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Evolution of large males is associated with female-skewed adult sex ratios in amniotes

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Body size often differs between the sexes (leading to sexual size dimorphism, SSD), as a consequence of differential responses by males and females to selection pressures. Adult sex ratio (ASR, the proportion of males in the adult population) should influence SSD because ASR relates to both the number of competitors and available mates, which shape the intensity of mating competition and thereby promotes SSD evolution. However, whether ASR correlates with SSD variation among species has not been yet tested across a broad range of taxa. Using phylogenetic comparative analyses of 462 amniotes (i.e., reptiles, birds, and mammals), we fill this knowledge gap by showing that male bias in SSD increases with increasingly female-skewed ASRs in both mammals and birds. This relationship is not explained by the higher mortality of the larger sex because SSD is not associated with sex differences in either juvenile or adult mortality. Phylogenetic path analysis indicates that higher mortality in one sex leads to skewed ASR, which in turn may generate selection for SSD biased toward the rare sex. Taken together, our findings provide evidence that skewed ASRs in amniote populations can result in the rarer sex evolving large size to capitalize on enhanced mating opportunities.

KEY WORDS: **Comparative method, mating competition, mating opportunity, sex-biased mortality, sexual selection.**

Sexual size dimorphism (SSD, measured as the size of males relative to females) is widespread in nature and is one of the most conspicuous phenotypic difference between the sexes (Darwin 1871; Andersson 1994; Fairbairn et al. 2007). It is the consequence of different optimal body size for the sexes resulting from opposing selection forces (some of which may influence only one of the sexes) that equilibrate differently in males and females (Blanckenhorn 2005).

A large volume of research has focused on how sex-specific behavior (e.g., mating system, parental care), ecological processes (e.g., abundance and quality of resources), and life history traits (e.g., fecundity in indeterminate growers) can generate size differences between the sexes (Andersson 1994; Blanckenhorn 2005). These studies have concluded that sexual selection is often a major driver of SSD evolution by either intrasexual competition for access to mates or intersexual mate choice, although

other evolutionary mechanisms (e.g., fertility selection and competition for resources) may also be important (Jehl and Murray 1986; Andersson 1994; Blanckenhorn 2005; Fairbairn et al. 2007; Clutton-Brock 2016). Strong sexual selection for large body size in one sex is particularly likely in species where that sex competes for mates by physical contests or endurance rivalry, as observed in several vertebrate taxa (e.g., reptiles, birds, and mammals; Jehl and Murray 1986; Andersson 1994; Cox et al. 2007; Székely et al. 2007; Clutton-Brock 2016).

Adult sex ratio (ASR), best measured as the proportion of males in the adult population (Ancona et al. 2017), is a key demographic property of populations that influences both the number of competitors for mates and the number of mates available to an individual (Murray 1984; Székely et al. 2014b; Jennions and Fromhage 2017; Schacht et al. 2017). For example, a male-skewed ASR means potentially more competitors and fewer available partners for males than for females. An increasing number of studies show that ASR covaries with several reproductive traits such as mating system, parental sex roles, divorce rate, extra-pair mating, and cooperative breeding both in nonhuman animals and humans (Liker et al. 2013, 2014; Schacht et al. 2014; Kappeler 2017; Komdeur et al. 2017; Eberhart-Phillips et al. 2018; Grant and Grant 2019). However, whether and how ASR is related to the evolution of SSD is still poorly understood.

Theories suggest that ASR can drive the evolution of SSD in at least two ways. First, the intensity of sexual competition may increase with the number of competitors. As Darwin (1871, p. 217) wrote: "*That some relation exists between polygamy and development of secondary sexual characters, appears nearly certain; and this supports the view that a numerical preponderance of males would be eminently favourable to the action of sexual selection*." According to his idea, highly skewed ASRs may intensify selection for competitive traits such as weapons and large body size in the more abundant sex. Thus this "mating competition hypothesis" predicts that the extent of male bias in SSD should increase with the degree of male skew in the ASR. Later work refined Darwin's (1871) original idea by suggesting that the operational sex ratio (OSR, the number of sexually active males per receptive female at a given time) rather than the ASR determines the intensity of mating competition in a population (Emlen and Oring 1977). Thus, according to this latter theory ASR would predict SSD if ASR covaries with OSR, for example, because OSR is in part determined by ASR (together with sex differences in behavior such as parental care; Kokko et al. 2012). Although the relationship between ASR and OSR is yet to be fully explored, their positive association has been demonstrated both by theoretical models (Kokko and Jennions 2008: fig. 4A; Fromhage and Jennions 2016: fig. 3C, D) and comparative analyses (Mitani et al. 1996, correlation between ASR and OSR in 18 primates: $r = 0.4$, $P = 0.002$; unpublished result using data from their table 1). Empirical studies commonly use ASR and OSR interchangeably in testing their relationship with SSD (Poulin 1997) and other proxies of sexual selection (Janicke and Morrow 2018).

Second, models of reproductive sex roles predict that ASR should influence the evolution of SSD because individuals of a given sex may allocate less to parental care when the sex ratio is skewed toward the opposite sex than when it is skewed toward their own sex (Queller 1997; McNamara et al. 2000). According to these models, males in female-skewed populations display a higher reproductive success due to increased probability of breeding with multiple partners and therefore may evolve to reduce parental care (Queller 1997: section 3; McNamara et al. 2000: section "Sex ratio"). This association between ASR and parental sex roles can drive the evolution of SSD because more elaborate trait expression in males is evolutionarily linked to female-biased care and stronger sexual selection on males (the so called "sex-role syndrome"; Janicke et al. 2016: fig. 3). Thus, this "mating opportunity hypothesis" predicts that the extent of male bias in mating competition, and hence in SSD, should decrease with increasing male skew in the ASR. A demographic analysis of mating systems by Murray (1984) also predicts that female-skewed ASRs should be associated with both polygyny and male-biased SSD, whereas male-skewed ASRs should be associated with polyandry and female-biased SSD.

Alternatively, SSD may drive changes in sex ratios through sex differences in mortality resulting from sexual competition. According to this "mortality cost hypothesis," the skewed ASR is a consequence rather than a cause of intense sexual selection, because when males allocate a lot to mating competition they may suffer increased mortality, which in turn leads to female-skewed ASR (Trivers 1972; Clutton-Brock et al. 1985; Liker and Székely 2005; Kalmbach and Benito 2007). This hypothesis predicts that in species exhibiting SSD, (1) the larger sex should have higher mortality due to the costs of being large, including the direct costs associated with competition (e.g., fights, displays), which leads to (2) decreasing male skew in the ASR with increasing degree of male bias in the SSD.

