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

András Liker[1,2,*], Veronika Bókony[3], Ivett Pipoly[1], Jean-Francois Lemaitre[4], Jean-Michel Gaillard[4], Tamás Székely[5,6], Robert P. Freckleton[7]

[1] MTA-PE Evolutionary Ecology Research Group, University of Pannonia, H-8210 Veszprém, Pf. 1158, Hungary
[2] Behavioral Ecology Research Group, Center for Natural Sciences, University of Pannonia, H-8210 Veszprém, Pf. 1158, Hungary
[3] Lendület Evolutionary Ecology Research Group, Plant Protection Institute, Centre for Agricultural Research, Eötvös Loránd Research Network, Herman Ottó u. 15, H-1022 Budapest, Hungary
[4] Université Lyon 1, CNRS, Laboratoire de Biométrie et Biologie Évolutive UMR 5558, F-69622, Villeurbanne, France
[5] Milner Centre for Evolution, Department of Biology and Biochemistry, University of Bath, Bath BA2 7AY, UK
[6] Department of Evolutionary Zoology and Human Biology, University of Debrecen, H-4032, Hungary
[7] Department of Animal and Plant Sciences, Alfred Denny Building, University of Sheffield, Western Bank, Sheffield S10 2TN, UK

[*] **Corresponding author**: András Liker, MTA-PE Evolutionary Ecology Research Group, University of Pannonia, Pf. 1158., H-8210 Veszprém, Hungary. Tel.: +36 88 624249, Fax: +36 88 624747, e-mail: aliker@almos.uni-pannon.hu

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Abstract

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 (the proportion of males in the adult population, ASR) 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-biased 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 towards 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.

Keywords: sexual selection, mating competition, mating opportunity, sex-biased mortality, comparative method

INTRODUCTION

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 intra-sexual competition for access to mates or inter-sexual 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 non-human 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 wrote (1871, p. 217): *"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 like 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 towards the opposite sex than when it is skewed towards 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. While 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. Since 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 in Supporting Information). 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, 122 mammals).

Body mass and SSD

Sex-specific body mass (g) was available for all birds and mammals in our dataset. Since 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 non-existent" 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, slope ± *SE*: 1.0096 ± 0.0102, 95% CI: 0.989 ≤ β ≤ 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 remains 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 were 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 ≥ 10 individuals and the dataset has ≥ 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 V.B. and I.P. (Pipoly et al. 2015; Bókony et al. 2019), birds by A.L. (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), mammals: ICC= 0.60 (Valentine Federico, J-F.L., J-M.G., A.L., I.P., T.S. unpublished 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 V.B. (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 A.L. 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-M.G. and J-F.L. (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 (phylogenetic generalized least squares models, juvenile mortality bias: slope ± *SE* = - 0.068 ± 0.101, $t = 0.7$, $P = 0.497$, $n = 100$; adult mortality bias: slope ± *SE* = - 0.05 ± 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 unpublished 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 non-acrobatic 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. 2019).

Statistical analyses

Phylogenetic generalized least squares (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). Since 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 the paper, 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 the paper). 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

385 determination system, and breeding latitude. In Multi-predictor model 2, we tested the ASR -
386 SSD relationships while controlling for the effects of sex differences in juvenile and adult
387 mortality rates, and mean mass. We built these two separate multi-predictor models because
388 we have much lower sample sizes for sex-specific mortalities than for the other predictors,
389 thus the statistical power would be reduced for variables of Multi-predictor model 1 if all
390 predictors were combined in a single model. We ran the models in two alternative versions in
391 which either SSD or ASR was the dependent variable, respectively, since we had no *a priori*
392 knowledge about the cause-effect direction of these relationships and results may differ
393 between these analyses if the two models have different values for Pagel's λ (see Appendix
394 S3).
395 We investigated whether the ASR – SSD relationship, which is the main focus of our
396 study, differed among taxa by testing the interaction between ASR and the taxonomic class.
397 To explore differences among taxa in the multivariate relationships, we repeated all analyses
398 separately for reptiles, birds and mammals. In taxon-specific Multi-predictor models 1, we
399 included reproductive mode for reptiles and display type for birds as further predictors. In
400 reptiles, we also tested whether the relationship between ASR and SSD is sensitive (1) to the
401 inclusion of species that have environmental sex determination, because ASR shows low
402 repeatability in such reptiles (Bókony et al. 2019), and (2) to the inclusion of species in which
403 the type of sex determination was inferred from data on related species in the genus or family.
404 Finally, we ran two additional separate analyses to test whether social mating system and
405 environmental harshness confounded the ASR - SSD relationship. All numeric variables were
406 standardized before analyses to make parameter estimates comparable, and model
407 assumptions were also checked and met. We report two-tailed statistics. Sample sizes differed
408 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, while Models 1b-c 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-c).

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 non-significant 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 non-directional 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

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 S1-4). 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 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 towards 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 significant, see above) also yielded results qualitatively consistent with the full dataset (Table S9).

DISCUSSION

Our analyses provided three major findings: (1) adult sex ratio is related to sexual size dimorphism 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 and 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-biased and male-biased populations, respectively; Murray 1984). Since 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, that 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 & 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 non-breeding 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-F.L., J-M.G., A.L., I.P., T.S. unpublished 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 3). 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 towards 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

608 population (Darwin 1871), theoretical and empirical work have suggested mechanisms that
609 can favor large size in the rare sex (Murray 1984; Klemme et al. 2007; Fitze and Le Galliard
610 2008; Dreiss et al. 2010). Further analyses of these processes and their application to species
611 with differing mating systems offer exciting opportunities for future investigations of the
612 interplay among sexual selection, SSD and ASR across the tree of life.
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869 **Table 1.** Phylogenetically corrected analyses of sexual size dimorphism (SSD) and adult sex
870 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**			

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873 Results of separate predictor and multi-predictor phylogenetic generalized least-squares
874 (PGLS) models with either (A) SSD ($\log_{10}$(male mass/female mass)) or (B) ASR (proportion
875 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, n is number of species.

* λ statistically different from 0, ǂ 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 S1. 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 *P*-value for Fisher's *C* statistic for model fit, with non-significant 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. The analyses include 97 species of reptiles, birds and mammals with data for all for variables.

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

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.

