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REVIEW

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Mechanisms of reproductive allocation as drivers of developmental plasticity in reptiles

James U. Van Dyke[1] | Oliver W. Griffith[2,3]

[1]School of Environmental Sciences, Institute of Land, Water & Society, Charles Sturt University, Albury, NSW, Australia

[2]Department of Ecology and Evolutionary Biology, Yale University, New Haven, Connecticut

[3]Yale Systems Biology Institute, Yale University, West Haven, Connecticut

Correspondence
James U. Van Dyke, Lecturer in Ecology, School of Environmental Sciences, Charles Sturt University, Albury, NSW 2640 Australia.
Email: jvandyke@csu.edu.au

Abstract

Developmental plasticity in offspring phenotype occurs as a result of the environmental conditions embryos experience during development. The nutritional environment provided to a fetus is an important source of developmental plasticity. Reptiles are a particularly interesting system to study this plasticity because of their varied routes of maternal nutrient allocation to reproduction. Most reptiles provide their offspring with all or most of the nutrients they require in egg yolk (lecithotrophy) while viviparous reptiles also provide their offspring with nutrients via a placenta (placentotrophy). We review the ways in which both lecithotrophy and placentotrophy can lead to differences in the nutrients embryonic reptiles receive, and discuss how these differences lead to developmental plasticity in offspring phenotype. We finish by reviewing the ecological and conservation consequences of nutritional-driven developmental plasticity in reptiles. If nutritional-driven developmental plasticity has fitness consequences, then understanding the basis of this plasticity has exciting potential to identify how reptile recruitment is affected by environmental changes in food supply. Such knowledge is critical to our ability to protect taxa threatened by environmental change.

KEYWORDS

conservation, lecithotrophy, matrotrophy, placenta, recruitment, vitellogenesis, yolk

1 | INTRODUCTION

Developmental plasticity is the generation of different phenotypes as a result of environmental conditions experienced by a developing embryo or fetus, independent of genotype (West-Eberhard, 2003). Developmental plasticity is important because it can impact the divergence of closely related species (West-Eberhard, 2005). Furthermore, phenotypic differences driven by developmental plasticity have major fitness implications for offspring (Van Buskirk & Steiner, 2009). In addition, there is potential for ecosystem-level consequences of plasticity within species, as a result of downstream changes to among-species interactions (Miner, Sultan, Morgan, Padilla, & Relyea, 2005). Humans are driving major environmental changes leading to species declines and extinctions (Dirzo et al., 2014), and there is an increasing need to understand the role of developmental plasticity in transducing or mitigating environmental effects. An example is how anthropogenic climate warming can lead to population biases in sex ratio in reptile species with temperature-dependent sex determination (Mitchell & Janzen, 2010). A strong bias in sex ratio would eventually lead to population collapse as a result of loss of offspring recruitment.

The assortment of nutrients an embryo receives during development is one of the major drivers of developmental plasticity. Embryonic nutrition has multi-generational fecundity consequences in *Anolis* lizards (Warner, Buckelew, Pearson, & Dhawan, 2015), impacts metabolism of adult rats (Gluckman et al., 2007), and has been linked to long-term health problems in humans (Bateson et al., 2004). Variation in embryonic nutrition arises as a result of an interaction among three factors: food constraints on mothers, the genetic and physiological mechanisms of maternal allocation, and, in placental animals, the ability of the embryo to manipulate maternal reproductive allocation mechanisms. Importantly, food constraints on mothers must always be viewed as a primary limitation on the nutrients they can allocate to their developing offspring. Even when food is abundant, assimilated nutrients must be allocated to a suite of competing physiological functions, including maintenance, activity, growth, and reproduction (Congdon, Dunham, & Tinkle, 1982; Dunham, Grant, & Overall, 1989).

Here, we review how variation in the mechanisms of maternal allocation drives developmental plasticity in reptiles, and discuss the potential consequences of the resulting plasticity for reptilian ecology and conservation. Reptiles are a fascinating system for studying maternal allocation and developmental plasticity because of their diversity

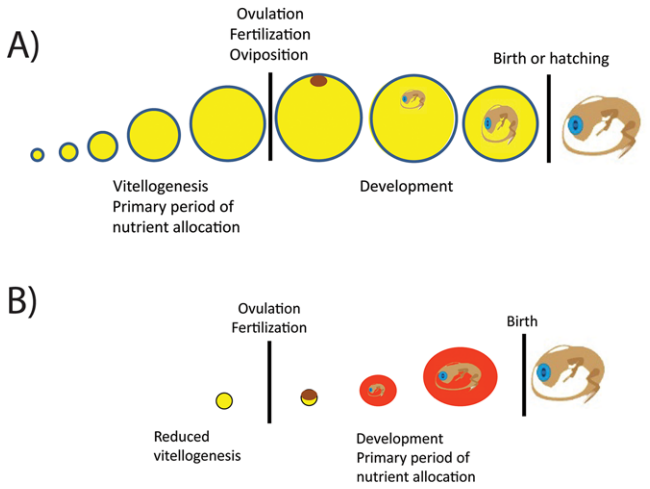

FIGURE 1 Diagrammatic representations of the mechanisms of reproductive allocation in reptiles. (A) Lecithotrophic reptiles slowly allocate yolk precursors to ovarian follicles, indicated by the growth in circle size and color yellow. After ovulation and fertilization (and oviposition in lecithotrophic viviparous species), the embryo consumes yolk as it develops, indicated by the decrease in size of the circle. (B) Placentotrophic reptiles, which are always viviparous), exhibit a reduced period of vitellogenesis, although this reduction is highly variable across species. The embryo then increases in size at some rate over the course of development as nutrients are provided across the placenta. The reduction on reliance on yolk by the embryo is indicated by the color red, as opposed to yellow, surrounding the embryo. In both diagrams, the brown oval early in development is meant to represent a blastodisc [Color figure can be viewed at wileyonlinelibrary.com]