Studies that have investigated the relationships between sex ratios, SSD, and sex-specific mortality have so far yielded inconsistent results. Although some studies found a positive link between SSD and ASR or OSR (i.e., an increasing male bias in SSD with increasing male skew in the sex ratios; Mitani et al. 1996; Poulin 1997), others reported negative associations (Clutton-Brock et al. 1977; Wittenberger 1978; Georgiadis 1985; Haro et al. 1994; Johansson et al. 2005; Lovich et al. 2014), or found no consistent relationships (Owen-Smith 1993; Hirst and Kiørboe 2014; Muralidhar and Johnson 2017). Similarly, mortality costs paid by the larger sex in dimorphic species were reported in some studies (Clutton-Brock et al. 1985; Promislow 1992; Promislow et al. 1992; Moore and Wilson 2002; Benito

and González-Solís 2007; Kalmbach and Benito 2007), whereas no consistent relationship between SSD and sex differences in mortality was found by others (Owens and Bennett 1994; Toïgo and Gaillard 2003; Lemaître and Gaillard 2013; Székely et al. 2014a; Tidière et al. 2015). Many of these studies focused on a narrow range of taxonomic groups and were based on a relatively small number of species (typically fewer than 50) in comparative analyses. Furthermore, none of the studies tested explicitly whether statistical models assuming that ASR drives variation in SSD (as proposed by the mating competition and mating opportunity hypotheses) or alternative models (like the mortality costs hypothesis) fit better to the data.

Here, we investigate the strength and direction of the relationship between ASR and SSD in populations of wild amniotes, using the largest existing comparative dataset on ASR compiled to date (462 species). First, we investigate whether SSD increases or decreases with ASR across species, as predicted by the mating competition and mating opportunity hypotheses, respectively. We also test whether the relationship is consistent among three major amniote taxa (reptiles, birds, and mammals) because these taxa differ in multiple ecological, behavioral, and life history traits. Because the extent and direction of SSD can be influenced by ecological, life history, and behavioral factors besides mating competition, we also control for several potential confounding variables in the analyses. Second, we study whether SSD drives ASR variation by generating sex-biased mortality as proposed by the mortality cost hypothesis. We test this latter hypothesis by investigating whether SSD is related to sex differences in juvenile or adult mortality, and by comparing path models representing different structural relationships between SSD, ASR, and sex-specific mortality.

Methods

DATA COLLECTION

Data were extracted from published sources (see Appendix S1). The initial dataset was based on Pipoly et al. (2015) that contains ASR and SSD for 344 amniote species. We excluded amphibians included in Pipoly et al. (2015) because sex-specific mortality data (see below) are very scarce for this taxon, especially in juveniles. The initial dataset was augmented with additional reptile and mammal species, and with information on sex-specific mortality. These additional data were taken from existing comparative datasets (Berger and Gompper 1999 and Bókony et al. 2019 for ASR in mammals and reptiles, respectively, and Székely et al. 2014a for mortality in birds) or from primary publications. In the latter case, we searched the literature through the search engines Web of Science and Google Scholar, using the search terms "sex ratio," "sex-specific mortality OR survival," or "male female mortality OR survival" together with taxonomic names. Data for different variables for the same species were often available only from different populations or studies. The final dataset includes 462 species with both ASR and SSD available (155 reptiles, 185 birds, and 122 mammals).

BODY MASS AND SSD

Sex-specific body mass (g) was available for all birds and mammals in our dataset. Because body mass data were missing for many reptiles, we also collected body length data (mm) for this taxon in the form of snout-vent length for squamates and crocodilians and plastron or carapace length for turtles. We estimated body mass from body length using published allometric equations (Appendix S2). We used estimated body mass for reptiles instead of body length in the combined analyses of all species because (1) data on mass are more readily available than data on body length in birds and mammals, which provided the majority of species, and (2) body mass is measured in a standardized way in all taxa, whereas the measurement of body length varies because different parts of the body are recorded as a proxy for length in different taxa. If multiple mass or length data were available for a species, we used the mean value. Average adult body mass was calculated as $\log_{10}$-transformed mean mass of the sexes.

We calculated SSD as $\log_{10}$(male mass/female mass). Earlier studies criticized measures of SSD that are based on male/female (or female/male) ratios and suggested other approaches, for example, to analyze male size as response variable in models that also include female size as a control variable (see Smith 1999 and Fairbairn 2007 for reviews). In his seminal paper, however, Smith (1999, p. 444) convincingly demonstrated that ratios can be safely used in the context of SSD analyses because "the risk of spurious correlation is negligible to nonexistent" due to the statistical properties of male and female size variables (i.e., their high correlation and approximately equal coefficients of variation, leading to an isometric relationship). We checked the assumption of isometry between male and female body mass in our dataset and found that male and female body mass (on a $\log_{10}$-$\log_{10}$ scale) are strongly correlated ($r = 0.994$) with a slope very close to and not different from 1 (phylogenetic generalized least squares [PGLS], slope $\pm$ *SE*: 1.010 ± 0.010, 95% CI: $0.989 \leq \beta \leq 1.029$, $n = 462$ species). Furthermore, Smith (1999, pp. 439–440) demonstrated that the approaches based on the log ratios versus male mass as response variable are statistically equivalent and suggested that the correct method is using log SSD ratio as response and controlling for log size. We thus followed this latter approach. However, because the measures of SSD remain a controversial issue among evolutionary ecologists (see, e.g., table 1 in Tidière et al. 2015 for a review of SSD metrics commonly used), we replicated the main analysis using an

alternative method (i.e., male size as response variable while controlling for female size in the model) to check the robustness of our results. All results remained qualitatively unchanged.

To test whether the results are sensitive to conversion of length to mass in reptiles, we replicated the main analyses (1) with SSD calculated from body length ($\log_{10}$(male length/female length)) of reptiles, and (2) with SSD calculated from body mass for a subset (31 species) of reptiles that has sex-specific mass data available from Myhrvold et al. (2015). Whatever approach was used to assess the degree of SSD, the results were qualitatively unchanged (see *Results*). In the main text, we thus report results based on body mass estimated from body length for reptiles.