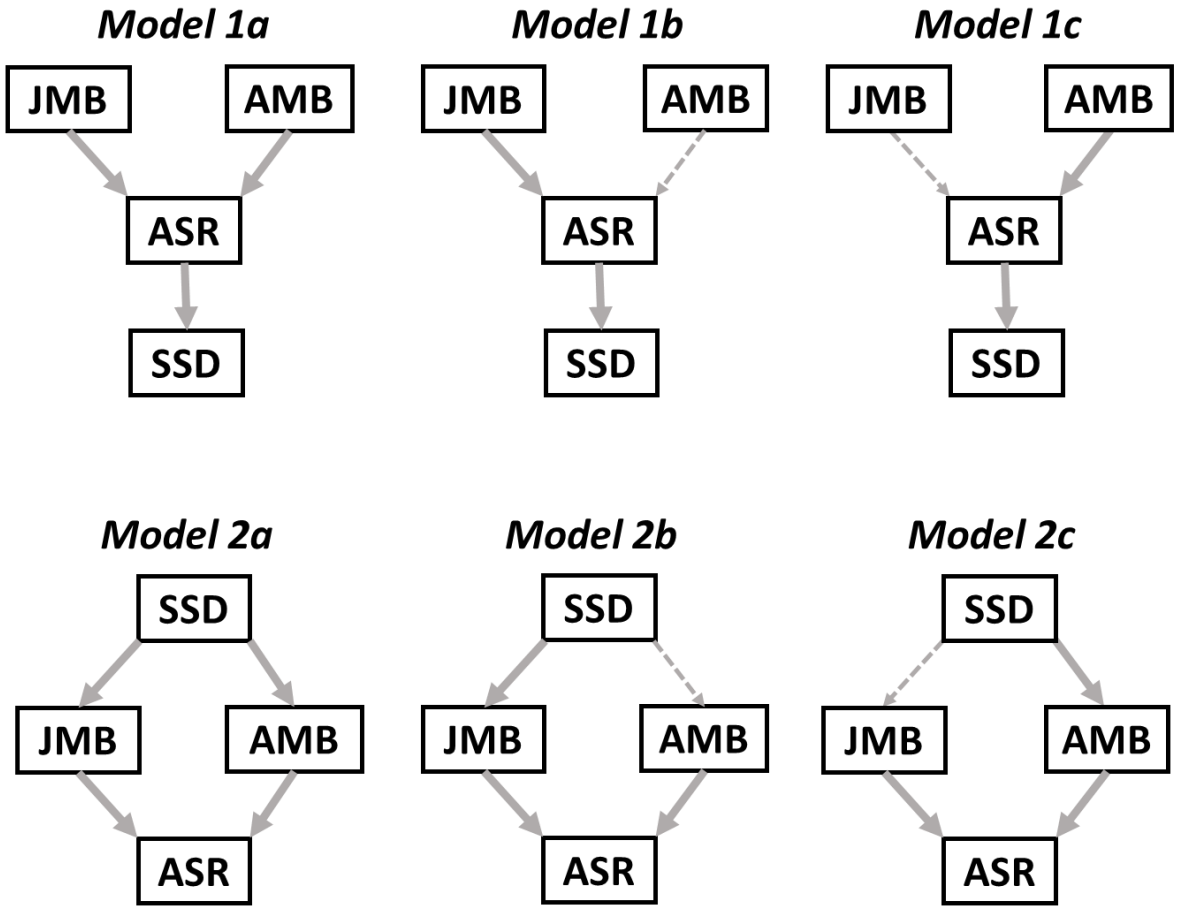
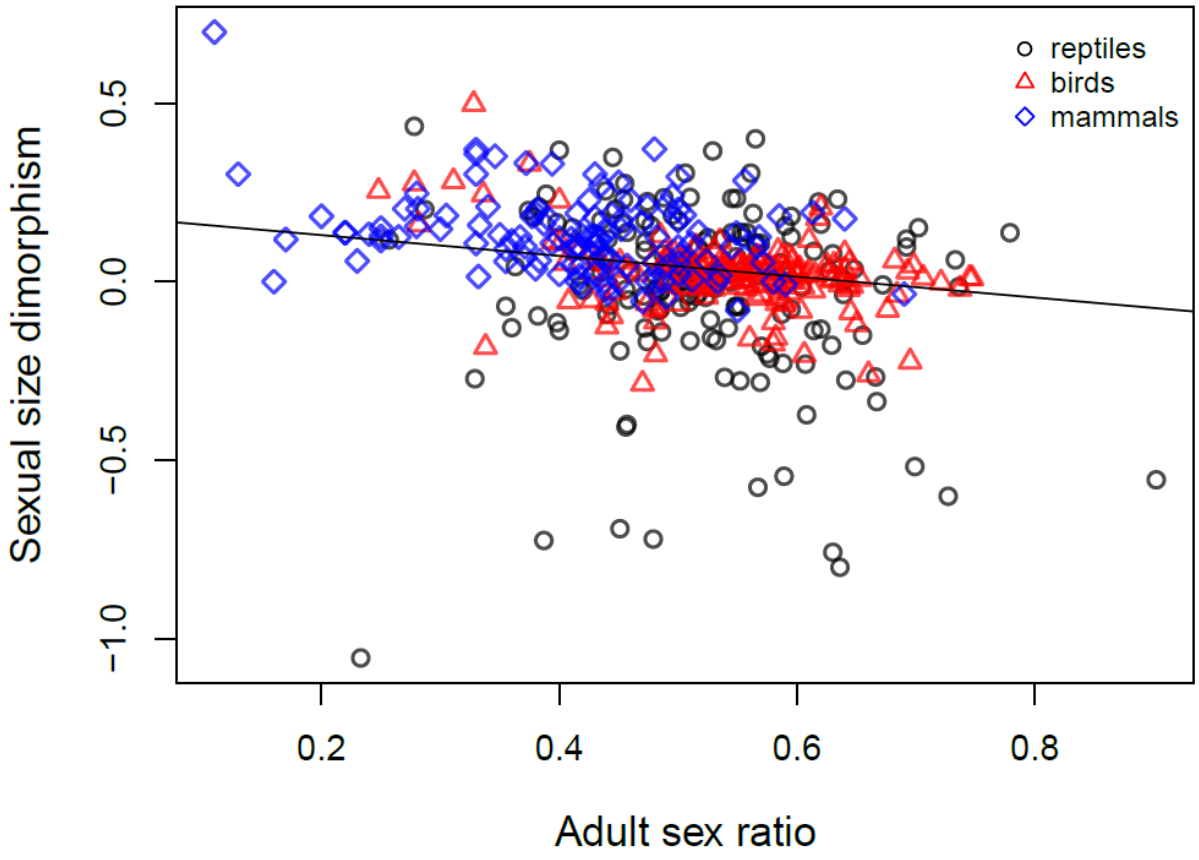


Figure 2. Sexual size dimorphism (SSD) in relation to adult sex ratio (ASR) in amniotes. SSD was calculated as $\log_{10}$(male mass/female mass); ASR is the proportion of males in the adult population. Each data point represents a species; the regression line is fitted by phylogenetic generalized least-squares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



