-size correlation.

Key words: body size, life-history theory, investment per offspring, egg size, propagule size, seed size, fecundity, condition-dependence, parental care, parent-offspring conflict.

CONTENTS

*Address for correspondence (Tel: 416-946-7229; E-mail: njal.rollinson@gmail.com).

I. INTRODUCTION

Reproductive allocation is an important determinant of fitness, but parents must also optimize the trade-off between fecundity (N) and investment per offspring (I). Most classic theories of offspring-size evolution predict the existence of a single optimal offspring size (I^*), which is a level of I that maximizes parental reproductive success in a given environment (Smith & Fretwell, 1974; Brockelman, 1975; McGinley, Temme & Geber, 1987; Lalonde, 1991; Hendry, Day & Cooper, 2001). This prediction is a direct consequence of three assumptions that underlie these models: first, there is a trade-off between I and N; second, the function relating I to offspring fitness (w) is concave-downward; and third, all parents in a given environment share these two functions (Smith & Fretwell, 1974). Parental reproductive success must therefore be stabilizing with respect to I (Fig. 1), resulting in the prediction of a single optimum for all parents (see glossary in Table 1).

Despite this simple prediction, I is generally variable within populations, which suggests that I is not optimized in the wild, at least not in the classic sense (Orzack & Sober, 1994; Bernardo, 1996). Variation itself may not be surprising, for a number of reasons. For instance, selection can fluctuate temporally (Siepielski, DiBattista & Carlson, 2009), or act on suites of correlated traits, such that optimization of any univariate trait among a set of correlated traits is generally not expected (Orzack & Sober, 1994; Abrams, 2001; Gilchrist & Kingsolver, 2001; Brooks *et al.*, 2005). What has long puzzled evolutionary ecologists, however, is that variation in I is not random, but is often patterned, where I is an increasing function of maternal size. We call this enigmatic association 'the maternal-size correlation'.

The maternal-size correlation is not a minor phenomenon exhibited by just a few species, it is pervasive in the wild and transcends modes of reproduction. For instance, Lim, Senior & Nakagawa (2014) review the strength of the maternal-size correlation (Pearson's r) for 231 animal species. They demonstrate that I typically increases with maternal size, and that the strength of the relationship between I and maternal size is remarkably consistent across taxa and environments, with a mean correlation coefficient of $r \approx 0.40$. Similarly, Roff (2002, p. 271) compiled data from just over 100 phenotypic regressions and found that a significant positive relationship between I and maternal size was observed in over 50% of cases; a negative relationship was observed in less than 3% of cases. Of the 79 insect species reviewed by Fox & Czesak (2000), at least 53% exhibited this positive relationship. Each of the five large-mammal species reviewed by Réale & Festa-Bianchet (2000) exhibited a positive correlation between offspring birth mass and maternal size. In dung beetles (*Onthophagus* sp.), initial egg size does *not* correlate with female size (Kishi, 2014), but interestingly, large females assemble relatively large amalgams of dung into which they deposit a single egg, resulting in a positive correlation between maternal size and size of subsequent juveniles (Hunt & Simmons, 2000).

Identifying the ultimate reason for positive genetic (Ebert, 1993; Su, Liljedahl & Gall, 1997; Czesak & Fox, 2003) and phenotypic (Fox & Czesak, 2000; Roff, 2002) associations between these traits is one of the more challenging problems facing life-history theory. Indeed, the perplexing correlation between maternal size and I has stimulated a great deal of theory, with a range of explanations being proposed (Table 2). It is possible that several of these disparate explanations are correct, and that the maternal-size correlation arises in different taxa for a number of independent reasons (Roff, 2002, p. 269). However, the prevalence of the pattern across such a range of taxa and life histories suggests that there may be a single explanation. In this review, we draw on classic life-history principles and the weight of empirical evidence to argue that a single unifying explanation may exist for the maternal-size correlation.

While many models currently exist, all models fall broadly within three categories (Table 2). Here, we attempt to narrow the scope of possible explanations by undertaking a systematic and critical review of each set of models and the related data. We then discuss which type of model offers the most promising general explanation for the maternal-size correlation, and we outline avenues of further inquiry. Ultimately, we aim to promote empirical and theoretical development of this problem, as we hope the next few years will see a resolution to this life-history puzzle.

Before we begin our review, we must first emphasize that several traits are confounded in correlations between I and maternal size. For example, maternal size is positively associated with maternal age in some species, especially those that grow indeterminately (Congdon *et al.*, 2013), and larger females also tend to be those in better condition (general health, nutritional status). Similarly, reproductive allocation (RA), which we define as the sum of energy allocated to a given reproductive event (e.g. total energy used to develop a clutch or brood of offspring), increases with maternal size in most groups (Visman *et al.*, 1996; Downhower & Charnov, 1998; Roff, 2002; Hendriks & Mulder, 2008). Based on the empirical data, the trait that affects I and results in the maternal-size correlation is rarely clear, and we discuss whether it is maternal size or not towards the end of this

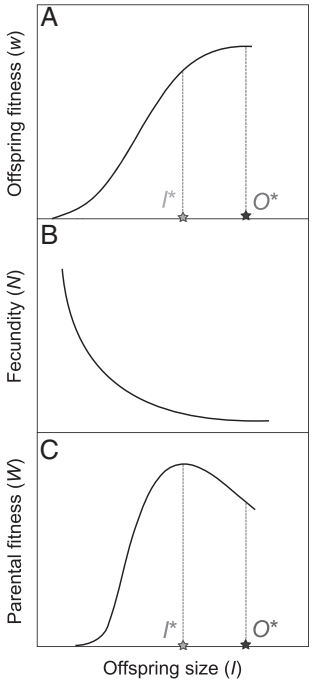

Fig. 1. Classic optimality theory. (A) The relationship between investment per offspring, I, and offspring fitness, w, is generally concave-downward. (B) Parents must trade off fecundity, N, against I. (C) Stabilizing selection on I exists at the parental level, which is the inevitable result of a concave-downward fitness function for I and a trade-off between N and I. Note that the level of I that maximizes the fitness of offspring, O^*, is greater than the level that maximizes parental fitness, I^*, reflecting parent–offspring conflict over I.

review. In our general discussion, we draw a distinction among age, maternal size, and *RA* when it is necessary to do so, but most theoretical models already draw a clear distinction among these effects on the predicted evolution of I (e.g. Hendry *et al.*, 2001; Kindsvater, Bonsall & Alonzo, 2011). When no distinction is made in our discussion, it should be assumed that we are discussing the correlation between I and maternal size in general.

II. MATERNAL SIZE AFFECTS THE RELATIONSHIP BETWEEN *I* AND OFFSPRING FITNESS

Classic offspring-size optimization is based in part on two functions: the investment-per-offspring–offspring fitness function, $w(I)$, and the offspring size–number trade-off function, $N(I)$ (Fig. 1). Smith & Fretwell's (1974) classic theory cannot predict among-individual variation in I^*, as it assumes that all females in a given environment share these two functions. However, if either of these functions differs among females, then different females will have different values of I^* (Pianka, 1976). A maternal-size correlation might arise, therefore, if one or both of these functions varies predictably with maternal size.

The first group of models we examine assume that maternal size affects the quality of the offspring environment, such that $w(I)$ varies predictably with maternal size. This situation might arise, for example, when offspring are produced in batches. In the absence of immediate dispersal, siblings may interact extensively and can collectively affect the quality of the environment they occupy. The four models reviewed in this section (Parker & Begon, 1986; McGinley, 1989; Hendry *et al.*, 2001; Kindsvater *et al.*, 2010) adopt this approach and assume that sibling interactions result in a non-linear relationship between N and parental fitness (W), which results in a positive correlation between I^* and N (Fig. 2A, B). This general idea is conceptually attractive in the context of the maternal-size correlation, primarily because the likelihood of sibling interactions increases with N, and maternal size often has a positive influence on N (Lim *et al.*, 2014), such that a correlation between maternal size and I^* is easy to envision. Below, we review the evidence for two types of sibling interactions that can predict a positive correlation

Table 1. Glossary of terms used in the present review

Term		Definition
I	Investment per offspring	The amount of energy invested in a given propagule
w	Offspring fitness	Offspring fitness
$w(I)$	The investment per offspring – offspring fitness function	How offspring fitness changes as a function of investment per offspring
I^*	Optimal investment per offspring	The level of investment per offspring that maximizes parental fitness
N	Fecundity	The number of propagules produced in a given reproductive event
$N(I)$	The offspring size–number trade-off function	How fecundity changes as a function of investment per offspring
RA	Reproductive allocation	The total amount of energy invested into reproductive activities in a given reproductive event
$RA = NI$	Smith & Fretwell's proportionality law	The classic assumption that reproductive allocation is the product of I and N; this assumption results in a particular form of $N(I)$
W	Parental fitness	Parental fitness
$W(I)$	The investment per offspring–parental fitness function	How parental fitness changes as a function of investment per offspring

Table 2. Summary of models that can predict a positive relationship between investment per offspring and maternal size, categorized by the school of thought to which each model appeals

Model	School[a]	Mechanism
Congdon *et al.* (1983)[b]	*Constraint*	Pelvic aperture limitation
Parker & Begon (1986)	$w(I)$	Sib interactions (negative density dependence)
McGinley (1989)	$w(I)$	Sib interactions (positive density dependence)
Venable (1992)	$w(I)$	Sib interactions (general density dependence)
Hendry *et al.* (2001)[c]	$w(I)$	Sib interactions and maternal nest-site choice
Sakai & Harada (2001)	$W(I)$	Metabolic costs and provisioning efficiency
Kindsvater *et al.* (2010)	$w(I)$	Female age, state, and negative sib interactions
Kindsvater *et al.* (2011)	$W(I)$	Survival cost to reproduction
Jørgensen *et al.* (2011)	$W(I)$	Survival cost to reproduction
Kindsvater & Otto (2014)	$W(I)$	Survival cost to reproduction
Filin (2015)	$W(I)$	Overhead costs and survival cost to reproduction

[a] *Constraint* refers to a 'non-adaptive' explanation; $w(I)$ refers to models that assume maternal body size influences the relationship between investment per offspring (I) and offspring fitness (w); $W(I)$ refers to how parental fitness (W) changes as a function of I. Some models fall into both adaptive schools [$w(I)$ and $W(I)$], and we attempted to categorize these models according to the school to which the model contributes a novel advance.
[b] This verbal model is the same as Congdon & Gibbons (1987).
[c] This model is the same model as Hendry & Day (2003).

