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11 Evolution of large males is associated with female-skewed adult 12 sex ratios in amniotes

13

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45

46

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66

67 **Abstract**

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

83

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

86

87 INTRODUCTION

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

94 A large volume of research has focused on how sex-specific behavior (e.g. mating
95 system, parental care), ecological processes (e.g. abundance and quality of resources), and life
96 history traits (e.g. fecundity in indeterminate growers) can generate size differences between
97 the sexes (Andersson 1994; Blanckenhorn 2005). These studies have concluded that sexual
98 selection is often a major driver of SSD evolution by either intra-sexual competition for
99 access to mates or inter-sexual mate choice, although other evolutionary mechanisms (e.g.
100 fertility selection and competition for resources) may also be important (Jehl and Murray
101 1986; Andersson 1994; Blanckenhorn 2005; Fairbairn et al. 2007; Clutton-Brock 2016).
102 Strong sexual selection for large body size in one sex is particularly likely in species where
103 that sex competes for mates by physical contests or endurance rivalry, as observed in several
104 vertebrate taxa (e.g. reptiles, birds, and mammals; Jehl and Murray 1986; Andersson 1994;
105 Cox *et al.* 2007; Székely *et al.* 2007; Clutton-Brock 2016).

106 Adult sex ratio (ASR), best measured as the proportion of males in the adult
107 population (Ancona et al. 2017) is a key demographic property of populations that influences
108 both the number of competitors for mates and the number of mates available to an individual
109 (Murray 1984; Székely et al. 2014b; Jennions and Fromhage 2017; Schacht et al. 2017). For
110 example, a male-skewed ASR means potentially more competitors and fewer available
111 partners for males than for females. An increasing number of studies show that ASR covaries

112 with several reproductive traits such as mating system, parental sex roles, divorce rate, extra-
113 pair mating and cooperative breeding both in non-human animals and humans (Liker et al.
114 2013, 2014; Schacht et al. 2014; Kappeler 2017; Komdeur et al. 2017; Eberhart-Phillips et al.
115 2018; Grant and Grant 2019). However, whether and how ASR is related to the evolution of
116 SSD is still poorly understood.

117 Theories suggest that ASR can drive the evolution of SSD in at least two ways. First,
118 the intensity of sexual competition may increase with the number of competitors. As Darwin
119 wrote (1871, p. 217): “*That some relation exists between polygamy and development of*
120 *secondary sexual characters, appears nearly certain; and this supports the view that a*
121 *numerical preponderance of males would be eminently favourable to the action of sexual*
122 *selection*”. According to his idea, highly skewed ASRs may intensify selection for
123 competitive traits such as weapons and large body size in the more abundant sex. Thus this
124 ‘mating competition hypothesis’ predicts that the extent of male-bias in SSD should increase
125 with the degree of male skew in the ASR. Later work refined Darwin’s (1871) original idea
126 by suggesting that the operational sex ratio (OSR, the number of sexually active males per
127 receptive female at a given time) rather than the ASR determines the intensity of mating
128 competition in a population (Emlen and Oring 1977). Thus, according to this latter theory
129 ASR would predict SSD if ASR covaries with OSR, for example because OSR is in part
130 determined by ASR (together with sex differences in behavior like parental care; Kokko et al.
131 2012). Although the relationship between ASR and OSR is yet to be fully explored, their
132 positive association has been demonstrated both by theoretical models (Kokko and Jennions
133 2008: Fig. 4a; Fromhage and Jennions 2016: Fig. 3c,d) and comparative analyses (Mitani et
134 al. 1996, correlation between ASR and OSR in 18 primates: $r = 0.4$, $P = 0.002$; unpublished
135 result using data from their Table 1). Empirical studies commonly use ASR and OSR

136 interchangeably in testing their relationship with SSD (Poulin 1997) and other proxies of
137 sexual selection (Janicke and Morrow 2018).

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

153 Alternatively, SSD may drive changes in sex ratios through sex differences in
154 mortality resulting from sexual competition. According to this ‘mortality cost hypothesis’, the
155 skewed ASR is a consequence rather than a cause of intense sexual selection, because when
156 males allocate a lot to mating competition they may suffer increased mortality, which in turn
157 leads to female-skewed ASR (Trivers 1972; Clutton-Brock et al. 1985; Liker and Székely
158 2005; Kalmbach and Benito 2007). This hypothesis predicts that in species exhibiting SSD
159 (1) the larger sex should have higher mortality due to the costs of being large, including the

160 direct costs associated with competition (e.g. fights, displays); which leads to (2) decreasing
161 male skew in the ASR with increasing degree of male bias in the SSD.

162 Studies that have investigated the relationships between sex ratios, SSD and sex-
163 specific mortality have so far yielded inconsistent results. While some studies found a
164 positive link between SSD and ASR or OSR (i.e. an increasing male bias in SSD with
165 increasing male skew in the sex ratios; Mitani *et al.* 1996; Poulin 1997), others reported
166 negative associations (Clutton-Brock *et al.* 1977; Wittenberger 1978; Georgiadis 1985; Haro
167 *et al.* 1994; Johansson *et al.* 2005; Lovich *et al.* 2014), or found no consistent relationships
168 (Owen-Smith 1993; Hirst and Kjørboe 2014; Muralidhar and Johnson 2017). Similarly,
169 mortality costs paid by the larger sex in dimorphic species were reported in some studies
170 (Clutton-Brock *et al.* 1985; Promislow 1992; Promislow *et al.* 1992; Moore and Wilson 2002;
171 Benito and González-Solís 2007; Kalmbach and Benito 2007), whereas no consistent
172 relationship between SSD and sex differences in mortality was found by others (Owens and
173 Bennett 1994; Toïgo and Gaillard 2003; Lemaître and Gaillard 2013; Székely *et al.* 2014a;
174 Tidière *et al.* 2015). Many of these studies focused on a narrow range of taxonomic groups
175 and were based on a relatively small number of species (typically fewer than 50) in
176 comparative analyses. Furthermore, none of the studies tested explicitly whether statistical
177 models assuming that ASR drives variation in SSD (as proposed by the mating competition
178 and mating opportunity hypotheses) or alternative models (like the mortality costs hypothesis)
179 fit better to the data.

180 Here we investigate the strength and direction of the relationship between ASR and
181 SSD in populations of wild amniotes, using the largest existing comparative dataset on ASR
182 compiled to date (462 species). First, we investigate whether SSD increases or decreases with
183 ASR across species, as predicted by the mating competition and mating opportunity
184 hypotheses, respectively. We also test whether the relationship is consistent among three

185 major amniote taxa (reptiles, birds, and mammals) because these taxa differ in multiple
186 ecological, behavioral and life-history traits. Since the extent and direction of SSD can be
187 influenced by ecological, life-history and behavioral factors besides mating competition, we
188 also control for several potential confounding variables in the analyses. Second, we study
189 whether SSD drives ASR variation by generating sex-biased mortality as proposed by the
190 mortality cost hypothesis. We test this latter hypothesis by investigating whether SSD is
191 related to sex differences in juvenile or adult mortality, and by comparing path models
192 representing different structural relationships between SSD, ASR and sex-specific mortality.
193

194 **METHODS**

195 **Data collection**

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

210

211 **Body mass and SSD**

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

224 We calculated SSD as $\log_{10}(\text{male mass} / \text{female mass})$. Earlier studies criticized
225 measures of SSD that are based on male/female (or female/male) ratios and suggested other
226 approaches, for example to analyze male size as response variable in models that also include
227 female size as a control variable (see Smith 1999 and Fairbairn 2007 for reviews). In his
228 seminal paper, however, Smith (1999, p. 444) convincingly demonstrated that ratios can be
229 safely used in the context of SSD analyses because "the risk of spurious correlation is
230 negligible to non-existent" due to the statistical properties of male and female size variables
231 (i.e. their high correlation and approximately equal coefficients of variation, leading to an
232 isometric relationship). We checked the assumption of isometry between male and female
233 body mass in our dataset and found that male and female body mass (on a $\log_{10} - \log_{10}$ scale)
234 are strongly correlated ($r = 0.994$) with a slope very close to and not different from 1

235 (phylogenetic generalized least squares, slope $\pm SE$: 1.0096 ± 0.0102 , 95% CI: $0.989 \leq \beta \leq$
236 1.029 , $n = 462$ species). Furthermore, Smith (1999, pp. 439-440) demonstrated that the
237 approaches based on the log ratios versus male mass as response variable are statistically
238 equivalent and suggested that the correct method is using log SSD ratio as response and
239 controlling for log size. We thus followed this latter approach. However, because the
240 measures of SSD remains a controversial issue among evolutionary ecologists (see e.g. Table
241 1 in Tidière et al. 2015 for a review of SSD metrics commonly used), we replicated the main
242 analysis using an alternative method (i.e. male size as response variable while controlling for
243 female size in the model) to check the robustness of our results. All results were qualitatively
244 unchanged.