945 **Figure 3**. Path diagram of the best-fitting phylogenetic path model (Model 1a in Table 2, *n* =
946 97 species of reptiles, birds and mammals). The model supports the scenario that sex-biased
947 juvenile and adult mortalities lead to skewed adult sex ratio, which in turn results in increased
948 size dimorphism by sexual selection. Width of the arrows is proportional to path coefficients
949 (see Table 2 for statistical details of the model). Bird pictures on the left illustrate the case
950 when differential mortality generates female-skewed ASR, which then leads to a more male-
951 biased SSD (i.e. larger body size in males relative to females). The path analyses were based
952 on the approach proposed by Santos (2012), see Appendix S3 for details.
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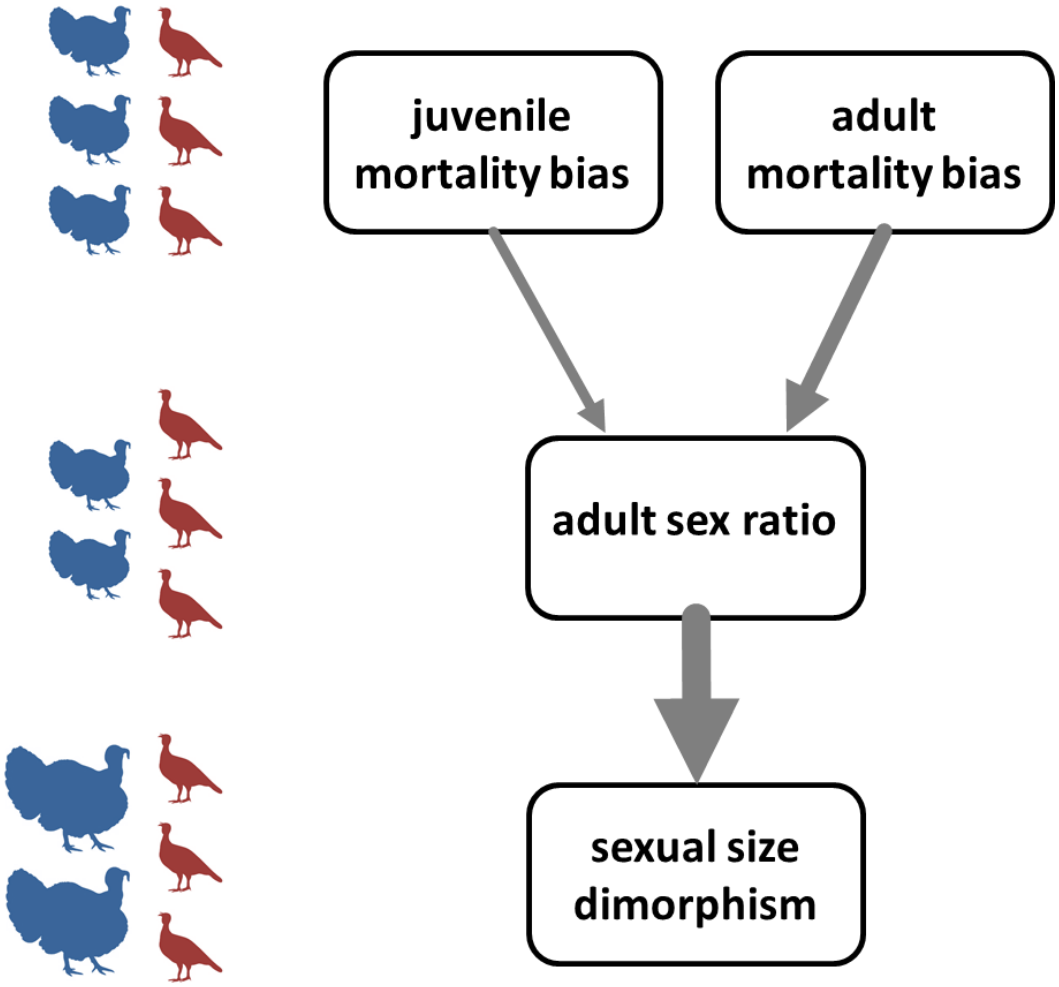

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Electronic Supporting Information: tables and figures

Table S1. Relationship between SSD, ASR and sex-biased mortalities in reptiles, using estimated body mass data for SSD calculation.

Predictors	$b \pm SE$	t	P	R^2	λ	n
(A) Response: sexual size dimorphism						
Separate predictor models:						
Model 1				*0.082*	*0.948 **	*155*
ASR	- 0.123 ± 0.075	1.641	0.103			
Mean body mass	0.668 ± 0.177	3.774	**< 0.001**			
Model 2				*0.005*	*0.0*	*17*
Juvenile mortality bias	- 0.414 ± 0.337	1.228	0.240			
Mean body mass	- 0.500 ± 0.440	1.136	0.275			
Model 3				*0.092*	*1.0 **	*62*
Adult mortality bias	- 0.151 ± 0.117	1.287	0.203			
Mean body mass	0.737 ± 0.317	2.324	**0.024**			
Multi-predictor model 1:				*0.116*	*0.956 **	*153*
ASR	- 0.090 ± 0.075	1.203	0.231			
Mean body mass	0.715 ± 0.178	4.019	**< 0.001**			
Latitude	- 0.175 ± 0.126	1.389	0.167			
Reproductive mode [1]	0.348 ± 0.313	1.112	0.268			
Sex determination, TSD [2]	- 0.463 ± 0.384	1.206	0.230			
Sex determination, ZW [2]	- 1.003 ± 0.313	2.344	**0.020**			
Multi-predictor model 2:				*< 0.001*	*0.0*	*17*
ASR	- 0.022 ± 0.252	0.086	0.933			
Mean body mass	- 0.452 ± 0.523	0.865	0.404			
Juvenile mortality bias	- 0.500 ± 0.374	1.339	0.205			
Adult mortality bias	0.284 ± 0.429	0.662	0.520			
(B) Response: adult sex ratio						
Separate predictor models:						
Model 1: SSD	- 0.074 ± 0.061	1.209	0.228	0.003	0.171 [#]	155
Model 2: Juvenile mortality bias	- 0.480 ± 0.415	1.156	0.266	0.021	0.0	17
Model 3: Adult mortality bias	- 0.159 ± 0.092	1.732	0.088	0.032	0.155 [#]	62
Multi-predictor model 1:				*0.078*	*0.0* [#]	*153*
SSD	- 0.049 ± 0.055	0.891	0.374			
Mean body mass	0.173 ± 0.108	1.599	0.112			
Latitude	- 0.001 ± 0.109	0.013	0.990			
Reproductive mode [1]	- 0.140 ± 0.216	0.650	0.517			
Sex determination, TSD [2]	0.209 ± 0.224	0.934	0.352			
Sex determination, ZW [2]	0.667 ± 0.216	3.091	**0.002**			
Multi-predictor model 2:				*0.165*	*0.0* [#]	*17*
SSD	- 0.028 ± 0.331	0.086	0.933			
Mean body mass	0.929 ± 0.556	1.671	0.121			
Juvenile mortality bias	- 0.044 ± 0.459	0.095	0.926			
Adult mortality bias	- 0.641 ± 0.465	1.377	0.194			

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961 * Pagel's lambda statistically different from 0, ♯ lambda statistically different from 1.
962 [1] Differences from oviparous species.
963 [2] Differences from XY species; overall effect of sex determination on SSD: $F_{2,146} = 2.8$, $P =$
964 0.066; on ASR: $F_{2,146} = 5.2$, $P = 0.006$.
965 For further explanation, see the footnotes of Table 1 in the main text.

Table S2. Relationship between SSD, ASR and sex-biased mortalities in reptiles, using body length data for SSD calculation.