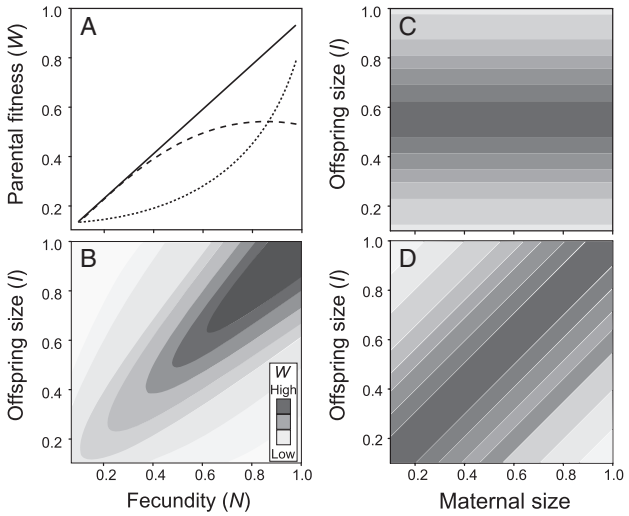

Fig. 2. (A) Sibling-interaction models assume that parental fecundity (N) affects the relationship between offspring size and fitness, which results in variation in optimal offspring size (I^*) at different levels of N. The type of sibling interaction (positive or negative) can be understood by visualizing how parental fitness (W) scales with N (concave-upward or concave-downward), while holding I fixed. A linear relationship is expected if no sibling interactions occur and I^* is the same at all levels of N. (B) Sibling interactions might generate a maternal-size correlation, although the causative agent in the correlation is not maternal size, it is N. Parental fitness increases with N, especially along a ridge of high fitness that corresponds with I^*. (C) In the classic model of offspring-size optimization, I^* does not vary with maternal size, as all individuals share the same fitness functions and trade-off functions in a given environment. Parental fitness does not vary with maternal size, holding N constant. (D) If the relationship between I and W varies with maternal size, then I^* will vary with maternal size. This is reflected by a ridge of high fitness running across the diagonal of the panel, while holding N constant.

between maternal size and I^*: positive and negative sibling interactions. We then assess existing evidence for sibling interactions as a cause of the maternal-size correlation.

One useful approach to envisioning the fitness consequences of sibling interactions is to consider how W changes as a function of N, while holding I constant. Negative sibling interactions reflect a concave-downward relationship between W and N (Fig. 2A), which occurs when competition among siblings for food or other resources depresses mean fitness of all individuals. Parker & Begon (1986) were the first to propose the idea of negative sibling interactions, and they consider a model where females must forage to obtain energy reserves and then find a site to reproduce. The model assumes that larger females can obtain more energy during foraging, which leads to a relatively large RA for larger females, and this RA must then be divided between I and N. When sibling interactions are negative, and assuming that larger offspring have greater fitness (e.g. Stanton, 1984; Hutchings, 1991), then females with greater RA are predicted to produce larger offspring to offset the decrease in environmental quality associated with increased N. Parker & Begon (1986) also point out that the costs of negative sibling interactions eventually become overwhelming, and that the production of multiple batches of offspring eventually becomes optimal. Although the precise nature of these switch-points depends on assumptions about foraging costs and the strength of sibling interactions, a general conclusion of the model is that females with greater RA should produce more batches of offspring than smaller females.

The model of Parker & Begon (1986) was the first explicitly to consider effects of the maternal phenotype on the evolution of I, and its rigorous and extensive treatment of the subject has stimulated a great deal of subsequent theory (Begon & Parker, 1986; Venable, 1992; Hendry *et al.*, 2001; Kindsvater *et al.*, 2010). For instance, Kindsvater *et al.* (2010) adopt a dynamical modelling approach to investigate how I^* varies

with maternal resource status, background adult mortality, negative sibling interactions, and a maximum female lifespan. Their results are very similar to those of Parker & Begon (1986). Specifically, older and larger females are predicted to have relatively more resources for reproduction, but because females are assumed to have a fixed, maximum lifespan, older (larger) females have relatively less time to accrue fitness. Older females may therefore maximize reproductive success in late life by increasing N and I simultaneously, a result that is attributable primarily to the confluence of a maximum lifespan, a lifetime of accumulated energy reserves, and negative sibling interactions.

Negative sibling interactions are fairly intuitive, but they are not the only type of sibling interaction that can affect $w(I)$. In fact, positive sibling interactions are also possible, and they occur when an increase in N has a positive average effect on the fitness of siblings, and hence a concave-upward relationship between W and N (Fig. 2A). This idea was first proposed by McGinley (1989) to help explain the maternal-size correlation for species that produce offspring in discrete batches. McGinley's (1989) model is particularly relevant to species that experience high depredation of offspring during a short period of time, such as in some turtles, where hatchlings emerge synchronously from a nest, then experience high rates of depredation during a short migration to water. In this model, W is initially determined by the incidence of depredation per batch, but following this initial stage, W is determined solely by the product of I and the number of surviving offspring. Assuming that a maximum rate of depredation exists during the initial stage, parents with lower *RA* may increase W by increasing N (and hence, decreasing I), as this maximizes the number of offspring that survive through the initial stage of depredation, even though w through the subsequent stage is relatively low. One result of this model is that I^* can be relatively low only when *RA* is low, which would ultimately generate a positive association between I (and I^*) and *RA* across parents in a population. Given that *RA* is often correlated with maternal body size, this model can predict maternal-size correlations.

Hendry *et al.* (2001) provide an interesting extension of sibling interaction models by introducing the additional possibility that maternal body size *per se* also affects offspring habitat quality *via* maternal nest-site choice. Given that sibling interactions and maternal size *per se* can have differing effects on $w(I)$, a positive correlation between I^* and maternal size will occur only when the net effect of maternal size on the quality of the offspring environment is negative (Hendry & Day, 2003). For instance, if larger females are more fecund and sibling interactions are negative, then a positive relationship between maternal size and I^* can arise only if the effect of maternal size *per se* improves the selective environment (e.g. by virtue of nest-site choice or the timing of reproduction) by an amount that does not exceed the negative effect of sibling interactions. The model provides a general framework in which to consider possible evolutionary outcomes for different fitness relationships among I and maternal traits.

The models of Parker & Begon (1986) and McGinley (1989) are among the most influential for the evolution of I (Venable, 1992; Hendry *et al.*, 2001; Hendry & Day, 2003; Kindsvater *et al.*, 2010), and there are specific cases in which the general tenets of these models are likely to apply. For instance, the model of Hendry *et al.* (2001) might be especially applicable to the plant kingdom, given that plant size and height can affect the strength of sibling interactions by affecting seed dispersal (Thomson *et al.*, 2011), and where selection operating through size-specific seed predation may act in the opposite direction from selection on post-germination viability (e.g. Gómez, 2004).

Some authors, however, have suggested that size-specific maternal influences on $w(I)$ can provide a general explanation for the maternal-size correlation (Einum & Fleming, 2002; Einum, Kinnison & Hendry, 2004; Rollinson & Brooks, 2008*a*; Marshall *et al.*, 2010). Yet there is very little evidence that maternal size has a predictable influence on $w(I)$ in any specific group, and there are further grounds upon which to question the generality of these models. For example, Acolas, Roussel & Baglinière (2008) provide an excellent illustration of how sibling interactions can fail to explain maternal-size correlations fully. These authors studied brown trout (*Salmo trutta*), a species in which negative density-dependent sibling interactions have long been suspected to cause a positive association between I^* and maternal size (Hendry *et al.*, 2001; Einum, Hendry & Fleming, 2002; Hendry & Day, 2003). Their study population is comprised of both sea-run (migratory) adults and lake-resident (non-migratory) adults; all adults interbreed and reproduce in the same spawning area at the same time, and juveniles of both forms share identical environments for at least 1 year of life. Despite much higher N produced by the migratory morph, the relationship between maternal size and I is relaxed in this morph (Fig. 3A), even though both morphs share a spawning environment and are not genetically differentiated. While this study is observational, no current model of sibling interactions can easily explain the marked difference between these morphs in the strength of the maternal-size correlation, especially given that N is far greater in the migratory morph.

More broadly, explicit tests of sibling interaction models have produced mixed results. While comparative data do suggest that sibling interactions might be associated with an increase in egg-size variation within populations of fish (Einum & Fleming, 2002; see also Schrader & Travis, 2012) and marine invertebrates (Marshall & Keough, 2007), the comparative nature of these studies limits the extent to which one can attribute causation to sibling interactions *per se*. In experimental studies, it is clear that increased offspring density can decrease w in some cases (Wall & Begon, 1986; Marshall, Cook & Emlet, 2006), and it is clear that relatively large offspring tend to fare better under competition (Stanton, 1984; Hutchings, 1991; Bashey, 2008; but see McIntyre & Gooding, 2000). But despite these observations, direct experimental manipulation has yet to provide clear support for any sibling interaction hypothesis (Einum & Fleming, 1999;

