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Sex-specific survival to maturity and the evolution of environmental sex determination

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Four decades ago, it was proposed that environmental sex determination (ESD) evolves when individual fitness depends on the environment in a sex-specific fashion—a form of condition-dependent sex allocation. Many biological processes have been hypothesized to drive this sex asymmetry, yet a general explanation for the evolution of sex-determining mechanisms remains elusive. Here, we develop a mathematical model for a novel hypothesis of the evolution of ESD, and provide a first empirical test using data across turtles. ESD is favored when the sex-determining environment affects annual survival rates equivalently in males and females, and males and females mature at different ages. We compare this hypothesis to alternative hypotheses, and demonstrate how it captures a crucially different process. This maturation process arises naturally from common life histories and applies more broadly to condition-dependent sex allocation. Therefore, it has widespread implications for animal taxa. Across turtle species, ESD is associated with greater sex differences in the age at maturity compared to species without ESD, as predicted by our hypothesis. However, the effect is not statistically significant and will require expanded empirical investigation. Given variation among taxa in sex-specific age at maturity, our survival-to-maturity hypothesis may capture common selective forces on sex-determining mechanisms.

KEY WORDS: **Condition-dependent sex allocation, GSD, reptile, sex determination, theoretical model, TSD.**

Across vertebrates, the sex-determining mechanism is an evolutionarily labile trait, with numerous bidirectional transitions between genotypic sex determination (GSD) and environmental sex determination (ESD; Bull 1983; Janzen and Paukstis 1991a; Janzen and Krenz 2004; Ezaz et al. 2009; Pen et al. 2010; Holleley et al. 2015). GSD involves activation of the sex-determining pathway by genes located on sex chromosomes. Under temperature-dependent sex determination (TSD), a form of ESD, the sex-determining pathway is initiated by incubation temperature during embryonic development.

The discovery of TSD in the mid-20th century has compelled numerous hypotheses for its evolution and maintenance (Janzen and Paukstis 1988, 1991a; Shine 1999). The primary adaptive hypothesis (Charnov and Bull 1977) suggests that TSD is a form of condition-dependent sex allocation that is favored over GSD when incubation temperature influences the fitness of individuals in a manner that differs between the sexes (Fig. 1A). Extensive empirical research on TSD has focused on revealing how this sex-specific effect of incubation temperature arises biologically (Shine 1999; Valenzuela 2004). Males and females may differ in how incubation temperature influences phenotypes (i.e., a temperature-by-sex effect on phenotypes relevant for fitness; Shine 1999). Support for this type of effect is taxonomically scattered and often species-specific (Joanen and McNease 1989; Janzen 1995; Spencer and Janzen 2014). More commonly, temperature influences phenotype similarly for the

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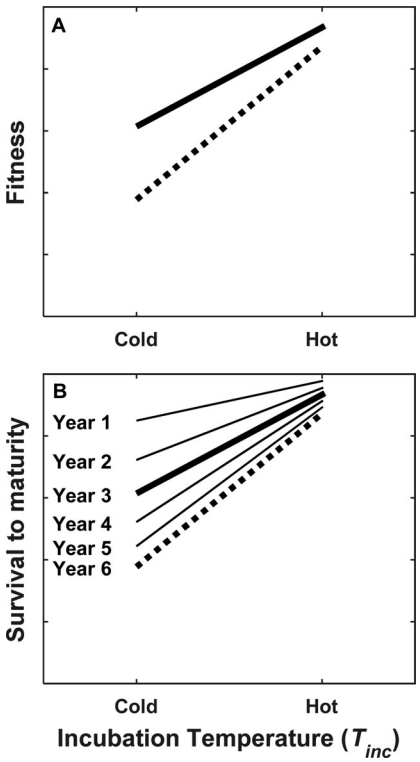

Figure 1. **Sex-differential fitness effects of incubation temperature select for TSD and arise when incubation temperature impacts annual survival and ages at maturity differs between the sexes. (A) hypothetical scenario illustrating selection for TSD: Incubation temperature more strongly influences the fitness of females (dotted) than that of males (solid). Note that we could set fitness in a given patch (e.g., cold patch) equal to 1 and compare relative fitness in other patches. In (B), small differences in annual survival between cold- and hot-incubated individuals are magnified across multiple years. Each line shows survival to the year indicated. If females reach sexual maturity later (year 6) than males (year 3), then temperature has a stronger effect on survival to maturity in females compared to males.**

two sexes (Deeming 2004; Warner and Shine 2005), and this phenotypic effect is assumed to influence fitness differentially for the two sexes (i.e., a phenotype-by-sex effect on fitness; Conover 1984; Warner and Shine 2008; Warner et al. 2009). For example, TSD may evolve in short-lived species when an effect of incubation temperature on body size impacts first-year reproductive success differently in males and females (Warner and Shine 2008; Warner et al. 2009; Pen et al. 2010). Still, explanations for the occurrence of TSD in any given taxon exhibit an ad hoc flavor, which may reflect a diversity of demographic processes producing selection for TSD.

Variation among species in sex-determining mechanism may also be explained via nonadaptive processes or evolutionary constraints imposed by underlying genetic and physiological architecture. For example, nonadaptive conversion from GSD to TSD may occur under climatic upheaval when extreme temperatures cause incidental sex reversal of the homogametic sex (e.g., ZZ females) and frequency-dependent selection drives to extinction the chromosome of the heterogametic sex (e.g., W chromosome; Grossen et al. 2010; Schwanz et al 2013; Holleley et al. 2015). Similarly, TSD can be maintained in long-lived animals with very low levels of selection under theoretical scenarios (Schwanz and Proulx 2008), raising the possibility that its persistence and taxonomic frequency in many reptile clades is due to nearly neutral processes (Janzen and Phillips 2006). Finally, sex chromosomes are often viewed as evolutionary traps that prevent transitions to other sex-determining mechanisms (Pokorná and Kratochvíl 2009; Gamble et al. 2015), although this is debated on empirical and theoretical grounds (Grossen et al. 2010; Schwanz et al. 2013; Bachtrog et al. 2014; Gamble et al. 2015; Holleley et al. 2015).

Although much conceptual focus for the adaptive evolution of TSD has been placed on the importance of temperature for reproductive success as an adult (Conover 1984,Warner et al. 2009), we argue that juvenile survival may be equally or more important in determining lifetime fitness (sensu Sæther et al 2013). Indeed, temperature need only influence survival to maturity differently for males and females to select for biased sex ratios under condition-dependent sex allocation (Schwanz et al. 2006). Here, we present a previously unappreciated biological process that provides a general explanation for the evolution of TSD. We demonstrate that two straightforward biological traits—temperature-dependence of annual juvenile survival and sex-differential age at maturity—jointly select for TSD, while either trait in isolation does not. In our novel "survival-to-maturity (SM)" hypothesis, sex differences in age at maturation can drive the evolution and maintenance of TSD even if incubation temperature affects the annual survival of juveniles in a similar manner in the two sexes. More broadly, our model can be applied to any condition-dependent sex-allocation strategy where an early-life environmental variable other than temperature (e.g., maternal provisioning) impacts annual survival rate postindependence.

Model

We present an optimality model for the evolution of TSD with sex-specific ages at maturity and temperature-dependent survival rates using a simple life history. We then demonstrate how these conclusions can be extended to more complex life histories and use this approach to distinguish our present hypothesis from alternative hypotheses presented in the literature.

SIMPLE LIFE HISTORY: NONOVERLAPPING GENERATIONS

Consider an organism that reaches sexual maturity after a specified number of years posthatching, and upon reaching sexual

Table 1. Three scenarios of incubation temperature and optimal primary sex ratios in a population with nonoverlapping generations.