245 To test whether the results are sensitive to conversion of length to mass in reptiles, we
246 replicated the main analyses (1) with SSD calculated from body length ($\log_{10}(\text{male length} /$
247 $\text{female length})$) of reptiles, and (2) with SSD calculated from body mass for a subset (31
248 species) of reptiles that has sex-specific mass data available from Myhrvold et al. (2015).
249 Whatever approach was used to assess the degree of SSD the results were qualitatively
250 unchanged (see Results). In the main text we thus report results based on body mass estimated
251 from body length for reptiles.

252

253 **Sex ratio**

254 We followed Wilson and Hardy (2002) and Ancona et al. (2017) in expressing ASR as the
255 proportion of males in the adult population. We defined the adult population here broadly as
256 adult individuals living in the study area during ASR sampling. Wilson and Hardy (2002)
257 showed that analyzing sex ratios as a proportion variable is appropriate when sex ratios are
258 estimated from samples of ≥ 10 individuals and the dataset has ≥ 50 sex ratio estimates. These
259 conditions were more than fully met in our analyses because sample sizes for ASR estimates

260 were always larger than 10 individuals per species (and typically much larger), and our
261 overall dataset included nine times more than the requirement of 50 species.

262 ASR data from Pipoly *et al.* (2015) were augmented with new species and updated
263 with more recent and/or better quality information (e.g. based on a more reliable method or a
264 larger sample size) for some reptiles. ASR estimates were collected by different observers for
265 the different taxa: reptiles by V.B. and I.P. (Pipoly *et al.* 2015; Bókony *et al.* 2019), birds by
266 A.L. (Liker *et al.* 2014), and mammals by Berger and Gompper (1999), Donald (2007) and
267 Anile and Devillard (2018). Details of data selection criteria are given in the original
268 publications (see also Ancona *et al.* 2017). Mean values were calculated for species with
269 multiple ASR data. ASR estimates are repeatable between populations of the same species as
270 measured by the intraclass correlation coefficient (ICC), although the magnitude of
271 repeatability varies among taxa: reptiles with genetic and environmental sex determination:
272 ICC= 0.55 and 0.14, respectively (Bókony *et al.* 2019), birds: ICC= 0.64 (Ancona *et al.* 2017),
273 mammals: ICC= 0.60 (Valentine Federico, J-F.L., J-M.G., A.L., I.P., T.S. unpublished result).
274 ASR estimates are not influenced by the sample size of the ASR studies (Székely *et al.* 2014a;
275 Bókony *et al.* 2019).

276

277 **Sex-specific mortality**

278 Annual mortality rates were collected from studies in which mortality (or survival) was
279 estimated for each of both sexes. Juvenile and adult mortality refer to age classes before and
280 after the age of first reproduction, respectively. For reptiles, data were collected by V.B.
281 (Bókony *et al.* 2019). Most adult mortality data on birds are taken from Székely *et al.* (2014a)
282 with the addition of new data for juvenile mortality by A.L. Reptile and bird mortality
283 includes estimates by various methods (e.g. capture-recapture, return rates, demographic
284 models), although we used better quality estimates (e.g. those from capture-recapture

285 analyses) whenever we had a choice (Székely *et al.* 2014a; Bókony *et al.* 2019). For
286 mammals, all sex-specific estimates were collected by J-M.G. and J-F.L. (Lemaître *et al.*
287 2020). Sex differences in juvenile and adult mortality rates were calculated as the magnitude
288 of male-biased mortality (i.e. $\log_{10}(\text{juvenile or adult male mortality} / \text{juvenile or adult female}$
289 $\text{mortality}))$, also referred to as ‘mortality bias’. These measures of mortality bias are not
290 related to the overall mortality rate of the species, as estimated by the average mortality rates
291 of the sexes (phylogenetic generalized least squares models, juvenile mortality bias: slope \pm
292 $SE = -0.068 \pm 0.101$, $t = 0.7$, $P = 0.497$, $n = 100$; adult mortality bias: slope $\pm SE = -0.05 \pm$
293 0.08 , $t = 0.7$, $P = 0.513$, $n = 230$).

294

295 **Other predictors**

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

307 Second, we controlled for the potential effects of environmental variation among
308 species by using two measures. Breeding latitude correlates with life-history traits in many
309 organisms (as shown in his pioneer work by Dobzhansky 1950) and may also influence the

310 potential for polygamy, hence also sexual selection (Fischer 1960; Isaac 2005;
311 Balasubramaniam and Rotenberry 2016). We used absolute values of the geographic latitude
312 of the ASR studies included in our dataset (i.e. average values for species with multiple ASR
313 estimates) to represent the distance from the Equator. When the authors did not report
314 latitude, we used Google Earth to estimate it as the center of the study sites based on the site
315 descriptions. For 30 birds and 10 mammals, accurate population locations were not reported,
316 hence, we used the latitudinal midpoint of the breeding ranges of these species (birds: V.
317 Remeš, A. Liker, R. Freckleton and T. Székely unpublished data, mammals: PanTHERIA
318 database).

319 In addition to latitude, we investigated environmental harshness as a second
320 environmental variable, which also has been hypothesized to influence SSD (Isaac 2005). We
321 quantified the harshness of the breeding environment using a proxy proposed by Botero *et al.*
322 (2014). This is the PC1 score extracted from Principal Component Analysis (PCA) performed
323 on a set of climatic and ecological variables (e.g. temperature and precipitation, net primary
324 productivity, habitat heterogeneity; see Botero *et al.* 2014 for a detailed description of the
325 variables and the analysis). The PC1 scores have higher values for a higher level of exposure
326 to drier, less productive environments, with colder, less predictable and more variable annual
327 temperatures (see Table 1 in Botero *et al.* 2014). In birds and mammals, we used the data
328 published in Botero *et al.* (2014), whereas for reptiles we calculated PC1 scores by
329 performing a PCA with the same set of variables.

330 Third, we characterized courtship displays in birds because earlier studies showed that
331 birds with aerial displays have less male-biased SSD compared to species with ground
332 displays, probably because selection favors male agility in aerially displaying species
333 constraining male body size (Jehl and Murray 1986; Székely *et al.* 2007). We followed
334 Székely *et al.* (2007) and divided species into two display groups: (1) mating displays that

335 may favor male agility, including species that mainly have aerial displays (both non-acrobatic
336 and acrobatic, categories 4 and 5 in Székely *et al.* 2007), and (2) displays that may not favor
337 male agility, including all other display types, typically performed on ground (categories 1-3
338 in Székely *et al.* 2007). Although SSD can also be influenced by display type and display
339 habitat in reptiles and mammals (e.g. see Agha *et al.* 2018), we were not able to collect
340 reliable data for these taxa, therefore we analyzed the effect of display type only in birds.

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

357 Finally, in reptiles, the evolution of viviparity and reduced reproductive frequency are
358 generally correlated with shifts toward female-biased SSD due to fecundity selection for large
359 female size (Pincheira-Donoso and Hunt 2017). To control for its potential effect on SSD, we

360 categorized the reproductive mode of reptiles as either viviparous or oviparous (Uetz et al.
361 2019).

362

363 **Statistical analyses**

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

378 First, using all species, we applied bivariate PGLS models to test interspecific
379 associations between ASR, SSD and sex differences in juvenile and adult mortality rates.
380 When SSD was the response variable in the model, we also included mean body mass as a
381 second predictor, as recommended by Smith (1999) (hence we termed these models as
382 'separate predictor models' instead of bivariate models in the rest of the paper). Then we built
383 two multi-predictor models. In Multi-predictor model 1, we tested the relationship between
384 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).

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

423 We followed the approach proposed by Santos (2012) for phylogenetic path analyses.
424 In the first step, we conducted phylogenetic transformation on the data to control for effects of
425 phylogenetic relatedness among species. For this purpose, we (1) determined λ separately for
426 each variable by maximum likelihood, (2) used this variable-specific λ value to re-scale the
427 phylogenetic tree to a unit tree, and (3) used the transformed tree to calculate phylogenetically
428 independent contrasts for the variable (using ‘pic’ function of the R package ‘ape’; Paradis
429 2012). We repeated this process for each variable, and the resulting phylogenetically
430 transformed values were used for fitting path models. In the second step of the analyses, we
431 evaluated model fit using d-separation method (Shipley 2016) as implemented in the R
432 package ‘piecewiseSEM’ (Lefcheck 2016). In this method, Fisher’s *C* statistic is used to test
433 the goodness of fit of the whole path model, and the model is rejected (i.e. it does not provide

434 a good fit to the data) if the result of this C statistic is statistically significant (and conversely
435 a statistically non-significant result means acceptable fit; Lefcheck 2016). We compared
436 model fit between the six path models by their AICc values. Note that this approach ensures
437 that the same variables (i.e. the contrasts with the same phylogenetic signal) are used in each
438 path model, and that the correlations are non-directional in the sense that for a pair of
439 variables X and Y , $r_{XY} = r_{YX}$ as assumed in path analysis (irrespective of the sign of the
440 correlation, i.e. whether it is positive or negative).