in maternal allocation mechanisms and their wide potential for nutritional impacts on developmental plasticity. Reptiles provide their offspring with nutrients via two potential mechanisms: lecithotrophy and placentotrophy (Figure 1), and we consider both of these mechanisms separately in our review. Lecithotrophic animals are those that provide nutrients to their offspring primarily or exclusively via yolk (Blackburn, 2000; Wourms, 1981). However, "lecithotrophy" is also used to describe the more general provisioning of nutrients prior to development (Uribe & Grier, 2011), including oil droplets in the ova of teleost fish. The vast majority of reptiles are lecithotrophic, including all oviparous species, and almost all viviparous species. Only the placentotrophic skinks within the taxa *Chalcides*, *Eumecia*, *Lubuya ivensii*, *Mabuya*, *Niveoscincus*, and *Pseudemoia* have reduced their reliance on lecithotrophy, and most of these remain at least partially lecithotrophic (Thompson, Stewart, & Speake, 2000). In oviparous species, lecithotrophy in practice also describes those species that deposit an albumen layer on eggs after ovulation: the Crocodila, Rhynchocephalia, and Testudinata. Squamate ova conspicuously lack an albumen layer (Packard, Tracy, & Roth, 1977). In oviparous reptiles, the eggshell is also deposited after ovulation, and is a source of ions for developing reptiles, especially calcium during development (Palmer & Guillette, 1991). Thus, in practice the descriptor "lecithotrophy" does include animals that use some post-ovulatory nutritional mechanisms. Still, in each of these cases, yolk is the primary source of embryonic nutrition, and will be our main focus in the lecithotrophy section of our review.

In contrast to lecithotrophy, placentotrophic animals are those that supplement embryonic nourishment through the transfer of nutrients across a placenta during embryogenesis. Placentotrophy is therefore assumed to be restricted to viviparous (live-bearing) species, which in reptiles are limited to about 20% of all species of squamates (lizards and snakes). Placentas in squamates are formed from the apposition of uterine tissue with a series of embryonic membranes, including the chorioallantoic and yolk sac membranes. While substantial transfer of macro-nutrients has evolved only five or six times in squamates (Blackburn, 2015b), in all viviparous squamates there seems to be some level of incipient placentotrophy (Hoffman, 1970; Stewart, 2013; Stewart, 1989; Stewart, 2015; Van Dyke & Beaupre, 2012; Yaron, 1977). In species with incipient placentotrophy, the surface epithelium of the uterus is responsible for the transport of post-ovulatory organic nutrients (Blackburn & Lorenz, 2003; Blackburn, Anderson, Johnson, Knight, & Gavelis, 2009; Blackburn, Gavelis, Anderson, Johnson, & Dunlap, 2010; Stewart & Brasch, 2003), but the makeup of these nutrients is unknown. It is likely that uterine shell glands, which mobilise calcium and eggshell proteins to the uterine lumen in oviparous reptiles, have a functional role in incipient transport of nutrients in viviparous squamates. (Cornetti et al., In Press; Herbert et al., 2006; Herbert, Murphy, & Thompson, 2010) In species that rely on a large amount of yolk initially in development but also exhibit a substantial increase in offspring mass compared to the mass of eggs at ovulation (ex., *Pseudemoia* sp.), the nutrient composition of placental transfer appears to match the composition of egg yolk at the macro and micronutrient scale (Thompson et al., 2000). In species that are (nearly) completely reliant on placentotrophy, (e.g., *Mabuya* sp.), placental transport is responsible for large quantities of lipid, protein, calcium, magnesium, potassium, and sodium, which replace yolk nearly completely (Ramirez-Pinilla, 2006; Ramirez-Pinilla, Rueda, & Stashenko, 2011).

2 | MATERNAL PLASTICITY IN LECITHOTROPHY

2.1 | Vitellogenesis

In lecithotrophic vertebrates, the egg yolk is a dense package that contains all, or nearly all, of the nutrients a developing embryo requires (energy, protein, micronutrients, etc.). Yolk is formed via vitellogenesis. During vitellogenesis, yolk precursor proteins (vitellogenin; VTG and very low density lipoprotein; VLDL) are formed in the maternal liver. Vitellogenesis is itself stimulated by estradiol secreted by the ovaries via activation of the hypothalamo-pituitary-gonadal axis, usually in response to seasonal, resource availability, and social cues (Van Dyke, 2014). Estradiol stimulates hepatic tissues to synthesize VTG, a lipoglycophosphoprotein, and VLDL, which are then transported via the bloodstream to the ovaries and sequestered into growing follicular oocytes via receptor-mediated endocytosis (Wallace, 1985). Within the oocytes, VTG is proteolysed by cathepsin D into yolk proteins including lipovitellins and phosvitins (Finn, 2007).