Scenario derivatives		Parameter space regions	r_c	r_h
I. $\frac{dW}{dr_c} > 0$	$\frac{dW}{d\hat{r}_h} = 0$	$\frac{P}{1-P} < \beta^{\alpha_m}$	1	$\frac{1}{2} - \frac{1}{2}\frac{P}{(1-P)}\frac{1}{\beta^{\alpha m}}$ equation (4.a)
II. $\frac{dW}{dr_c} > 0$	$\frac{dW}{d\hat{r}_h} < 0$	$\beta^{\alpha_m} \leq \frac{P}{1-P} \leq \beta^{\alpha_f}$	1	0
III. $\frac{dW}{d\hat{r}_h} = 0$	$\frac{dW}{d\hat{r}_h} < 0$	$\beta^{\alpha_f} < \frac{P}{1-P}$	$\frac{1}{2} + \frac{1}{2}\frac{(1-P)}{P}\beta^{\alpha_f}$ equation (4.b)	0

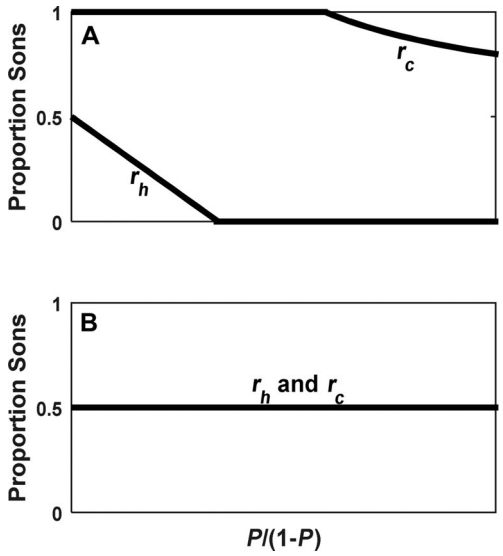

Figure 2. Offspring sex ratios when developing in cold and hot patches with and without TSD. (A) When the conditions of incubation temperature-dependent annual survival and sex difference in age at maturity are met, TSD is the ESS, here shown as biased primary sex ratios for hot and cold patches. (B) when age at maturity is equal for males and females, TSD is not favored (both patches produce 50% males).

maturity, breeds one time and dies. Eggs are incubated in one of two patch types—a "cold" patch or a "hot" patch. Patch temperature influences the survival of a juvenile, such that annual survival of a juvenile from a hot patch ($s\beta$) is higher than the annual survival of a juvenile from a cold patch (s, assuming $\beta > 1$; see Fig. 1B, "Year 1" line). Oviposition sites are limited and patch frequencies are fixed such that not all females can oviposit in the preferred hot patches. Survival to the age at maturity (S_{α_i}, where α_i is the age at maturity for i sex [male, female]) is the product of each annual survival probability. Thus, survival to maturity of an individual from a cold patch is $S_{\alpha_i,c} = s^{\alpha_i}$, and survival to maturity of an individual from a hot patch is $S_{\alpha_i,h} = (s\beta)^{\alpha_i}$.

We model the effect of different ages at maturation on the evolution of TSD using the Shaw and Mohler (1953) equation. We measure the fitness of a mutant mother whose offspring develop as $\hat{r}_c$proportion sons (when nesting in cold patches) and $\hat{r}_h$ (hot patches) in a population of wild-type mothers with r_c and r_h proportion sons. If offspring sex is also related to production costs (e.g., sex determination is linked to egg size; Radder et al. 2009), mother–offspring conflict over the sex ratio could arise and alter theoretical predictions (Kuijper and Pen 2014). However, for this model, we ignore these complications. A mother produces b number of offspring, with P probability of developing in a cold patch, and 1-P probability of developing in a hot patch. The mutant mother's fitness (W) is her relative representation of grandchildren in a wild-type population:

$$W = \left[\frac{P\hat{r}_c bs^{\alpha_m} + (1-P)\hat{r}_h bs^{\alpha_m}\beta^{\alpha_m}}{Pr_c bs^{\alpha_m} + (1-P)r_h bs^{\alpha_m}\beta^{\alpha_m}} + \frac{P(1-\hat{r}_c)bs^{\alpha_f} + (1-P)(1-\hat{r}_h)bs^{\alpha_f}\beta^{\alpha_f}}{P(1-r_c)bs^{\alpha_f} + (1-P)(1-r_h)bs^{\alpha_f}\beta^{\alpha_f}}\right]. \quad (1)$$

A wild-type organism has $W = 2$, so the sex ratios r_c and r_h are evolutionarily stable if no mutant values ($\hat{r}_c$,$\hat{r}_h$) yield $W > 2$. Thus, at the Evolutionarily Stable Strategy (ESS) conditions: (1) W is maximized with respect to $\hat{r}_c$ and $\hat{r}_h$, (2) $\hat{r}_c = r_c$ and $\hat{r}_h = r_h$, and (3) $W = 2$. We can find the ESS of equation (1) by considering the derivatives $dW/d\hat{r}_c$ and $dW/d\hat{r}_h$ and setting $\hat{r}_c = r_c$ and $\hat{r}_h = r_h$:

$$\frac{dW}{d\hat{r}_c} = \frac{P}{Pr_c + (1-P)r_h\beta^{\alpha_m}} + \frac{-P}{P(1-r_c) + (1-P)(1-r_h)\beta^{\alpha_f}} \text{ and} \quad (2.a)$$

$$\frac{dW}{d\hat{r}_h} = \frac{(1-P)\beta^{\alpha_m}}{Pr_c + (1-P)r_h\beta^{\alpha_m}} + \frac{-(1-P)\beta^{\alpha_f}}{P(1-r_c) + (1-P)(1-r_h)\beta^{\alpha_f}}. \quad (2.b)$$

If we define the population's contribution of males (M) and females (F) as $M = Pr_c + (1-P)r_h\beta^{\alpha_m}$ and $F = P(1-r_c) + (1-P)(1-r_h)\beta^{\alpha_f}$, we find

$$\frac{dW}{d\hat{r}_c} = \frac{P}{M} - \frac{P}{F} \text{ and } \frac{dW}{d\hat{r}_h} = \frac{(1-P)\beta^{\alpha_m}}{M} - \frac{(1-P)\beta^{\alpha_f}}{F}. \quad (3.a–b)$$

Analyzing equations (3.a and 3.b), we find that TSD evolves if two conditions are met: (1) age at maturity differs for males and females (e.g., $\alpha_m < \alpha_f$) and (2) an annual survival advantage accrues during immaturity to individuals developing in one thermal patch over the other (e.g., $\beta > 1$ in Fig. 1B). When these conditions are met, the two derivatives (eqs. 3.a and 3.b) cannot simultaneously equal zero. Thus, three TSD scenarios (Table 1; Fig. 2A) are possible based on the frequency of cold and hot patches. When cold patches are rare, cold patches always produce males ($r_c = 1$), whereas hot patches overproduce females ($r_h < 1/2$; scenario I). When hot patches are rare, hot patches always produce females ($r_h = 0$), whereas cold patches overproduce males ($r_c > 1/2$; scenario III). At intermediate frequency of patches, cold patches produce all males and hot patches produce all females (scenario II). In all cases, cold patches produce more males than warm patches.

The intuitive explanation for this outcome is that the difference in survival to maturity between cold- and hot-incubated individuals is amplified as the age at maturity increases (Fig. 3). This means that, if males and females mature at different ages, temperature exerts a sex-differential effect on juvenile survival (hence fitness). If age at maturity for males (α_m) is earlier than for females (α_f; $\alpha_m < \alpha_f$), then a hot incubation temperature has a stronger benefit for females than for males ($(S\beta)^{\alpha_f} / S^{\alpha_f} > (S\beta)^{\alpha_m} / S^{\alpha_m}$), and individuals are selected to develop as females at hot temperatures and males at cold temperatures (Fig. 2A). Indeed, the survival parameter itself (s) cancels out, so we can say more generally that TSD evolves whenever the survival advantage of patch type differs between the sexes (i.e., when $\beta^{\alpha_f} \neq \beta^{\alpha_m}$). The greater the incubation temperature effect on annual survival (β) and the greater the disparity in age at maturity, the stronger selection for TSD will be.