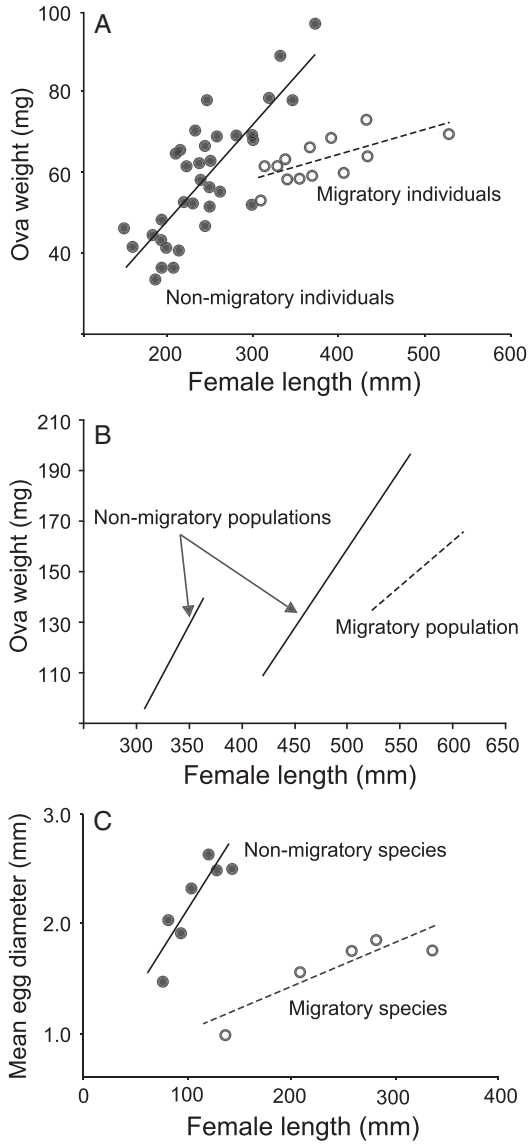

Fig. 3. Recurrent patterns of covariance in maternal size and investment per offspring (I) at multiple ecological scales. The slope of the relationship between I and maternal size is shallower in fishes that must migrate from the ocean into freshwater to spawn. This pattern persists in different species and at different ecological scales. (A) Within a single interbreeding population of brown trout (*Salmo trutta*). Each data point is a unique individual (redrawn from Acolas *et al.*, 2008). (B) Three distinct populations of masu salmon [*Oncorhynchus masou*; regression parameters from Table 2 in Tamate & Maekawa (2000)]. (C) Across 12 species of galaxiids (*Galaxias* spp.), each data point is a unique species [redrawn from Closs *et al.* (2013)].

Lalonde, 2005; Takahashi, Makino & Sakai, 2005; Plaistow, Lapsley & Benton, 2006; Plaistow *et al.*, 2007; Rollinson & Hutchings, 2010; Eberhart & Tielbörger, 2012), or hypotheses invoking maternal influences on $w(I)$ that are independent of sibling interactions (Rollinson & Hutchings, 2011; Louhi *et al.*, 2015). Furthermore, maternal-size correlations are often observed in species that spread propagules (seeds, eggs, etc.) across space and time (e.g. Atlantic cod *Gadus morhua*: Kjesbu *et al.*, 1996; Marteinsdóttir & Steinarsson, 1998; common barnacle *Balanus balanoides*: Barnes & Barnes, 1965; striped bass *Morone saxatilis*: Zastrow, Houde & Saunders, 1989), thereby removing potential for sibling interactions *per se*. Therefore, even if sibling interactions or other maternal influences on $w(I)$ can help explain the maternal-size correlation in some groups, this explanation is probably not general. In sum, models predicting that the maternal-size correlation arises from size-specific maternal influences on $w(I)$ appear to lack generality and empirical support. So while these models might help explain the evolution of I in some populations or groups, it is not clear how these models help explain the maternal-size correlation more broadly.

III. THE FITNESS BENEFITS OF *I* VARY WITH MATERNAL SIZE

Recently, there has been a burst of models that explore how mothers of different sizes can accrue different fitness benefits from a given level of I (Table 2). The concepts underlying this line of inquiry involve a substantial departure from those underlying sibling-interaction models: whereas sibling models assume maternal influences on $w(I)$, these more recent models assume that intrinsic qualities of a female vary with her size, and that these intrinsic qualities affect the fitness benefits she receives at a given level of I, such that maternal size influences $W(I)$ (Fig. 2C, D). Below, we explore what intrinsic qualities of a mother might vary predictably with her size, and how these qualities result in size-related variation in $W(I)$.

(1) Age- and stage-specific adult survival

Here we examine a series of related models that explain the maternal-size correlation by linking I with parental survival. While parental survival ultimately drives the maternal-size correlation in these models, the underlying logic is broadly similar to classic theories that describe correlated evolution of offspring size and parental care (Shine, 1978; Sargent, Taylor & Gross, 1987; Nussbaum & Schultz, 1989).

The evolution of parental care often coincides with the evolution of larger egg size (fishes: Gross & Sargent, 1985; anurans: Summers, Sea McKeon & Heying, 2006; but not in insects: Gilbert & Manica, 2010). The 'safe harbor' models of Shine (1978); Sargent *et al.* (1987), and Nussbaum & Schultz (1989) provide an explanation for this pattern. Safe harbor models partition offspring ontogeny into the embryo stage and the juvenile stage, but the focus of these models is on the extent of parental care afforded during the embryo stage. The models rest on two critical assumptions. First, larger embryos have a relatively high rate of embryo mortality, which might arise because of a constant embryo mortality but increased development time for large embryos (Gillooly *et al.*, 2002) or positive size-specific depredation of embryos (Shine, 1989). Second, parental care can lower the instantaneous

rate of mortality during the embryo stage. When these two assumptions hold, then a positive relationship between the amount of parental care and I^* can evolve (Sargent *et al.*, 1987; Nussbaum & Schultz, 1989).

The safe harbor concept was recently extended by Jørgensen, Auer & Reznick (2011) to help explain the maternal-size correlation. From first principles, Jørgensen *et al.* (2011) derive a sigmoidal relationship for $w(I)$ (as in Fig. 1A), and using parameters estimated from the literature, they show that adult survival should also be positively related to adult size. Adult survival is important for species that guard their eggs or that are viviparous, given that parental survival during the period of care becomes a critical component of w. When positive size-specific survival of adults exists, then larger parents provide better parental care, a relationship that arises from lower parental mortality during the period of care and hence higher average w for larger parents. Furthermore, assuming that larger offspring take longer to develop (Sargent *et al.*, 1987; Gillooly *et al.*, 2002), there may be a negative relationship between I and parental survival. As a result, Jørgensen *et al.* (2011) predict that I^* can increase with parental body size because only larger parents with greater survival prospects can afford to gestate or guard large offspring. Thus, Jørgensen *et al.* (2011) demonstrate that $W(I)$ can change predictably with parental size, as the ability of parents to avoid mortality is size-specific, and small parents suffer a relatively greater survival cost when caring for larger offspring.

The model developed by Jørgensen *et al.* (2011) applies to discrete reproductive events, but it was not developed to predict patterns of offspring provisioning across a mother's lifetime. More recently, Kindsvater & Otto (2014) extend the work of Jørgensen *et al.* (2011) and model how trade-offs between current and future reproduction can result in age- or stage-specific values of I^* (see also Kindsvater *et al.*, 2010, 2011). When there is a direct cost of I to maternal survival (e.g. increased depredation), then the production of large offspring may not increase lifetime reproductive success of the parent, even though larger offspring have greater fitness at independence. However, as the adult ages, increasing emphasis is placed on current reproduction, as future reproductive prospects decrease (Williams, 1966; Creighton, Heflin & Belk, 2009). Consequently, $W(I)$ is predicted to change with the residual reproductive value of the parent, and in particular, I^* can be relatively large when a female's residual reproductive value is relatively low (Kindsvater & Otto, 2014). Collapsing this effect across all life stages results in age- or condition-dependent variation in I^*, which for many species can arise in the form of a maternal-size correlation. Interestingly, even when the survival cost associated with a given level of I is the same for all parents in a population (*cf.* Jørgensen *et al.*, 2011), this model can still predict age- or stage-dependent variation in $W(I)$, resulting in positive correlations between these traits and I^* (Kindsvater & Otto, 2014). This result arises from the model because residual reproductive value always varies with age or stage.

The models of Jørgensen *et al.* (2011) and Kindsvater & Otto (2014) focus on an association between I and extrinsic mortality factors, which ultimately results in maternal-size correlations by virtue of age- or size-related variation in $W(I)$ (see also Kindsvater *et al.*, 2010, 2011). Importantly, assigning survival costs to N in these models does not predict maternal-size correlations as readily as when survival costs are assigned to I (Kindsvater *et al.*, 2011; see also Sakai & Harada, 2001; Filin, 2015). This underlines the importance of the assumed cost to I, such that the generality of these models depends on a broadly applicable mechanism that links I with maternal survival. It is easy to envision a clear mechanism in some cases. For instance, larger seeds, eggs, or embryos may develop more slowly (Gillooly *et al.*, 2002), which may exacerbate mortality risk for parents provisioning or guarding these offspring (Jørgensen *et al.*, 2011; Kindsvater & Otto, 2014). In other cases, however, the mechanism is less clear. For instance, many fishes provision thousands of very small embryos simultaneously, and in the absence of a survival cost to N, it is difficult to envision how variation in I can be linked with variation in maternal survival. Furthermore, an age- or condition-dependence of I^* is rooted in decreases in residual reproductive value with age or condition, but some evidence suggests that maternal-size correlations can be very strong in semelparous species (Heath & Blouw, 1998). At present then, models that link variation in I with variation in extrinsic mortality provide an interesting framework to explore the evolution of I, but their importance in understanding the maternal-size correlation in general is not yet clear, as little work has been invested in understanding how variation in I might be associated with variation in parental survival.

(2) Condition-dependent offspring provisioning

That I must trade off against N (Lack, 1947) is central to every theoretical model of offspring-size evolution developed to date. With few exceptions, theoretical and empirical studies model $N(I)$ following the optimality model of Smith & Fretwell (1974), simply as $N = RA/I$ (for exceptions, see León & Nóbrega, 2000; Sakai & Harada, 2001; Kindsvater & Otto, 2014; Filin, 2015). However, despite its ubiquity in models, the only empirical support for $N = RA/I$ is that it adequately describes the patterns of I and N observed across species of mammals (Charnov & Ernest, 2006) and lizards (Warne & Charnov, 2008). Furthermore, the term RA in Smith & Fretwell's (1974) proportionality law reflects only the energy which is directly transferred to offspring, as if the trade-off involved dividing a pie into pieces (Fox & Czesak, 2000). But it is almost certain that a great deal of energy expended during reproduction is not directly allocated to offspring, and it is only relatively recently that theorists have begun to examine the consequences of $RA \neq NI$, and what mechanisms might be responsible for these added reproductive costs (Sikes, 1998; León & Nóbrega, 2000).