SEX RATIO

We followed Wilson and Hardy (2002) and Ancona et al. (2017) in expressing ASR as the proportion of males in the adult population. We defined the adult population here broadly as adult individuals living in the study area during ASR sampling. Wilson and Hardy (2002) showed that analyzing sex ratios as a proportion variable is appropriate when sex ratios are estimated from samples of $\geq$10 individuals and the dataset has $\geq$50 sex ratio estimates. These conditions were more than fully met in our analyses because sample sizes for ASR estimates were always larger than 10 individuals per species (and typically much larger), and our overall dataset included nine times more than the requirement of 50 species.

ASR data from Pipoly et al. (2015) were augmented with new species and updated with more recent and/or better-quality information (e.g., based on a more reliable method or a larger sample size) for some reptiles. ASR estimates were collected by different observers for the different taxa: reptiles by VB and IP (Pipoly et al. 2015; Bókony et al. 2019), birds by AL (Liker et al. 2014), and mammals by Berger and Gompper (1999), Donald (2007), and Anile and Devillard (2018). Details of data selection criteria are given in the original publications (see also Ancona et al. 2017). Mean values were calculated for species with multiple ASR data. ASR estimates are repeatable between populations of the same species as measured by the intraclass correlation coefficient (ICC), although the magnitude of repeatability varies among taxa — reptiles with genetic and environmental sex determination: ICC = 0.55 and 0.14, respectively (Bókony et al. 2019), birds: ICC = 0.64 (Ancona et al. 2017), and mammals: ICC = 0.60 (Valentine Federico, J-FL, J-MG, AL, IP, and TS unpubl. result). ASR estimates are not influenced by the sample size of the ASR studies (Székely et al. 2014a; Bókony et al. 2019).

SEX-SPECIFIC MORTALITY

Annual mortality rates were collected from studies in which mortality (or survival) was estimated for each of both sexes. Juvenile and adult mortality refer to age classes before and after the age of first reproduction, respectively. For reptiles, data were collected by VB (Bókony et al. 2019). Most adult mortality data on birds are taken from Székely et al. (2014a) with the addition of new data for juvenile mortality by AL. Reptile and bird mortality includes estimates by various methods (e.g., capture-recapture, return rates, demographic models), although we used better-quality estimates (e.g., those from capture-recapture analyses) whenever we had a choice (Székely et al. 2014a; Bókony et al. 2019). For mammals, all sex-specific estimates were collected by J-MG and J-FL (Lemaître et al. 2020). Sex differences in juvenile and adult mortality rates were calculated as the magnitude of male-biased mortality (i.e., $\log_{10}$(juvenile or adult male mortality/juvenile or adult female mortality)), also referred to as "mortality bias." These measures of mortality bias are not related to the overall mortality rate of the species, as estimated by the average mortality rates of the sexes (PGLS models, juvenile mortality bias: slope $\pm$ SE = –0.068 $\pm$ 0.101, t = 0.7, P = 0.497, n = 100; adult mortality bias: slope $\pm$ SE = –0.05 $\pm$ 0.08, t = 0.7, P = 0.513, n = 230).

OTHER PREDICTORS

We controlled for the potential effects of ecological variables and life history traits related to either ASR or SSD (or both) that may confound the assessment of their relationship. First, we collected data on the type of sex determination system because it is associated with both ASR (Pipoly et al. 2015) and SSD (Adkins-Regan and Reeve 2014). We divided the species into three categories according to the Tree of Sex database (Ashman et al. 2014): male-heterogametic (XY) or female-heterogametic (ZW) genetic sex determination, or temperature-dependent sex determination (TSD). For species that were not included in the Tree of Sex database, we assumed the same type of sex determination as reported for the genus (or family, respectively; Bókony et al. 2019) when the genus (or family) to which it belongs had invariable sex determination system. All birds were assigned to ZW, and all mammals to XY sex determination (Ashman et al. 2014).

Second, we controlled for the potential effects of environmental variation among species by using two measures. Breeding latitude correlates with life history traits in many organisms (as shown in his pioneer work by Dobzhansky 1950) and may also influence the potential for polygamy, hence also sexual selection (Fischer 1960; Isaac 2005; Balasubramaniam and Rotenberry 2016). We used absolute values of the geographic latitude of the ASR studies included in our dataset (i.e., average values for species with multiple ASR estimates) to represent the distance from the Equator. When the authors did not report latitude, we used Google Earth to estimate it as the center of the study sites based on the site descriptions. For 30 birds and 10 mammals, accurate population locations were not reported; hence, we used the latitudinal midpoint of the breeding ranges of these species

(birds: V. Remeš, A. Liker, R. Freckleton, and T. Székely unpubl. data, mammals: PanTHERIA database).

In addition to latitude, we investigated environmental harshness as a second environmental variable, which also has been hypothesized to influence SSD (Isaac 2005). We quantified the harshness of the breeding environment using a proxy proposed by Botero et al. (2014). This is the PC1 score extracted from Principal Component Analysis (PCA) performed on a set of climatic and ecological variables (e.g., temperature and precipitation, net primary productivity, habitat heterogeneity; see Botero et al. 2014 for a detailed description of the variables and the analysis). The PC1 scores have higher values for a higher level of exposure to drier, less productive environments, with colder, less predictable, and more variable annual temperatures (see table 1 in Botero et al. 2014). In birds and mammals, we used the data published in Botero et al. (2014), whereas for reptiles we calculated PC1 scores by performing a PCA with the same set of variables.

Third, we characterized courtship displays in birds because earlier studies showed that birds with aerial displays have less male-biased SSD compared to species with ground displays, probably because selection favors male agility in aerially displaying species constraining male body size (Jehl and Murray 1986; Székely et al. 2007). We followed Székely et al. (2007) and divided species into two display groups: (1) mating displays that may favor male agility, including species that mainly have aerial displays (both nonacrobatic and acrobatic, categories 4 and 5 in Székely et al. 2007), and (2) displays that may not favor male agility, including all other display types, typically performed on ground (categories 1–3 in Székely et al. 2007). Although SSD can also be influenced by display type and display habitat in reptiles and mammals (e.g., see Agha et al. 2018), we were not able to collect reliable data for these taxa, therefore we analyzed the effect of display type only in birds.