Notably, most of our knowledge of this pathway comes from model species of lampreys, teleost fish, *Xenopus*, and *Gallus*, and specific

details of vitellogenesis in non-avian reptiles have been rarely documented (Nainan et al., 2009), aside from its endocrine regulation (Callard, Ho, Gapp, Kleis, & Heisermann, 1985; Callard, Riley, & Perez, 1990; Ho, 1987; Ho, Kleis, McPherson, Heisermann, & Callard, 1982). However, several interesting differences occur among taxa. For example, in chickens, VLDL represents 60% of yolk dry mass and contributes 23% of yolk protein and 93% of lipid, whereas VTG contributes 48% of yolk protein and 7% of lipid (Burley, Back, Wellington, & Grigg, 1988; Thompson & Speake, 2003). In crocodilians, VLDL accounts for only 30% of yolk dry mass and VTG is much more abundant (Burley et al., 1988; White, 1991). VLDL has not been isolated from the yolks of squamates or turtles (Thompson & Speake, 2003) and was noticeably not reported in the yolk of *Anolis pulchellus* (De Morales, Valles, & Baerga-Santini, 1987). VLDL has been isolated from the plasma of vitellogenic female turtles (Duggan et al., 2001; Lance, Place, Grumbles, & Rostal, 2002) and the snake *Bothrops jararaca* (Janiero-Cinquini, Bijovsky, Leinz, & Winter, 1995), Thus, the nutritional roles of both VTG and VLDL may be variable in reptiles. Speake and Thompson (1999) suggest that these differences relate to the different energy and nutritional requirements of different taxa. VLDL is a major source of nonpolar lipids, including triacylglycerol, and bird embryos need large amounts of nonpolar lipids to fuel their higher metabolic costs of embryogenesis (Speake & Thompson, 1999). Indeed, bird eggs typically contain a lipid:protein ratio of approximately 1:1, while that of reptile eggs are variable, but average 1:2 (Speake & Thompson, 1999). These ratios reflect the statistical difference in energy density of egg yolks (birds: 29.2 kj/g; reptiles: 26.9 kj/g; Booth & Thompson, 1991). The bioenergetic requirements of development in these taxa may explain the considerable differences in VLDL and VTG fractions between birds and non-avian reptiles, but similar hypotheses could also explain smaller differences observed across reptile species.

2.2 | VTG and VLDL structure and function

In most lecithotrophic reptiles, VTG must provide nearly all of the nutrients embryos need to complete development. Because VLDL has been identified in crocodilian yolks, it likely provides nutrition in their embryos in addition to that provided by VTG. Both molecules can be envisioned as capsules or spheres. VTG is a polypeptide dimer of the molecules lipovitellin and phosvitin (Raag, Appelt, Xuong, & Banaszak, 1988). Together, both polypeptides contain over 1800 amino acids and the VTG amino acid sequence may differ by more than 40% across vertebrate species (Nardelli et al., 1987; White, 1991). The lipovitellin component is essentially a polypeptide capsule that carries lipids (Banaszak, Sharrock, & Timmins, 1991; Raag et al., 1988). The fractions of fatty acids carried in lipovitellin are plastic and vary with diet in chickens (Naber & Biggert, 1989). In contrast, the phosvitin component of VTG is heavily phosphorylated and may carry up to 90% of all of the phosphorous in yolk (Samaraweera, Zhang, Lee, & Ahn, 2011). The phosphate molecules themselves act as chelating factors that have a strong affinity to bivalent ions, including calcium, magnesium, and iron (Grizzuti & Perlmann, 1973; Samaraweera et al., 2011; Taborsky, 1963).

Essentially, VTG acts as a remarkably efficient combined transporter/resource. It provides a mechanism of transporting lipids, phosphates, and ions from the liver to the oocyte, but also simultaneously provides the amino acids the eventual embryo will need, via its built-in polypeptide structure. Its secondary structure (i.e., amino acid sequence) is probably tuned by selection to ensure that all amino acids necessary for embryonic development are provided to the oocyte. Simultaneously, the VTG tertiary structure is probably semi-independently tuned by selection to provide cavities and attachment points for carrying other necessary nutrients (e.g., Finn, 2007). When viewed on a per-molecule basis, the polypeptide secondary structure of a VTG molecule is for the most part fixed within species as its amino acid sequence is encoded in the genome. Thus, the number and identity of amino acids that an oocyte receives should be relatively constant per molecule of VTG. In contrast, the relative composition of lipids, phosphate, and ions is not genetically fixed, and probably depends at least somewhat on the availability of these nutrients to the liver once vitellogenesis is initiated (e.g., Naber & Biggert, 1989). Thus, the per-VTG-molecule composition of non-protein nutrients can be highly variable among molecules, oocytes, or mothers.

