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1 **Female reproductive senescence**
2 **across mammals: a high diversity of**
3 **patterns modulated by life history**
4 **and mating traits**

5

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21 ABSTRACT

22

23 Senescence patterns are highly variable across the animal kingdom. However, while empirical
24 evidence of actuarial senescence in vertebrates is accumulating in the wild and life history
25 correlates of actuarial senescence are increasingly identified, both the extent and variation of
26 reproductive senescence across species remain poorly studied. Here, we performed the first
27 large-scale analysis of female reproductive senescence across 101 mammalian species that
28 encompassed a wide range of Orders. We found evidence of reproductive senescence in
29 68.31% of the species, which demonstrates that reproductive senescence is pervasive in
30 mammals. As expected from allometric rules, the onset of reproductive senescence occurs
31 later and the rate of reproductive senescence decreases with increasing body mass and
32 delayed age at first reproduction. Moreover, for a given pace of life, females displaying a high
33 level of multiple mating and/or with induced ovulation senesce earlier than females displaying
34 a low level of multiple mating and/or with spontaneous ovulation. These results suggest that
35 both female mating behavior and reproductive physiology shape the diversity of reproductive
36 senescence patterns across mammals. We propose future avenues of research regarding the
37 role played by environmental conditions or reproductive features (e.g. type of placentation) on
38 the evolution of reproductive senescence.

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41 *Keywords:* Aging, Comparative biology, Fertility, Life-history, Reproduction

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46 **1. Introduction**

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48 From an evolutionary biology perspective, the process of senescence corresponds to a
49 decrease in the age-specific contribution to fitness (Cohen et al., 2020; Gaillard and
50 Lemaître, 2020; Monaghan et al., 2008). Therefore, senescence is mostly studied through a
51 decline in demographic rates with increasing age (i.e. actuarial and reproductive senescence).
52 Historically, senescence was thought to not show up in the wild because animals were
53 expected to die from harsh environmental conditions or anthropogenic effects (e.g. hunting)
54 before experiencing a decline in age-specific survival or reproductive rates (Nussey et al.,
55 2013). Thanks to the increasing number of long-term field studies - where individuals are
56 monitored from birth to death (Clutton-Brock & Sheldon 2010), and the subsequent
57 demographic analyses of these populations, the view that demographic senescence does not
58 occur in the wild has been totally revisited (Nussey et al. 2013). Yet, after pioneer descriptive
59 studies suggesting that mortality increases in late life (e.g. Caughley, 1966), empirical
60 evidence currently indicates that actuarial senescence is pervasive across vertebrates with
61 determinate growth in the wild (Brunet-Rossinni and Austad, 2006; Nussey et al., 2013),
62 whereas reproductive senescence was considered to be inexistent in the wild for a much
63 longer period (e.g. Caughley, 1976). On the contrary, the negative effects of age on
64 reproductive performance have been documented in laboratory rodents (e.g. Leslie and
65 Ranson, 1940) and human populations (e.g. Tietze, 1957) for a while. The first reports of
66 reproductive senescence in the wild only emerged in the late 70s (Coulson and Horobin,
67 1976; Ollason and Dunnet, 1978; Perrins and Moss, 1974; Sinclair, 1977), following the
68 insightful work by Emlen (1970), who stated that age-specific reproductive rates should
69 increase to a peak, then stay relatively constant during a prime-age stage (i.e. the period of
70 adulthood between the beginning of the reproductive peak and the onset of reproductive
71 senescence) before declining in late life.

72 Empirical evidence of reproductive senescence from wild populations of vertebrates is
73 now compelling (Nussey et al., 2013). So far, these case studies have been almost entirely
74 performed on females (but see Lemaître and Gaillard, 2017 for a review of evidence in
75 males), simply because accurate paternity assignments in the wild require the use of
76 molecular tools. Age-specific declines in a wide array of traits reflecting female reproductive
77 success have been documented, such as breeding proportions (e.g. Photopoulou et al., 2017),
78 birth rates (e.g. Lee et al., 2016), number of offspring produced at birth (e.g. Sparkman et al.,
79 2017) or at the end of parental care (i.e. at weaning or fledgling, e.g. Thorley et al., 2020), and