Fourth, we tested for the potential effect of social mating system, because the scope for mating competition may be more limited in monogamous than in polygamous species (Andersson 1994). Thus, although there is ASR variation among monogamous species that can generate some variation in mating competition and/or opportunity, the relationship between ASR and SSD is expected to be weaker in monogamous than in polygamous species. To test this idea, we characterized social mating system for birds and mammals, because we found reliable information in these taxa for most species (Liker et al. 2014; Lukas and Clutton-Brock 2013). Although socially polygamous mating systems differ from promiscuous mating system, we pooled these mating systems because sexual selection is consistently stronger in polygamous than in monogamous species, whereas the relative intensity of sexual selection in polygynous versus promiscuous species is not easy to assess. We thus categorized species as either socially monogamous or polygamous (most often polygynous) according to the sources, as previously done (see, e.g., Lukas and Clutton-Brock 2013). In birds, social mating system was originally scored on a five-point scale (Liker et al. 2014), and here we considered a species monogamous if it had score 0 or 1 (polygamy frequency $<1\%$) for both sexes.

Finally, in reptiles, the evolution of viviparity and reduced reproductive frequency are generally correlated with shifts toward female-biased SSD due to fecundity selection for large female size (Pincheira-Donoso and Hunt 2017). To control for its potential effect on SSD, we categorized the reproductive mode of reptiles as either viviparous or oviparous (Uetz et al. 2021).

STATISTICAL ANALYSES

PGLS models were built to conduct bivariate and multi-predictor analyses. To control for phylogenetic relationships among taxa, we used the composite phylogeny applied in Pipoly et al. (2015) with the addition of new species according to the family-level (Sarre et al. 2011) and other recent phylogenies (Squamata: Nicholson et al. 2012, Pyron et al. 2013, Gamble et al. 2014; Testudines: Barley et al. 2010, Guillon et al. 2012, Spinks et al. 2014; Crocodylia: Oaks 2011; mammals: Fritz et al. 2009, Meredith et al. 2011). Because composite phylogenies do not have true branch lengths, we used three methods to generate branch lengths (Nee's method, Pagel's method, and unit branch lengths, using the PDAP:PDTREE module of Mesquite; Midford et al. 2011), and repeated key analyses with these alternative trees. We present results with Nee's branch lengths in this article, except for the sensitivity analyses (see *Results*). Freckleton et al. (2002) showed that PGLS is relatively insensitive to branch length assumptions. In each model, we used the maximum-likelihood estimate of phylogenetic dependence (Pagel's λ). PGLS models were run using the "caper" R package (Orme et al. 2013).

First, using all species, we applied bivariate PGLS models to test interspecific associations between ASR, SSD, and sex differences in juvenile and adult mortality rates. When SSD was the response variable in the model, we also included mean body mass as a second predictor, as recommended by Smith (1999) (hence, we termed these models as "separate predictor models" instead of bivariate models in the rest of this article). Then we built two multi-predictor models. In Multi-predictor model 1, we tested the relationship between ASR and SSD while controlling for potential confounding effects of mean mass, sex determination system, and breeding latitude. In Multi-predictor model 2, we tested the ASR-SSD relationships while controlling for the effects of sex differences in juvenile and adult mortality rates, and mean mass. We built these two separate multi-predictor models because we have much lower sample sizes for sex-specific mortalities than for the other predictors, thus the statistical power would be reduced for variables of Multi-predictor model 1 if all predictors

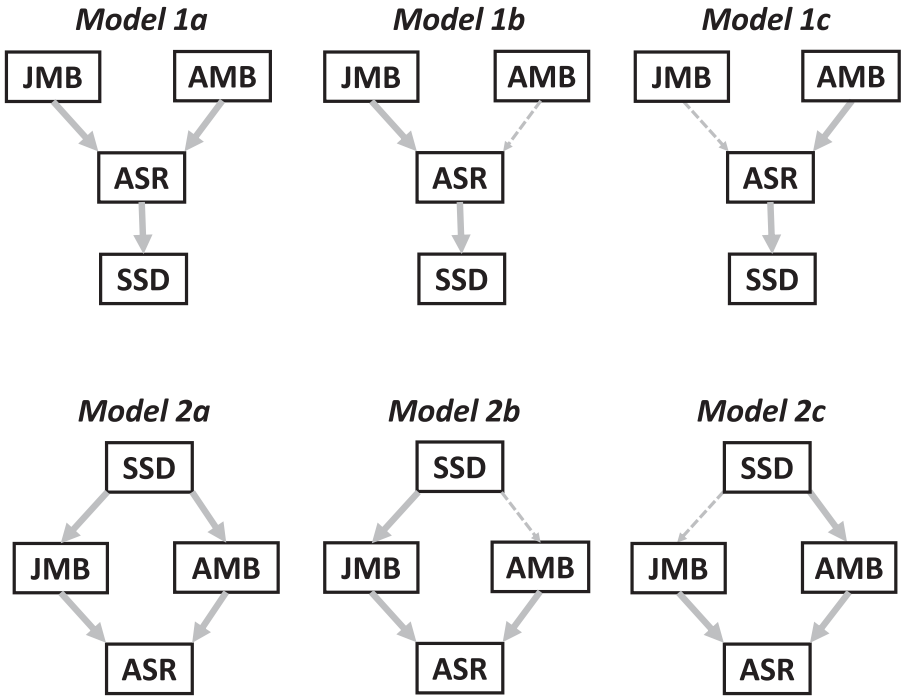

Figure 1. Path models tested in the phylogenetic path analyses. SSD = sexual size dimorphism; ASR = adult sex ratio; JMB = juvenile mortality bias; AMB = adult mortality bias. Dashed arrows indicate paths with coefficients set to zero to keep the variable in the model. Models 1a-c and 2a-c represent relationships as predicted by the mating opportunity hypothesis and the mortality cost hypothesis, respectively.

were combined in a single model. We ran the models in two alternative versions in which either SSD or ASR was the dependent variable, respectively, because we had no a priori knowledge about the cause-effect direction of these relationships and results may differ between these analyses if the two models have different values for Pagel's λ (see Appendix S3).