In contrast, when age at maturity is the same for both sexes ($\alpha_m = \alpha_f$), or if there is no effect of incubation temperature on annual survival ($\beta = 1$), $\beta^{\alpha_m} = \beta^{\alpha_f}$ and the derivatives can simultaneously equal zero. Solving for r_c and r_h, we find:

$$r_c = \frac{1}{2} + \frac{1}{2}\frac{(1-P)}{P}\beta^{\alpha_m}(1-2r_h) \tag{5.a}$$

and

$$r_h = \frac{1}{2} + \frac{1}{2}\frac{P}{(1-P)}\frac{1}{\beta^{\alpha_f}}(1-2r_c). \tag{5.b}$$

One solution is that, for all proportions of hot and cold patches, 50% males are produced in both hot and cold patches ($r_c = 1/2$ and $r_h = 1/2$; Fig. 2B). TSD will not be favored in this scenario.

GENERALIZED LIFE HISTORY: OVERLAPPING GENERATIONS

Our model can be generalized across more complex life histories and compared to alternative evolutionary models using R_0 as the fitness measure (R_0 = juvenile survival × fecundity × expected adult life span, see proofs in Charnov 1997; Schwanz et al. 2006). We modify our notation slightly to accommodate comparison with alternative hypotheses. Each life-history component can differ according to i sex (male, female) and j incubation temperature (hot, cold). If we allow overlapping generations, with adult annual survival $p_{i,j}$ (such that expected adult life span is given by $E_{i,j} = 1/(1 - p_{i,j})$), and annual fertility, $b_{i,j}$, then the relative fitness advantage of males in the hot patch compared to the cold patch is as follows:

$$\frac{R_{0,m,h}}{R_{0,m,c}} = \frac{{s_{m,h}}^{(\alpha_{m,h})} b_{m,h} / \left(1 - p_{m,h}\right)}{{s_{m,c}}^{(\alpha_{m,c})} b_{m,c} / (1 - p_{m,c})}. \tag{6.a}$$

Similarly, the relative fitness of females in the hot patch is as follows:

$$\frac{R_{0,f,h}}{R_{0,f,c}} = \frac{{s_{f,h}}^{(\alpha_{f,h})} b_{f,h} / \left(1 - p_{f,h}\right)}{{s_{f,c}}^{(\alpha_{f,c})} b_{f,c} / \left(1 - p_{f,c}\right)}. \tag{6.b}$$

These are general equations that can be leveraged to understand the fitness differentials associated with any specific sex × temperature life-history effect, assuming stable age distribution.

SM hypothesis

Under the assumptions of our SM hypothesis (Table 2), equations [6.a and 6.b] can be rewritten and simplified as:

$$\frac{R_{0,m,h}}{R_{0,m,c}} = \left(\frac{s_h}{s_c}\right)^{\alpha_m} \tag{7.a}$$

and

$$\frac{R_{0,f,h}}{R_{0,f,c}} = \left(\frac{s_h}{s_c}\right)^{\alpha_f}, \tag{7.b}$$

which is equivalent to saying that the relative fitness advantage of males in the hot patch compared to the cold patch is β^{α_m}, and the relative fitness of females in the hot patch is β^{α_f}.

Thus, examining a generalized life history with R_0 as the fitness measure leads to the same formulation of relative male and female fitness as the simple life history. As proven above, TSD evolves when $\beta^{\alpha_f} \neq \beta^{\alpha_m}$ or when (1) $\frac{s_h}{s_c} \neq 1$ and (2) $\alpha_f \neq \alpha_m$ (assumptions SM-a and SM-b, Table 2). The difference in sex-specific fitness depends entirely on the quantitative effect of incubation temperature on juvenile survival rate (and that this effect persists until both sexes have matured), and the quantitative difference in age at maturity between males and females (Fig. 4A). Specifically, as the benefit of hot incubation temperatures for juvenile survival rate increases (lines in Fig. 4A), it acts synergistically with difference in age at maturity between the

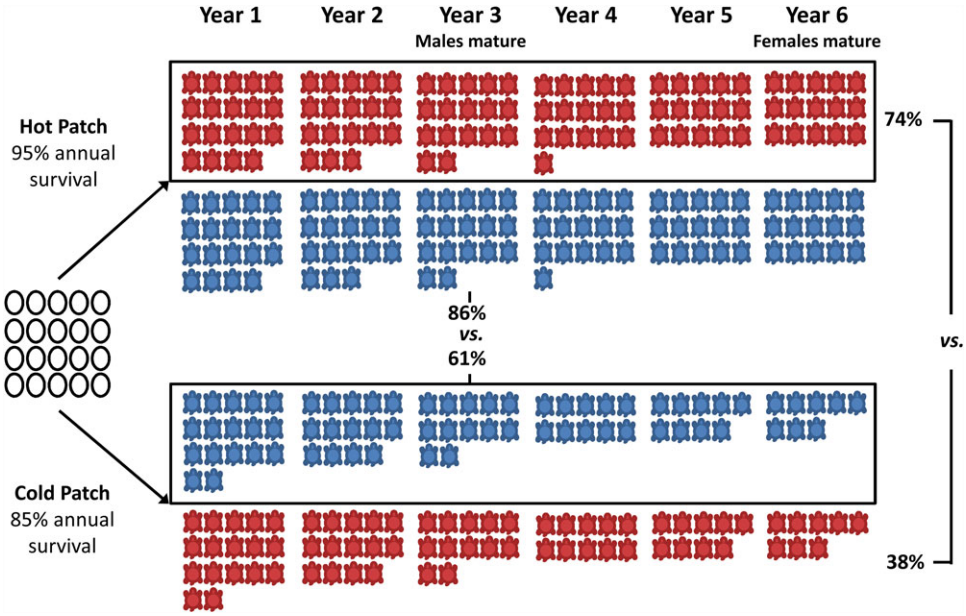

Figure 3. Schematic of "SM" hypothesis for the evolution of TSD. TSD evolves when incubation temperature of embryos impacts annual survival for the rest of the individuals' lives and the two sexes mature at different ages. It is assumed that cold patches are unavoidable. Survival of a clutch of eggs is shown whether incubated in hot or cold patches and whether developing as all males (blue) or all females (red). Annual survival is assessed each year, here shown for the first six years of life. For the sex that matures later (here, females mature at six years), survival to the age at maturity is much higher when incubated at hot temperature than cold temperatures. In contrast, survival to the age at maturity is not as strongly impacted by incubation temperature for the sex that matures earlier (here, males mature at three years). The optimal strategy (black boxes) is to develop as a female at warm temperatures to benefit from the stronger SM advantage, and develop as a male at cold temperatures to accept the relatively small cost of SM while benefitting from frequency-dependent selection on sex.

sexes (x-axis in Fig. 4A) to produce strong selection for TSD. When females mature later than males (right side of Fig. 4A), their fitness benefits from hot incubation temperatures exceed those of males, and selection favors the development of females at hot temperatures and males at cold temperatures. The opposite pattern of TSD is favored when males mature later than females (left side of Fig. 4A).