Predictors	*b* ± *SE*	*t*	*P*	R^2	*λ*	*n*
(A) Response: sexual size dimorphism						
Separate predictor models:						
Model 1				*0.073*	*0.935 **	*155*
ASR	- 0.008 ± 0.005	1.587	0.114			
Mean body mass	0.040 ± 0.011	3.562	< **0.001**			
Model 2				*0.073*	*0.0*	*17*
Juvenile mortality bias	- 0.035 ± 0.024	1.472	0.163			
Mean body mass	- 0.046 ± 0.031	1.485	0.160			
Model 3				*0.086*	*1.0 **	*62*
Adult mortality bias	- 0.010 ± 0.007	1.402	0.166			
Mean body mass	0.044 ± 0.020	2.156	**0.035**			
Multi-predictor model 1:				*0.122*	*0.952 **	*153*
ASR	- 0.048 ± 0.042	1.126	0.262			
Mean body mass	0.391 ± 0.100	3.891	< **0.001**			
Latitude	- 0.103 ± 0.071	1.459	0.147			
Reproductive mode [1]	0.179 ± 0.177	1.015	0.312			
Sex determination, TSD [2]	- 0.223 ± 0.216	1.032	0.304			
Sex determination, ZW [2]	- 0.633 ± 0.241	2.628	**0.010**			
Multi-predictor model 2:				*< 0.001*	*0.0*	*17*
ASR	- 0.059 ± 0.161	0.368	0.720			
Mean body mass	- 0.347 ± 0.334	1.038	0.320			
Juvenile mortality bias	- 0.354 ± 0.239	1.484	0.164			
Adult mortality bias	0.092 ± 0.274	0.337	0.742			
(B) Response: adult sex ratio						
Separate predictor models:						
Model 1: SSD	- 0.131 ± 0.109	1.209	0.229	0.003	0.169 ǂ	155
Model 2: Juvenile mortality bias	- 0.480 ± 0.415	1.156	0.266	0.021	0.0	17
Model 2: Adult mortality bias	- 0.159 ± 0.092	1.732	0.088	0.032	0.155 ǂ	62
Multi-predictor model 1:				*0.078*	*0.0* ǂ	*153*
SSD	- 0.085 ± 0.098	0.870	0.386			
Mean body mass	0.172 ± 0.109	1.588	0.114			
Latitude	- 0.001 ± 0.109	0.007	0.994			
Reproductive mode [1]	- 0.141 ± 0.216	0.654	0.514			
Sex determination, TSD [2]	0.214 ± 0.223	0.958	0.340			
Sex determination, ZW [2]	0.667 ± 0.216	3.089	**0.002**			
Multi-predictor model 2:				*0.174*	*0.0* ǂ	*17*
SSD	- 0.188 ± 0.512	0.368	0.720			
Mean body mass	0.867 ± 0.570	1.522	0.154			
Juvenile mortality bias	- 0.096 ± 0.463	0.208	0.839			
Adult mortality bias	- 0.624 ± 0.457	1.366	0.197			

* Pagel's lambda statistically different from 0, ǂ lambda statistically different from 1.
[1] Differences from oviparous species.

971 [2] Differences from XY species; overall effect of sex determination on SSD: $F_{2,146} = 3.7$, $P =$
972 0.028; on ASR: $F_{2,146} = 5.2$, $P = 0.006$.
973 For further explanation, see the footnotes of Table 1 in the main text.
974

Table S3. Relationship between SSD, ASR and sex-biased mortalities in birds.

Predictors	$b \pm SE$	t	P	R^2	λ	n
(A) Response: sexual size dimorphism						
Separate predictor models:						
Model 1				*0.250*	*0.812 *#*	*185*
ASR	- 0.242 ± 0.037	6.625	< **0.001**			
Mean body mass	0.293 ± 0.105	2.798	**0.006**			
Model 2				*0.130*	*0.095 **	*47*
Juvenile mortality bias	0.066 ± 0.073	0.898	0.374			
Mean body mass	0.735 ± 0.270	2.722	**0.009**			
Model 3				*0.072*	*0.708 *#*	*123*
Adult mortality bias	0.068 ± 0.051	1.335	0.184			
Mean body mass	0.372 ± 0.130	2.870	**0.005**			
Multi-predictor model 1:				*0.272*	*0.720 *#*	*178*
ASR	- 0.242 ± 0.038	6.390	< **0.001**			
Mean body mass	0.261 ± 0.100	2.599	**0.010**			
Latitude	- 0.020 ± 0.034	0.584	0.560			
Display type, agile [1]	- 0.338 ± 0.090	3.748	< **0.001**			
Multi-predictor model 2:				*0.386*	*1.0 **	*47*
ASR	- 0.346 ± 0.080	4.318	< **0.001**			
Mean body mass	0.424 ± 0.246	1.719	0.093			
Juvenile mortality bias	- 0.032 ± 0.065	0.489	0.627			
Adult mortality bias	- 0.068 ± 0.089	0.763	0.450			
(B) Response: adult sex ratio						
Separate predictor models:						
Model 1: SSD	- 0.746 ± 0.114	6.520	< **0.001**	0.184	0.480 *#	185
Model 2: Juvenile mortality bias	- 0.354 ± 0.115	3.084	**0.003**	0.156	0.0 #	47
Model 3: Adult mortality bias	- 0.384 ± 0.079	4.866	< **0.001**	0.157	0.0 #	123
Multi-predictor model 1:				*0.239*	*0.244 #*	*178*
SSD	- 0.717 ± 0.116	6.183	< **0.001**			
Mean body mass	- 0.191 ± 0.136	1.406	0.161			
Latitude	- 0.127 ± 0.058	2.201	**0.029**			
Display type, agile [1]	- 0.589 ± 0.161	3.667	< **0.001**			
Multi-predictor model 2:				*0.397*	*0.0 #*	*47*
SSD	- 0.382 ± 0.153	2.499	**0.016**			
Mean body mass	- 0.128 ± 0.198	0.646	0.522			
Juvenile mortality bias	- 0.199 ± 0.109	1.831	0.074			
Adult mortality bias	- 0.468 ± 0.139	3.368	**0.002**			

* Pagel's lambda statistically different from 0, # lambda statistically different from 1.
[1] Difference from non-agile species.
For further explanation, see the footnotes of Table 1 in the main text.

Table S4. Relationship between SSD, ASR and sex-biased mortalities in mammals.