We investigated whether the ASR-SSD relationship, which is the main focus of our study, differed among taxa by testing the interaction between ASR and the taxonomic class. To explore differences among taxa in the multivariate relationships, we repeated all analyses separately for reptiles, birds, and mammals. In taxon-specific Multi-predictor models 1, we included reproductive mode for reptiles and display type for birds as further predictors. In reptiles, we also tested whether the relationship between ASR and SSD is sensitive (1) to the inclusion of species that have environmental sex determination, because ASR shows low repeatability in such reptiles (Bókony et al. 2019), and (2) to the inclusion of species in which the type of sex determination was inferred from data on related species in the genus or family. Finally, we ran two additional separate analyses to test whether social mating system and environmental harshness confounded the ASR-SSD relationship. All numeric variables were standardized before analyses to make parameter estimates comparable, and model assumptions were also checked and met. We report two-tailed statistics. Sample sizes differed between models because not all variables were available for all species (see Appendix S1).

In addition to PGLS models, we used phylogenetic path analyses (Santos 2012; Gonzalez-Voyer and von Hardenberg 2014) to compare two sets of path models corresponding to different hypotheses for the relationships linking ASR, SSD, and sex differences in mortality. Although path analyses — unlike experiments — cannot infer causality, it is a suitable method to compare alternative scenarios representing different causal relationships between variables (Shipley 2016). Model 1 assumes that sex-biased mortality influences ASR, which in turn influences SSD through its effects on mating competition (as proposed by the mating opportunity hypothesis; Fig. 1). Three variants of this model were tested: Model 1a assumes that sex differences in both juvenile and adult mortality rates influence ASR, whereas Models 1b and 1c include only one of these mortality effects. Model 2 assumes that SSD has sex-specific effects on juvenile and/or adult mortality, which then drives ASR variation (representing the mortality cost hypothesis; Fig. 1). We tested all the three variants of this latter scenario, assuming SSD effects on both juvenile and adult mortality (Model 2a) or only on one mortality component (Models 2b and 2c).

We followed the approach proposed by Santos (2012) for phylogenetic path analyses. In the first step, we conducted phylogenetic transformation on the data to control for effects of

phylogenetic relatedness among species. For this purpose, we (1) determined λ separately for each variable by maximum likelihood, (2) used this variable-specific λ value to re-scale the phylogenetic tree to a unit tree, and (3) used the transformed tree to calculate phylogenetically independent contrasts for the variable (using "pic" function of the R package "ape"; Paradis 2012). We repeated this process for each variable, and the resulting phylogenetically transformed values were used for fitting path models. In the second step of the analyses, we evaluated model fit using d-separation method (Shipley 2016) as implemented in the R package "piecewiseSEM" (Lefcheck 2016). In this method, Fisher's *C* statistic is used to test the goodness of fit of the whole path model, and the model is rejected (i.e., it does not provide a good fit to the data) if the result of this *C* statistic is statistically significant (and conversely a statistically nonsignificant result means acceptable fit; Lefcheck 2016). We compared model fit between the six path models by their AICc values. Note that this approach ensures that the same variables (i.e., the contrasts with the same phylogenetic signal) are used in each path model, and that the correlations are nondirectional in the sense that for a pair of variables *X* and *Y*, $r_{XY} = r_{YX}$ as assumed in path analysis (irrespective of the sign of the correlation, i.e., whether it is positive or negative).

To test the robustness of the results, we repeated the path analyses using two other methods. First, we repeated the above procedure (i.e., followed Santos 2012) except that we used the covariance matrix comparison method for model fit instead of d-separation, as implemented in the R package "lavaan" (Rosseel 2012). Second, we repeated the analyses using the method developed by von Hardenberg and Gonzalez-Voyer (2013). Unlike Santos' (2012) method, in this latter approach a single value of Pagel's λ is estimated for the residuals of a regression of each pair of traits in a directional model, rather than a value of λ for each variable (see the *Discussion* and Appendix S3). We used the R package "phylopath" (van der Bijl 2018) for this latter analysis, which relies on the d-separation method for model fitting (similarly to "piecewiseSEM," see above). We provide additional analyses to test the robustness of the path analysis' results in Appendix S3.

Results

MATING COMPETITION VERSUS MATING OPPORTUNITY HYPOTHESES

Consistent with the mating opportunity hypothesis, and in contrast to the mating competition hypothesis, we found a negative relationship between our measures of ASR and SSD: the size of males relative to females increases when ASR becomes more female-skewed (Fig. 2; Table 1). This correlation was statistically significant when all species were analyzed together and

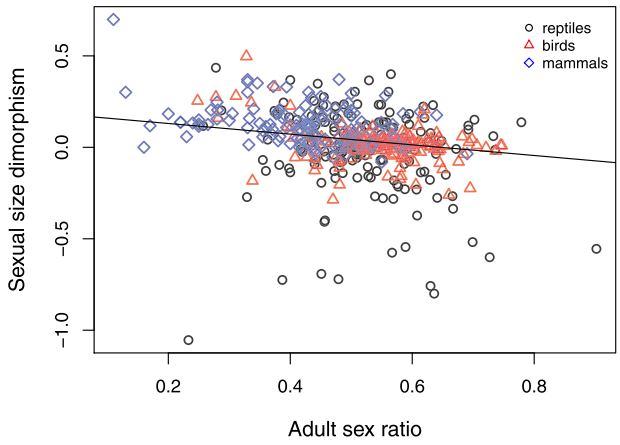

Figure 2. Sexual size dimorphism (SSD) in relation to adult sex ratio (ASR) in amniotes. SSD was calculated as $\log_{10}$(male mass/female mass); ASR is the proportion of males in the adult population. Each data point represents a species; the regression line is fitted by phylogenetic generalized least squares (PGLS) model (see Table 1 for statistics).

did not differ among the three amniote classes (ASR × class interaction on SSD: $F_{2,456} = 0.935$, $P = 0.393$). The increase of SSD with increasingly female-skewed ASR was statistically significant within birds and mammals but was not in reptiles when the three taxa were analyzed separately (Fig. S1; Tables S1-S4). These results remained consistent when we used SSD estimates based on length instead of estimated mass in reptiles (Tables S1, S2, and S5), when SSD for reptiles were estimated from published body mass data (Table S5), and also when male mass was used as response variable (Table S5).