In the context of maternal-size correlations, Sakai & Harada (2001) were the first to develop a model where $RA \neq NI$. Theirs is, in essence, a straightforward expansion of Smith & Fretwell's (1974) proportionality law, with three

main components to the model: first, a fixed pool of resources is assumed, from which any number of offspring can draw nourishment; second, maintenance respiration costs are incurred by the parent during provisioning, which reduces the amount of nutrients that are available to offspring; third, post-provisioning offspring fitness is governed by a monotonically increasing sigmoid function common to all parents (as in Fig. 1A). They find that when resource transport rate to offspring is limited by how efficiently individual offspring can extract resources from the parent, I^* is larger when resource transport rate is greater, such that parental reproductive success is maximized at relatively large values of I and relatively low values of N. Interestingly, empirical data suggest that offspring developing in large or high-condition mothers are able to extract resources more quickly than those developing in smaller or low-condition mothers (Sakai & Harada, 2001; Sakai & Sakai, 2005). This supports the idea that the best strategy for large or high-condition parents is indeed to increase I at the expense of N for a given pool of resources, which would result in the maternal-size correlation.

Two important lessons can be learned from their study. First, I^* is affected by efficiency in the conversion of reproductive resources into I, a result that persists despite the fact that all females share a common relationship for $w(I)$ (*cf*. Parker & Begon, 1986). Second, the existence of condition-dependence in offspring provisioning efficiency suggests that $W(I)$ varies predictably with maternal size (see also Sakai & Sakai, 2005). Given the universal nature of baseline metabolic costs and the potential generality of condition-dependent offspring provisioning, this model represents a promising framework in which to explore maternal-size correlations. Nevertheless, the mechanism underlying condition-dependent provisioning is not yet clear, and Sakai & Harada (2001) fail to predict the maternal-size correlation when this condition-dependence is absent from their model. The generality of this model therefore depends on identifying a general reason why condition-dependent provisioning occurs in the first place.

(3) Overhead costs to reproduction

Sakai & Harada (2001) modified Smith & Fretwell's (1974) proportionality law ($N = RA/I$) partly by incorporating baseline metabolic costs during the period of offspring provisioning. However, there can be other costs associated with reproduction, such as the development of reproductive structures that support offspring provisioning (León & Nóbrega, 2000; Day & Rowe, 2002; Filin, 2015). Energetic costs that are required for an individual to become reproductively active but that are not directly translated into I or N are typically called 'overhead costs', as only energy that remains after the 'overhead' is paid can be translated into I or N. The problem of overhead costs in the evolution of I has only recently received theoretical attention (Sikes, 1995, 1998; León & Nóbrega, 2000; Filin, 2015), but this small body of literature provides further reason to question the proportionality law assumed by Smith & Fretwell (1974).

Filin (2015) was the first to examine overhead costs in the context of the maternal-size correlation. In his model, fitness is accrued over a mother's lifetime, and maternal starvation can occur if energy reserves fall below a certain threshold between reproductive bouts. Furthermore, maternal size is composed of maternal structural size, which cannot be allocated to reproduction, and maternal reserves, which can fluctuate and are recoverable. We note that Filin's (2015) work is complex, and that the overarching conclusions depend strongly on how specific types of overhead costs are incorporated into the model. Nevertheless, several general conclusions arise from this interesting work. For instance, a fixed overhead cost, or a cost that depends only on the maternal phenotype (e.g. maternal structural size), does not induce among-individual variation in I^*. Similarly, overhead costs proportional to N or to the product of N and I do not induce among-individual variation in I^*; costs such as these might be associated with shelling eggs or developing dispersal structures for seeds. Conversely, overhead costs that scale positively with I will tend to result in a joint increase in N and I^* with maternal energetic reserves, a result that arises because the *per capita* overhead cost associated with I becomes less significant as N increases. To explain this result, Filin (2015) suggests that a cost associated with I can be envisioned in terms of increased metabolic expenditure during offspring provisioning with increasing I, such that underlying metabolic costs incurred during provisioning are effectively diminished when offspring are all provisioned simultaneously. This finding is very similar to that of Sakai & Harada (2001).

The salient feature of Filin's (2015) model is that when overhead costs are associated with I, the maternal-size correlation is produced, whereas the correlation is not produced when overhead costs are associated with N. The model therefore seems promising, as overhead costs are likely a very general feature of reproduction, at least in plants and animals. Nevertheless, it is unclear what particular overhead costs might operate generally, and more specifically, what overhead cost(s) might scale with I in broad taxa. While Filin (2015) suggests that the overhead cost that scales with I might comprise underlying metabolic costs that occur during offspring provisioning (*cf*. Sakai & Harada, 2001), this definition of overhead costs conflates baseline metabolic costs (which always operate) with specific costs that are directly associated with reproduction. Drawing a distinction between these two types of costs is important in this case, as can be seen contrasting the findings of Sakai & Harada (2001) and Filin (2015). Generic overhead costs, like those modeled by Filin (2015), and metabolic costs that act in combination with features of the maternal phenotype (e.g. condition-dependent provisioning) (Sakai & Harada, 2001) can apparently have very similar effects on variation in I^*, yet they comprise very distinct mechanisms and processes. To elucidate the specific mechanism by which the maternal-size correlation is produced might therefore require assessing the relative importance of overhead costs, as defined herein, and other types of costs that act alone or in combination with

features of the maternal phenotype. We return to this topic in Section V, where we discuss avenues of future research.

IV. ADULT MORPHOLOGY INFLUENCES *I**

There are a number of 'non-adaptive' explanations for maternal-size correlations, where positive correlations between *I* and maternal size *per se* are attributed to an inability of small females to produce larger, more fit offspring (Clark, Ewert & Nelson, 2001; Plaistow *et al.*, 2007; Davis *et al.*, 2012). These explanations typically assume that maternal body size or morphology is optimized, then *I* becomes subject to limitations imposed by optimal maternal size. In other words, it is assumed that there is a morphological 'constraint' on *I*, such as the scaling of the width of the oviducts or the ovipositor to maternal size (Clark *et al.*, 2001; Yanagi & Tuda, 2012). The term 'constraint' can be meaningful under the strictest definition of optimality (Orzack & Sober, 1994; Abrams, 2001; Rollinson & Hutchings, 2013*b*), but it adds confusion to the study of maternal-size correlations. It is not a matter of adaptive or non-adaptive processes, it is a question of what evolutionary forces are present. Invoking a 'constraint' is akin to arguing that a particular species faces a particular selective force that will affect the optimization of *I*. For this reason, we avoid using the term 'constraint' and interpret arguments that have been made previously in an adaptive framework (see also Rollinson & Brooks, 2008*a*,*b*).

A classic explanation for the maternal-size correlation, which applies primarily to reptiles, relates to the size of the pelvic aperture (opening) through which eggs must pass during oviposition. Verbal arguments suggest that there is selection for a small pelvic aperture, to facilitate locomotion, which in turn affects *I** (Congdon & Gibbons, 1987; see also Zug, 1972). The *I* that maximizes fitness of small females is therefore the largest egg they can possibly produce, and *I* must increase with maternal body size up to the point where the aperture is large enough to allow *I* to be optimized with respect to external environmental conditions (Lovich *et al.*, 2012; Macip-Ríos, Sustaita-Rodriguez & Casas-Andreu, 2013). In the field of evolutionary ecology, this hypothesis was championed by Congdon, Gibbons & Greene (1983) and later by Congdon & Gibbons (1987) and Sinervo & Licht (1991). A similar hypothesis has long been invoked in the field of evolutionary anthropology, where a trade-off between bipedal locomotion and the size of the human brain at birth is thought to explain why humans are born in an altricial state (Krogman, 1951).

Several observations lend support to the prediction that *I** depends on body size because of concurrent selection for aperture width, as suggested by Congdon & Gibbons (1987). In lizards, for example, direct experimental evidence suggests that the width of the pelvic aperture does impose an upper limit on the size of the egg that a mother can produce (Sinervo & Licht, 1991). Comparative data in turtles also show that pelvic aperture width is larger in females than in males (Long & Rose, 1989), and in at least one species, pelvic kinesis allows an egg that is normally larger than the pelvic aperture to pass at oviposition (Hofmeyr, Henen & Loehr, 2005). Finally, egg size increases asymptotically with female size in at least three populations of turtles (Rollinson & Brooks, 2008*a*; Rollinson, Farmer & Brooks, 2012), suggesting that egg size becomes optimized with respect to external environmental conditions only after females reach a certain threshold size.

While there are clear associations between maternal morphology and *I*, there is little reason to suspect that maternal-size correlations are an emergent property of selection for features of maternal structural size. For instance, even in the classic case of turtles, maternal-size correlations exist in populations where there is no correspondence between aperture size and egg size (Iverson & Smith, 1993; Clark *et al.*, 2001). In light of this, Rollinson & Brooks (2008*a*,*b*) point out that phenotypic correlations do not determine the direction of causality, and that even if aperture width is important in terrestrial locomotion, aperture width might encroach on an egg size that has already been optimized by some other adaptive process, such as sibling competition (Parker & Begon, 1986) or by the combined effects of sibling competition and maternal nest-site choice (Hendry *et al.*, 2001). Indeed, even Krogman's (1951) long-standing aperture hypothesis was recently supplanted by a metabolic explanation (Dunsworth *et al.*, 2012), undermining the importance of trade-offs between bipedal locomotion and the size of the pelvic girdle in determining human birth mass. Thus, so-called 'constraints' on offspring size are perhaps overemphasized as an explanation for the positive scaling of *I* and maternal size *per se*.

Unknown morphological factors are sometimes invoked to explain why the mass of individual offspring scales positively with maternal size across species (e.g. Davis *et al.*, 2012; Caval-Holme, Payne & Skotheim, 2013). Perhaps this is because comparative studies that seek ecological explanations for interspecific variation in species-mean *I* often find that mean maternal body size of a species is the only important predictor of species-mean *I* (Rahn, Paganelli & Ar, 1975; Dixon & Hemptinne, 2001; Gilbert & Manica, 2010; Davis *et al.*, 2012; Caval-Holme *et al.*, 2013), suggesting that adult morphology somehow has an overarching influence on the evolution of *I*. Yet one can question a direct effect of maternal size on offspring size, at least in a universal sense, on the simple grounds that positive interspecific correlations are weak or lacking across amphibians, fish, crustaceans, and plants (Visman *et al.*, 1996). An alternative view is that these interspecific scaling patterns reflect adaptive, correlated evolution of *I* and maternal size. For instance, initial size can presumably have a positive influence on adult size and hence adult components of fitness in some groups, and this generates an expectation of correlated evolution of *I* and maternal size. Indeed, amniotes and groups with protracted parental care tend to show relatively strong interspecific scaling relationships between species-mean *I* and species mean body size (Visman *et al.*, 1996), suggesting a stronger correlated evolution of these two traits when life-histories are amenable to maternal influences on fitness components of adults.