In contrast to VTG, VLDL is a droplet of lipid, with a core of triacylglycerol and cholesteryl ester surrounded by a phospholipid bilayer, free cholesterol, and apoproteins (Anton et al., 2003; Martin, Augustyniak, & Cook, 1964). In non-avian reptiles, VLDL has only been reported in crocodilian yolk, but it could occur in the yolks of other reptiles that have not been adequately investigated. In birds, VLDL particles are large spherical droplets that contain large amounts of triacylglycerol and cholesterol ester, surrounded by phospholipid and free cholesterol (Speake & Thompson, 1999). The lipid droplets are embedded or even partially surrounded with apoproteins (Anton et al., 2003; Martin et al., 1964; Walzem, 1996) which help transport the lipid from liver to oocyte and facilitate receptor-mediated endocytosis (Speake & Thompson, 1999). In birds and crocodilians, VLDL likely functions to provide the embryo with large amounts of lipids, both for structural and bioenergetic purposes (Speake & Thompson, 1999). In reptiles that lack VLDL, VTG must provide all of the lipids the embryo requires (Speake & Thompson, 1999). In species that use it, the large size of some of the apoproteins found in VLDL suggest that it could also function as an accessory amino acid transporter, alongside VTG. Like VTG, the genetic basis of VLDL-associated apoproteins (Kirghgessner et al., 1987) probably constrains variability in what amino acids are provided on a per-molecule basis. In contrast, the fatty acid composition of VLDL is highly plastic with diet (Speake & Thompson, 1999).

2.3 | How can yolk composition vary?

In this section, we are primarily concerned with how the production of VTG and VLDL could lead to variation in yolk composition within an individual female reptile. Presumably, this variation can occur either as a result of maternal manipulations to maximize offspring fitness (Mousseau & Fox, 1998), or as a result of environmental constraints on maternal nutrition (Geister, Lorenz, Hoffman, & Fischer, 2008). Environmental constraints can occur as a result of dietary restrictions or as a result of competitive use by competing biological functions (Dunham et al., 1989).

The model of oocyte production via hepatic vitellogenesis and ovarian uptake from circulation suggests that the organic nutrient component of yolk is largely a matrix of discrete VTG-derived and possibly VLDL-derived molecules. Thus, one of the most important ways that a female reptile can vary her yolk composition is to allocate varying numbers of discrete VTG/VLDL molecules into different oocytes. This decision likely underlies the variation in egg size (via yolk dry mass) that has been frequently reported in reptiles, both within and among females (Bernardo, 1996b; Finkler & Claussen, 1997; Wallace et al., 2006). Selective pressures on egg size, and co-variation among egg size, egg number, body size, and age, are fundamental questions in life history evolution and have received considerable attention in the literature (Bowden, Harms, Paitz, & Janzen, 2004; Congdon & Gibbons, 1987; Dunham & Miles, 1985; Dunham, Miles, & Reznick, 1988; Ford & Seigel, 1989; Lack, 1954). In contrast, the genetic and physiological mechanisms underlying this variation, or how mothers actually manipulate egg size during vitellogenesis, are virtually unknown. We hypothesize that it occurs as a result of (1) the total number of VTG/VLDL molecules produced in the liver in a reproductive bout; (2) the number of ovarian follicles recruited in a reproductive bout; (3) the number of VTG/VLDL receptors produced in each ovarian follicle; and (4) the amount of time each VTG/VLDL receptor is active in each follicle. Thus, there are a number of potential mechanisms that a mother could use to determine how many VTG/VLDL molecules are allocated to each oocyte. Based on optimal egg size theory and morphological constraints, we assume that natural selection fine-tunes these mechanisms to produce a mean offspring size phenotype that mothers might "bet-hedge" around, along with clutch size, to maximize potential offspring survivorship (Bernardo, 1996b; Congdon & Gibbons, 1987; Smith & Fretwell, 1974).

In addition to effects on egg size, altering the number of VTG/VLDL molecules allocated to a given oocyte is probably also the only way to adjust what amino acids are provided to an oocyte via yolk. The amino acid compositions of both molecules are determined by the mother's VTG/VLDL genes, and are thus not likely to vary (White, 1991). Thus, if a mother aimed to manipulate the amount of a particular amino acid allocated to an oocyte, she would probably have to produce additional whole VTG molecules, rather than allocate only that amino acid. The polypeptide synthesis of VTG and VLDL likely uses free amino acids that are hydrolyzed from dietary or stored proteins, or synthesized de novo (White, 1991). Essential amino acids would have to be acquired from diet, but non-essential amino acids can be produced de novo via transamination. A lack of a particular essential amino acid can thus be a limiting factor in VTG/VLDL synthesis.

The second way a female reptile could vary her yolk composition would be to alter the contents of individual VTG/VLDL molecules during vitellogenesis in the liver. Amino acid compositions of VTG/VLDL probably cannot be modified this way, and the broader lipid fractions of VTG and VLDL appear to be relatively fixed (Speake & Thompson, 2000; Walzem, 1996). For example, chicken egg lipids are consistently made up of about 71% triacylglycerol, 21% polar lipids, 6% free cholesterol, 1% cholesteryl ester, and 1% free fatty acids (Speake & Thompson, 1999). However, the composition of fatty acids within each lipid fraction are much more variable (Speake & Thompson, 1999; Speake, Herbert, & Thompson, 2004; Thompson et al., 1999) and appear to change with diet (Naber & Biggert, 1989; Noble, McCartney, & Ferguson, 1993; Speake, Noble, McCartney, & Ferguson, 1994; Speake, Surai, & Bortolotti, 2002). Some fatty acids, including palmitic, stearic, palmitoleic, and oleic acids, are nonessential and are likely derived both from de novo synthesis from carbohydrates and from the mother's diet (Speake & Thompson, 1999). Thus, these should not be limiting and may be readily available for allocation into VTG/VLDL molecules. Other fatty acids, including linoleic and α-linoleic acids are essential and may only be available for allocation if present in the diet (Speake & Thompson, 1999). Either mechanism could play a role in maternal effects: synthesis of non-essential fatty acids can allow females to manipulate their density within individual VTG/VLDL molecules within yolk, whereas dietary acquisition of essential fatty acids can limit the number of VTG/VLDL molecules that were adequately provisioned for the embryo.