These results are robust because the sign of the slope of the ASR-SSD relationship and its statistical significance were not sensitive to branch length assumptions (Table S6), and to the inclusion of other predictors (Table 1). In multi-predictor models (Table 1), mean body mass was positively related to SSD, supporting the Rensch rule (Abouheif and Fairbairn 1997), and the type of sex determination influenced ASR variation as previously reported by Pipoly et al. (2015). Nevertheless, ASR remained negatively associated with SSD when the effects of mass and sex determination systems were accounted for (Table 1). This result also did not change when environmental variation was included in the models using either breeding latitude (Table 1) or environmental harshness (Table S5). Finally, excluding reptiles with TSD (that have the lowest consistency in ASR; Bókony et al. 2019) or with assumed sex determination also did not influence the relationship (Table S5).

The multi-predictor model for birds showed that species with aerial courtship displays have lowered SSD as found in earlier studies (Jehl and Murray 1986; Székely et al. 2007); however, the relationship between ASR and SSD remained statistically

Table 1. Phylogenetically corrected analyses of sexual size dimorphism (SSD) and adult sex ratio (ASR) in amniotes (reptiles, birds, and mammals).

Predictors	$b \pm SE$	t	P	R^2	λ	n
(A) Response: Sexual size dimorphism						
Separate predictor models:						
Model 1				0.119	0.868*‡	462
ASR	–0.168 ± 0. 035	4.835	**<0.001**			
Mean body mass	0.515 ± 0. 086	5.980	**<0.001**			
Model 2				0.129	0.703*‡	100
Juvenile mortality bias	0.041 ± 0.065	0.629	0.531			
Mean body mass	0.529 ± 0.131	4.051	**<0.001**			
Model 3				0.095	0.932*	230
Adult mortality bias	–0.021 ± 0.047	0.454	0.650			
Mean body mass	0.596 ± 0. 117	5.090	**<0.001**			
Multi-predictor model 1:				0.126	0.869*‡	457
ASR	–0.160 ± 0.035	4.555	**<0.001**			
Mean body mass	0.515 ± 0.087	5.950	**<0.001**			
Latitude	0.004 ± 0.038	0.103	0.918			
Sex determination, TSD[1]	–0.297 ± 0.251	1.184	0.237			
Sex determination, ZW[1]	–0.685 ± 0.264	2.592	**0.010**			
Multi-predictor model 2:				0.273	0.841*	97
ASR	–0.271 ± 0.061	4.452	**<0.001**			
Mean body mass	0.377 ± 0.134	2.824	**0.006**			
Juvenile mortality bias	0.001 ± 0.060	0.011	0.992			
Adult mortality bias	–0.019 ± 0.067	0.277	0.783			
(B) Response: Adult sex ratio						
Separate predictor models:						
Model 1: SSD	–0.234 ± 0.051	4.593	**<0.001**	0.042	0.359*‡	462
Model 2: Juvenile mortality bias	–0.214 ± 0.099	2.151	**0.034**	0.035	0.281*‡	100
Model 3: Adult mortality bias	–0.257 ± 0.060	4.313	**<0.001**	0.071	0.288*‡	230
Multi-predictor model 1:				0.071	0.247*‡	457
SSD	–0.188 ± 0.050	3.727	**<0.001**			
Mean body mass	–0.106 ± 0.080	1.330	0.184			
Latitude	–0.095 ± 0.045	2.135	**0.033**			
Sex determination, TSD[1]	0.481 ± 0.221	2.178	**0.030**			
Sex determination, ZW[1]	0.712 ± 0.205	3.471	**<0.001**			
Multi-predictor model 2:				0.402	0.030‡	97
SSD	–0.457 ± 0.120	3.794	**<0.001**			
Mean body mass	–0.249 ± 0.108	2.316	**0.023**			
Juvenile mortality bias	–0.146 ± 0.086	1.702	0.092			
Adult mortality bias	–0.259 ± 0.100	2.591	**0.011**			

Results of separate predictor and multi-predictor phylogenetic generalized least-squares (PGLS) models with either (A) SSD ($\log_{10}$(male mass/female mass)) or (B) ASR (proportion of males in the adult population) as dependent variable. Separate predictor models with SSD as dependent variable also include $\log_{10}$(mean mass) as predictor (see *Methods*). Mortality biases were calculated as $\log_{10}$(male mortality/female mortality) for juveniles and adults, respectively. $b \pm SE$ is the model's parameter estimate with its standard error (intercepts are not shown), t and P are the associated test statistic and its significance, λ is Pagel's lambda, and n is number of species.

*λ statistically different from 0,

‡λ statistically different from 1.

[1] Differences from species with XY sex determination; overall effect of sex determination on SSD: $F_{2,451} = 3.411$, $P = 0.034$; on ASR: $F_{2,451} = 6.135$, $P = 0.002$.

Table 2. Phylogenetic path models of the mating opportunity hypothesis (Models 1a-c) and the mortality cost hypothesis (Models 2a-c) in amniotes (reptiles, birds, and mammals).

Model/Path	Path coefficient ± *SE*	Z	*P*
Model 1a	$P_C = 0.972$, $df = 4$, AICc = 15.8, ΔAICc = 0.0		
AMB → ASR	–0.340 ± 0.113	–3.000	**0.004**
JMB → ASR	–0.205 ± 0.104	–1.970	0.052
ASR → SSD	–0.425 ± 0.074	–5.723	**<0.001**
Model 1b	$P_C = 0.065$, $df = 6$, AICc = 25.7, ΔAICc = 9.9		
(AMB → ASR)[1]	0	–	–
JMB → ASR	–0.258 ± 0.107	–2.417	**0.018**
ASR → SSD	–0.425 ± 0.074	–5.723	**<0.001**
Model 1c	$P_C = 0.376$, $df = 6$, AICc = 19.9, ΔAICc = 4.1		
AMB → ASR	–0.378 ± 0.113	–3.334	**0.001**
(JMB → ASR)[1]	0	–	–
ASR → SSD	–0.425 ± 0.074	–5.723	**<0.001**
Model 2a	$P_C = 0.0$, $df = 4$, AICc = 59.0, ΔAICc = 43.2		
SSD → AMB	0.171 ± 0.105	1.631	0.106
SSD → JMB	0.111 ± 0.115	0.958	0.341
AMB → ASR	–0.340 ± 0.113	–3.000	**0.004**
JMB → ASR	–0.205 ± 0.104	–1.970	0.052
Model 2b	$P_C = 0.0$, $df = 4$, AICc = 50.4, ΔAICc = 34.6		
SSD → JMB	0.111 ± 0.115	0.958	0.341
AMB → ASR	–0.340 ± 0.113	–3.000	**0.004**
JMB → ASR	–0.205 ± 0.104	–1.970	0.052
Model 2c	$P_C = 0.0$, AICc = 50.4, ΔAICc = 34.6		
SSD → AMB	0.171 ± 0.105	1.631	0.106
AMB → ASR	–0.340 ± 0.113	–3.000	**0.004**
JMB → ASR	–0.205 ± 0.104	–1.970	0.052