441 To test the robustness of the results, we repeated the path analyses using two other
442 methods. First, we repeated the above procedure (i.e. followed Santos 2012) except that we
443 used the covariance matrix comparison method for model fit instead of d-separation, as
444 implemented in the R package ‘lavaan’ (Rosseel 2012). Second, we repeated the analyses
445 using the method developed by von Hardenberg and Gonzalez-Voyer (2013). Unlike Santos’
446 (2012) method, in this latter approach a single value of Pagel’s λ is estimated for the residuals
447 of a regression of each pair of traits in a directional model, rather than a value of λ for each
448 variable (see the Discussion and Appendix S3). We used the R package ‘phylopath’ (van der
449 Bijl 2018) for this latter analysis, which relies on the d-separation method for model fitting
450 (similarly to ‘piecewiseSEM’, see above). We provide additional analyses to test the
451 robustness of the path analysis’ results in Appendix S3.

452

453 **RESULTS**

454 **Mating competition versus mating opportunity hypotheses**

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

459 did not differ among the three amniote classes (ASR \times class interaction on SSD: $F_{2,456} =$
460 0.935, $P = 0.393$). The increase of SSD with increasingly female-skewed ASR was
461 statistically significant within birds and mammals but was not in reptiles when the three taxa
462 were analyzed separately (Fig. S1, Tables S1-4). These results remained consistent when we
463 used SSD estimates based on length instead of estimated mass in reptiles (Tables S1, S2 and
464 S5), when SSD for reptiles were estimated from published body mass data (Table S5), and
465 also when male mass was used as response variable (Table S5).

466 These results are robust because the sign of the slope of the ASR - SSD relationship
467 and its statistical significance were not sensitive to branch length assumptions (Table S6), and
468 to the inclusion of other predictors (Table 1). In multi-predictor models (Table 1), mean body
469 mass was positively related to SSD, supporting the Rensch rule (Abouheif and Fairbairn
470 1997), and the type of sex determination influenced ASR variation as previously reported by
471 Pipoly *et al.* (2015). Nevertheless, ASR remained negatively associated with SSD when the
472 effects of mass and sex determination systems were accounted for (Table 1). This result also
473 did not change when environmental variation was included in the models using either
474 breeding latitude (Table 1) or environmental harshness (Table S5). Finally, excluding reptiles
475 with TSD (that have the lowest consistency in ASR; Bókony *et al.* 2019) or with assumed sex
476 determination also did not influence the relationship (Table S5).

477 The multi-predictor model for birds showed that species with aerial courtship displays
478 have lowered SSD as found in earlier studies (Jehl and Murray 1986; Székely *et al.* 2007);
479 however, the relationship between ASR and SSD remained statistically significant and
480 negative when this effect was included in the model (Table S3). Furthermore, data in birds
481 and mammals showed that, as expected, the relationship was weaker in monogamous than in
482 polygamous species, although the same trend occurred in both mating systems (Table S7).

483 Finally, reproductive mode was not associated with SSD or ASR in reptiles in our dataset
484 (Tables S1-2).

485

486 **Mating opportunity versus mortality costs hypotheses**

487 Both the mating opportunity hypothesis and the mortality cost hypothesis predict female-
488 skewed ASRs in species with male-biased SSD. However, our results are more consistent
489 with the mating opportunity hypothesis for two reasons. First, ASR but not SSD was
490 associated with the extent of sex differences in juvenile or adult mortality, and ASR remained
491 strongly and negatively correlated with SSD when sex differences in juvenile and adult
492 mortality were statistically controlled for (Table 1). Second, phylogenetic path analyses
493 showed that models of the mating opportunity hypothesis provided better fit to the data
494 (Models 1a-c, Fisher' C statistic: $P = 0.07 - 0.97$) than models corresponding to the mortality
495 cost hypothesis (Models 2a-c, $P < 0.001$; Table 2). The strongest support was for Model 1a
496 because it had the lowest AICc ($\Delta AICc = 4.1 - 43.2$; Table 2). This model proposes that sex-
497 biased mortality in both juveniles and adults generates skewed ASR, which in turn leads to
498 SSD biased towards the rarer sex (Fig. 3). These results are robust because we obtained the
499 same results when the analyses were repeated using two other implementations of the path
500 analysis (see Table S8 for the results obtained using 'phylopath', and Appendix S3 for the
501 results obtained using 'lavaan'). Finally, path analyses that excluded reptiles (for which the
502 ASR - SSD relationship was not statistically significant, see above) also yielded results
503 qualitatively consistent with the full dataset (Table S9).

504

505 **DISCUSSION**

506 Our analyses provided three major findings: (1) adult sex ratio is related to sexual size
507 dimorphism among amniote species, although the association is the opposite of the one

508 proposed by Darwin; (2) sex-biased mortality is unrelated to the extent of SSD in amniotes;
509 and (3) confirmatory path analyses indicate that sex-biased mortality influences ASR, which
510 in turn induces changes in SSD. Collectively, these findings support the mating opportunity
511 hypothesis, indicating that selection is likely to favor an increased resource allocation toward
512 mating competition (by growing and maintaining a large body mass) in the rarer sex, which
513 has a higher chance of getting mates than the other sex.

514 Theoretical models show that skewed ASRs can promote evolutionary changes that
515 may generate this association between ASR and SSD. First, models of sex role evolution
516 showed that skewed ASR can result in divergences in reproductive roles between the sexes
517 leading to less parental care and more frequent desertion and remating in the rarer sex and
518 opposite changes (i.e. more parental care and less frequent remating) in the more abundant
519 sex (Queller 1997; McNamara et al. 2000). Similarly, a demographic analysis based on the
520 relationships between mating systems and sex ratio, sex-specific patterns of survivorship, age
521 of first reproduction, and annual fecundity predicts that skewed ASRs promote the evolution
522 of polygamy (i.e. polygyny and polyandry in female-biased and male-biased populations,
523 respectively; Murray 1984). Since both frequent remating and polygamy can intensify sexual
524 selection, the above effects of skewed ASR can promote the evolution of SSD by favoring
525 increased body size in the rare sex. In line with the predictions of these models, an increasing
526 number of recent studies in birds and humans show that polygyny is more frequent and
527 parental care by males is reduced in female-skewed populations (Liker et al. 2013, 2014,
528 2015; Remeš et al. 2015; Schacht and Borgerhoff Mulder 2015; Eberhart-Phillips et al. 2018;
529 Grant and Grant 2019). Our results are also concordant with experimental studies in voles and
530 lizards, which reported that female-skewed ASRs exert directional selection for large body
531 size in males (Klemme et al. 2007; Fitze and Le Galliard 2008), and increase variance in male
532 reproductive success (Dreiss et al. 2010).

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

543 The association between intense sexual selection in males and female-skewed ASRs
544 was proposed decades ago by avian evolutionary ecologists (e.g. Mayr 1939), although it was
545 usually explained by the mortality cost hypothesis (Wittenberger 1976). Our analyses do not
546 support this latter hypothesis because sex-biased SSD is not associated with sex-biased
547 juvenile or adult mortality in the studied amniote species, and the results of the confirmatory
548 path analyses are also inconsistent with the mortality cost hypothesis. We propose that the
549 lack of relationship between SSD and sex differences in mortality may be explained by
550 variation in the environmental context (Lemaître et al. 2020). Studies in birds and mammals
551 showed that having a large body size may only be costly in terms of mortality in populations
552 subjected to harsh environmental conditions (Toïgo and Gaillard 2003; Kalmbach and Benito
553 2007; Jones et al. 2009; Clutton-Brock 2017). The effect of SSD may thus be reduced or
554 absent when the sex-specific mortality estimates correspond to average conditions, that may
555 often be the case in wild populations.

556 The ASR - SSD relationship may also be influenced by sex differences in the time of
557 maturation because longer maturation time in the larger sex can result in a shortage of that sex

558 in the adult population (Lovich et al. 2014) because immature life stages are generally
559 characterized by higher mortality (e.g. Gaillard et al. 2000). Furthermore, Fromhage &
560 Jennions (2016) showed that female-skewed sex ratios at maturation (MSR) can result in the
561 evolution of increased female care and male allocation to traits facilitating mating success.
562 Thus, if variation in ASR is determined at least in part by MSR, then the effects of sex-biased
563 MSR on sex roles can contribute to the observed association of ASR with the intensity of
564 mating competition, and, hence, SSD. This latter mechanism would deserve further
565 investigations.