For the remaining two hypotheses, we posit more broadly that TSD is favored over GSD whenever the relative fitness of males from hot and cold patches does not equal the relative fitness of females from hot and cold patches (assuming stable-age distribution):

$$\frac{R_{0,m,h}}{R_{0,m,c}} \neq \frac{R_{0,f,h}}{R_{0,f,c}}. \tag{8}$$

Temperature-dependent fertility (TF) hypothesis

An early hypothesis for the evolution of TSD proposed that the sex × temperature effect on fitness arises when (1) incubation temperature affects postincubation body size or growth (e.g., via seasonal time of hatching) and (2) body size influences adult fertility more in one sex (e.g., females) compared to the other (Conover 1984). TSD should evolve such that the sex that gains the most in fertility from larger adult body size develops at the incubation temperature associated with greater adult body size (also known as the "sexual dimorphism hypothesis;" Janzen and Paukstis 1991b). These effects explain the occurrence of TSD in silverside fish (Conover 1984), but has not received support across turtles in general (Janzen and Paukstis 1991b).

Applying the parameter assumptions for this model (Table 2) to equations [6.a and 6.b] and simplifying leads to:

$$\frac{R_{0,m,h}}{R_{0,m,c}} = \frac{b_{m,h}}{b_{m,c}} \tag{9.a}$$

and

$$\frac{R_{0,m,h}}{R_{0,m,c}} = \frac{b_{f,h}}{b_{f,c}}. \tag{9.b}$$

A formal proof for a similar condition-dependent sex allocation model can be found in Schwanz et al. (2006). As long as the fertility of one sex is more strongly influenced by incubation temperature than is the fertility of the other sex (assumption TF-a, Table 2), TSD is favored (Fig. 4B, where fitness ratio ≠ 1). For example, when male fertility is not influenced by incubation temperature (Fig. 4B, top line), the strength of selection for TSD increases as the benefit of hot temperatures for female fertility

Table 2. Comparison of the hypothesis developed in this article with two popular hypotheses for the adaptive evolution of TSD.

Hypothesis	Assumptions	Parameters
SM[1] TSD is favored when annual juvenile survival rate depends on incubation temperature (T_{inc}) and the sexes mature at different ages	(SM-a) Annual juvenile survival ($s_{i,j}$) differs for T_{inc}, but not sexes	(a) $s_{f,j} = s_{m,j} = s_j \;\; s_h \neq s_c$
	(SM-b) Age at maturity ($\alpha_{i,j}$) differs for sex, but not T_{inc}	(b) $\alpha_{i,h} = \alpha_{i,c} = \alpha_i \;\; \alpha_f \neq \alpha_m$
	(SM-c) Fertility ($b_{i,j}$) and adult survival ($p_{i,j}$) are not influenced by T_{inc} or sex	(c) All $p_{i,j} = p$, all $b_{i,j} = b$
TF[2] TSD is favored when incubation temperature influences fertility differently for males and females	(TF-a) Adult fertility may vary by T_{inc} and sex, with T_{inc} effects	(a) $\frac{b_{f,h}}{b_{f,c}} \neq \frac{b_{m,h}}{b_{m,c}}$
	(TF-b) Annual juvenile mortality, age at maturity, and adult life span are not influenced by T_{inc} or sex	(b) all $s_{i,j} = s$, all $\alpha_{i,j} = \alpha$, all $p_{i,j} = p$
TM[3] TSD is favored when incubation temperature influences effective age at maturity differently for males and females	(TM-a) Age at maturity may vary by T_{inc} and sex, with T_{inc} effects differing between sexes	(a) $\alpha_{f,h} - \alpha_{f,c} \neq \alpha_{m,h} - \alpha_{m,c}$
	(TM-b) Total life span may be finite such that mean survival rate and adult life span are reduced when maturity is delayed	(b) $\frac{(1-p_{f,c})}{(1-p_{f,h})} \neq \frac{(1-p_{m,c})}{(1-p_{m,h})}$
	(TM-c) Annual juvenile mortality and adult fertility not influenced by T_{inc} or sex	(c) All $s_{i,j} = s$, all $b_{i,j} = b$

The comparison demonstrates how the hypotheses differ in the mechanism by which incubation temperature has a differential effect on male and female relative lifetime fitness. All parameters are for *i* sex (male, female) and *j* incubation temperature (hot, cold).

[1]Hypothesis presented herein.

[2]Conover (1984) and Janzen and Paukstis (1991bb).

[3]Daan et al. (1996), Pen et al. (1999), Warner et al. (2009), and Pen et al. (2010).

increases (x-axis). There is no sex-differential fitness advantage, and no selection for TSD, when incubation temperatures influence fertility the same for the two sexes (Fig. 4B, when fitness ratio = 1 moving down the lines and across the x-axis). Note that age at maturity and survival to maturity do not feature in the solution, but that incubation temperature must have a sex-specific influence on fertility regardless of how many years after hatching individuals mature.

Temperature-dependent maturation (TM) hypothesis

A recent hypothesis that we will call the TM hypothesis (Warner et al. 2009) shares features with the SM and TF hypotheses, yet has distinct selective forces. As in the TF hypothesis, the TM hypothesis proposes that incubation temperature influences postincubation body size equally for both sexes—warm-incubated offspring hatch early and have a long season to grow, whereas cool-incubated offspring hatch late and have little opportunity to grow before winter. Age at maturity also features in the TM hypothesis as an important biological mechanism—size impacts the age at maturity in one sex (females) more than the other (males). Age at maturity is taken to be the age at first successful breeding, regardless of gonadal development.

There is support for this hypothesis in dragon lizards (Warner and Shine 2008; Warner et al. 2009). Specifically, TSD is more often associated with species where all males are likely to be too small to secure matings at one year of age (due to strong, size-based intrasexual competition), and early-hatched females, but not late-hatched females, are large enough to produce eggs at one year of age.

The TM hypothesis draws upon models of seasonal sex ratios in birds (Daan et al. 1996; Pen et al. 1999). An important feature of seasonal sex ratio models is whether one assumes that average annual adult survival ($p_{i,j}$) and the resulting adult reproductive life span are independent of age at maturity (Pen et al. 1999) or whether delaying age at maturity leads to a decrease in adult reproductive life span (Warner et al. 2009). The former is a typical theoretical simplification of invariant adult survival rate. The latter may occur when senescence in viability occurs and there is a finite total life span, so that delaying maturity means losing a year of reproduction from a finite number of years. At the extreme (low