Predictors	*b* ± *SE*	*t*	*P*	R^2	*λ*	*n*
(A) Response: sexual size dimorphism						
Separate predictor models:						
Model 1				*0.143*	*0.313 *#*	*122*
ASR	- 0.170 ± 0.058	2.946	**0.004**			
Mean body mass	0.385 ± 0.129	2.979	**0.004**			
Model 2				*0.078*	*0.233 #*	*36*
Juvenile mortality bias	0.089 ± 0.123	0.719	0.477			
Mean body mass	0.515 ± 0.233	2.214	**0.034**			
Model 3				*0.056*	*0.217 #*	*45*
Adult mortality bias	0.025 ± 0.103	0.244	0.809			
Mean body mass	0.424 ± 0.202	2.093	**0.042**			
Multi-predictor model 1:				*0.166*	*0.342 *#*	*120*
ASR	- 0.153 ± 0.058	2.646	**0.009**			
Mean body mass	0.418 ± 0.131	3.191	**0.002**			
Latitude	0.106 ± 0.053	2. 016	**0.046**			
Multi-predictor model 2:				*0.250*	*0.0 #*	*33*
ASR	- 0.374 ± 0.129	2.900	**0.007**			
Mean body mass	0.209 ± 0.237	0.880	0.386			
Juvenile mortality bias	0.088 ± 0.116	0.762	0.452			
Adult mortality bias	- 0.016 ± 0.119	0.134	0.894			
(B) Response: adult sex ratio						
Separate predictor models:						
Model 1: SSD	- 0.460 ± 0.130	3.539	< **0.001**	0.087	0.252 *#	122
Model 2: Juvenile mortality bias	- 0.032 ± 0.166	0.195	0.847	< 0.001	0.0 #	36
Model 3: Adult mortality bias	- 0.076 ± 0.155	0.493	0.624	< 0.001	0.0 #	45
Multi-predictor model 1:				*0.093*	*0.320 *#*	*120*
SSD	- 0.375 ± 0.140	2.670	**0.009**			
Mean body mass	- 0.314 ± 0.209	1.500	0.136			
Latitude	- 0.075 ± 0.083	0.907	0.366			
Multi-predictor model 2:				*0.293*	*0.0 #*	*33*
SSD	- 0.617 ± 0.213	2.900	**0.007**			
Mean body mass	- 0.494 ± 0.294	1.678	0.104			
Juvenile mortality bias	- 0.043 ± 0.150	0.285	0.778			
Adult mortality bias	0.022 ± 0.153	0.142	0.888			

* Pagel's lambda statistically different from 0, # lambda statistically different from 1.
For further explanation, see the footnotes of Table 1 in the main text.

Table S5. Sensitivity analyses of the relationship between sexual size dimorphism (SSD, dependent variable in all models) and adult sex ratio (ASR). Table shows results when (A) male mass (instead of $\log_{10}$(male mass / female mass) is used as response variable, (B) reptiles are included with SSD based on body length, (C) reptiles are included with SSD calculated from sex-specific body mass, (D) reptiles with temperature-dependent sex determination (TSD) are excluded, (E) reptiles with assumed sex determination, based on related species, are excluded, and (F) environmental harshness is included in the model.

Predictors	*b* ± *SE*	*t*	*P*	R^2	λ	*n*
(A) Male body mass as response variable (all species):				*0.957*	*0.846 *#*	*462*
ASR	- 0.020 ± 0.004	4.953	< **0.001**			
Female body mass	1.008 ± 0.010	100.658	< **0.001**			
(B) Reptiles' SSD calculated from body length (all species):				*0.139*	*0.703 *#*	*462*
ASR	- 0.234 ± 0.038	6.231	< **0.001**			
Mean body mass	0.473 ± 0.085	5.575	< **0.001**			
(C) Reptiles' SSD calculated from body mass[1] (all species):				*0.144*	*0.761 *#*	*338*
ASR	- 0.271 ± 0.050	5.437	< **0.001**			
Mean body mass	0.495 ± 0.112	4.426	< **0.001**			
(D) TSD reptiles excluded (all species):				*0.132*	*0.791 *#*	*402*
ASR	- 0.250 ± 0.043	5.767	< **0.001**			
Mean body mass	0.507 ± 0.105	4.814	< **0.001**			
(E) Reptiles with assumed sex determination excluded[2] (all species):				*0.125*	*0.860 *#*	*409*
ASR	- 0.167 ± 0.036	4.669	< **0.001**			
Mean body mass	0.502 ± 0.088	5.710	< **0.001**			
(F) Effect of environmental harshness[3]:						
birds and mammals:				*0.141*	*0.763 *#*	*219*
ASR	- 0.164 ± 0.037	4.440	< **0.001**			
Environmental harshness	0.039 ± 0.045	0.855	0.394			
Mean body mass	0.263 ± 0.088	2.966	**0.003**			
reptiles:				*0.026*	*0.957 **	*58*
ASR	- 0.081 ± 0.144	0.562	0.576			
Environmental harshness	0.105 ± 0.064	1.624	0.110			
Mean body mass	0.294 ± 0.302	0.975	0.334			
all species:				*0.111*	*0.867 *#*	*277*
ASR	- 0.153 ± 0.038	4.012	< **0.001**			
Environmental harshness	0.076 ± 0.033	2.295	**0.023**			
Mean body mass	0.297 ± 0.091	3.256	**0.001**			

* Pagel's lambda statistically different from 0, # lambda statistically different from 1

[1] Sex-specific body mass data from Myhrvold et al. (2015).

[2] Sex determination mechanism assumed to be the same type as reported for the genus or family (see Methods).

[3] The influence of environmental harshness was tested in birds and mammals using data from Botero et al. (2014), in reptiles using data calculated in this study (following the method of Botero et al 2014), and in all species by pooling the harshness scores from the two studies.

Table S6. Analyses of the relationship between SSD (dependent variable) and ASR with branch lengths calculated by three different methods for the phylogeny used in the PGLS models. The analyses included reptiles, birds, and mammals.

Predictors	$b \pm SE$	t	P	R^2	λ	n
(A) Nee's method				*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**			
(B) Pagel's method				*0.124*	*0.869 *#*	*462*
ASR	- 0.166 ± 0. 034	4.826	< **0.001**			
Mean body mass	0.564 ± 0. 090	6.282	< **0.001**			
(C) Unit branch length				*0.148*	*1.0 **	*462*
ASR	- 0.179 ± 0. 032	5.577	< **0.001**			
Mean body mass	0.565 ± 0. 085	6.682	< **0.001**			

* Pagel's lambda statistically different from 0, # lambda statistically different from 1
[1] See Methods for details of branch length calculations

Table S7. Analyses of the relationship between SSD (dependent variable) and ASR in socially monogamous and socially polygamous species, respectively. The analyses included birds and mammals.

Predictors	$b \pm SE$	t	P	R^2	λ	n
(A) Monogamy				*0.022*	*1.0 **	*109*
ASR	- 0.066 ± 0.038	1.717	0.089			
Mean body mass	0.087 ± 0.083	1.044	0.299			
(B) Polygamy				*0.222*	*0.418 *#*	*162*
ASR	- 0.223 ± 0.048	4.607	< **0.001**			
Mean body mass	0.399 ± 0.105	3.790	< **0.001**			

* Pagel's lambda statistically different from 0, # lambda statistically different from 1
[1] When monogamous and polygynous species are analyzed together, there is a statistically significant interaction between the effects of mating system and ASR ($b \pm SE = 0.218 \pm 0.087$, $t = 2.510$, $P = 0.013$).

Table S8. Results of the phylogenetic path analyses using the R package 'phylopath'. Models represent the mating opportunity hypothesis (Models 1a-c) and the mortality cost hypothesis (Models 2a-c). Analyses based on data of all species (birds, mammals, and reptiles; *n*= 97 species).