Model structures are shown in Figure 1. SSD = sexual size dimorphism; ASR = adult sex ratio; JMB and AMB = juvenile and adult mortality biases, respectively (variables are explained in footnotes of Table 1). P_C is the *P*-value for Fisher's *C* statistic for model fit, with nonsignificant values (>0.05) indicating an acceptable fit. ΔAICc indicates difference in AICc values between the most supported model (lowest AICc, Model 1a) and the focal models. ΔAICc > 2 indicates substantially higher support for the best model than for the other models. *Z* and *P* values are the test statistic and the associated significance for each path. The analyses include 97 species of reptiles, birds, and mammals with data for all variables.

[1] **Path coefficient set to zero to keep the variable in the model.**

significant and negative when this effect was included in the model (Table S3). Furthermore, data in birds and mammals showed that, as expected, the relationship was weaker in monogamous than in polygamous species, although the same trend occurred in both mating systems (Table S7). Finally, reproductive mode was not associated with SSD or ASR in reptiles in our dataset (Tables S1-2).

MATING OPPORTUNITY VERSUS MORTALITY COSTS HYPOTHESES

Both the mating opportunity hypothesis and the mortality cost hypothesis predict female-skewed ASRs in species with male-biased SSD. However, our results are more consistent with the mating opportunity hypothesis for two reasons. First, ASR but not SSD was associated with the extent of sex differences in juvenile or adult mortality, and ASR remained strongly and negatively correlated with SSD when sex differences in juvenile and adult mortality were statistically controlled for (Table 1). Second, phylogenetic path analyses showed that models of the mating opportunity hypothesis provided better fit to the data (Models 1a-c, Fisher' *C* statistic: $P = 0.07$-0.97) than models corresponding to the mortality cost hypothesis (Models 2a-c, $P < 0.001$; Table 2). The strongest support was for Model 1a because it had the lowest AICc (ΔAICc = 4.1-43.2; Table 2). This model proposes that sex-biased mortality in both juveniles and adults generates skewed ASR, which in turn leads to SSD biased toward the rarer sex (Fig. 3). These results are robust because we obtained the same results when the analyses were repeated using two other implementations of the path analysis (see Table S8 for the results obtained using "phylopath," and Appendix S3 for the results obtained using "lavaan"). Finally, path analyses that excluded reptiles (for which the ASR-SSD relationship was not statistically

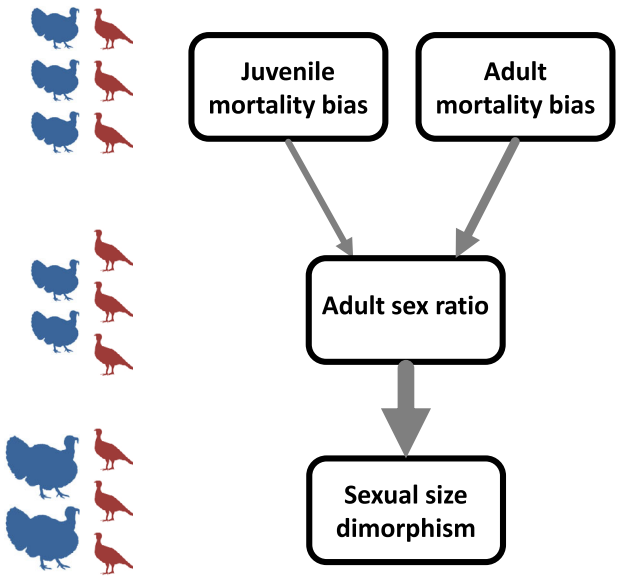

Figure 3. Path diagram of the best-fitting phylogenetic path model (Model 1a in Table 2; $n = 97$ species of reptiles, birds, and mammals). The model supports the scenario that sex-biased juvenile and adult mortalities lead to skewed adult sex ratio, which in turn results in increased size dimorphism by sexual selection. Width of the arrows is proportional to path coefficients (see Table 2 for statistical details of the model). Bird pictures on the left illustrate the case when differential mortality generates female-skewed ASR, which then leads to a more male-biased SSD (i.e., larger body size in males relative to females). The path analyses were based on the approach proposed by Santos (2012).

significant, see above) also yielded results qualitatively consistent with the full dataset (Table S9).

Discussion

Our analyses provided three major findings: (1) ASR is related to SSD among amniote species, although the association is the opposite of the one proposed by Darwin; (2) sex-biased mortality is unrelated to the extent of SSD in amniotes; and (3) confirmatory path analyses indicate that sex-biased mortality influences ASR, which in turn induces changes in SSD. Collectively, these findings support the mating opportunity hypothesis, indicating that selection is likely to favor an increased resource allocation toward mating competition (by growing and maintaining a large body mass) in the rarer sex, which has a higher chance of getting mates than the other sex.

Theoretical models show that skewed ASRs can promote evolutionary changes that may generate this association between ASR and SSD. First, models of sex role evolution showed that skewed ASR can result in divergences in reproductive roles between the sexes leading to less parental care and more frequent desertion and remating in the rarer sex and opposite changes (i.e., more parental care and less frequent remating) in the more abundant sex (Queller 1997; McNamara et al. 2000). Similarly, a demographic analysis based on the relationships between mating systems, sex ratio, sex-specific patterns of survivorship, age of first reproduction, and annual fecundity predicts that skewed ASRs promote the evolution of polygamy (i.e., polygyny and polyandry in female-skewed and male-skewed populations, respectively; Murray 1984). Because both frequent remating and polygamy can intensify sexual selection, the above effects of skewed ASR can promote the evolution of SSD by favoring increased body size in the rare sex. In line with the predictions of these models, an increasing number of recent studies in birds and humans show that polygyny is more frequent and parental care by males is reduced in female-skewed populations (Liker et al. 2013, 2014, 2015; Remeš et al. 2015; Schacht and Borgerhoff Mulder 2015; Eberhart-Phillips et al. 2018; Grant and Grant 2019). Our results are also concordant with experimental studies in voles and lizards, which reported that female-skewed ASRs exert directional selection for large body size in males (Klemme et al. 2007; Fitze and Le Galliard 2008), and increase variance in male reproductive success (Dreiss et al. 2010).