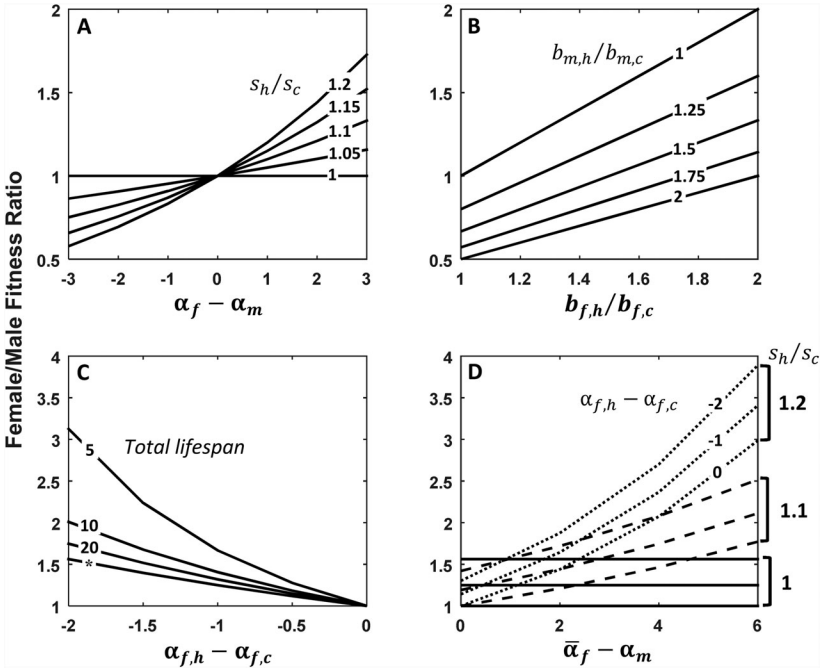

Figure 4. Selection differentials under three hypotheses for the evolution of TSD. Each panel shows the ratio of the temperature advantage for females (eq. (8), right side) to the temperature advantage for males (eq. (8), left side) on the *y*-axis (note variation in scale across figure panels). A value of 1 indicates no sex-specific fitness as a function of temperature, thus no selection for TSD. Values greater than 1 indicate selection for females to develop preferentially at warm temperatures, whereas values less than 1 indicate the opposite selection. (A) According to the SM hypothesis developed in this article, selection depends on the difference in ages at maturity for males and females (*x*-axis) and the benefit of incubating at warm temperatures for juvenile survival rate (*lines*, s_h/s_c; $\alpha_m = 4$ for figure). (B) Under the TF hypothesis, selection depends on the benefit in fertility females receive from incubating at warm temperatures (*x*-axis) compared to the corresponding benefit males receive (*lines*, $b_{m,h}/b_{m,c}$). (C) Under the TM hypothesis, selection depends on whether incubation temperature has a greater effect on age at maturation for females (*x*-axis) compared to males ($\alpha_{m,h} - \alpha_{m,c} = 1$), with the additional possibility that total life span is finite and is a linear function of age at maturation (*lines*, total life span in years, *adult survival is unaffected by age at maturity; $s = 0.8$; $\alpha_{f,h} = 1$). (D) The SM and TM hypotheses may simultaneously apply to a population. Increasing the difference in age at maturity between males and females has a strong effect (*x*-axis) when incubation temperature has a large influence on survival rates (*line groups*: s_h/s_c). The difference in female age at maturity due to incubation temperature (*within group lines*, $\alpha_{f,h} - \alpha_{f,c}$) has a fairly constant effect of increasing the female:male fitness ratio ($s_c = 0.8$; $s_h = 0.8, 0.88, 0.96$; $\alpha_m = 3$; $\bar{\alpha}_f = 3, 5, 7, 9$, with $\alpha_{f,h}$ and $\alpha_{f,c}$ distributed evenly around the mean).

overlap in generations), seasonal sex ratios can disrupt the stable age distribution required for R_0 to be a viable fitness measure (Werren and Charnov 1978), so a formal model is required to validate the following formulation. We specify age at maturity (and adult life span) varying according to sex and incubation temperature (Table 2; eqs. 6.a and 6.b):

$$\frac{R_{0,m,h}}{R_{0,m,c}} = s^{(\alpha_{m,h}-\alpha_{m,c})} \left(\frac{(1-p_{m,c})}{(1-p_{m,h})} \right), \qquad (10.a)$$

$$\frac{R_{0,f,h}}{R_{0,f,c}} = s^{(\alpha_{f,h}-\alpha_{f,c})} \left(\frac{(1-p_{f,c})}{(1-p_{f,h})} \right), \qquad (10.b)$$

where $p_{i,j}$ is a function of $\alpha_{i,j}$. When $p_{i,j}$ is not related to $\alpha_{i,j}$, the $p_{i,j}$ cancel out of both equations and TSD is favored when the differences in age at maturity between incubation temperatures are not equal for the sexes ($\alpha_{f,h} - \alpha_{f,c} \neq \alpha_{m,h} - \alpha_{m,c}$; assumption TM-a, Table 2). For example, if all males mature at the same age (the ratio of fitness of males from hot and cold temperatures is 1), but hot-incubated females mature one year earlier than cold-incubated females (the female fitness ratio is $1/s$, a value greater than 1), then females gain more in lifetime fitness from hot incubation compared to cold incubation than do males (Fig. 4C, bottom line). If these are the only effects, then the driving fitness impact of incubation temperature is through its influence on juvenile survival.

If, in addition, maturing earlier (smaller $\alpha_{i,j}$) allows an extra year of reproduction within an individual's lifetime (larger mean $p_{i,j}$), then the impact of incubation temperature on relative fitness is amplified (Fig. 4C, upper lines). As total life span becomes

short (e.g., five years; Fig. 4C, top line), sex differences in TM lead to a strong fitness differential between females and males. In this conceptualization (e.g., Warner et al. 2009), the driving fitness effects include juvenile survival and adult life span. Note that survival rates are invariant between the sexes and incubation temperatures, and that it is the *difference* in ages at maturity in each sex that matter (absolute age at maturity matters if $p_{i,j}$ is a function of $\alpha_{i,j}$).

Combined maturity

It is highly plausible that the effect of incubation temperature on size and/or quality simultaneously impacts annual survival rates as well as age- and sex-specific reproductive success, manifest as an earlier age at maturity and higher adult fertility for one or both sexes. These combined effects can be examined numerically using equations (6.a and 6.b). We briefly consider the interactive effects of the two hypotheses related to age at maturity (SM and TM). Specifically, incubation temperature impacts juvenile survival rate, the sexes mature at different mean ages, and incubation temperature influences the age at maturity for females but not males. Equations (6.a and 6.b) are rewritten as:

$$\frac{R_{0,m,h}}{R_{0,m,c}} = \left(\frac{s_h}{s_c}\right)^{\alpha_m} \tag{11.a}$$

and

$$\frac{R_{0,f,h}}{R_{0,f,c}} = \frac{{s_h}^{(\alpha_{f,h})}}{{s_c}^{(\alpha_{f,c})}} \tag{11.b}$$

As shown for the SM hypothesis (Fig. 4A), the advantage of hot incubation temperatures for survival rate interacts strongly with the sex difference in age at maturity (Fig. 4D; compare line style groups). If, in addition, hot-incubated females mature earlier than cold-incubated females (lines within line groups), there is an increase in the female:male fitness differential; however, within-sex difference in maturity interacts very little with between-sex difference in average age at maturity. Interestingly, when mean age at maturity is similar for the two sexes (e.g., x-axis $= 0$), the survival rate advantages and female maturation advantages interact. When hot-incubated females mature earlier than cold-incubated females (e.g., by two years—the top line of each group), increasing the juvenile survival advantage (solid vs. dashed vs. dotted lines) decreases selection for TSD.

EMPIRICAL TEST OF THE SM HYPOTHESIS

Testing any of these hypotheses or trying to distinguish among them is exceptionally challenging. Such analyses would best be accomplished by directly testing the assumptions for each hypothesis listed in Table 2 or following the experimental protocol presented in Figure 5. However, gathering the necessary life-history details for a single species is not trivial, and the challenges of gathering such information for multiple species to acquire generality are extraordinary. Because of these limitations, we examine the likelihood of the "SM" hypothesis as a general explanation for variation in sex-determining mechanisms at a broad scale, by examining interspecific variation in age at maturity and using comparative methods to test whether sex differences in age at maturity in turtle species are associated with sex-determining mechanism.

We compiled data for sex-determining mechanisms (TSD or GSD), sex-specific ages at maturity, and body sizes for turtles from the primary literature (Table S1). Differences in the age at maturity between males and females may be causally linked to average body size and sexual size dimorphism (Shine 1990), which themselves could provide alternative biological processes selecting for TSD (Lovich et al. 2014). Although direction of size dimorphism was previously shown not to be associated with sex-determining mechanisms (Janzen and Pauksits 1991b), we used our updated data to examine whether body size variables predict sex-determining mechanisms.