566 Although the relationship between ASR and SSD is not statistically significant in
567 reptiles, it is qualitatively consistent with our findings in birds and mammals. Other selective
568 processes (e.g. fertility selection for large female size in indeterminate growers, Cox *et al.*
569 2007) might have masked the influence of sexual selection on SSD in reptiles. Consistent
570 with this explanation, selection often favors delayed maturation in female reptiles, which
571 enables them to produce larger clutches, which in turn also influences their body size and the
572 extent of SSD (Shine 2005; Agha et al. 2018). Follow-up studies using different proxies of
573 sexual selection are needed to investigate further how sexual selection is related to ASR in
574 reptiles.

575 Biased estimates of ASR may generate spurious relationship with SSD, which may
576 potentially affect our results. For example, the larger sex may have lower detectability in
577 polygamous species if some members of that sex are excluded from breeding sites (Ancona et
578 al. 2017). However, highly polygamous species in which populations have been thoroughly
579 surveyed showed skewed ASR even when all individuals in the population were accurately
580 counted (Granjon et al. 2017), and fairly consistent ASR estimates were obtained when both
581 breeding and non-breeding individuals were included (Emlen and Wrege 2004). In general,
582 ASR estimates show a moderate but statistically significant repeatability across populations in

583 most of the studied taxa, except reptiles with temperature-dependent sex determination
584 (Ancona et al. 2017; Bókony et al. 2019; Valentine Federico, J-F.L., J-M.G., A.L., I.P., T.S.
585 unpublished result), and in 80% of bird species the direction of ASR skew is the same for all
586 repeated estimates (Székely et al. 2014a).

587 The paths of causality in comparative data are difficult to untangle. Path analysis is a
588 valuable tool for contrasting different causal models, although it cannot reveal causality
589 (Shipley 2016). Path analysis assumes that each variable includes independent variations or
590 ‘errors’ and that these errors are independent among variables. This is not true for
591 comparative data, because the errors will be correlated across species. Our approach follows
592 Santos (2012), an innovative but overlooked method that satisfies the assumptions of path
593 analysis better than an alternative method based on phylogenetic regressions proposed by von
594 Hardenberg and Gonzalez-Voyer (2013). This latter approach is problematic because it is not
595 robust to changes in the specification of the model: if variable Y is regressed on X and λ
596 estimated, then the estimates of the partial correlations and λ may be different from those
597 obtained if Y is regressed on X with λ estimated (Appendix 3). The approach we have taken
598 avoids this problem. However, there is still room for methodological improvement. For
599 instance, our approach has the drawback of being a ‘subtractive’ comparative method (sensu
600 Harvey and Pagel 1991). The question of how to robustly fit complex path models for data on
601 multiple traits with different levels of phylogenetic signal is not straightforward.

602

603 *Concluding remarks*

604 Our findings indicate that sex-specific selection for large body size is associated with skewed
605 ASRs across amniotes, and this process appears to produce SSD biased towards the rare sex
606 in birds and mammals. Although this conclusion contrasts with Darwin’s initial suggestion
607 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.
613

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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	A	n
(A) Response: sexual size dimorphism						
Separate predictor models:						
<i>Model 1</i>						
ASR	- 0.168 ± 0.035	4.835	< 0.001	0.119	0.868 **	462
Mean body mass	0.515 ± 0.086	5.980	< 0.001			
<i>Model 2</i>						
Juvenile mortality bias	0.041 ± 0.065	0.629	0.531	0.129	0.703 **	100
Mean body mass	0.529 ± 0.131	4.051	< 0.001			
<i>Model 3</i>						
Adult mortality bias	- 0.021 ± 0.047	0.454	0.650	0.095	0.932 *	230
Mean body mass	0.596 ± 0.117	5.090	< 0.001			
Multi-predictor model 1:						
ASR	- 0.160 ± 0.035	4.555	< 0.001	0.126	0.869 **	457
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:						
ASR	- 0.271 ± 0.061	4.452	< 0.001	0.273	0.841 *	97
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:						
<i>Model 1: SSD</i>						
SSD	- 0.234 ± 0.051	4.593	< 0.001	0.042	0.359 **	462
<i>Model 2: Juvenile mortality bias</i>						
Juvenile mortality bias	- 0.214 ± 0.099	2.151	0.034	0.035	0.281 **	100
<i>Model 3: Adult mortality bias</i>						
Adult mortality bias	- 0.257 ± 0.060	4.313	< 0.001	0.071	0.288 **	230
Multi-predictor model 1:						
SSD	- 0.188 ± 0.050	3.727	< 0.001	0.071	0.247 **	457
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:						
SSD	- 0.457 ± 0.120	3.794	< 0.001	0.402	0.030 #	97
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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Results of separate predictor and multi-predictor phylogenetic generalized least-squares (PGLS) models with either (A) SSD ($\log_{10}(\text{male mass}/\text{female mass})$) or (B) ASR (proportion of males in the adult population) as dependent variable. Separate predictor models with SSD

876 as dependent variable also include $\log_{10}(\text{mean mass})$ as predictor (see Methods). Mortality
877 biases were calculated as $\log_{10}(\text{male mortality}/\text{female mortality})$ for juveniles and adults,
878 respectively. $b \pm SE$ is the model's parameter estimate with its standard error (intercepts are
879 not shown), t and P are the associated test statistic and its significance, λ is Pagel's lambda, n
880 is number of species.
881 * λ statistically different from 0, # λ statistically different from 1.
882 ¹ Differences from species with XY sex determination; overall effect of sex determination on
883 SSD: $F_{2,451} = 3.411$, $P = 0.034$; on ASR: $F_{2,451} = 6.135$, $P = 0.002$.

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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).

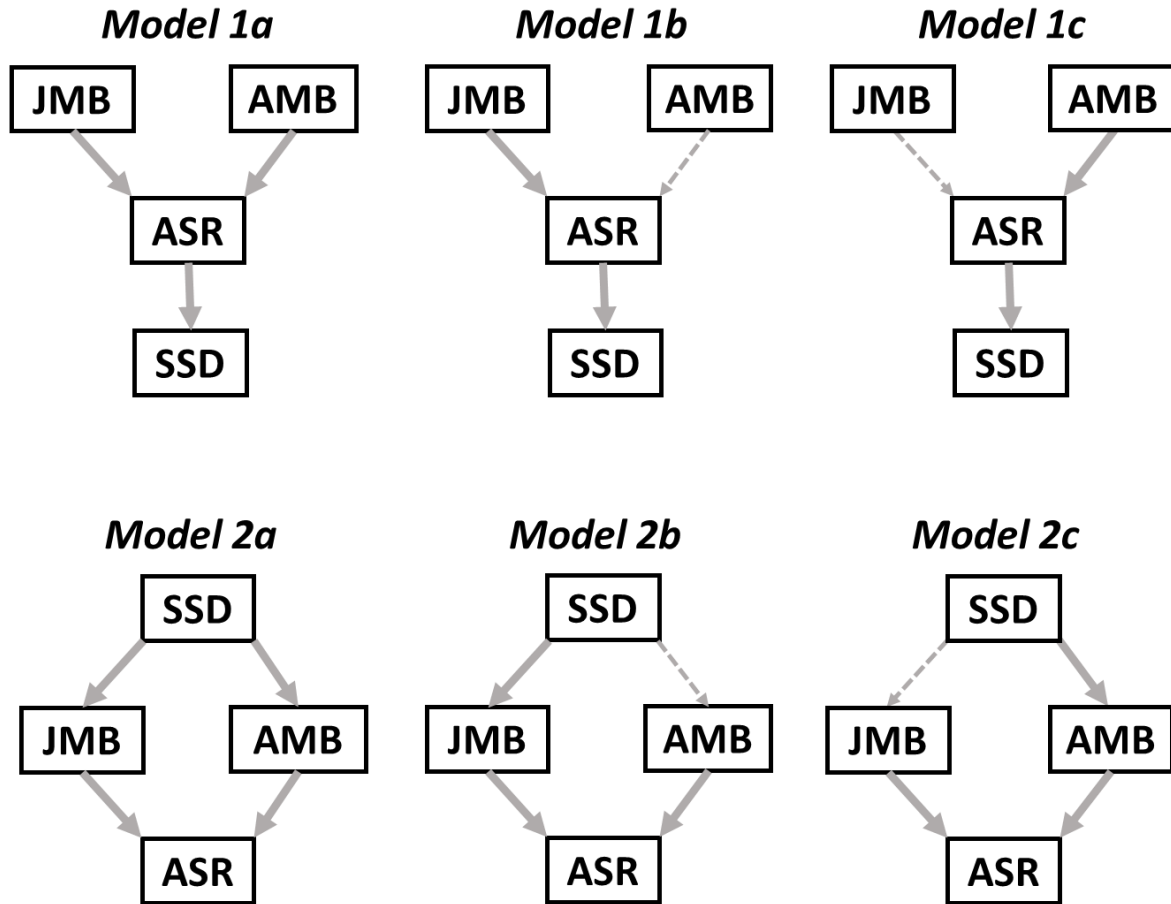
Model/Path	Path coefficient \pm SE	Z	P_{887}
Model 1a	$P_C = 0.972, df = 4, AICc = 15.8, \Delta AICc = 0.0$		
AMB \rightarrow ASR	- 0.340 \pm 0.113	- 3.000	0.004
JMB \rightarrow ASR	- 0.205 \pm 0.104	- 1.970	0.052
ASR \rightarrow SSD	- 0.425 \pm 0.074	- 5.723	< 0.001
Model 1b	$P_C = 0.065, df = 6, AICc = 25.7, \Delta AICc = 9.9$		
(AMB \rightarrow ASR) ¹	0	-	-
JMB \rightarrow ASR	- 0.258 \pm 0.107	- 2.417	0.018
ASR \rightarrow SSD	- 0.425 \pm 0.074	- 5.723	< 0.001
Model 1c	$P_C = 0.376, df = 6, AICc = 19.9, \Delta AICc = 4.1$		
AMB \rightarrow ASR	- 0.378 \pm 0.113	- 3.334	0.0010
(JMB \rightarrow ASR) ¹	0	-	-
ASR \rightarrow SSD	- 0.425 \pm 0.074	- 5.723	< 0.001
Model 2a	$P_C = 0.0, df = 4, AICc = 59.0, \Delta AICc = 43.2$		
SSD \rightarrow AMB	0.171 \pm 0.105	1.631	0.106
SSD \rightarrow JMB	0.111 \pm 0.115	0.958	0.341
AMB \rightarrow ASR	- 0.340 \pm 0.113	- 3.000	0.004
JMB \rightarrow ASR	- 0.205 \pm 0.104	- 1.970	0.052
Model 2b	$P_C = 0.0, df = 4, AICc = 50.4, \Delta AICc = 34.6$		
SSD \rightarrow JMB	0.111 \pm 0.115	0.958	0.341
AMB \rightarrow ASR	- 0.340 \pm 0.113	- 3.000	0.004
JMB \rightarrow ASR	- 0.205 \pm 0.104	- 1.970	0.052
Model 2c	$P_C = 0.0, AICc = 50.4, \Delta AICc = 34.6$		
SSD \rightarrow AMB	0.171 \pm 0.105	1.631	0.106
AMB \rightarrow ASR	- 0.340 \pm 0.113	- 3.000	0.004
JMB \rightarrow ASR	- 0.205 \pm 0.104	- 1.970	0.052