Model	*k*	*q*	*C*	*P*	*CICc*	*ΔCICc*
Model 1a	**3**	**7**	**6.4**	**0.383**	**21.6**	**0.0**
Model 1b	4	6	18.7	0.017	31.6	10.0
Model 1c	**4**	**6**	**11.2**	**0.188**	**24.2**	**2.6**
Model 2a	2	8	32.4	<0.001	50.0	28.4
Model 2b	3	7	34.8	<0.001	50.0	28.4
Model 2c	3	7	36.6	<0.001	51.9	30.3

Model structures are shown in Figure 1. SSD: sexual size dimorphism, ASR: adult sex ratio, JMB and AMB: juvenile and adult mortality biases, respectively. The table shows the number of independence claims (*k*), the number of parameters (*q*), Fisher's C statistic (*C*) and its accompanying probability (*P*), C-statistic information criterion corrected for small sample sizes (CICc), and the difference in CICc from the top model (ΔCICc). A *P*-value less than 0.05 indicates a poor model fit (i.e. rejection of the model), whereas a ΔCICc > 2 indicates substantial support for the top path model over the alternative models.

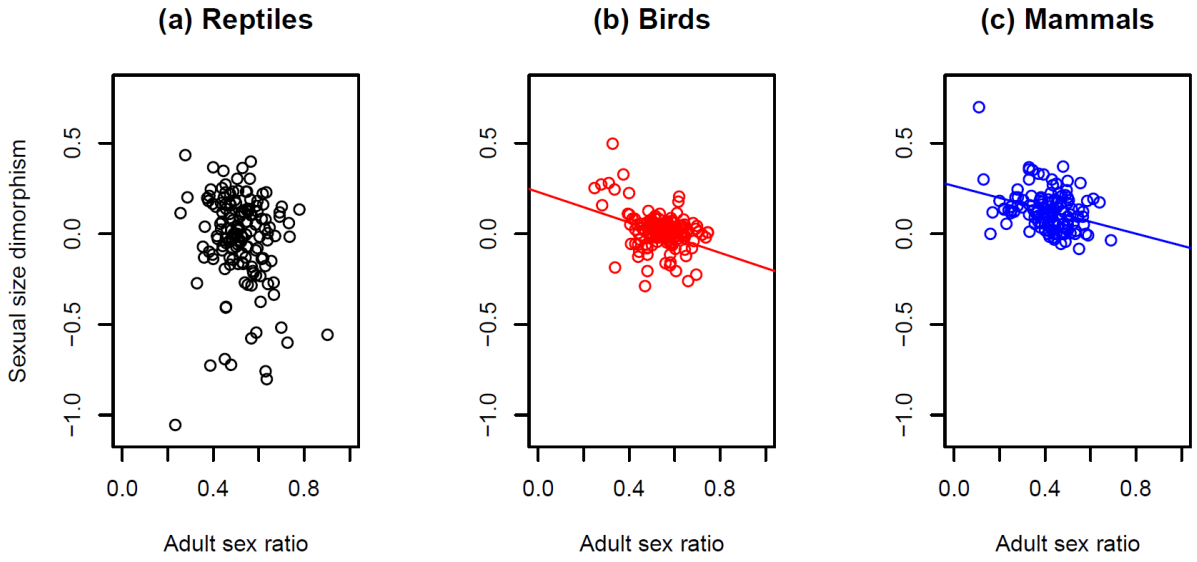
Table S9. Phylogenetic path models representing the mating opportunity hypothesis (Models 1a-c) and the mortality cost hypothesis (Models 2a-c). Analyses with data of birds and mammals (i.e. excluding reptiles; *n*= 81 species).

Model/Path	Path coefficient ± *SE*	*Z*	*P*
Model 1a	***P_C= 0.991, df= 4, AICc= 15.9, ΔAICc= 0.0***		
AMB → ASR	- 0.321 ± 0.121	- 2.662	**0.009**
JMB → ASR	- 0.210 ± 0.109	- 1.920	0.059
ASR → SSD	- 0.719 ± 0.122	- 5.887	**< 0.001**
Model 1b	***P_C= 0.142, df= 6, AICc= 23.7, ΔAICc= 7.8***		
(AMB → ASR)[1]	0	-	
JMB → ASR	- 0.257 ± 0.112	- 2.289	**0.025**
ASR → SSD	- 0.719 ± 0.122	- 5.887	**< 0.001**
Model 1c	***P_C= 0.428, df= 6, AICc= 19.7, ΔAICc= 3.8***		
AMB → ASR	- 0.358 ± 0.121	- 2.958	**0.004**
(JMB → ASR)[1]	0	-	
ASR → SSD	- 0.719 ± 0.122	- 5.887	**< 0.001**
Model 2a	*P_C= 0.0, df= 4, AICc= 59.7, ΔAICc= 43.8*		
SSD → AMB	0.117 ± 0.077	1.503	0.127
SSD → JMB	0.102 ± 0.086	1.187	0.239
AMB → ASR	- 0.321 ± 0.121	- 2.662	**0.009**
JMB → ASR	- 0.210 ± 0.109	- 1.920	0.059
Model 2b	*P_C= 0.0, df= 4, AICc= 50.5, ΔAICc= 34.6*		
SSD → JMB	0.102 ± 0.086	1.187	0.239
AMB → ASR	- 0.321 ± 0.121	- 2.662	**0.009**
JMB → ASR	- 0.210 ± 0.109	- 1.920	0.059
Model 2c	*P_C= 0.0, AICc= 50.5, ΔAICc= 34.6*		
SSD → AMB	0.117 ± 0.077	1.503	0.137
AMB → ASR	- 0.321 ± 0.121	- 2.662	**0.009**
JMB → ASR	- 0.210 ± 0.109	- 1.920	0.059

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 *P*-value for Fisher's *C* statistic for model fit, with non-significant 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 model.

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

Figure S1. Sexual size dimorphism in relation to adult sex ratio in (a) reptiles (PGLS, b ± *SE* = - 0.123 ± 0.075, *P* = 0.103, *n* = 155 species), (b) birds (*b* ± *SE* = - 0.242 ± 0.037, *P* < 0.001, *n* = 185), and (c) mammals (*b* ± *SE* = - 0.170 ± 0.058, *P* = 0.004, *n* = 122). Each data point represents a species, and lines show statistically significant regressions fitted by PGLS (see Tables S1-4 for further statistical details).



Electronic Supporting Information: Appendix S1

References for data sources are provided in a separate excel file. The full dataset will be published together with references after the manuscript is accepted for publication.

Electronic Supporting Information: Appendix S2

Parameters of the allometric equations between body length and body mass (log10(mass in g) = *a* + *b**log10(length in mm)) used for the calculation of mass estimates in reptiles. *n* is the number of species included in the analyses.

Taxon	**Intercept (*a*)**	**Slope (*b*)**	***n***	**Reference**
Snakes	-5.773	2.786	336	Feldman and Meiri (2013)
Squamates and crocodilians	-4.52	2.923	600	Meiri (2010)
Turtles	-3.535	2.887	199	Regis and Meik (2017)

References:

Feldman, A., and S. Meiri. 2013. Length-mass allometry in snakes. Biol. J. Linn. Soc. 108:161–172.

Meiri, S. 2010. Length-weight allometries in lizards. J. Zool. 281:218–226.

Regis, K. W., and J. M. Meik. 2017. Allometry of sexual size dimorphism in turtles: a comparison of mass and length data. PeerJ 5:e2914.