Concomitantly examining these relationships provides an avenue for assessing the general applicability of the three hypotheses presented in this study. Different ages at maturity for males and females are (1) a necessary driver in the SM hypothesis, (2) not addressed in the TF hypothesis, and (3) an outcome of the TM hypothesis, although reduced if one sex matures across multiple ages. Sex differences in adult body size are (1) a potential outcome of different ages at maturity in the SM hypothesis, (2) a driver in the TF hypothesis, and (3) a potential outcome in the TM hypothesis that decreases in likelihood as variation in age at maturity increases and as adult life span increases, overwhelming size differences at maturity. Thus, if any of the three hypotheses provides a general explanation for sex-determining mechanisms in turtles, we expect significant associations with differences in age at maturity or body size between the two sexes. To find broad support for the SM hypothesis, age at maturity must differ between the sexes more in TSD species than in GSD species.

We created data quality filters for the age at maturity data. Because sexual maturity varies among populations of a given species, sex-specific estimates were used only when presented for the same geographic location. When data on both male and female sexual maturity were available for multiple populations, we chose data from the population closest to the center of the species' range under the assumption that phenotypes at the center of the range most closely match optimal phenotypes (García-Ramos and Kirkpatrick 1997).

Phylogenetic generalized least squares models that account for species interdependence were used to determine whether sex-determining mechanisms are associated with: (1) differences in the age at first reproduction between males and females (Diff_maturity = female age at maturity – male age at matu-

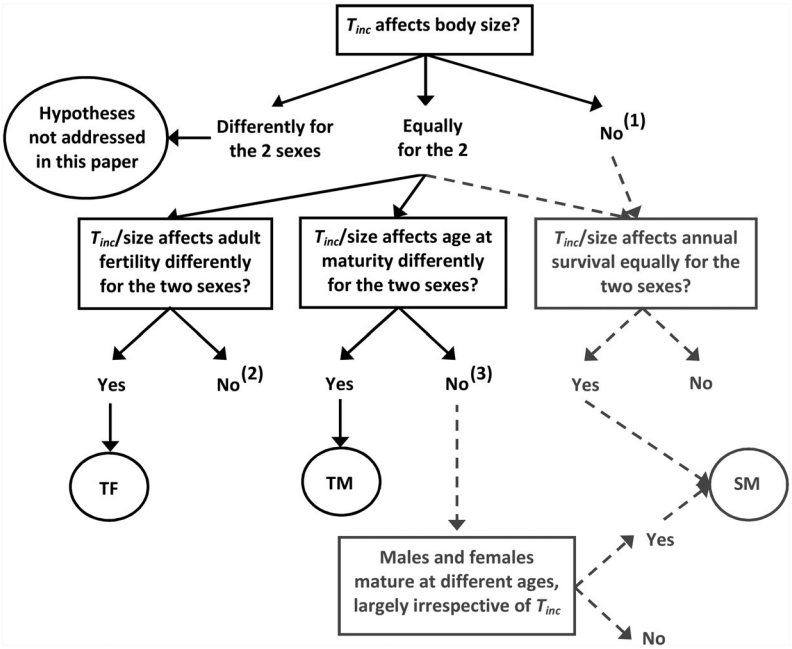

Figure 5. **The importance of distinguishing the SM hypothesis from the TF and TM hypotheses is demonstrated with hypothetical experimental results. A researcher is studying a species with TSD and wants to test the adaptive significance of this sex-determining mechanism for the species, focusing on sex-differential fitness effects of incubation temperature (Charnov and Bull 1977). For this hypothetical scenario, we imagine the researcher is able to generate males and females across a range of incubation temperatures and starts by measuring body size. If the researcher is not considering the SM hypothesis (conceptual framework with solid arrows), then results in line with point (1) or points (2) and (3) would lead to the conclusion that there is no support for adaptive evolution of TSD. However, these results are consistent with the SM hypothesis (dashed), and additional data allow further testing the model.**

rity); (2) differences in body size between males and females (i.e., sexual size dimorphism; Diff_size = female size – male size); and/or (3) mean body size inclusive of both females and males (Mean_size). In addition, we tested whether Diff_maturity is predicted by Diff_size and Mean_size.

First, nonphylogenetic generalized least squares models were generated and their residuals were tested for Brownian motion, Ornstein–Uhlenbeck, and white noise models of evolution on pruned ultrametric trees with branch lengths derived from the most recent comprehensive molecular phylogeny of cheloni-ans(Guillon et al. 2012). Akaike information criteria indicated that the Ornstein–Uhlenbeck model of evolution best described covariance structures. Thus, before running phylogenetic generalized least squares models, α was adjusted accordingly. Analyses were conducted using the Ape, Geiger, nlme, and phytools packages of the R programming language (R Development Core Team 2014).

In all cases where the sexes differed in age at maturity in our dataset, males matured earlier than females. There was substantial variation among species in sex-specific age at maturity (females matured between zero and eight years older than males; Table S1), providing ample opportunity for survival effects to accumulate differently between males and females in some species, but not in others. As predicted by the SM hypothesis, the mean difference in age at maturity between males and females was approximately one year greater for species with TSD compared to species with GSD (Fig. S1B). In addition, the proportion of species with an age difference more than and equal to two years was higher in TSD representatives (86%) than in GSD representatives (50%; Fig. S1C; Table S1). However, the difference in age at maturity was not supported with statistical significance in the phylogenetic generalized least squares models, likely due to the low number of species with GSD in the comparative test (Table 3; Fig. S1).

As expected from previous studies, sex-determining mechanisms did not covary with either sexual size dimorphism or mean body size (Table 3; Figs. S2 and S3), although sexual maturity was related to both factors (Table 3). Thus, we found little support that any single hypotheses described herein provides a general explanation for patterns of sex-determining mechanism across turtle taxa.

Discussion

Sex-determining mechanisms have evolved independently numerous times in animals. The most compelling adaptive hypothesis for the evolution of TSD was proposed nearly four decades ago (Charnov and Bull 1977). These authors suggested that TSD is a type of condition-dependent sex allocation that is favored

Table 3. Results of phylogenetic generalized least squares models on Diff_maturity ($N = 28$), Diff_size ($N = 55$), and Mean_size ($N = 55$); SDM = sex-determining mechanism. * = model residuals with statistically significant ($P < 0.05$) phylogenetic signal (Bloomberg's K).

Model	Source	$_{num}$DF	$_{den}$DF	F	P
Diff_maturity ($K = 0.057$)	Intercept	1	27	66.09	<0.0001
	SDM	1	27	0.88	0.3571
Diff_maturity ($K = 0.052$)	Intercept	1	26	102.53	<0.0001
	Diff_size	1	26	15.21	0.0006
	Mean_size	1	26	0.05	0.8329
Diff_size ($^{*}K = 0.031$)	Intercept	1	53	71.27	<0.0001
	SDM	1	53	0.15	0.7009
Mean_size ($^{*}K = 0.026$)	Intercept	1	53	72.50	<0.0001
	SDM	1	53	1.48	0.2287

over GSD when fitness depends on incubation temperature in a sex-specific manner. Testing the hypothesis, however, has been hampered by the difficulty of measuring lifetime fitness and the experimental challenge of decoupling temperature and sex.

Empirical support for adaptive hypotheses of TSD has been contentious, being both scattered taxonomically and species-specific (Conover 1984; Janzen 1995; Warner and Shine 2008). Moreover, nonadaptive or nearly neutral processes have also been invoked to explain the evolution and maintenance of TSD (Janzen and Phillips 2006; Grossen et al. 2010; Holleley et al. 2015). Here, we demonstrate mathematically that a simple biological process related to age at first reproduction provides a broadly inclusive explanation for the adaptive evolution of TSD across all taxa. In this "SM" hypothesis (Fig. 3), incubation temperature affects annual juvenile survival. The annual effect is multiplied across successive years of immaturity, so that survival from hatching to a given year depends more strongly on temperature as more years pass. When males and females mature at different ages, the combined impact of incubation temperature on survival to maturity differs between the two sexes, thus establishing a (temperature-dependent) phenotype × sex interaction on fitness. If females mature later than males and annual survival is higher for warmer natural incubation temperatures, then we would expect females to develop at warm temperatures and males to develop at cold temperatures. Alternatively, if one of those patterns is reversed, we would predict the opposite pattern of TSD.