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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. $\Delta AICc$ indicates difference in $AICc$ values between the most supported model (lowest $AICc$, Model 1a) and the focal models. $\Delta AICc > 2$ 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.

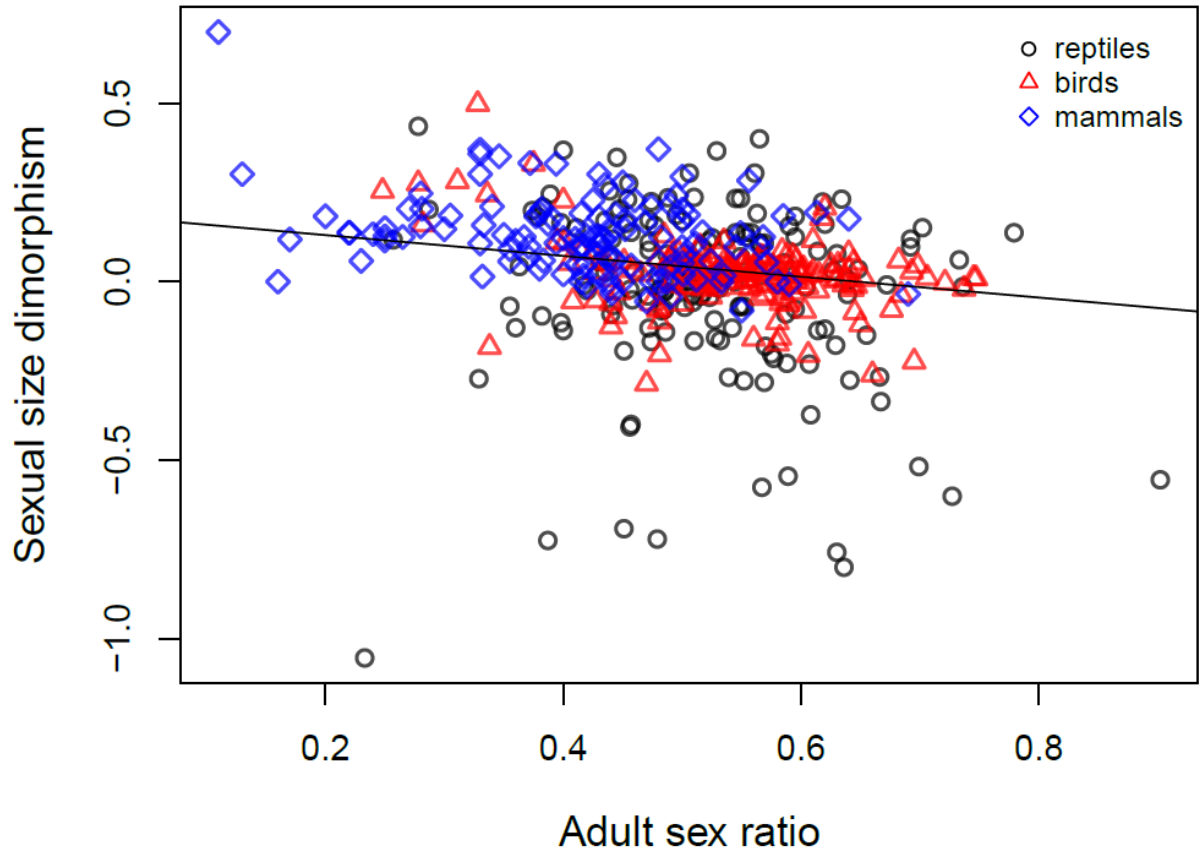
¹ Path coefficient set to zero to keep the variable in the model.

927 **Figure 1.** Path models tested in the phylogenetic path analyses. SSD: sexual size dimorphism,
 928 ASR: adult sex ratio, JMB: juvenile mortality bias, AMB: adult mortality bias. Dashed arrows
 929 indicate paths with coefficients set to zero to keep the variable in the model. Models 1a-c and
 930 2a-c represent relationships as predicted by the mating opportunity hypothesis and the
 931 mortality cost hypothesis, respectively.
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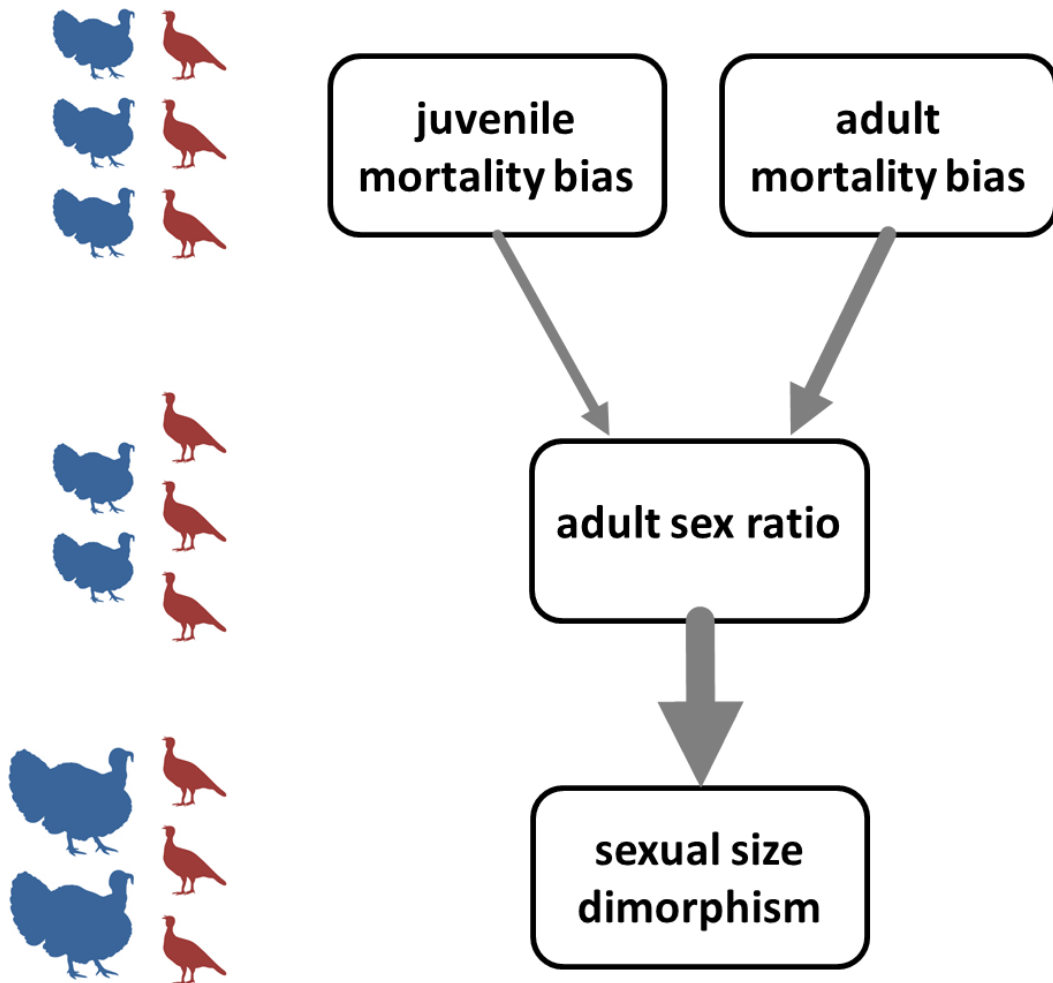
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937 **Figure 2.** Sexual size dimorphism (SSD) in relation to adult sex ratio (ASR) in amniotes.
938 SSD was calculated as $\log_{10}(\text{male mass}/\text{female mass})$; ASR is the proportion of males in the
939 adult population. Each data point represents a species; the regression line is fitted by
940 phylogenetic generalized least-squares (PGLS) model (see Table 1 for statistics).
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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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Electronic Supporting Information: tables and figures