In addition to the organic nutrients present in yolk, it may also be possible for the non-organic nutrients to vary in abundance, especially metallic ions. Yolk (probably VTG) carries large amounts of calcium (Packard & Packard, 1991; Packard & Packard, 1988), and dietary restrictions on calcium are a major constraint for egg production in birds (Graveland & Drent, 1997; Tilgar, Mand, & Magi, 2002). In oviparous species, the primary constraint of calcium limitation can be on eggshell formation rather than vitellogenesis (Graveland, 1996). Both yolk and eggshell are major calcium sources for oviparous embryos, so while calcium limitations during vitellogenesis can reduce calcium content of yolk, it could be compensated by the calcium present in the eggshell.

3 | MATERNAL PLASTICITY IN PLACENTOTROPHY

In reptiles, placental nutrient transport likely occurs via a number of potential mechanisms, including apocrine, holocrine, and merocrine secretion, and membrane bound solute carriers (Griffith & Wagner, 2017; Van Dyke, Brandley, & Thompson, 2014a). In the most widely studied species, *Pseudemoia entrecasteauxii*, placentotrophy appears to use apocrine secretion and membrane-bound solute carriers (Biazik, Thompson, & Murphy, 2010; Biazik, Thompson, & Murphy, 2009). Lipids and proteins are packaged in vesicles (Adams, Biazik, Thompson, & Murphy, 2005; Griffith, Ujvari, Belov, & Thompson, 2013), and smaller molecules such as small ions, and small organic molecules like amino acids are likely transported by solute carriers (Griffith, Brandley, Belov, & Thompson, 2016b). The mode of secretion likely depends on the nutrients provided, and the location of the transport.

The amount of lipid and protein resources transported across the placenta in some placentotrophic lizards is a plastic trait, which can be impacted by maternal diet, basking opportunities, and maternal stress (Itonaga, Jones, & Wapstra, 2012; Swain & Jones, 2000; Van Dyke, Griffith, & Thompson, 2014b). Furthermore, placentotrophy can be modulated by interactions of these variables, for example, in *Niveoscincus metallicus* increased maternal diet only increases placental

provisioning when adequate basking opportunities are provided (Swain & Jones, 2000). Placentotrophy as a plastic trait allows mothers to nourish embryos (1) when nutrients are most available, (2) when mothers can be more assured that the offspring will be viable at birth, and (3) when nutrients are most needed by the embryo for growth and development.

3.1 | The genetic basis of plasticity in placentotrophy

Plasticity in placentotrophy ultimately relies on the mother's ability to control nutrient transport in one of the following three ways: (1) by adjusting the uterine machinery that facilitates transport (e.g., the solute carriers); (2) by adjusting the placental surface area that facilitates transport; and (3) by adjusting the circulating nutrient substrates for transport. No one has investigated whether any of these are altered in squamate pregnancy to modify differential transport of nutrients.

In mammals with an invasive hemochorial placenta, like humans and mice, embryonic tissues have direct access to maternal blood and so nutrient transfer is predominantly regulated by embryonic factors such as trophoblast size and regulation of trophoblast-expressed transporters including GLUT3 and SLC38A4 (Constancia et al., 2005). The genetic basis of this embryonic regulation is not completely known, but some important pathways have been identified, including the insulin-like growth factor signaling pathway. Wholesale suppression of embryonic insulin-like growth factor 2 (*IGF2*) results in a smaller offspring as a result of reduced placenta growth. However, suppression of *IGF2* in the trophoblast alone results in reduced placental growth, but not reduced offspring size, because somatic *IGF2* expression somehow communicates to the trophoblast to tell it to increase the expression of nutrient transport proteins GLUT3 and SLC38A4 (Fowden, Ward, Wooding, Forhead, & Constancia, 2006). In the horse, pig, and cow, where there are breed-specific differences in offspring weight, embryo transfer experiments allow us to identify maternal/embryonic effects that alter placentotrophy. The single biggest factor that alters placentotrophy from these studies is placental size and surface area (Fowden et al., 2006).

In all placentotrophic reptiles there seems to be uterine or trophoblast innovations that increase placental surface area (Blackburn, 1992; Stewart, 2013). These include additional folding and interdigitation of specific regions of the uterus. This increased folding is most significant in the placentotrophic lizards in the genera *Chalcides*, *Mabuya*, and *Pseudemoia*, which have specialized placental regions (e.g., the placentome) where there is a high degree of interdigitated folding between the uterus and chorioallantoic membrane (Adams et al., 2005; Blackburn & Callard, 1997; Vieira, de Perez, & Ramirez-Pinilla, 2007). Similarly, the placentotrophic *Lubuya ivensii* exhibits invasive components in its chorion and choriovitelline membrane, which also increase surface area (Blackburn & Flemming, 2012). In the highly placentotrophic genus, *Mabuya*, this increase in placental complexity is achieved in part by the recruitment of retroviral envelope proteins to the placenta (Cornelis et al., 2017; Denner, 2017). Future work is needed to test whether the plasticity in placentotrophy observed in placentotrophic squamates has genetic or physiological bases. Differences in placental complexity (degree of interdigitation and surface area), nutrient transport regulation, and/or in maternal mobilization of resources could all be physiological factors correlated with plasticity in placentotrophy.