Finally, when invoking morphological constraints imposed by maternal body size to explain variation in I, we must also remember that maternal body size is usually correlated with suites of life-history traits, such as age at maturity and *RA*. A limited number of phylogenetically explicit analyses have shown that evolutionary changes in species-mean I are positively associated with changes in *RA* (Beck & Beck, 2005, 2009; Ikeda *et al.*, 2008). In each of these analyses, the positive relationship persisted even after controlling for species-mean body size. Within species, there is also strong evidence of a correlated evolution of I and *RA*, although the direction of this relationship is variable. For instance, selection experiments have suggested that selection on I can result in changes in *RA*, although the direction of response in *RA* may or may not be the same as the direction of selection (Schwarzkopf *et al.*, 1999, with qualifications in Roff, 2002), and sometimes *RA* remains unchanged after selection on I (Czesak & Fox, 2003; Fischer *et al.*, 2006). Furthermore, reciprocal transplant experiments on Trinidadian guppies (*Poecilia reticulata*) demonstrate that when adult predation pressure is increased, *RA* evolves towards larger values and I towards smaller values (Reznick, Bryga & Endler, 1990; also see Winkler & Wallin, 1987; León & Nóbrega, 2000). The point is that maternal structural size can easily be overemphasized in its purported evolutionary relationship with I. While maternal size is easy to measure, it is clear that maternal size correlates with life-history traits that also coevolve with I, and here we have provided examples of evolutionary associations between I and *RA* both within and across species.

V. IN PURSUIT OF A UNIFYING EXPLANATION

We believe there is a unifying explanation for the maternal-size correlation, for several reasons. First, the maternal-size correlation appears to be observed in nearly all plant and animal taxa (Roff, 2002; Lim *et al.*, 2014). Second, the effect size of this correlation (Pearson's r) is essentially invariant across taxa and is similar even for amniotes *versus* non-amniotes, and captive *versus* wild animals (Lim *et al.*, 2014). Third, as we emphasize below (Fig. 3), the qualitative pattern of the maternal-size correlation is similar both within and among species (Blueweiss *et al.*, 1978; Visman *et al.*, 1996; Roff, 2002), across modes of migration (Tamate & Maekawa, 2000; Acolas *et al.*, 2008; Closs, Hicks & Jellyman, 2013), and across modes of parity (Heath & Blouw, 1998; Crespi & Teo, 2002). All of this suggests that some general mechanism is operating.

We have examined two types of adaptive explanations for the maternal-size correlation. This first series of models assumes that maternal size alters $w(I)$, either indirectly by virtue of sibling interactions or more directly by virtue of size-specific maternal influences on the offspring environment. The second series of models assumes that intrinsic qualities of a female, such as her residual reproductive value or her ability to escape depredation, have the result of altering $W(I)$. Having examined all theoretical models as well as the empirical evidence in support of these models, we synthesize this information in the present section and attempt to narrow the scope of possible explanations. First, we focus on what trait is actually mediating the maternal-size correlation. Next, we suggest what we believe to be promising avenues of future inquiry, and we highlight important observations that could inform both empirical and theoretical development of this problem.

(1) Maternal size or unmeasured traits?

The maternal-size correlation is likely driven by maternal nutritional status or maternal condition, rather than maternal size *per se*. Recent theory supports this conclusion (Filin, 2015), but an equally compelling line of evidence lies in experimental observation of maternal provisioning patterns under food stress. Roff (2002, p. 271) reviewed 16 studies in which maternal nutritional status was varied prior to reproduction, and in 69% of cases (11 of 16 studies) both I and N increased with increasing food. There is no clear basis for such an observation in classic theory, as increased resource status of the mother is generally predicted to affect N, not I (Smith & Fretwell, 1974; Brockelman, 1975; McGinley *et al.*, 1987). Similarly, there is a general propensity for I to decline seasonally in many species and populations, particularly in species that breed several times per season (reviewed by McGinley *et al.*, 1987; Green, 2008), and this decline may also be related to maternal resource status. Although a body of theory deals with plasticity of I in relation to the quality of the external environment (McGinley *et al.*, 1987; Schultz, 1991; Fischer, Taborsky & Kokko, 2011), declines in I with declining maternal resource status have never been fully explained or reconciled with theory (but see Filin, 2015). Interestingly, these declines parallel the maternal-size correlation, given that maternal body size is thought primarily to reflect variation in past and present nutritional status (e.g. Price, Kirkpatrick & Arnold, 1988; Schluter, Price & Rowe, 1991). The general tendency for I to vary with maternal nutritional status might, then, occur for the same proximate reason as the maternal-size correlation (Filin, 2015).

(2) Avenues of future inquiry

A majority of studies in the field of offspring-size evolution have focused exclusively on how $w(I)$ responds to the external environment, and this overrepresentation might have arisen because there is a clear and straightforward relationship between variation in $w(I)$ and variation in I^* (Brockelman, 1975; McGinley *et al.*, 1987; Einum & Fleming, 2000, 2004; Hendry *et al.*, 2001; Rollinson & Hutchings, 2013*a*,*b*). It is therefore not surprising that maternal-size correlations were first explained by virtue of size-specific maternal influences on $w(I)$ (Parker & Begon, 1986), an idea that has been persistent in the literature (Hendry *et al.*, 2001; Einum & Fleming, 2002; Einum *et al.*, 2002; Hendry & Day, 2003; Marshall *et al.*, 2010; Rollinson & Hutchings, 2010).

In the present review, we have emphasized that the maternal-size correlation is probably not related to maternal influences on $w(I)$. This is because an association between variation in $w(I)$ and maternal body size is unlikely in many species, and in fact, even when these associations are likely, direct evidence fails to support the hypothesis (Takahashi *et al.,* 2005; Acolas *et al.,* 2008; Rollinson & Hutchings, 2011; Louhi *et al.,* 2015). Similarly, explaining the correlation by suggesting that maternal body size imposes 'constraints' on I is conceptually unsatisfactory (Congdon & Gibbons, 1987; Sinervo & Licht, 1991; Rollinson & Brooks, 2008*a*), and at any rate, such morphological 'constraints' are also unlikely to apply broadly in many groups, such as in plants and fishes (Einum *et al.,* 2004; Marshall *et al.,* 2010).

We also explored another general hypothesis for the maternal-size correlation: namely that $W(I)$ varies directly with female size or condition, a hypothesis pioneered by Sakai & Harada (2001) and that is receiving increasing attention in the theoretical literature (Jørgensen *et al.,* 2011; Kindsvater *et al.,* 2011; Kindsvater & Otto, 2014; Filin, 2015). This exciting area of research has provided a number of new insights into the origin of the maternal-size correlation. In particular, a recurrent finding of these models is that costs associated with N are generally less effective in producing the maternal-size correlation than are costs associated with I (Sakai & Harada, 2001; Kindsvater *et al.,* 2011; Filin, 2015). To this end, costs have been assigned to I in several ways, including a direct negative influence on maternal survival (Jørgensen *et al.,* 2011; Kindsvater *et al.,* 2011; Kindsvater & Otto, 2014), an indirect influence on survival by virtue of associations between I and overhead costs (Filin, 2015), and metabolic costs associated with the time required to provision large offspring (Sakai & Harada, 2001). An interesting new challenge for empiricists and theorists, then, is to identify which type of cost is most broadly applicable, and what mechanism is associated with generating these costs in the first place.

We suggest that it may be particularly useful to pursue overhead costs that are associated with I. While modelling generic overhead costs can produce maternal-size correlations (Filin, 2015), biological realism and a resolution to this life-history puzzle can only be achieved by identifying and understanding the mechanism underlying these costs. It is also important to draw a distinction between overhead costs that can produce the maternal-size correlation, and underlying metabolic costs that interact with aspects of the maternal phenotype to produce the correlation. Both mechanisms affect $W(I)$ by altering $N(I)$, but these mechanisms can be distinguished conceptually, and doing so is of interest in clarifying how the maternal-size correlation arises (Sakai & Harada, 2001; Filin, 2015).

More broadly, there is currently very little experimental work on what costs alter $N(I)$ (Sikes, 1995, 1998; Sakai & Sakai, 2005), and a fruitful goal for empiricists might be to achieve a better understanding such costs. This is no trivial task, however, as it requires an understanding of resource acquisition and subsequent allocation along the RA–N–I allocation hierarchy (de Jong & van Noordwijk, 1992; Worley, Houle & Barrett, 2003; King, Roff & Fairbairn, 2011*a*,*b*; Roff & Fairbairn, 2012). While experimental investigation of these costs is daunting, we point out that positive associations between maternal nutritional status and I, those that are observed in manipulative experiments (e.g. Roff, 2002), might arise by the same mechanism as the maternal-size correlation. Therefore, a complimentary avenue of inquiry might be to investigate the enigmatic association between maternal nutritional status and I using experimental manipulation, within the conceptual context of determining the mechanism involved in the maternal-size correlation. Experimental manipulations of nutritional status are tangible and can be performed in a variety of species, such that this approach may prove to be tremendously important in the pursuit of a general explanation for the maternal-size correlation.