958 **Table S1.** Relationship between SSD, ASR and sex-biased mortalities in reptiles, using
959 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:						
<i>Model 1</i>						
ASR	- 0.123 ± 0.075	1.641	0.103	0.082	0.948 *	155
Mean body mass	0.668 ± 0.177	3.774	< 0.001			
<i>Model 2</i>						
Juvenile mortality bias	- 0.414 ± 0.337	1.228	0.240	0.005	0.0	17
Mean body mass	- 0.500 ± 0.440	1.136	0.275			
<i>Model 3</i>						
Adult mortality bias	- 0.151 ± 0.117	1.287	0.203	0.092	1.0 *	62
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:						
<i>Model 1: SSD</i>						
SSD	- 0.074 ± 0.061	1.209	0.228	0.003	0.171 #	155
<i>Model 2: Juvenile mortality bias</i>						
Juvenile mortality bias	- 0.480 ± 0.415	1.156	0.266	0.021	0.0	17
<i>Model 3: Adult mortality bias</i>						
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.

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

Predictors	$b \pm SE$	t	P	R^2	λ	n
(A) Response: sexual size dimorphism						
Separate predictor models:						
<i>Model 1</i>						
ASR	- 0.008 ± 0.005	1.587	0.114	0.073	0.935 *	155
Mean body mass	0.040 ± 0.011	3.562	< 0.001			
<i>Model 2</i>						
Juvenile mortality bias	- 0.035 ± 0.024	1.472	0.163	0.073	0.0	17
Mean body mass	- 0.046 ± 0.031	1.485	0.160			
<i>Model 3</i>						
Adult mortality bias	- 0.010 ± 0.007	1.402	0.166	0.086	1.0 *	62
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:						
<i>Model 1: SSD</i>						
SSD	- 0.131 ± 0.109	1.209	0.229	0.003	0.169 #	155
<i>Model 2: Juvenile mortality bias</i>						
Juvenile mortality bias	- 0.480 ± 0.415	1.156	0.266	0.021	0.0	17
<i>Model 2: Adult mortality bias</i>						
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	- 0.085 ± 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 #	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			

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* Pagel's lambda statistically different from 0, # 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

975 **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						
<i>Separate predictor models:</i>						
<i>Model 1</i>						
ASR	- 0.242 ± 0.037	6.625	< 0.001	0.250	0.812 **	185
Mean body mass	0.293 ± 0.105	2.798	0.006			
<i>Model 2</i>						
Juvenile mortality bias	0.066 ± 0.073	0.898	0.374	0.130	0.095 *	47
Mean body mass	0.735 ± 0.270	2.722	0.009			
<i>Model 3</i>						
Adult mortality bias	0.068 ± 0.051	1.335	0.184	0.072	0.708 **	123
Mean body mass	0.372 ± 0.130	2.870	0.005			
<i>Multi-predictor model 1:</i>						
ASR	- 0.242 ± 0.038	6.390	< 0.001	0.272	0.720 **	178
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			
<i>Multi-predictor model 2:</i>						
ASR	- 0.346 ± 0.080	4.318	< 0.001	0.386	1.0 *	47
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						
<i>Separate predictor models:</i>						
<i>Model 1: SSD</i>						
SSD	- 0.746 ± 0.114	6.520	< 0.001	0.184	0.480 **	185
<i>Model 2: Juvenile mortality bias</i>						
Juvenile mortality bias	- 0.354 ± 0.115	3.084	0.003	0.156	0.0 #	47
<i>Model 3: Adult mortality bias</i>						
Adult mortality bias	- 0.384 ± 0.079	4.866	< 0.001	0.157	0.0 #	123
<i>Multi-predictor model 1:</i>						
SSD	- 0.717 ± 0.116	6.183	< 0.001	0.239	0.244 #	178
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			
<i>Multi-predictor model 2:</i>						
SSD	- 0.382 ± 0.153	2.499	0.016	0.397	0.0 #	47
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			

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

978 ¹ Difference from non-agile species.

979 For further explanation, see the footnotes of Table 1 in the main text.

980

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

Predictors	$b \pm SE$	t	P	R^2	λ	n
(A) Response: sexual size dimorphism						
<i>Separate predictor models:</i>						
<i>Model 1</i>						
ASR	- 0.170 ± 0.058	2.946	0.004	0.143	0.313 **	122
Mean body mass	0.385 ± 0.129	2.979	0.004			
<i>Model 2</i>						
Juvenile mortality bias	0.089 ± 0.123	0.719	0.477	0.078	0.233 #	36
Mean body mass	0.515 ± 0.233	2.214	0.034			
<i>Model 3</i>						
Adult mortality bias	0.025 ± 0.103	0.244	0.809	0.056	0.217 #	45
Mean body mass	0.424 ± 0.202	2.093	0.042			
<i>Multi-predictor model 1:</i>						
ASR	- 0.153 ± 0.058	2.646	0.009	0.166	0.342 **	120
Mean body mass	0.418 ± 0.131	3.191	0.002			
Latitude	0.106 ± 0.053	2.016	0.046			
<i>Multi-predictor model 2:</i>						
ASR	- 0.374 ± 0.129	2.900	0.007	0.250	0.0 #	33
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						
<i>Separate predictor models:</i>						
<i>Model 1: SSD</i>	- 0.460 ± 0.130	3.539	< 0.001	0.087	0.252 **	122
<i>Model 2: Juvenile mortality bias</i>	- 0.032 ± 0.166	0.195	0.847	< 0.001	0.0 #	36
<i>Model 3: Adult mortality bias</i>	- 0.076 ± 0.155	0.493	0.624	< 0.001	0.0 #	45
<i>Multi-predictor model 1:</i>						
SSD	- 0.375 ± 0.140	2.670	0.009	0.093	0.320 **	120
Mean body mass	- 0.314 ± 0.209	1.500	0.136			
Latitude	- 0.075 ± 0.083	0.907	0.366			
<i>Multi-predictor model 2:</i>						
SSD	- 0.617 ± 0.213	2.900	0.007	0.293	0.0 #	33
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			

982
 983 * Pagel's lambda statistically different from 0, # lambda statistically different from 1.
 984 For further explanation, see the footnotes of Table 1 in the main text.
 985
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987 **Table S5.** Sensitivity analyses of the relationship between sexual size dimorphism (SSD,
988 dependent variable in all models) and adult sex ratio (ASR). Table shows results when (A)
989 male mass (instead of $\log_{10}(\text{male mass} / \text{female mass})$ is used as response variable, (B)
990 reptiles are included with SSD based on body length, (C) reptiles are included with SSD
991 calculated from sex-specific body mass, (D) reptiles with temperature-dependent sex
992 determination (TSD) are excluded, (E) reptiles with assumed sex determination, based on
993 related species, are excluded, and (F) environmental harshness is included in the model.
994

Predictors	$b \pm SE$	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 mass¹ (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 excluded² (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 harshness³:						
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			

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

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

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

1000 ³ The influence of environmental harshness was tested in birds and mammals using data from
1001 Botero et al. (2014), in reptiles using data calculated in this study (following the method of
1002 Botero et al 2014), and in all species by pooling the harshness scores from the two studies.