The "SM" hypothesis has potential to explain the occurrence of TSD across taxa. In reptiles, incubation temperature has diverse and nearly ubiquitous impacts on hatchling phenotypes relevant for survival (e.g., morphology, locomotion, and antipredator behavior), as well as survival itself (Janzen 1995; Deeming 2004; Warner and Shine 2005). Perhaps counterintuitively, these thermal effects need not differ for males and females for our hypothesis to hold (Rhen and Lang 1995). Crucially, for our hypothesis to be correct, the temperature effect on survival must persist during the ages when one sex has matured, but the other has not. Although this age may be many years after hatching for long-lived species, the assumption is no greater than those made for many alternative hypotheses. For example, the additional two hypotheses contrasted in this article rely on size differences established by incubation temperature persisting up to and beyond maturity. Such size differences are at least as likely to influence annual survival as they are to influence maturation or fertility. Unfortunately, little is known regarding whether the phenotypic and survival effects persist beyond the limited temporal scale of most experiments (one month to one year). In particular, post-hatching survival is a fruitful target for increased empirical research to assess the general relevance of the hypothesis (Janzen 1995; Andrews et al. 2000; Booth et al. 2004; Freedberg et al. 2004; Hare et al. 2004).

If temperature effects on annual survival are near universal for ectothermic animals, then variation in sex-determining mechanisms would be explained largely by the extent of sex differences in age at maturity. GSD would be expected when males and females have similar ages at maturity, whereas TSD would be expected when males and females have greater differences in ages at maturity. We provided a first test of this prediction with a comparative analysis across chelonian taxa, and found little support that our hypothesis provides a universal explanation for patterns of sex-determining mechanism in this clade. Species with TSD had greater differences in age at maturity between males and females than did species with GSD, as predicted, but there are currently too few taxa with GSD and known ages at maturity for both sexes to provide a robust statistical test.

With so few data in the GSD group, the results may also be influenced by a few taxa (Maddison and Fitzjohn 2015). Increasing the number of species for which sex-specific age at maturity is known would greatly improve our ability to test the hypotheses, particularly if nonchelonian (e.g., lizard) taxa could be included in the comparative analysis.

We additionally examined the role of sexual size dimorphism and associated size-related fitness in selecting for TSD (Janzen and Paukstis 1988 and here), but found even less support for these factors being related to sex-determining mechanism. These results also hold when sea turtles are excluded (Fig. S3, Table S2).

Although none of the hypotheses seem to hold broad explanatory power across turtles, each hypothesis may still capture important selective forces within individual species. TM may create selection in one species, whereas SM creates selection in another, or the two may operate together. Identifying the selection acting within any one species requires focused experimental work (e.g., Table 2, Fig. 5). Importantly, we have assumed that incubation temperature influences annual survival or body size equivalently across turtle taxa. These assumptions need to be addressed. The data we have compiled point to obvious target taxa whose sex-determining mechanisms seem supportive of the SM hypothesis (e.g., *Glyptemys* vs. other emydid species) or incongruous (e.g., the GSD species *Apalone mutica*; the TSD species *Kinosternon flavescens*). Potentially, additional variation can be explained by incorporating data on natural nest temperatures and the comparative strength of phenotypic effects of temperature. Collecting these additional data would further allow us to examine whether some of the phylogenetic signal may reflect constraints on transitions between sex-determining mechanisms and nonadaptive forces acting on the evolution of TSD and GSD (Schwanz and Proulx 2008; Pokorná and Kratochvíl 2009; Grossen et al. 2010; Gamble et al. 2015; Holleley et al. 2015).

The distinction between a role for size-related traits or survival to maturity per se is important in comparing alternative hypotheses for the adaptive evolution of TSD. We considered two common, traditional hypotheses that are linked critically to differences in body size and its impact on reproductive potential. In the "TF" hypothesis, sex-specific differences in the importance of body size for adult fertility provide the sex-by-temperature fitness effect. In the TM hypothesis, body size influences the ability to breed for the first time (i.e., age at maturity) more in one sex than the other. In contrast, the "SM" hypothesis does not rely on sexual dimorphism, and any scatter in the link between size and age at maturity is inconsequential. It is difficult, however, to use these data to disprove the TM hypothesis because the variable maturation age in one sex should generate noise in the size and age at maturity data.

Viewing the "SM" hypothesis as a novel selective process is not a trivial, theoretical exercise. Its distinctness is of vital importance for empiricists, as demonstrated using a hypothetical empirical study (Fig. 5). Standard avenues of inquiry into the adaptive nature of TSD (e.g., effects of incubation temperature on growth, maturation, and fertility) could lead a researcher to erroneously conclude that TSD has no adaptive relevance if negative results arise. Only when the results are considered in the context of our "SM" hypothesis is an additional sex-specific effect of incubation temperature apparent (see Fig. 5). Although other processes operating in conjunction may be important, our "SM" hypothesis provides a sufficient and general explanation for the otherwise persistent conundrum of TSD in long-lived vertebrates.

Moreover, the "SM" hypothesis adds a novel dimension and important distinction for empirical research on condition-dependent sex allocation in taxa with GSD. Our hypothesis would apply when early-life conditions (e.g., maternal condition) influence the postindependence juvenile survival rate differently for males and females and the two sexes mature at different mean ages. The hypothesis contrasts with the common view of the Trivers–Willard hypothesis (Trivers and Willard 1973), which focuses on the sex-specific impact of condition for reproductive success as an adult (but see the broader, "reproductive value" view, Leimar 1996).

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LITERATURE CITED

Andrews, R. M., T. Mathies, and D. A. Warner. 2000. Effect of incubation temperature on morphology, growth, and survival of juvenile sceloporus undulates. Herpetol. Monogr. 14:420–431.

Bachtrog, D., J. E. Mank, C. L. Peichel, M. Kirkpatrick, S. P. Otto, T.-L. Ashman, M. W. Hahn, J. Kitano, I. Mayrose, R. Ming et al. 2014. Sex determination: why so many ways of doing it? PLOS Biol. 12:e1001899.

Booth, D. T., E. Burgess, J. McCosker, and J. M. Lanyon. 2004. The influence of incubation temperature on post-hatching fitness characteristics of turtles. Int. Congr. Ser. 1275:226–233.

Bull, J. J. 1983. Evolution of sex determining mechanisms. Benjamin/Cummings, Menlo Park, CA.

Charnov, E. L. 1997. Trade-off-invariant rules for evolutionarily stable life histories. Nature 387:393–394.

Charnov, E. L., and J. Bull. 1977. When is sex environmentally determined? Nature 266:828–830.

Conover, D. O. 1984. Adaptive significance of temperature-dependent sex determination in a fish. Am. Nat. 123:297–313.

Daan, S., C. Dijkstra, and F. J. Weissing. 1996. An evolutionary explanation for seasonal trends in avian sex ratios. Behav. Ecol. 7:426–430.

Deeming, D. C. 2004. Post-hatching phenotype effects of incubation in reptiles. Pp. 229–251 *in* D. C. Deeming, ed. Reptilian incubation: environment, evolution and behavior. Nottingham Univ. Press, Nottingham.

Ezaz, T., S. D. Sarre, D. O'Meally, J. A. Marshall Graves, and A. Georges. 2009. Sex chromosome evolution in lizards: independent origins and rapid transitions. Cytogen. Genome Res. 127:249–260.