The maternal-size correlation might also be clarified by a meta-analysis of regression slopes. Even though Pearson's r values are already known to be similar across taxa (Lim *et al.,* 2014), a phylogenetic analysis of the average change in I with a change in maternal size could reveal different types of associations in different taxa or environments. There are indeed examples of species (or perhaps environments) in which there is clearly very little relationship between I and maternal size (Fischer, Zwaan & Brakefield, 2002). For instance, in pipefishes (Syngnathidae), males provide all of the postzygotic parental care, and care takes the form of 'pouch brooding' in some species, while in other species paternal care consists of 'ventral brooding'. Interestingly, there is a positive relationship between female size and egg size in pouch-brooding species, but the maternal-size correlation is conspicuously absent in species with ventral brooding (Fig. 4A). Similarly, all species of pacific salmon (*Oncorhynchus* spp.) are semelparous, and these semelparous species show maternal-size correlations that are far stronger (i.e. a steeper slope) than the correlations observed in their iteroparous counterparts. Surprisingly, this association is observed both within populations (Heath & Blouw, 1998) and across species (Crespi & Teo, 2002), underlining the point that parallel associations between maternal and offspring size recur at multiple ecological scales (Fig. 3). More broadly, these examples demonstrate that maternal-size correlations are not invariant and that exceptions do exist. If other general exceptions or deviations from the rule can be identified and explored more thoroughly, then the biology of these groups might prove very informative in understanding maternal-size correlations.

Finally, it is not our goal to discourage the development of theory that may help understand better how maternal-size correlations arise or are moderated by particular biological features of specific taxa. While specific mechanisms that are rooted in overhead or metabolic costs might ultimately explain the correlation very broadly, the presence of these mechanisms does not preclude the existence of other adaptive mechanisms. For instance, models that rely on the presence of

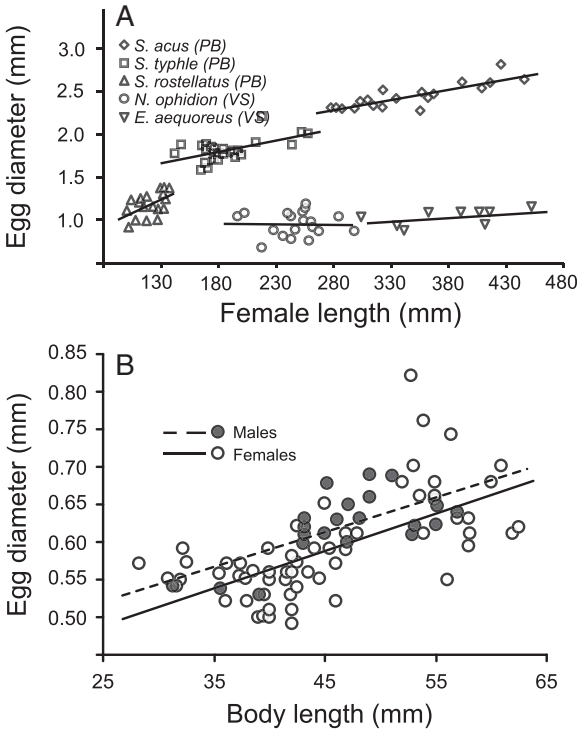

Fig. 4. (A) Relationships between maternal size and egg diameter in pipefish (Syngnathidae), a family in which males provide postzygotic parental care in the form of nourishment, oxygenation and osmoregulation. There is a positive correlation between maternal size and egg size in pouch-brooding species, *PB* (*Syngnathus acus*, *S. typhle*, *S. rostellatus*), but no relationship is apparent in ventral-brooding species, *VS* (*Nerophis ophidion*, *Entelurus aequoreus*). Redrawn from Goncalves *et al.* (2011). (B) In the marbled goby, *Pomatoschistus marmoratus*, males construct nests and provide postzygotic parental care in the form of egg guarding and egg fanning. Nest surface area, the number of eggs being guarded, and the size of eggs being guarded all increase with paternal size. Depicted is the relationship between egg size and female size, and the relationship between egg size and the size of males guarding the eggs. Redrawn from Mazzoldi *et al.* (2002).

parental care to produce the correlation may not be general (e.g. Jørgensen *et al.*, 2011), but nevertheless it is possible that the quantity and quality of parental care might moderate the strength or extent of maternal-size correlations in some groups. As an example, in the marbled goby (*Pomatoschistus marmoratus*), males construct nests and provide parental care to embryos. Female size is positively correlated both with I and with the size of the male that subsequently defends these embryos (Fig. 4B), suggesting size-assortative mating (Mazzoldi, Poltronieri & Rasotto, 2002). If male size is positively related to the quality of parental care, e.g. if male size *per se* increases male survival during incubation, then I^* might also increase with parental size. In this case, the model of Jørgensen *et al.* (2011) seems consistent with this pattern, such that this model might ultimately help clarify the nuances of the maternal-size correlation when parental care is present.

VI. CONCLUSIONS

(1) A general explanation for the positive correlation between maternal size and I is highly plausible. The best line of evidence for a unifying explanation is the pervasive nature of the correlation, coupled with the similarity of the effect size in all taxa (Lim *et al.*, 2014). It is also compelling that *decreases* in I with maternal size are exceptionally rare (Roff, 2002).

(2) Maternal size *per se* is probably not causatively related to variation in I, and the trait that effects the maternal-size correlation is likely maternal nutritional status or maternal condition, which is usually correlated with maternal size. In this respect, the average effect size estimated from associations between 'maternal size' and I [Pearson's $r \approx 0.40$ on average across 200+ species (Lim *et al.*, 2014)] might be greatly underestimated, because in most analyses, maternal size is the measured trait, but maternal condition is the true explanatory variable.

(3) Models that assume maternal influences on $w(I)$ lack generality. Therefore, the maternal-size correlation is probably not related to sibling interactions or size-specific maternal influences on the offspring environment, at least not across diverse taxa.

(4) Explaining the correlation by suggesting that maternal body size imposes 'constraints' on I is conceptually unsatisfactory, and this idea suffers from the same lack of generality as models assuming that $w(I)$ varies with female size.

(5) A promising avenue of inquiry is to investigate further why $W(I)$ might vary with maternal size. In particular, realism can be added to $N(I)$ by exploring what particular overhead costs are associated with I, or by exploring the mechanism(s) underlying condition-dependence in offspring provisioning efficiency.

(6) Using manipulative experiments to elucidate why females with high nutritional status typically produce large offspring might help explain what mechanism(s) underlie the maternal-size correlation. Furthermore, identifying taxonomic groups or populations that either deviate from the rule or are bonafide exceptions to the rule (e.g. Figs 3 and 4) might help resolve what proximate and ultimate mechanism(s) underlie this life-history puzzle.

VII. ACKNOWLEDGEMENTS

We thank J. Porter, two anonymous reviewers, and members of the Rowe lab for thought-provoking discussions on this topic. Funding to N.R. was provided by a University of Toronto Ecology and Evolutionary Biology Postdoctoral Fellowship and by a Natural Sciences and Engineering Council of Canada Postdoctoral Fellowship. Funding to L.R. was provided by NSERC, and the Canada Research Chairs Program.

VIII. REFERENCES

Abrams, P. (2001). Adaptationism, optimality models, and tests of adaptive scenarios. In *Adaptationism and Optimality* (eds S. H. Orzack and E. Sober), pp. 273–302. Cambridge University Press, New York.

Acolas, M., Roussel, J.-M. & Baglinière, J.-L. (2008). Linking migratory patterns and diet to reproductive traits in female brown trout (*Salmo trutta* L.) by means of stable isotope analysis on ova. *Ecology of Freshwater Fish* **17**, 382–393.

Barnes, H. & Barnes, M. (1965). Egg size, nauplius size, and their variation with local, geographical, and specific factors in some common cirripedes. *Journal of Animal Ecology* **34**, 391–402.

Bashey, F. (2008). Competition as a selective mechanism for larger offspring size in guppies. *Oikos* **117**, 104–113.

Beck, C. W. & Beck, R. E. (2005). The effect of packing constraints on optimal investment in offspring. *Evolutionary Ecology Research* **7**, 1077–1088.

Beck, C. W. & Beck, R. E. (2009). Trade-offs among life-history traits; a comment on Wang et al. (2009). *Journal of Zoology* **279**, 319–320.

Begon, M. & Parker, G. A. (1986). Should egg size and clutch size decrease with age? *Oikos* **47**, 293–302.

Bernardo, J. (1996). The particular maternal effects of propagule size, especially egg size: patterns, models, quality of evidence and interpretation. *American Zoologist* **36**, 216–236.

Blueweiss, L., Fox, H., Kudzma, V. & Nakashima, D. (1978). Relationships between body size and some life history parameters. *Oecologia* **37**, 257–272.

Brockelman, W. Y. (1975). Competition, the fitness of offspring, and optimal clutch size. *American Naturalist* **109**, 677–699.

Brooks, R., Hunt, J., Blows, M. W., Smith, M. J., Bussière, L. F. & Jennions, M. D. (2005). Experimental evidence for multivariate stabilizing sexual selection. *Evolution* **59**, 871–880.

Caval-Holme, F., Payne, J. & Skotheim, J. M. (2013). Constraints on the adult-offspring size relationship in protists. *Evolution* **67**, 3537–3544.

Charnov, E. L. & Ernest, S. K. M. (2006). The offspring-size/clutch-size trade-off in mammals. *American Naturalist* **167**, 578–582.

Clark, P. J., Ewert, M. A. & Nelson, C. E. (2001). Physical apertures as constraints on egg size and shape in the Common Musk Turtle, *Sternotherus odoratus*. *Functional Ecology* **15**, 70–77.

Closs, G. P., Hicks, A. S. & Jellyman, P. G. (2013). Life histories of closely related amphidromous and non-migratory fish species: a trade-off between egg size and fecundity. *Freshwater Biology* **58**, 1162–1177.

Congdon, J. & Gibbons, J. (1987). Morphological constraint on egg size: a challenge to optimal egg size theory? *Proceedings of the National Academy of Sciences of the United States of America* **84**, 4145–4147.

Congdon, J. D., Gibbons, J. W., Brooks, R. J., Rollinson, N. & Tsaliagos, R. N. (2013). Indeterminate growth in long-lived freshwater turtles as a component of individual fitness. *Evolutionary Ecology* **27**, 445–459.

Congdon, J., Gibbons, J. & Greene, J. L. (1983). Parental investment in the chicken turtle (*Deirochelys reticularia*). *Ecology* **64**, 419–425.

Creighton, J. C., Heflin, N. D. & Belk, M. C. (2009). Cost of reproduction, resource quality, and terminal investment in a burying beetle. *American Naturalist* **174**, 673–684.

Crespi, B. B. J. & Teo, R. (2002). Comparative phylogenetic analysis of the evolution of semelparity and life history in salmonid fishes. *Evolution* **56**, 1008–1020.