1003 **Table S6.** Analyses of the relationship between SSD (dependent variable) and ASR with
 1004 branch lengths calculated by three different methods for the phylogeny used in the PGLS
 1005 models. The analyses included reptiles, birds, and mammals.
 1006

Predictors	$b \pm SE$	t	P	R^2	λ	n
(A) Nee's method						
ASR	- 0.168 ± 0. 035	4.835	< 0.001	0.119	0.868 **#	462
Mean body mass	0.515 ± 0. 086	5.980	< 0.001			
(B) Pagel's method						
ASR	- 0.166 ± 0. 034	4.826	< 0.001	0.124	0.869 **#	462
Mean body mass	0.564 ± 0. 090	6.282	< 0.001			
(C) Unit branch length						
ASR	- 0.179 ± 0. 032	5.577	< 0.001	0.148	1.0 *	462
Mean body mass	0.565 ± 0. 085	6.682	< 0.001			

1007
 1008 * Pagel's lambda statistically different from 0, # lambda statistically different from 1
 1009 ¹ See Methods for details of branch length calculations
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1014 **Table S7.** Analyses of the relationship between SSD (dependent variable) and ASR in
 1015 socially monogamous and socially polygamous species, respectively. The analyses included
 1016 birds and mammals.
 1017

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

1018
 1019 * Pagel's lambda statistically different from 0, # lambda statistically different from 1
 1020 ¹ When monogamous and polygynous species are analyzed together, there is a statistically
 1021 significant interaction between the effects of mating system and ASR ($b \pm SE = 0.218 \pm 0.087$,
 1022 $t = 2.510$, $P = 0.013$).

1023 **Table S8.** Results of the phylogenetic path analyses using the R package ‘phylopath’. Models
 1024 represent the mating opportunity hypothesis (Models 1a-c) and the mortality cost hypothesis
 1025 (Models 2a-c). Analyses based on data of all species (birds, mammals, and reptiles; $n= 97$
 1026 species).
 1027

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

1029 Model structures are shown in Figure 1. SSD: sexual size dimorphism, ASR: adult sex ratio,
 1030 JMB and AMB: juvenile and adult mortality biases, respectively. The table shows the number
 1031 of independence claims (*k*), the number of parameters (*q*), Fisher’s *C* statistic (*C*) and its
 1032 accompanying probability (*P*), C-statistic information criterion corrected for small sample
 1033 sizes (*CICc*), and the difference in *CICc* from the top model (Δ *CICc*). A *P*-value less than
 1034 0.05 indicates a poor model fit (i.e. rejection of the model), whereas a Δ *CICc* > 2 indicates
 1035 substantial support for the top path model over the alternative models.
 1036
 1037

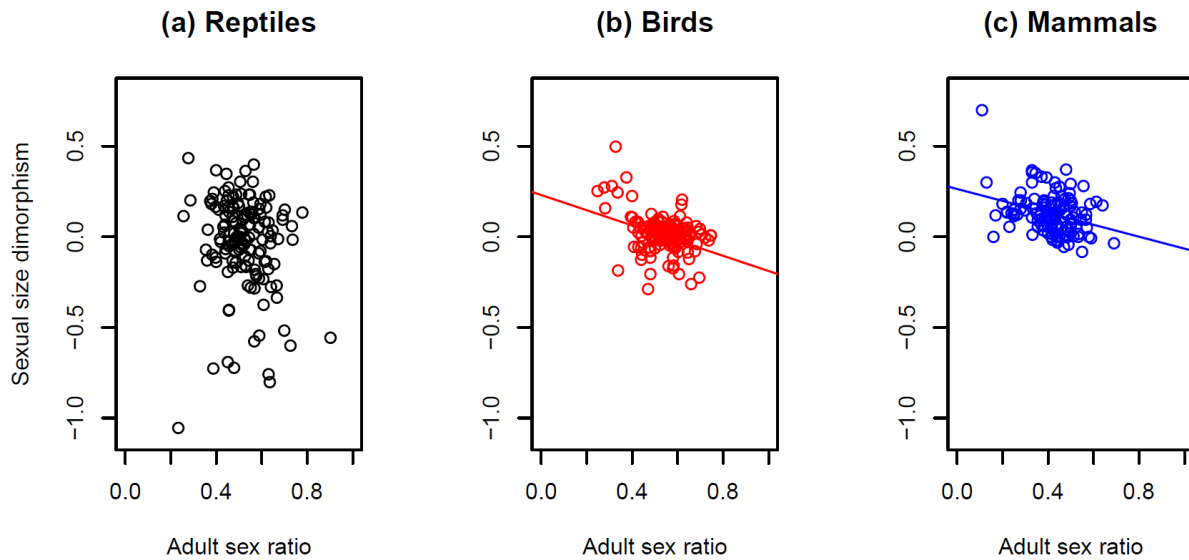
1038 **Table S9.** Phylogenetic path models representing the mating opportunity hypothesis (Models
 1039 1a-c) and the mortality cost hypothesis (Models 2a-c). Analyses with data of birds and
 1040 mammals (i.e. excluding reptiles; $n= 81$ species).
 1041

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

1074 Model structures are shown in Figure 1. SSD: sexual size dimorphism, ASR: adult sex ratio,
 1075 JMB and AMB: juvenile and adult mortality biases, respectively (variables are explained in
 1076 footnotes of Table 1). P_C is P -value for Fisher's C statistic for model fit, with non-significant
 1077 values (> 0.05) indicating an acceptable fit. $\Delta AICc$ indicates difference in $AICc$ values
 1078 between the most supported model (lowest $AICc$, Model 1a) and the focal models. $\Delta AICc > 2$
 1079 indicates substantially higher support for the best model than for the other model.

1080 ¹ Path coefficient set to zero to keep the variable in the model.
 1081

1082 **Figure S1.** Sexual size dimorphism in relation to adult sex ratio in (a) reptiles (PGLS, $b \pm SE$
1083 $= -0.123 \pm 0.075$, $P = 0.103$, $n = 155$ species), (b) birds ($b \pm SE = -0.242 \pm 0.037$, $P < 0.001$,
1084 $n = 185$), and (c) mammals ($b \pm SE = -0.170 \pm 0.058$, $P = 0.004$, $n = 122$). Each data point
1085 represents a species, and lines show statistically significant regressions fitted by PGLS (see
1086 Tables S1-4 for further statistical details).
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Electronic Supporting Information: Appendix S1

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1092 References for data sources are provided in a separate excel file. The full dataset
1093 will be published together with references after the manuscript is accepted for
1094 publication.
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Electronic Supporting Information: Appendix S2

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Parameters of the allometric equations between body length and body mass ($\log_{10}(\text{mass in g}) = a + b \cdot \log_{10}(\text{length in mm})$) 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 crocodylians	-4.52	2.923	600	Meiri (2010)
Turtles	-3.535	2.887	199	Regis and Meik (2017)

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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

1143 dependence to be modelled through estimating Pagel's λ , which accounts for varying
1144 contributions of phylogeny to trait variation.

1145 However the analysis of such data is complex. Consider a simple example, in which
1146 we have three variables x , y , and z . If we model $x \rightarrow y$ (i.e. where x is the predictor and y is
1147 the response variable) using the PGLS- λ approach, we might well estimate a different
1148 correlation than if we model $y \rightarrow x$. This is because in PGLS the estimate of λ depends on the
1149 direction of the relationship, and this affects the estimate of the correlation. In path analysis
1150 correlations between pairs of variables are the input and the directionality of the statistical
1151 model should not be an issue. Specifically, the net correlation r_{xz} should then be the product
1152 of the component pairwise correlations, i.e. $r_{xz} = r_{xy} \times r_{yz}$. However, if we use PGLS and $\lambda \neq 0$
1153 and $\lambda \neq 1$ (as in most cases in our analyses, see Table 1 in the main text), then in general $r_{xz} =$
1154 $r_{xy} \times r_{yz} \neq r_{yx} \times r_{yz} = r_{xz}$. We would therefore prefer to avoid an arbitrary decision about the
1155 directionality of the model to affect the estimate of the coefficient.

1156 In our analyses we overcame the above difficulties by avoiding the use of bivariate
1157 PGLS models to estimate correlations between the variables in the path models. We followed
1158 the approach proposed by Santos (2012), in which first we calculated λ for each individual
1159 variable and conducted a phylogenetic transformation on the variable using that estimate of λ .
1160 Then we fitted path models to these already transformed data using ordinary fitting methods
1161 developed for fitting path models to non-phylogenetic data. Details of the analyses are
1162 described in the Methods section of the main text.