3.2 | Maternal-fetal conflict and embryonic control

Plasticity in placental nutrient provisioning is distinct from lecithotrophy because it is susceptible to embryonic manipulation, rather than managed by the mother alone. This susceptibility occurs because placental nutrients are transported during (rather than prior to) embryonic development, and because transport is typically achieved in part by cooperation with the fetus. The mechanisms by which embryos manipulate placentotrophic provisions are better understood in mammals than in reptiles (Blackburn, 2015a). Hormones and growth factors produced by the trophoblast (the embryonic placental tissue) are a major source of embryonic regulation of placental nutrient transfer in humans and mice. Many of the hormones secreted into the placenta by viviparous mammals are also present in the embryonic placenta of viviparous squamates, including *IGF2* and progesterone (Girling & Jones, 2003; Griffith, Brandley, Whittington, Belov, & Thompson, 2017). Thus, there is potential for similar embryonic regulation of placental development to occur in reptiles. In viviparous mammals, *IGF2* is a particularly important growth factor. In viviparous mammals *IGF2* is an imprinted gene which is only expressed from the paternally inherited allele. Overexpression of *IGF2* in mouse and human results in fetal overgrowth, while *IGF2* deficient embryos have significantly smaller placentas which transfer fewer resources through the bulk of pregnancy (Wilkins & Haig, 2003). While genomic imprinting has not been found in any non-mammalian viviparous vertebrates, this may be due to methodological constraints, as only candidate gene approaches have been used to search for them (Griffith, Brandley, Belov, & Thompson, 2016a).

4 | DEVELOPMENTAL AND ECOLOGICAL CONSEQUENCES OF PLASTICITY IN REPRODUCTIVE ALLOCATION

Broadly speaking, the developmental, and subsequently ecological, consequences of plasticity in maternal allocation in reptiles have received limited attention in the literature. Far more research has focused on how differences in egg size and number, presumably caused by interactions of genetic and plastic differences in reproductive allocation, and environmental effects, can impact offspring size, survivorship, and/or biology (Aubret, Bonnet, Shine, & Maumelat, 2003; Congdon & Gibbons, 1987; Packard, Miller, Packard, & Birchard, 1999; Roosenburg & Dunham, 1997; Sinervo & Licht, 1991). More recent investigations have begun to investigate how differences in allocation of particular nutrient classes can affect offspring phenotype. For example, in the placentotrophic *Niveoscincus ocellatus*, protein content of pregnant mothers' diets interacts with that of postpartum offspring diets to impact offspring growth and sprint speed (Cadby, Jones, & Wapstra, 2011). Notably, neither offspring size nor sprint speed at

birth is affected by maternal diet only. Instead, litter size is decreased by extra protein, and maternal growth is increased. Together, these results suggest that bulk protein availability to pregnant mothers may impact offspring phenotype, but the underlying mechanisms remain unclear. While a protein-rich diet may increase the total amount of amino acids available to a mother to allocate to her offspring, it may not provide the essential amino acids needed to directly benefit any single individual offspring. In addition to increasing studies on bulk nutrient allocation, a growing body of literature has investigated how allocation of accessory hormones into egg yolk affects offspring phenotype, particularly sex in species with temperature-dependent sex determination (Bowden, Ewert, & Nelson, 2000; Bowden, Ewert, Freedberg, & Nelson, 2002; Crews, 1996; Paitz, Sawa, & Bowden, 2012). In this section, we review the current knowledge of how maternal plasticity in reproductive allocation impacts developmental plasticity and ecology of individual offspring in reptiles.

4.1 | Developmental consequences

The physiology of reproductive allocation allows several predictions about how variations in nutrient abundance affect developmental success and/or growth of embryos. First, because the polypeptide secondary structures of VTG and VLDL in lecithotrophic species are likely to be fixed (White, 1991), at least within species, scarcity in essential amino acids required for these structures may be directly limiting to VTG or VLDL synthesis. Scarcity of essential fatty acids may not limit the number of VTG or VLDL molecules produced, but could reduce the abundance of those fatty acids within each VTG or VLDL molecule. How subsequent reductions in abundance of VTG or VLDL molecules, or a lack of essential fatty acids within those molecules, impacts an embryo is unknown in reptiles. We hypothesize that such scarcities should result either in fewer ovarian follicles being fully provisioned and ovulated, and/or reductions in the nutrients available to each ovum. The former case would simply result in smaller clutch sizes. In the latter case, reductions in nutrients available to an ovum should result in smaller egg/offspring size. Yolkectomy experiments indicate that even large reductions in yolk can still allow complete development of viable reptile embryos (Sinervo, 1990; Sinervo & Huey, 1990; Sinervo, Doughty, Huey, & Zamudio, 1992). Thus, reductions in the provisioning of nutrients, and resulting smaller egg yolk size, may not alone lead to reductions in fecundity. Instead, the fitness consequences of small offspring (e.g., Janzen, 1993a) may select for a minimum offspring size, below which a female's fitness is unlikely to benefit (Shine, 1978). However, there probably is some species-specific lower limit of nutrient allocation, below which an embryo cannot successfully complete development.