Theoretical models predict that the effects of ASR may depend on other life history and behavioral traits of the populations. For example, Fromhage and Jennions (2016) highlighted the importance of the specific processes generating ASR skews for the outcomes of sex role evolution, and that a coevolutionary feedback between parental care and sexually selected traits can greatly amplify sex role divergence. In addition, sexual competition for mates may favor different traits in species with distinct ecology and behavior, leading to inconsistent relationships between sex differences in mating competition and sexual dimorphisms in behavioral or morphological trait across species (Clutton-Brock 2017). Collectively, these factors may account for the relatively low amount of variation in SSD explained by ASR in some of our analyses.

The association between intense sexual selection in males and female-skewed ASRs was proposed decades ago by avian evolutionary ecologists (e.g., Mayr 1939), although it was usually explained by the mortality cost hypothesis (Wittenberger 1976). Our analyses do not support this latter hypothesis because sex-biased SSD is not associated with sex-biased juvenile or adult mortality in the studied amniote species, and the results of the confirmatory path analyses are also inconsistent with the mortality cost hypothesis. We propose that the lack of relationship between SSD and sex differences in mortality may be explained by variation in the environmental context (Lemaître et al. 2020). Studies in birds and mammals showed that having a large body size may only be costly in terms of mortality in populations subjected to harsh environmental conditions (Toïgo and Gaillard 2003; Kalmbach and Benito 2007; Jones et al. 2009;

Clutton-Brock 2017). The effect of SSD may thus be reduced or absent when the sex-specific mortality estimates correspond to average conditions, which may often be the case in wild populations.

The ASR-SSD relationship may also be influenced by sex differences in the time of maturation because longer maturation time in the larger sex can result in a shortage of that sex in the adult population (Lovich et al. 2014) because immature life stages are generally characterized by higher mortality (e.g., Gaillard et al. 2000). Furthermore, Fromhage and Jennions (2016) showed that female-skewed sex ratios at maturation (MSR) can result in the evolution of increased female care and male allocation to traits facilitating mating success. Thus, if variation in ASR is determined at least in part by MSR, then the effects of sex-biased MSR on sex roles can contribute to the observed association of ASR with the intensity of mating competition, and, hence, SSD. This latter mechanism would deserve further investigations.

Although the relationship between ASR and SSD is not statistically significant in reptiles, it is qualitatively consistent with our findings in birds and mammals. Other selective processes (e.g., fertility selection for large female size in indeterminate growers; Cox et al. 2007) might have masked the influence of sexual selection on SSD in reptiles. Consistent with this explanation, selection often favors delayed maturation in female reptiles, which enables them to produce larger clutches, which in turn also influences their body size and the extent of SSD (Shine 2005; Agha et al. 2018). Follow-up studies using different proxies of sexual selection are needed to investigate further how sexual selection is related to ASR in reptiles.

Biased estimates of ASR may generate spurious relationship with SSD, which may potentially affect our results. For example, the larger sex may have lower detectability in polygamous species if some members of that sex are excluded from breeding sites (Ancona et al. 2017). However, highly polygamous species in which populations have been thoroughly surveyed showed skewed ASR even when all individuals in the population were accurately counted (Granjon et al. 2017), and fairly consistent ASR estimates were obtained when both breeding and nonbreeding individuals were included (Emlen and Wrege 2004). In general, ASR estimates show a moderate but statistically significant repeatability across populations in most of the studied taxa, except reptiles with temperature-dependent sex determination (Ancona et al. 2017; Bókony et al. 2019; Valentine Federico, J-FL, J-MG, AL, IP, and TS unpubl. result), and in 80% of bird species the direction of ASR skew is the same for all repeated estimates (Székely et al. 2014a).

The paths of causality in comparative data are difficult to untangle. Path analysis is a valuable tool for contrasting different causal models, although it cannot reveal causality (Shipley 2016). Path analysis assumes that each variable includes independent variations or "errors" and that these errors are independent among variables. This is not true for comparative data, because the errors will be correlated across species. Our approach follows Santos (2012), an innovative but overlooked method that satisfies the assumptions of path analysis better than an alternative method based on phylogenetic regressions proposed by von Hardenberg and Gonzalez-Voyer (2013). This latter approach is problematic because it is not robust to changes in the specification of the model: if variable Y is regressed on X and λ estimated, then the estimates of the partial correlations and λ may be different from those obtained if Y is regressed on X with λ estimated (Appendix S3). The approach we have taken avoids this problem. However, there is still room for methodological improvement. For instance, our approach has the drawback of being a "subtractive" comparative method (sensu Harvey and Pagel 1991). The question of how to robustly fit complex path models for data on multiple traits with different levels of phylogenetic signal is not straightforward.

Concluding Remarks

Our findings indicate that sex-specific selection for large body size is associated with skewed ASRs across amniotes, and this process appears to produce SSD biased toward the rare sex in birds and mammals. Although this conclusion contrasts with Darwin's initial suggestion that intense sexual selection among males occurs when there is a surplus of males in the population (Darwin 1871), theoretical and empirical works have suggested mechanisms that can favor large size in the rare sex (Murray 1984; Klemme et al. 2007; Fitze and Le Galliard 2008; Dreiss et al. 2010). Further analyses of these processes and their application to species with differing mating systems offer exciting opportunities for future investigations of the interplay among sexual selection, SSD, and ASR across the tree of life.

AUTHOR CONTRIBUTIONS

AL conceived the study. AL, RPF, and TS designed the analyses. AL, IP, VB, J-FL, and J-MG collected data. AL conducted the analyses with input from RPF. All authors wrote this article.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

DATA ARCHIVING

All data and their references are provided in Appendix S1 archived in Dryad (https://doi.org/10.5061/dryad.5qfttdz56).

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Supporting Information

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

Table S1.Relationship between SSD, ASR, and sex-biased mortalities in reptiles, using estimated body mass data for SSD calculation.
Table S2.Relationship between SSD, ASR, and sex-biased mortalities in reptiles, using body length data for SSD calculation.
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Appendix 3. Methodological notes on path analyses applied to comparative data, and additional path analyses.