1163

1164 **2. The problem of comparing non-nested path models by AIC statistics**

1165 In phylogenetic comparative studies the direction of causality between variables is often
1166 unknown, and different evolutionary hypotheses may propose opposing cause - effect
1167 relationships (like the mating competition and the mortality cost hypotheses in our study, see
1168 Fig. S1). These hypotheses may be represented by different path models, and then their fit to
1169 the data can be compared by some comparative fit indices, most commonly by AIC (West et
1170 al. 2012). However, simulations suggest that conclusions of path model comparisons based on
1171 information theory approach (like AIC) can be unreliable (Preacher and Merkle 2012). In
1172 addition the competing models can be non-nested (non-hierarchical) (e.g. Models 1a versus 2a
1173 in Fig. S1), for which AIC-based comparison should be applied with caution (Kline 2015).

1174 To explore the problem of model comparison in the context of our study, first we
1175 fitted our path models to the real dataset by two alternative methods: (1) by covariance matrix

1176 comparison, as implemented in the R package *lavaan* (Rosseell 2012), and (2) by piecewise
1177 structural equation modelling (or d-separation) method, as implemented in the *piecewiseSEM*
1178 (Lefcheck 2016) package. We compared path coefficient estimates and various model fit
1179 indices between these two methods to evaluate whether they produce consistent conclusions.
1180 Second, we used the same two methods and R implementations to fit the models to simulated
1181 datasets, and tested which of the methods produces more reliable (less biased) model
1182 comparisons.

1183

1184 2.1. Fitting path models to real data

1185 The general steps of model fitting procedure we followed in this study are described in the
1186 Methods section of the main text. We performed model fitting with the two R packages
1187 *piecewiseSEM* and *lavaan*. In *piecewiseSEM* and *lavaan* the global model fit for each
1188 individual path model is evaluated by Fisher's *C* and χ^2 statistics, respectively, where a
1189 statistically non-significant result means acceptable fit. In *lavaan*, several other measures for
1190 model fit of individual models are also available, and here we report four of the most widely
1191 used indices (TLI, CFI, RMSEA, SRMR). It has been proposed that that the values of TLI
1192 and CFI > 0.95, RMSEA < 0.06, and SRMR < 0.08 indicate acceptable/good fit of models to
1193 the data (West et al. 2012).

1194 We found that the two methods produced highly consistent estimates for the
1195 standardized path coefficients in all path models (*piecewiseSEM*: Table 1 in the main text,
1196 *lavaan*: Table S9 below). The effect of juvenile mortality on ASR was marginally not
1197 significant in most *piecewiseSEM* models whereas it was significant with all *lavaan* models.
1198 For all other relationships the two methods produced consistent results.

1199 The two methods also produced highly consistent results for model fit as evaluated by
1200 global fit indices (i.e. *C* and χ^2 statistics, respectively, see Table S10). The only difference
1201 was that for Model 1b *piecewiseSEM* indicated 'marginally acceptable' model fit whereas
1202 *lavaan* indicated poor model fit for this path model. The other fit indices (TLI, CFI, RMSEA,
1203 and SRMR) suggest conclusions that are fully consistent with *C* statistics and χ^2 tests, i.e.
1204 acceptable fit for Models 1a and 1c by all of these indices and unacceptable fit for all other
1205 models (Table S10).

1206

1207

1208

1209 **Table S9.** Estimates of standardized path coefficients for the six path models representing
 1210 various relationships between SSD, ASR, and sex biases in adult (AMB) and juvenile (JMB)
 1211 mortality, obtained by the R package *lavaan* (see Fig. S1 for model details). Significant
 1212 relationships are highlighted in bold.
 1213

Model/Path	Path coefficient ± SE	Z	P
<i>Model 1a</i>			
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
<i>Model 1b</i>			
(AMB → ASR) ¹	0	-	-
JMB → ASR	- 0.258 ± 0.105	- 2.443	0.015
ASR → SSD	- 0.657 ± 0.107	- 6.144	0.000
<i>Model 1c</i>			
AMB → ASR	- 0.378 ± 0.112	- 3.370	0.001
(JMB → ASR) ¹	0	-	-
ASR → SSD	- 0.657 ± 0.107	- 6.144	0.000
<i>Model 2a</i>			
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
<i>Model 2b</i>			
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
<i>Model 2c</i>			
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

1214
 1215 ¹ Path coefficient set to zero
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1223 **Table S10.** Fit indices for the six path models, obtained by *piecewiseSEM* and *lavaan*. Values
 1224 indicating acceptable fit are highlighted in bold.
 1225

Model	piecewiseSEM			lavaan						
	<i>C</i>	<i>df</i>	<i>P_c</i>	χ^2	<i>df</i>	<i>P_{χ^2}</i>	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

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1228 2.2. AIC-based model comparisons using real and simulated data

1229 To assess which of these models provides the best account of the data, first we calculated the
 1230 AIC value for each model (in *piecewiseSEM* this is corrected for small sample size, i.e. AIC_c)
 1231 using the real dataset. Second, we used simulated data to test which of the two methods
 1232 produces less biased conclusions. For this latter purpose, we generated simulated datasets
 1233 using the R function ‘rnorm’. The simulated datasets have the same number of variables and
 1234 sample size as the phylogenetically transformed real dataset. We fitted path models with both
 1235 *piecewiseSEM* and *lavaan* to obtain the AIC (or AIC_c) values. Then we compared Model 1a
 1236 (the model that got the highest support for model fit by the global fit indices, see Table S10)
 1237 to the other five models (Models 1b,1c, 2a, 2b, and 2c), thus conducted five pairwise
 1238 comparisons, repeated with the two methods. These paired comparisons between models
 1239 mimic the comparison we conducted with the real dataset in our study (Table 2 in the main
 1240 text). We calculated Δ AIC for each comparison as the difference between AIC values of the
 1241 two models (i.e. AIC of compared model - AIC of Model 1a, thus a positive Δ AIC value
 1242 indicates better fit for Model 1a). We repeated this procedure with 1000 simulated datasets
 1243 that resulted in 1000 Δ AIC values for each pairwise comparison. To assess whether the
 1244 comparison of two particular models produces biased results with simulated data we
 1245 calculated (1) the mean Δ AIC value of the 1000 runs (Δ AIC_{simulation}), and (2) the probability
 1246 that the simulated Δ AIC was larger than the Δ AIC value we got with the real dataset
 1247 ($P_{\geq\Delta$ AIC_{sim}}).

1248 Using real data, *piecewiseSEM* gave the lowest AIC_c for Model 1a (Table S11), a
 1249 result consistent with global model fit evaluation (see Table S10). ΔAIC_c values suggested
 1250 strong support for this model in all comparisons ($\Delta AIC_c \geq 4.1$, Table S11). In contrast,
 1251 *lavaan* results were inconsistent with global model fit evaluation because it gave very strong
 1252 support for Model 2c (Table S11), a model that had an unacceptable fit by all fit indices (see
 1253 Table S10).

1254

1255 **Table S11.** AIC-based model comparison using real and simulated data by the two methods.
 1256 AIC_c (*piecewiseSEM*) and AIC (*lavaan*) values provided for all models are based on analyses
 1257 of our real data. ΔAIC_{data} and $\Delta AIC_{simulation}$ show differences from Model 1a in pairwise
 1258 comparisons, based on analyses of real or simulated data, respectively. $P_{\geq \Delta AIC_{sim}}$ indicates the
 1259 probability that analyses of random data result in as large or larger AIC differences in support
 1260 for Model 1a than the ΔAIC values obtained with real data.

1261

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

1262

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1264 Using simulated data, we found that *piecewiseSEM* produced less biased results than *lavaan*.
 1265 First, in most cases mean simulated ΔAIC values were small and there was no strong bias in
 1266 favour of one specific model (see $\Delta AIC_{simulation}$ in Table S11), as one would expect with
 1267 random data. The only exception was the comparison between Model 1a and Model 2a in
 1268 which simulated ΔAIC produced by *piecewiseSEM* was 7.4, favouring Model 1a. Importantly,
 1269 however, these simulations indicated only a low probability for random data resulting in as
 1270 large or larger AIC differences (43.2) in support for Model 1a than the ΔAIC values we
 1271 obtained with real data (see low $P_{\geq \Delta AIC_{sim}}$ values in Table S11), suggesting that support for
 1272 Model 1a was unlikely the result of biased AIC estimates.

1273

1274 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

1275 and 2c in Table S9). On the other hand, for nested model comparisons (i.e. with Models 1b
1276 and 1c) *lavaan* produced unbiased results similarly to those we got with *piecewiseSEM*
1277 (Table S11).

1278 These analyses suggest that the two methods gave consistent results for (1) path
1279 coefficients estimates and for (2) evaluating model fit of individual path models by global fit
1280 indices (using *C* statistics in *piecewiseSEM*, and χ^2 , TLI, CFI, RMSEA, and SRMR in
1281 *lavaan*). On the other hand, simulation results indicate that AIC-based model comparisons are
1282 less biased when performed by the piecewise structural equation modelling method, at least
1283 for comparisons between non-nested models.

1284

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