Czesak, M. E. M. & Fox, C. W. C. (2003). Evolutionary ecology of egg size and number in a seed beetle: genetic trade-off differs between environments. *Evolution* **57**, 1121–1132.

Davis, R. B., Javoiš, J., Pienaar, J., Õunap, E. & Tammaru, T. (2012). Disentangling determinants of egg size in the Geometridae (Lepidoptera) using an advanced phylogenetic comparative method. *Journal of Evolutionary Biology* **25**, 210–219.

Day, T. & Rowe, L. (2002). Developmental thresholds and the evolution of reaction norms for age and size at life-history transitions. *American Naturalist* **159**, 338–350.

Dixon, A. F. G. & Hemptinne, J.-L. (2001). Body size distribution in predatory ladybird beetles reflects that of their prey. *Ecology* **82**, 1847–1856.

Downhower, J. F. & Charnov, E. L. (1998). A resource range invariance rule for optimal offspring size predicts patterns of variability in parental phenotypes. *Proceedings of the National Academy of Sciences of the United States of America* **95**, 6208–6211.

Dunsworth, H. M., Warrener, A. G., Deacon, T., Ellison, P. T. & Pontzer, H. (2012). Metabolic hypothesis for human altriciality. *Proceedings of the National Academy of Sciences of the United States of America* **109**, 15212–15216.

Eberhart, A. & Tielbörger, K. (2012). Maternal fecundity does not affect offspring germination – An empirical test of the sibling competition hypothesis. *Journal of Arid Environments* **76**, 23–29.

Ebert, D. (1993). The trade-off between offspring size and number in *Daphnia magna*: the influence of genetic, environmental and maternal effects. *Archiv für Hydrobiologie* **90**, 453–473.

Einum, S. & Fleming, I. A. (1999). Maternal effects of egg size in brown trout (*Salmo trutta*): norms of reaction to environmental quality. *Proceedings of the Royal Society B: Biological Sciences* **266**, 2095–2100.

Einum, S. & Fleming, I. A. (2000). Highly fecund mothers sacrifice offspring survival to maximize fitness. *Nature* **405**, 565–567.

Einum, S. & Fleming, I. A. (2002). Does within-population variation in fish egg size reflect maternal influences on optimal values? *American Naturalist* **160**, 756–765.

Einum, S. & Fleming, I. A. (2004). Environmental unpredictability and offspring size: conservative versus diversified bet-hedging. *Evolutionary Ecology Research* **6**, 443–455.

Einum, S., Hendry, A. P. & Fleming, I. A. (2002). Egg-size evolution in aquatic environments: does oxygen availability constrain size? *Proceedings of the Royal Society B: Biological Sciences* **269**, 2325–2330.

Einum, S., Kinnison, M. T. & Hendry, A. P. (2004). Evolution of egg size and number. In *Evolution Illuminated: Salmon and their Relatives* (eds A. P. Hendry and S. C. Stearns), pp. 128–153. Oxford University Press, Oxford.

Filin, I. (2015). The relation between maternal phenotype and offspring size, explained by overhead material costs of reproduction. *Journal of Theoretical Biology* **364**, 168–178.

Fischer, K., Bot, A. N. M., Brakefield, P. M. & Zwaan, B. J. (2006). Do mothers producing large offspring have to sacrifice fecundity? *Journal of Evolutionary Biology* **19**, 380–391.

Fischer, B., Taborsky, B. & Kokko, H. (2011). How to balance the offspring quality-quantity tradeoff when environmental cues are unreliable. *Oikos* **120**, 258–270.

Fischer, K., Zwaan, B. & Brakefield, P. (2002). How does egg size relate to body size in butterflies? *Oecologia* **131**, 375–379.

Fox, C. W. & Czesak, M. E. (2000). Evolutionary ecology of progeny size in arthropods. *Annual Review of Entomology* **45**, 341–369.

Gilbert, J. D. J. & Manica, A. (2010). Parental care trade-offs and life-history relationships in insects. *The American Naturalist* **176**, 212–226.

Gilchrist, G. W. & Kingsolver, J. G. (2001). Is optimality over the hill? The fitness landscapes of idealized organisms. In *Adaptationism and Optimality* (eds S. H. Orzack and E. Sober), pp. 219–241. Cambridge University Press, New York.

Gillooly, J. F., Charnov, E. L., West, G. B., Savage, V. M. & Brown, J. H. (2002). Effects of size and temperature on developmental time. *Nature* **417**, 70–73.

Gómez, J. M. (2004). Bigger is not always better: conflicting selective pressures on seed size in *Quercus ilex*. *Evolution* **58**, 71–80.

Goncalves, I. B., Ahnesjö, I., Kvarnemo, C. & Braga Goncalves, I. (2011). The relationship between female body size and egg size in pipefishes. *Journal of Fish Biology* **78**, 1847–1854.

Green, B. S. (2008). Maternal effects in fish populations. *Advances in Marine Biology* **54**, 1–105.

Gross, M. R. & Sargent, R. C. (1985). The evolution of male and female parental care in fishes. *American Zoologist* **25**, 807–822.

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

Hendriks, A. & Mulder, C. (2008). Scaling of offspring number and mass to plant and animal size: model and meta-analysis. *Oecologia* **155**, 705–716.

Hendry, A. P. & Day, T. (2003). Revisiting the positive correlation between female size and egg size. *Evolutionary Ecology Research* **5**, 421–429.

Hendry, A. P., Day, T. & Cooper, A. B. (2001). Optimal size and number of propagules: allowance for discrete stages and effects of maternal size on reproductive output and offspring fitness. *American Naturalist* **157**, 387–407.

Hofmeyr, M. D., Henen, B. T. & Loehr, V. J. (2005). Overcoming environmental and morphological constraints: egg size and pelvic kinesis in the smallest tortoise, *Homopus signatus*. *Canadian Journal of Zoology* **83**, 1343–1352.

Hunt, J. & Simmons, L. W. (2000). Maternal and paternal effects on offspring phenotype in the dung beetle *Onthophagus taurus*. *Evolution* **54**, 936–941.

Hutchings, J. A. (1991). Fitness consequences of variation in egg size and food abundance in brook trout *Salvelinus fontinalis*. *Evolution* **45**, 1162–1168.

Ikeda, H., Kagaya, T., Kubota, K. & Abe, T. (2008). Evolutionary relationships among food habit, loss of flight, and reproductive traits: life-history evolution in the Silphinae (Coleoptera: Silphidae). *Evolution* **62**, 2065–2079.

Iverson, J. B. & Smith, G. R. (1993). Reproductive ecology of the painted turtle (*Chrysemys picta*) in the Nebraska sandhills and across its range. *Copeia* **1993**, 1–21.

de Jong, G. & van Noordwijk, A. J. (1992). Acquisition and allocation of resources: genetic (co)variances, selection, and life histories. *American Naturalist* **139**, 749–770.

Jørgensen, C., Auer, S. K. & Reznick, D. N. (2011). A model for optimal offspring size in fish, including live-bearing and parental effects. *American Naturalist* **177**, E119–E135.

Kindsvater, H. K., Alonzo, S. H., Mangel, M. & Bonsall, M. B. (2010). Effects of age- and state-dependent allocation on offspring size and number. *Evolutionary Ecology Research* **12**, 327–346.

Kindsvater, H. K., Bonsall, M. B. & Alonzo, S. H. (2011). Survival costs of reproduction predict age-dependent variation in maternal investment. *Journal of Evolutionary Biology* **24**, 2230–2240.

Kindsvater, H. K. & Otto, S. P. (2014). The evolution of offspring size across life-history stages. *American Naturalist* **184**, 543–555.

King, E. G., Roff, D. A. & Fairbairn, D. J. (2011*a*). Trade-off acquisition and allocation in Gryllus firmus: a test of the Y model. *Journal of Evolutionary Biology* **24**, 256–264.

King, E. G., Roff, D. A. & Fairbairn, D. J. (2011*b*). The evolutionary genetics of acquisition and allocation in the wing dimorphic cricket, *Gryllus firmus*. *Evolution* **65**, 2273–2285.

Kishi, S. (2014). Brood ball size but not egg size correlates with maternal size in a dung beetle, *Onthophagus atripennis*. *Ecological Entomology* **39**, 355–360.

Kjesbu, O., Solemdal, P., Bratland, P. & Fonn, M. (1996). Variation in annual egg production in individual captive Atlantic cod (*Gadus morhua*). *Canadian Journal of Fisheries and Aquatic Sciences* **620**, 610–620.

Krogman, W. (1951). The scars of human evolution. *Scientific American* **185**, 54–57.

Lack, D. (1947). The significance of clutch-size. *Ibis* **89**, 302–352.

Lalonde, R. (1991). Optimal offspring provisioning when resources are not predictable. *American Naturalist* **138**, 680–686.

Lalonde, R. (2005). Egg size variation does not increase offspring performance under interspecific competition in *Nasonia vitripennis*, a gregarious parasitoid. *Journal of Animal Ecology* **74**, 630–635.

León, J. A. & Nóbrega, J. R. (2000). Comparative statics of joint reproductive allocation. *Journal of Theoretical Biology* **205**, 563–579.

Lim, J. N., Senior, A. M. & Nakagawa, S. (2014). Heterogeneity in individual quality and reproductive trade-offs within species. *Evolution* **68**, 2306–2318.

Long, D. R. & Rose, F. L. (1989). Pelvic girdle relationships in three turtle species. *Journal of Herpetology* **23**, 315–318.

Louhi, P., Robertsen, G., Fleming, I. A. & Einum, S. (2015). Can timing of spawning explain the increase in egg size with female size in salmonid fish? *Ecology of Freshwater Fish* **24**, 23–31.

Lovich, J. E., Madrak, S. V., Drost, C. A., Monatesti, A. J., Casper, D. & Znari, M. (2012). Optimal egg size in a suboptimal environment: reproductive ecology of female Sonora mud turtles (*Kinosternon sonoriense*) in central Arizona, USA. *Amphibia-Reptilia* **33**, 161–170.

Macip-Ríos, R., Sustaita-Rodriguez, V. H. & Casas-Andreu, G. (2013). Evidence of pelvic and nonpelvic constraint on egg size in two species of *Kinosternon* from Mexico. *Chelonian Conservation and Biology* **12**, 218–226.