A second possibility is that reductions in some essential nutrients might not impact the abundance of VTG or VLDL molecules in a way that would affect egg or offspring size, but could still impact offspring phenotype. Notably, such impacts can occur both in the allocation of non-protein nutrients bound to VTG or VLDL in lecithotrophic species, or of any nutrients freely transported across a placenta in placentotrophic species. For example, reduced allocations of trace metals, like zinc, can result in congenital malformations, as in rats (Hurley, 1969). Currently, what nutrients are essential and cause physiological problems if deficient in embryonic reptiles is poorly known. However, fatty acids are a useful starting point for investigation. Many 18-carbon polyunsaturated fatty acids are essential, and their abundances vary widely across potential food types (Speake & Thompson, 1999). Thus, their abundance might vary widely in eggs across mothers, or possibly even within mothers depending on the frequency of dietary shifts a female experience during a reproductive allocation period (Van Dyke, Beck, Jackson, & Hopkins, 2013). Another fatty acid, docosahexaenoic acid (DHA; 22:6*n*-3) is produced as a result of desaturation/elongation of essential fatty acids, such as α-linoleic acid (18:3*n*-3), and is important for the development of the brain and retina (Speake & Thompson, 1999). Deficiencies in DHA have been linked to visual loss in fish (Bell et al., 1995), and both brain and retinal deficiencies in monkeys (Neuringer, Connor, & Van Petten CB, 1984; Neuringer, Connor, Lin, Barstad, & Luck, 1986). Deficiencies in DHA have even been linked to behavioral changes in rats (Moriguchi, Sheaff Greiner, & Salem, 2000). Deficiencies in DHA (or other nutrients) may also be associated with low hatching success in captive alligators (Noble et al., 1993). Such deficiencies of fatty acids, or other nutrients important for brain development, might also underlie cognitive differences observed in juvenile White's skinks (*Liopholis whitii*) from mothers fed restricted diets (Munch et al., 2018).

4.2 | Ecological implications

Ultimately, whether and how plasticity in reproductive nutrient allocation affects developmental plasticity in reptiles is important not just for advancing our understanding of reptile biology, but also for improving conservation of threatened reptile species. Clearly, if plasticity in reproductive allocation affects the number of offspring produced then there are direct consequences for population dynamics. The best example of these impacts is when local changes in food supply lead to changes in adult fecundity (Beaupre & Douglas, 2009; Bonnet, Naulleau, Shine, & Lourdais, 2001; Congdon, 1989; Douglas, 2010; Seigel & Ford, 1992; Van Dyke et al., 2014b; Winne, Willson, & Gibbons, 2006). Likewise, if plasticity in reproductive allocation affects the "quality" of individual offspring in a way that impacts their likelihood to recruit or survive to adulthood, then there are also consequences for population dynamics.

How plasticity in reproductive allocation impacts individual offspring in a way that affects their recruitment is more cryptic than direct impacts on maternal fecundity. These impacts are often studied under the umbrella term of maternal effects. Maternal effects are the non-genetic influence mothers have on offspring phenotype, which include the effects of plasticity in reproductive allocation (Bernardo, 1996a; Newcombe, Hunt, Mitchell, & Moore, 2015; Roach & Wulff, 1987). Because maternal effects impact offspring phenotype independently of genetics, they are a direct cause of developmental plasticity. Maternal effects in reproductive allocation are a well-recognized concept among biologists, and in reptile studies have often focused on among-mother differences in egg size and/or offspring biology (Bernardo, 1996b; Rhen & Lang, 1995; Valenzuela, 2001), including as a result of food manipulations (Warner & Lovern, 2014). Furthermore, a

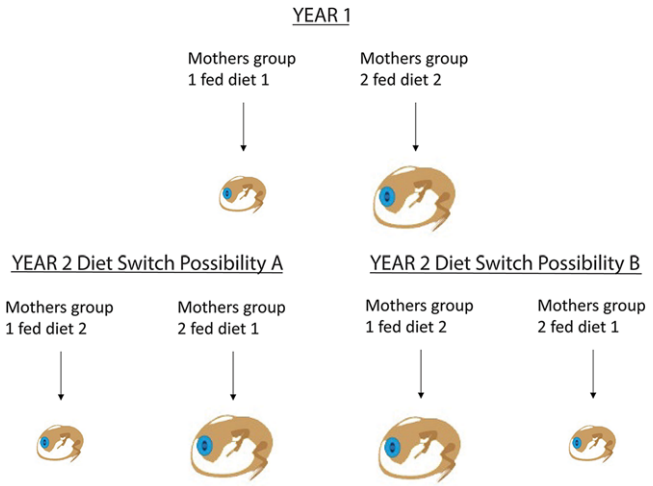

FIGURE 2 Experimental paradigm for studying reproductive allocation effects on developmental plasticity via diet switching experiments. In year 1, diet differences experienced by two groups of females are associated with compositional differences in offspring, and potentially biological differences indicative of developmental plasticity. Offspring differences here are indicated by size differences. In year 2, the mothers' diets are switched and two outcomes are theoretically possible, A and B. In Switch A, the offspring differences observed in year 1 remain fixed and are thus not likely the result of nutritional effects. In Switch B, the offspring differences reverse coincident with the diet switch, and are likely due to nutritional effects [Color figure can be viewed at wileyonlinelibrary.com]

growing body of evidence also suggests that offspring quality differences, measured as offspring performance and survival, occur as a result of differences in maternal food availability during reproduction (Wang, Li, Zeng, Liang, & Du, 2017; Warner & Lovern, 2014). Differences in maternal food availability can even lead to changes in sex-allocation in lizard species with temperature-dependent sex determination, in a complex interaction between incubation temperature and yolk nutrition (Warner, Lovern, & Shine, 2007).