Freedberg, S., A. L. Stumpf, M. A. Ewert, and C. E. Nelson. 2004. Developmental environment has long-lasting effects on behavioral performance in two turtles with environmental sex determination. Evol. Ecol. Res. 6:739–747.

Gamble, T., J. Coryell, T. Ezaz, J. Lynch, D. P. Scantlebury, and D. Zarkower. 2015. Restriction site-associated DNA sequencing (RAD-Seq) reveals an extraordinary number of transitions among gecko sex-determining systems. Mol. Biol. Evol. 32:1296–1309.

García-Ramos, G., and M. Kirkpatrick. 1997. Genetic models of adaptation and gene flow in peripheral populations. Evolution 51:21–28.

Grossen, C., S. Neuenschwander, and N. Perrin. 2010. Temperature-dependent turnovers I sex-determination mechanisms: a quantitative model. Evolution 65:64–78.

Guillon, J.-M., L. Guéry, V. Hulin, and M. Girondot. 2012. A large phylogeny of turtles (Testudines) using molecular data. Contrib. Zool. 81:147–158.

Halámková, L., J. A. Schulte II, and T. A. Langen. 2013. Patterns of sexual size dimorphism in Chelonia. Biol. J. Linn. Soc. 108:396–413.

Hare, K. M., C. G. Longson, S. Pledger, and C. H. Daugherty. 2004. Size, growth, and survival are reduced at cool incubation temperatures in the temperate lizard *Oligosoma suteri* (Lacertilia: Scincidae). Copeia 2004:383–390.

Holleley, C. E., D. O'Meally, S. D. Sarre, J. A. Marshall Graves, T. Ezaz, K. Matsubara, B. Azad, X. Zhang, and A. Georges. 2015. Sex reversal triggers the rapid transition from genetic to temperature-dependent sex. Nature 523:79–82.

Janzen, F. J. 1995. Experimental evidence for the evolutionary significance of temperature-dependent sex determination. Evolution 49:864–873.

Janzen, F. J., and J. G. Krenz. 2004. Phylogenetics: which was first, TSD or GSD? Pp. 121–130 *in* N. Valenzuela and V. Lance, eds. Temperature-dependent sex determination in vertebrates. Smithsonian Institute Press, Washington, DC.

Janzen, F. J., and G. L. Paukstis. 1988. Environmental sex determination in reptiles. Nature 332:790.

———. 1991a. Environmental sex determination in reptiles: ecology, evolution, and experimental design. Q. Rev. Biol. 66:149–179.

———. 1991b. A preliminary test of the adaptive significance of environmental sex determination in reptiles. Evolution 45:435–440.

Janzen, F. J., and P. C. Phillips. 2006. Exploring the evolution of environmental sex determination, especially in reptiles. J. Evol. Biol. 19:1775–1784.

Joanen, T., and L. L. McNease. 1989. Ecology and physiology of nesting and early development of the American alligator. Am. Zool. 29:987–998.

Kuijper, B., and I. Pen. 2014. Conflict over condition-dependent sex allocation can lead to mixed sex-determination systems. Evolution 68:3229–3247.

Leimar, O. 1996. Life-history analysis of the Trivers and Willard sex-ratio problem. Behav. Ecol. 7:316–325.

Lovich, J. E., J. W. Gibbons, and M. Agha. 2014. Does the timing of attainment of maturity influence sexual size dimorphism and adult sex ratio in turtles? Biol. J. Linn. Soc. 112:142–149.

Maddison, W. P., and R. G. Fitzjohn. 2015. The unsolved challenge to phylogenetic correlation tests for categorical characters. Syst. Biol. 64:127–136.

Pen, I., F. J. Weissing, and S. Daan. 1999. Seasonal sex ratio trend in the European Kestrel: an evolutionarily stable strategy analysis. Am. Nat. 153:384–397.

Pen, I., T. Uller, B. Feldmeyer, A. Harts, G. M. While, and E. Wapstra. 2010. Climate-driven population divergence in sex-determining systems. Nature 468:436–438.

Pokorná, M., and L. Kratochvíl. 2009. Phylogeny of sex-determining mechanisms in squamate reptiles: are sex chromosomes an evolutionary trap? Zool. J. Linn. Soc. 156:168–183.

Radder, R. S., D. A. Pike, A. E. Quinn, and R. Shine. 2009. Offspring sex in a lizard depends on egg size. Curr. Biol. 19:1105–1105.

Rhen, T., and J. W. Lang. 1995. Phenotypic plasticity from growth in the common snapping turtle: effects of incubation temperature, clutch and their interaction. Am. Nat. 146:726–747.

R Development Core Team. 2014. R: a language and environment for statistical computing. Version 3.1. R Foundation for Statistical Computing Vienna. Available at http://cran.R-project.org.

Sæther, B.-E., et al. 2013. How life history influences population dynamics in fluctuating environments. Am. Nat. 182:743–759.

Schwanz, L. E., and S. R. Proulx. 2008. Mutual information reveals variation in temperature-dependent sex determination in response to environmental fluctuation, lifespan and selection. Proc. R. Soc. Lond. B 275:2441–2448.

Schwanz, L. E., J. G. Bragg, and E. L. Charnov. 2006. Maternal condition and facultative sex ratios in populations with overlapping generations. Am. Nat. 168:521–530.

Schwanz, L. E., T. Ezaz, B. Gruber, and A. Georges. 2013. Novel evolutionary transitions between sex-determining mechanisms. J Evol. Biol. 26:2544–2557.

Shaw, R. F., and J. D. Mohler. 1953. The selective significance of the sex ratio. Am. Nat. 87:337–342.

Shine, R. 1990. Proximate determinant of sexual differences in adult body size. Am. Nat. 135:278–283.

———. 1999. Why is sex determined by nest temperature in many reptiles? Trends Ecol. Evol. 14:186–189.

Spencer, R.-J., and F. J. Janzen 2014. A novel hypothesis for the adaptive maintenance of environmental sex determination in a turtle. Proc. R. Soc. Lond. B 281:20140831.

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

Valenzuela, N. 2004. Evolution and maintenance of temperature-dependent sex determination. Pp. 131–147 *in* N. Valenzuela and V. Lance, eds. Temperature dependent sex determination in vertebrates. Smithsonian Books, Washington, DC.

Warner, D. A., and R. Shine. 2005. The adaptive significance of temperature-dependent sex determination: experimental tests with a short-lived lizard. Evolution 59:2209–2221.

———. 2008. The adaptive significance of temperature-dependent sex determination in a reptile. Nature 451:566–568.

Warner, D. A., T. Uller, and R. Shine. 2009. Fitness effects of the timing of hatching may drive the evolution of temperature-dependent sex determination in short-lived lizards. Evol. Ecol. 23:281–294.

Werren, J., and E. L. Charnov. 1978. Facultative sex ratios and population dynamics. Nature 272:349–350.

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Supporting Information

Additional Supporting Information may be found in the online version of this article at the publisher's website:

Figure S1. Pruned phylogeny, after Guillon et al. (2012), used in phylogenetic generalized least squares models on sex-determining mechanism (GSD vs. TSD) with Diff_maturity and size-related predictors (A).
Figure S2. Pruned phylogeny, after Guillon et al. (2012), used in phylogenetic generalized least squares models on sex-determining mechanism (GSD vs. TSD) with only size-related predictors (A).
Figure S3. Pruned phylogeny, after Guillon et al. (2012), used in phylogenetic generalized least squares models on sex-determining mechanism (GSD vs. TSD) with only size-related predictors and with sea turtles excluded (A).
Table S1. Data on SDM, sex differences in age of maturity (Diff_maturity) and body size (Diff_size), and mean body size including both sexes.