Marshall, D. J., Cook, C. N. & Emlet, R. B. (2006). Offspring size effects mediate competitive interactions in a colonial marine invertebrate. *Ecology* **87**, 214–225.

Marshall, D. J., Heppell, S. S., Munch, S. B. & Warner, R. R. (2010). The relationship between maternal phenotype and offspring quality: do older mothers really produce the best offspring? *Ecology* **91**, 2862–2873.

Marshall, D. J. & Keough, M. J. (2007). The evolutionary ecology of offspring size in marine invertebrates. *Advances in Marine Biology* **53**, 1–60.

Marteinsdóttir, G. & Steinarsson, A. (1998). Maternal influence on the size and viability of Iceland cod *Gadus morhua* eggs and larvae. *Journal of Fish Biology* **52**, 1241–1258.

Mazzoldi, C., Poltronieri, C. & Rasotto, M. (2002). Egg size variability and mating system in the marbled goby *Pomatoschistus marmoratus* (Pisces: Gobiidae). *Marine Ecology Progress Series* **233**, 231–239.

McGinley, M. A. (1989). The influence of a positive correlation between clutch size and offspring fitness on the optimal offspring size. *Evolutionary Ecology* **3**, 150–156.

McGinley, M. A., Temme, D. H. & Geber, M. A. (1987). Parental investment in offspring in variable environments: theoretical and empirical considerations. *American Naturalist* **130**, 370–398.

McIntyre, G. S. & Gooding, R. H. (2000). Effects of maternal age on larval competitiveness in house flies. *Heredity* **85**, 480–489.

Nussbaum, R. & Schultz, D. (1989). Coevolution of parental care and egg size. *American Naturalist* **133**, 591–603.

Orzack, S. H. & Sober, E. (1994). Optimality models and the test of adaptationism. *American Naturalist* **143**, 361–380.

Parker, G. A. & Begon, M. (1986). Optimal egg size and clutch size: effects of environment and maternal phenotype. *American Naturalist* **128**, 573–592.

Pianka, E. R. (1976). Natural selection of optimal reproductive tactics. *American Zoologist* **16**, 775–784.

Plaistow, S. J., Lapsley, C. T. & Benton, T. G. (2006). Context-dependent intergenerational effects: the interaction between past and present environments and its effect on population dynamics. *American Naturalist* **167**, 206–215.

Plaistow, S. J., St. Clair, J. J. H., Grant, J. & Benton, T. G. (2007). How to put all your eggs in one basket: empirical patterns of offspring provisioning throughout a mother's lifetime. *American Naturalist* **170**, 520–529.

Price, T., Kirkpatrick, M. & Arnold, S. J. (1988). Directional selection and the evolution of breeding date in birds. *Science* **240**, 798–799.

Rahn, H., Paganelli, C. V. & Ar, A. (1975). Relation of avian egg weight to body weight. *The Auk* **1975**, 750–765.

Réale, D. & Festa-Bianchet, M. (2000). Mass-dependent reproductive strategies in wild bighorn ewes: a quantitative genetic approach. *Journal of Evolutionary Biology* **13**, 679–688.

Reznick, D. A., Bryga, H. & Endler, J. A. (1990). Experimentally induced life-history evolution in a natural population. *Nature* **346**, 357–359.

Roff, D. A. (2002). *Life History Evolution*. Sinauer Associates, Inc., Sunderland.

Roff, D. A. & Fairbairn, D. J. (2012). The evolution of trade-offs under directional and correlational selection. *Evolution* **66**, 2593–2960.

Rollinson, N. & Brooks, R. J. (2008*a*). Optimal offspring provisioning when egg size is 'constrained': a case study with the painted turtle *Chrysemys picta*. *Oikos* **117**, 144–151.

Rollinson, N. & Brooks, R. J. (2008*b*). Sources and significance of among-individual reproductive variation in a northern population of painted turtles (*Chrysemys picta*). *Copeia* **2008**, 533–541.

Rollinson, N., Farmer, R. G. & Brooks, R. J. (2012). Widespread reproductive variation in North American turtles: temperature, egg size and optimality. *Zoology* **115**, 160–169.

Rollinson, N. & Hutchings, J. A. (2010). Why does egg size increase with maternal size ? Effects of egg size and egg density on offspring phenotypes in Atlantic salmon (*Salmo salar*). *Evolutionary Ecology Research* **12**, 949–960.

Rollinson, N. & Hutchings, J. A. (2011). Body size-specific maternal effects on the offspring environment shape juvenile phenotypes in Atlantic salmon. *Oecologia* **166**, 889–898.

Rollinson, N. & Hutchings, J. A. (2013*a*). Environmental quality predicts optimal egg size in the wild. *American Naturalist* **182**, 76–90.

Rollinson, N. & Hutchings, J. A. (2013*b*). The relationship between offspring size and fitness: integrating theory and empiricism. *Ecology* **94**, 315–324.

Sakai, S. & Harada, Y. (2001). Why do large mothers produce large offspring ? Theory and a test. *American Naturalist* **157**, 348–359.

Sakai, S. & Sakai, A. (2005). Nature of size–number trade–off: test of the terminal-stream-limitation model for seed production of *Cardiocrium cordatum*. *Oikos* **108**, 105–114.

Sargent, R. C., Taylor, P. D. & Gross, M. R. (1987). Parental care and the evolution of egg size in fishes. *American Naturalist* **129**, 32–46.

Schluter, D., Price, T. D. & Rowe, L. (1991). Conflicting selection pressures and life-history trade-offs. *Proceedings of the Royal Society B: Biological Sciences* **246**, 11–17.

Schrader, M. & Travis, J. (2012). Variation in offspring size with birth order in placental fish: a role for asymmetric sibling competition? *Evolution* **66**, 272–279.

Schultz, D. L. (1991). Parental investment in temporally varying environments. *Evolutionary Ecology* **5**, 414–427.

Schwarzkopf, L., Blows, M. W., Caley, J. M. & Caley, M. (1999). Life-history consequences of divergent selection on egg size in *Drosophila melanogaster*. *American Naturalist* **154**, 333–340.

Shine, R. (1978). Propagule size and parental care: the 'safe harbor' hypothesis. *Journal of Theoretical Biology* **75**, 417–424.

Shine, R. (1989). Alternative models for the evolution of offspring size. *American Naturalist* **134**, 311–317.

Siepielski, A. M., DiBattista, J. D. & Carlson, S. M. (2009). It's about time: the temporal dynamics of phenotypic selection in the wild. *Ecology Letters* **12**, 1261–1276.

Sikes, R. S. (1995). Costs of lactation and optimal litter size in northern grasshopper mice (*Onychomys leucogaster*). *Journal of Mammalogy* **76**, 348–357.

Sikes, R. S. (1998). Unit pricing: economics and the evolution of litter size. *Evolutionary Ecology* **12**, 179–190.

Sinervo, B. & Licht, P. (1991). Proximate constraints on the evolution of egg size, egg number and total clutch mass in lizards. *Science* **252**, 1300–1302.

Smith, C. & Fretwell, S. (1974). The optimal balance between size and number of offspring. *American Naturalist* **108**, 499–506.

Stanton, M. L. (1984). Seed variation in wild radish: effect of seed size on components of seedling and adult fitness. *Ecology* **65**, 1105–1112.

Su, G., Liljedahl, L. & Gall, G. (1997). Genetic and environmental variation of female reproductive traits in rainbow trout (*Oncorhynchus mykiss*). *Aquaculture* **154**, 115–124.

Summers, K., Sea McKeon, C. & Heying, H. (2006). The evolution of parental care and egg size: a comparative analysis in frogs. *Proceedings of the Royal Society B: Biological Sciences* **273**, 687–692.

Takahashi, K., Makino, T. T. & Sakai, S. (2005). Effects of sib-competition on female reproductive success in *Salvia lutescens* Koidz. var. crenata. *Evolutionary Ecology Research* **7**, 1201–1212.

Tamate, T. & Maekawa, K. (2000). Interpopulation variation in reproductive traits of female masu salmon, *Oncorhynchus masou*. *Oikos* **90**, 209–218.

Thomson, F. J., Moles, A. T., Auld, T. D. & Kingsford, R. T. (2011). Seed dispersal distance is more strongly correlated with plant height than with seed mass. *Journal of Ecology* **99**, 1299–1307.

Venable, L. D. (1992). Size-number trade-offs and the variation of seed size with plant resource status. *American Naturalist* **140**, 287–304.

Visman, V., Pesant, S., Dion, J., Shipley, B. & Peters, R. R. H. (1996). Joint effects of maternal and offspring sizes on clutch mass and fecundity in plants and animals. *Ecoscience* **3**, 173–182.

Wall, R. & Begon, M. (1986). Population density, phenotype and mortality in the grasshopper *Chorthippus brunneus*. *Ecological Entomology* **11**, 445–456.

Warne, R. W. & Charnov, E. L. (2008). Reproductive allometry and the size-number trade-off for lizards. *American Naturalist* **172**, E80–E98.
Williams, G. C. (1966). Natural selection, the costs of reproduction, and a refinement of Lack's principle. *American Naturalist* **100**, 687–690.
Winkler, D. W. & Wallin, K. (1987). Offspring size and number: a life history model linking effort per offspring and total effort. *American Naturalist* **129**, 708–720.
Worley, A. C., Houle, D. & Barrett, S. C. H. (2003). Consequences of hierarchical allocation for the evolution of life-history traits. *American Naturalist* **161**, 153–167.
Yanagi, S.-I. & Tuda, M. (2012). Female size constrains egg size via the influence of reproductive organ size and resource storage in the seed beetle *Callosobruchus chinensis*. *Journal of Insect Physiology* **58**, 1432–1437.
Zastrow, C. E., Houde, E. D. & Saunders, E. H. (1989). Quality of striped bass (*Morone saxatilis*) eggs in relation to river source and female weight. *Rapports et Proces-Verbaux des Reunions du Conseil International pour l'Exploration de la Mer* **191**, 34–42.
Zug, G. R. (1972). Walk pattern analysis of cryptodiran turtle gaits. *Animal Behaviour* **20**, 439–443.

(*Received 23 March 2015; revised 3 July 2015; accepted 23 July 2015; published online 20 August 2015*)