Electronic Supporting Information: Appendix S3

Methodological notes on path analyses applied to comparative data

Several approaches have been proposed for applying path analysis in phylogenetic comparative studies (von Hardenberg and Gonzalez-Voyer 2013). However, some methodological aspects of this method still pose challenges for its phylogenetic applications. Here we discuss two of such aspects: (1) the problem of using bivariate phylogenetic generalized least squares (PGLS) models to estimate correlations between variables in the path models, and (2) the reliability of AIC statistics to compare non-nested path models. We suggest ways to avoid these problems which may help further applications of path analysis to phylogenetic data.

1. The problem of using PGLS in path analyses

The analysis of multivariate dependent data is a notoriously thorny problem. In this dataset we, as in common with many similar analyses, had to deal with the problem of phylogenetic non-independence (Harvey and Pagel 1991). Techniques for analyzing cause-effect relationships using linear models are well developed (Felsenstein 1985; Harvey and Pagel 1991; Hansen and Martins 1996; Pagel 1997; Freckleton et al. 2002). These techniques are designed for analysis of data in which there is a dependent variable of interest, which is modelled as a function of independent predictors. In these models the effect of phylogeny is accounted for by modelling phylogenetic dependence in the residual term, and we have used these for several analyses (e.g. Table 1 in the main text). However, analyses of data in which variables are treated as multivariate responses are much less common. Phylogenetic principal components analysis is one exception, although recent research has stressed that this may be more complex than previously realized (Uyeda et al. 2015).

Path analysis is a method of multivariate trait analysis that allows complex dependencies among variables to be modelled. von Hardenberg & Gonzalez-Voyer (2013) presented a method for performing path analysis on phylogenetically dependent data. This approach accounts for phylogenetic non-independence through constructing a series of bivariate PGLS models in which one variable is treated as the dependent variable, and the other is the independent variable. Importantly, this approach permits variable levels of

dependence to be modelled through estimating Pagel's λ, which accounts for varying contributions of phylogeny to trait variation.

However the analysis of such data is complex. Consider a simple example, in which we have three variables *x*, *y*, and *z*. If we model $x \rightarrow y$ (i.e. where *x* is the predictor and *y* is the response variable) using the PGLS-λ approach, we might well estimate a different correlation than if we model $y \rightarrow x$. This is because in PGLS the estimate of λ depends on the direction of the relationship, and this affects the estimate of the correlation. In path analysis correlations between pairs of variables are the input and the directionality of the statistical model should not be an issue. Specifically, the net correlation r_{xz} should then be the product of the component pairwise correlations, i.e. $r_{xz} = r_{xy} \times r_{yz}$. However, if we use PGLS and $\lambda \neq 0$ and $\lambda \neq 1$ (as in most cases in our analyses, see Table 1 in the main text), then in general $r_{xz} = r_{xy} \times r_{yz} \neq r_{yx} \times r_{yz} = r_{xz}$. We would therefore prefer to avoid an arbitrary decision about the directionality of the model to affect the estimate of the coefficient.

In our analyses we overcame the above difficulties by avoiding the use of bivariate PGLS models to estimate correlations between the variables in the path models. We followed the approach proposed by Santos (2012), in which first we calculated λ for each individual variable and conducted a phylogenetic transformation on the variable using that estimate of λ. Then we fitted path models to these already transformed data using ordinary fitting methods developed for fitting path models to non-phylogenetic data. Details of the analyses are described in the Methods section of the main text.

2. The problem of comparing non-nested path models by AIC statistics

In phylogenetic comparative studies the direction of causality between variables is often unknown, and different evolutionary hypotheses may propose opposing cause - effect relationships (like the mating competition and the mortality cost hypotheses in our study, see Fig. S1). These hypotheses may be represented by different path models, and then their fit to the data can be compared by some comparative fit indices, most commonly by AIC (West et al. 2012). However, simulations suggest that conclusions of path model comparisons based on information theory approach (like AIC) can be unreliable (Preacher and Merkle 2012). In addition the competing models can be non-nested (non-hierarchical) (e.g. Models 1a versus 2a in Fig. S1), for which AIC-based comparison should be applied with caution (Kline 2015).

To explore the problem of model comparison in the context of our study, first we fitted our path models to the real dataset by two alternative methods: (1) by covariance matrix

comparison, as implemented in the R package *lavaan* (Rosseell 2012), and (2) by piecewise structural equation modelling (or d-separation) method, as implemented in the *piecewiseSEM* (Lefcheck 2016) package. We compared path coefficient estimates and various model fit indices between these two methods to evaluate whether they produce consistent conclusions. Second, we used the same two methods and R implementations to fit the models to simulated datasets, and tested which of the methods produces more reliable (less biased) model comparisons.

2.1. Fitting path models to real data

The general steps of model fitting procedure we followed in this study are described in the Methods section of the main text. We performed model fitting with the two R packages *piecewiseSEM* and *lavaan*. In *piecewiseSEM* and *lavaan* the global model fit for each individual path model is evaluated by Fisher's *C* and χ^2 statistics, respectively, where a statistically non-significant result means acceptable fit. In *lavaan*, several other measures for model fit of individual models are also available, and here we report four of the most widely used indices (TLI, CFI, RMSEA, SRMR). It has been proposed that that the values of TLI and CFI > 0.95, RMSEA < 0.06, and SRMR < 0.08 indicate acceptable/good fit of models to the data (West et al. 2012).

We found that the two methods produced highly consistent estimates for the standardized path coefficients in all path models (*piecewiseSEM*: Table 1 in the main text, *lavaan*: Table S9 below). The effect of juvenile mortality on ASR was marginally not significant in most *piecewiseSEM* models whereas it was significant with all *lavaan* models. For all other relationships the two methods produced consistent results.

The two methods also produced highly consistent results for model fit as evaluated by global fit indices (i.e. *C* and χ^2 statistics, respectively, see Table S10). The only difference was that for Model 1b *piecewiseSEM* indicated 'marginally acceptable' model fit whereas *lavaan* indicated poor model fit for this path model. The other fit indices (TLI, CFI, RMSEA, and SRMR) suggest conclusions that are fully consistent with *C* statistics and χ^2 tests, i.e. acceptable fit for Models 1a and 1c by all of these indices and unacceptable fit for all other models (Table S10).

Table S9. Estimates of standardized path coefficients for the six path models representing various relationships between SSD, ASR, and sex biases in adult (AMB) and juvenile (JMB) mortality, obtained by the R package *lavaan* (see Fig. S1 for model details). Significant relationships are highlighted in bold.