Even when they are not studied directly, maternal effects are almost always accounted for in studies on reptile offspring biology, independently of egg size, when they are included in statistical analyses as random "clutch effects" (Ashmore & Janzen, 2003; Hurlbert, 1984; Janzen, 1993b; Steen, Van Dyke, Jackson, & Hopkins, 2015; Van Dyke, Plummer, & Beaupre, 2011; Warner, Jorgensen, & Janzen, 2010; Webb, Brown, & Shine, 2001). We hypothesize that plastic differences in reproductive nutrient allocation underlie many of these maternal or clutch effects in reptiles. These differences arise as a result of genetic differences in nutrient allocation among females, as a result of nutritional differences females experience during reproductive allocation, or as an interaction between the two.

How can our hypothesis be tested? First, egg and/or offspring compositional differences must be established, either among or within females. If among-mother differences are consistent, regardless of their diets, then those differences may be genetically driven. In contrast, if those differences only occur as a result of dietary differences, then they are likely environmentally driven (Figure 2). Generalist species may be particularly useful for this type of analysis. For example, generalist turtle species like the Australian *Emydura macquarii* (Spencer, Thompson, & Hume, 1998) or American *Trachemys scripta* (Bjorndal, 1991) would be useful for manipulating diet and nutrition, such that some females experienced highly carnivorous diets, and others experienced highly herbivorous diets. Diet switches within females could then be used to separate the effects of reproductive allocation plasticity on offspring from other maternal effects (Figure 2). The primary constraint on these types of projects is the long period between reproductive bouts (~1 year), compared to other model species, which may not be suitable in the modern funding environment. In contrast, reptiles that are less generalist in their diet may be not tractable study species. For example, Australian skinks of the genera *Acritoscincus*, *Lampropholis*, *Niveoscincus*, and *Pseudemoia* are all largely insectivorous, and the fatty acid profiles of their yolk, at least, are very similar across species (Speake & Thompson, 1999; Speake et al., 2004).

Second, once compositional differences are established, their effects on the offspring must be investigated. Given the consistent appearance of maternal effects in many studies of offspring development, physiology, and performance, there are many potential metrics to be investigated. Studies of invertebrates have used a similar approach, and are good models to follow to test for general trends across taxa (Stachlschmidt & Adamo, 2015). Recent reptile studies are even beginning to report such maternal- and diet-driven effects on behavior and cognition (Munch et al., 2018). If these differences have fitness consequences, then they have exciting potential to further our understanding of how reptile recruitment is affected by environmental changes in their food supply. Furthermore, such knowledge would enhance our ability to manage species threatened by environmental change, via conservation physiology approaches (Wikelski & Cooke, 2006). Management options here include in situ approaches to improve nutritional availability for threatened species, as has been suggested for the greater sage grouse (Gregg & Crawford, 2009). Improving embryonic nutrition would also be useful for ex situ headstarting and breeding projects, where diets of mothers are manipulated in captivity to improve the potential for offspring recruitment after release. Such considerations have recently been suggested for headstarting of endangered ploughshare tortoises (Currylow et al., 2017).

5 | CONCLUSION

In our review, we have summarized knowledge about the mechanisms by which both lecithotrophic and placentotrophic reptiles allocate nutrients to their offspring, described how these lead to developmental plasticity, and argued for further study on their consequences for reptile ecology and conservation. Based on our review, there is clearly much work to be done to continue advancing our knowledge on the mechanisms underlying developmental plasticity in reptiles. Our knowledge on the genetic and physiological mechanisms of vitellogenesis and placentotrophy are largely based on inferences from other model species, and whether they are consistent in reptiles remains to be tested. Until this is done, the actual processes of vitellogenesis and placentotrophy remain incompletely understood. Likewise, the basic biochemistry of VTG has only been investigated in a handful of

vertebrate species, and the generality of these results across taxa, or even across nutritional status within taxa, is unknown. These are thus important gaps in our knowledge, which hamper our ability to understand how reproductive allocation leads to plasticity in offspring traits. For this field to progress, these gaps require extensive further study. Furthermore, given the current state of anthropogenic environmental change, we show the ultimate impacts of reproductive allocation on reptile phenotype and fitness are of paramount importance for further study. Both turtles and viviparous squamates have been identified as vertebrate taxa among the most at-risk of extinction (Sinervo et al., 2010; Turtle Conservation Coalition, 2011). Thus, our review ultimately shows that even basic-science studies that further understanding of reptile reproductive physiology may have eventual applications to improving conservation outcomes of these declining species.

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ORCID

James U. Van Dyke http://orcid.org/0000-0002-3933-111X

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