Model/Path	**Path coefficient ± SE**	**Z**	**P**
Model 1a			
AMB → ASR	**- 0.340 ± 0.112**	**- 3.048**	**0.002**
JMB → ASR	**- 0.205 ± 0.102**	**- 2.002**	**0.045**
ASR → SSD	**- 0.657 ± 0.107**	**- 6.144**	**0.000**
Model 1b			
(AMB → ASR)[1]	0	-	-
JMB → ASR	**- 0.258 ± 0.105**	**- 2.443**	**0.015**
ASR → SSD	**- 0.657 ± 0.107**	**- 6.144**	**0.000**
Model 1c			
AMB → ASR	**- 0.378 ± 0.112**	**- 3.370**	**0.001**
(JMB → ASR)[1]	0	-	-
ASR → SSD	**- 0.657 ± 0.107**	**- 6.144**	**0.000**
Model 2a			
SSD → AMB	0.117 ± 0.070	1.680	0.093
SSD → JMB	0.089 ± 0.077	1.157	0.247
AMB → ASR	**- 0.340 ± 0.110**	**- 3.092**	**0.002**
JMB → ASR	**- 0.205 ± 0.101**	**- 2.031**	**0.042**
Model 2b			
SSD → JMB	0.089 ± 0.077	1.157	0.247
AMB → ASR	**- 0.340 ± 0.110**	**- 3.092**	**0.002**
JMB → ASR	**- 0.205 ± 0.101**	**- 2.031**	**0.042**
Model 2c			
SSD → AMB	0.117 ± 0.070	1.680	0.093
AMB → ASR	**- 0.340 ± 0.110**	**- 3.092**	**0.002**
JMB → ASR	**- 0.205 ± 0.101**	**- 2.031**	**0.042**

[1] Path coefficient set to zero

Table S10. Fit indices for the six path models, obtained by *piecewiseSEM* and *lavaan*. Values indicating acceptable fit are highlighted in bold.

Model	piecewiseSEM				lavaan						
	C	***df***	P_c		χ^2	***df***	P_{χ^2}	**TLI**	**CFI**	**RMSEA**	**SRMR**
1a	**0.29**	**4**	**0.972**		**0.02**	**2**	**0.991**	**1.119**	**1.000**	**0.000**	**0.004**
1b	**11.6**	**6**	**0.065**		8.9	3	0.031	0.764	0.858	0.143	0.101
1c	**6.2**	**6**	**0.376**		**3.9**	**3**	**0.267**	**0.962**	**0.977**	**0.057**	**0.065**
2a	34.7	4	0.000		30.6	2	0.000	-0.978	0.341	0.386	0.154
2b	34.7	4	0.000		30.6	2	0.000	-0.719	0.313	0.386	0.154
2c	34.7	4	0.000		30.6	2	0.000	-0.661	0.336	0.386	0.154

2.2. AIC-based model comparisons using real and simulated data

To assess which of these models provides the best account of the data, first we calculated the AIC value for each model (in *piecewiseSEM* this is corrected for small sample size, i.e. AIC_c) using the real dataset. Second, we used simulated data to test which of the two methods produces less biased conclusions. For this latter purpose, we generated simulated datasets using the R function 'rnorm'. The simulated datasets have the same number of variables and sample size as the phylogenetically transformed real dataset. We fitted path models with both *piecewiseSEM* and *lavaan* to obtain the AIC (or AIC_c) values. Then we compared Model 1a (the model that got the highest support for model fit by the global fit indices, see Table S10) to the other five models (Models 1b,1c, 2a, 2b, and 2c), thus conducted five pairwise comparisons, repeated with the two methods. These paired comparisons between models mimic the comparison we conducted with the real dataset in our study (Table 2 in the main text). We calculated ΔAIC for each comparison as the difference between AIC values of the two models (i.e. AIC of compared model - AIC of Model 1a, thus a positive ΔAIC value indicates better fit for Model 1a). We repeated this procedure with 1000 simulated datasets that resulted in 1000 ΔAIC values for each pairwise comparison. To assess whether the comparison of two particular models produces biased results with simulated data we calculated (1) the mean ΔAIC value of the 1000 runs ($\Delta AIC_{simulation}$), and (2) the probability that the simulated ΔAIC was larger than the ΔAIC value we got with the real dataset ($P_{\geq \Delta AIC\_sim}$).

Using real data, *piecewiseSEM* gave the lowest AIC_c for Model 1a (Table S11), a result consistent with global model fit evaluation (see Table S10). ΔAICc values suggested strong support for this model in all comparisons (ΔAICc ≥ 4.1, Table S11). In contrast, *lavaan* results were inconsistent with global model fit evaluation because it gave very strong support for Model 2c (Table S11), a model that had an unacceptable fit by all fit indices (see Table S10).

Table S11. AIC-based model comparison using real and simulated data by the two methods. AIC_c (*piecsewiseSEM*) and AIC (*lavaan*) values provided for all models are based on analyses of our real data. ΔAIC_{data} and $\Delta AIC_{simulation}$ show differences from Model 1a in pairwise comparisons, based on analyses of real or simulated data, respectively. $P_{\geq \Delta AIC\_sim}$ indicates the probability that analyses of random data result in as large or larger AIC differences in support for Model 1a than the ΔAIC values obtained with real data.

Model	**piecewiseSEM**					**lavaan**			
	AIC_c	**ΔAIC_{data}**	**$\Delta AIC_{simulation}$**	**$P_{\geq \Delta AIC\_sim}$**		**AIC**	**ΔAIC_{data}**	**$\Delta AIC_{simulation}$**	**$P_{\geq \Delta AIC\_sim}$**
1a	**15.8**	**0.0**	-	-		382.4	0.0	-	-
1b	25.7	9.9	-0.2	0.003		389.3	6.9	-1.0	0.004
1c	19.9	4.1	-0.9	0.042		384.3	1.9	-1.1	0.041
2a	59.0	43.2	7.4	0.0		521.3	138.9	274.3	1.0
2b	50.4	34.6	-0.1	0.0		360.0	-22.4	273.4	1.0
2c	50.4	34.6	0.4	0.0		**341.7**	**-40.7**	273.4	1.0

Using simulated data, we found that *piecewiseSEM* produced less biased results than *lavaan*. First, in most cases mean simulated ΔAIC values were small and there was no strong bias in favour of one specific model (see $\Delta AIC_{simulation}$ in Table S11), as one would expect with random data. The only exception was the comparison between Model 1a and Model 2a in which simulated ΔAIC produced by *piecewiseSEM* was 7.4, favouring Model 1a. Importantly, however, these simulations indicated only a low probability for random data resulting in as large or larger AIC differences (43.2) in support for Model 1a than the ΔAIC values we obtained with real data (see low $P_{\geq \Delta AIC\_sim}$ values in Table S11), suggesting that support for Model 1a was unlikely the result of biased AIC estimates.

In contrast, simulations showed that *lavaan* produced highly biased ΔAIC values in all non-nested comparisons (see the high $\Delta AIC_{simulation}$ and $P_{\geq \Delta AIC\_sim}$ values for Models 2a, 2b

and 2c in Table S9). On the other hand, for nested model comparisons (i.e. with Models 1b and 1c) *lavaan* produced unbiased results similarly to those we got with *piecsewiseSEM* (Table S11).

These analyses suggest that the two methods gave consistent results for (1) path coefficients estimates and for (2) evaluating model fit of individual path models by global fit indices (using *C* statistics in *piecewiseSEM*, and χ^2, TLI, CFI, RMSEA, and SRMR in *lavaan*). On the other hand, simulation results indicate that AIC-based model comparisons are less biased when performed by the piecewise structural equation modelling method, at least for comparisons between non-nested models.

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