80 offspring survival (e.g. Karniski et al., 2018). Interestingly, it has been suggested that female
81 reproductive senescence patterns can markedly differ across species (Baudisch and Stott,
82 2019; Jones et al., 2014), even between closely related species. For instance, a population-
83 level comparison of reproductive senescence in terms of breeding success across three
84 albatross species has revealed that the age at the onset of reproductive senescence was much
85 earlier in the wandering albatross (*Diomedea exulans*) than in both the black-browed albatross
86 (*Thalassarche melanophris*) and the grey-headed albatross (*Thalassarche chrysostoma*) (Froy
87 et al., 2017). Likewise, the southern fulmar (*Fulmarus glacialisoides*) showed clear evidence of
88 senescence in breeding success, whereas the snow petrel (*Pagodroma nivea*) did not (Berman
89 et al., 2009). Despite these reports of contrasted patterns of age-specific decline in
90 reproductive performance with increasing age, there has been so far no attempt to quantify the
91 occurrence and the variation in reproductive senescence across a wide range of species, or to
92 identify the ecological and biological factors underlying the diversity of patterns. This
93 contrasts with the increasing number of comparative analyses that focused on actuarial
94 senescence (Bronikowski et al., 2011; Lemaître et al., 2020c; Péron et al., 2019b; Ricklefs,
95 1998, 2006; Ricklefs and Scheuerlein, 2001), which have notably revealed that life-history
96 strategies (e.g. Garratt et al., 2013; Ricklefs, 2010) and environmental conditions (Colchero et
97 al., 2019; Lemaître et al., 2013; Tidière et al., 2016) modulate both the onset and the rate of
98 actuarial senescence. So far, most comparative analyses of age-specific reproductive data in
99 mammals have focused on the evolution of post-reproductive lifespan (e.g. Alberts et al.,
100 2013; Cohen, 2004; Ellis et al., 2018) and did not investigate among-species differences in the
101 onset or rate of reproductive senescence. Only Jones and colleagues (2008) analyzed
102 interspecific differences in female reproductive senescence patterns (i.e. measured as the age-
103 specific decline in the number of recruited offspring) across 19 species of birds and mammals.
104 They found that the rate of reproductive senescence decreased with increasing generation
105 time, a reliable measure of the species position along the slow-fast continuum (Gaillard et al.,
106 2005). Thus, slow-living species had a lower rate of reproductive senescence than fast-living
107 species (Jones et al., 2008; see also Gaillard et al., 2016).

108 The aim of this work is twofold. First, we assess whether female reproductive
109 senescence is the rule rather than the exception across mammals by accurately quantifying
110 reproductive senescence using a large sample of mammalian species that display a high
111 diversity of lifestyles and life history strategies. This first step allowed quantifying among-
112 species variation in both the onset and the rate of reproductive senescence. Second, we
113 investigate the role of several ecological, biological and life history traits in shaping the

114 diversity of reproductive senescence patterns observed across mammalian females. Among
115 these factors, we focus on the role played by the phylogeny, the species position along the
116 slow-fast continuum, the mating behavior and the ovulation mode.

117 Following comparative analyses of many life history traits (e.g. Kamilar and Cooper,
118 2013; Wootton, 1987), notably the rate of actuarial senescence (e.g. Lemaître et al., 2020b),
119 we expected closely related species to share more similar reproductive senescence patterns
120 than distant species across the phylogeny. We also expected the onset and the rate of
121 senescence to occur earlier and to increase, respectively, with increasingly fast-living life
122 history as a direct consequence of the covariation among all biological times (i.e. life history
123 traits expressed in time units that positively covary across species, see Gaillard et al., 2016;
124 Ronget and Gaillard, 2020). Moreover, we investigated whether the number of mating
125 partners and the ovulation mode influenced the timing and intensity of reproductive
126 senescence. In most mammals, females generally copulate with more than one male during a
127 given reproductive event (Gomendio et al., 1998; Hayssen and Orr, 2017; Soulsbury, 2010).
128 The propensity of females to mate repeatedly within a reproductive event increases from
129 monogamous species to polyandrous or polygynandrous species, which is considered as a
130 way to increase fitness through genetic benefits (e.g. increased genetic diversity among
131 offspring, Jennions and Petrie, 2000; Stockley, 2003). However, multiple mating promotes
132 sperm competition (i.e. when sperm from two or more males compete to fertilize a given set
133 of ova, Parker, 1970), which in turn can lead to intense sexual conflicts between sexes
134 (Stockley, 1997). Physiological and subsequent fertility costs associated to these sexual
135 conflicts have been widely documented (see Arnqvist and Rowe, 2005; Stockley, 1997 for
136 reviews) and might increase with female age. Thus, the risk of contracting infectious diseases
137 increases steadily with age in species displaying high levels of multiple mating (Nunn et al.,
138 2014) and the negative consequences of such infections in terms of female fertility are likely
139 to be amplified at old ages due to the progressive (and taxonomically widespread) decline un
140 immune performance throughout the lifetime (Peters et al., 2019). We thus tested whether the
141 degree of multiple mating by females leads to an earlier and/or steeper reproductive
142 senescence. We also investigated whether the ovulation mode influences mammalian
143 reproductive senescence. Ovulation is a physiological process governed by complex
144 interactions between hormonal levels and external cues (e.g. photoperiod, mating) (Hayssen
145 and Orr, 2017). Mammalian females are typically divided between spontaneous and induced
146 ovulators (Soulsbury and Iossa, 2010). When spontaneous, ovulation is mostly triggered by
147 endogenous hormonal changes, whereas with induced ovulation, the mating event triggers a

148 physiological cascade that leads to ovulation. Comparative analyses performed so far have
149 revealed that the intensity of sperm competition is stronger in species with spontaneous
150 ovulation (Iossa et al., 2008; Soulsbury and Iossa, 2010). Since sperm competition can be
151 associated with long-term aging costs in females, including a decline in age-specific
152 physiological performance and fertility in females (Lemaître et al., 2020a; Stockley, 1997),
153 we expect females from species with spontaneous ovulation to suffer from an earlier and/or
154 steeper reproductive senescence than females from species with induced ovulation.

155

156 **2. Material and methods**

157

158 **2.1. Data collection**

159 Age-specific reproductive data for females were extracted from an unpublished database
160 (entitled ‘Malddaba’) built and managed by VR, JFL and JMG, and currently under
161 development. This database contains sex- and age-specific demographic data for wild
162 populations of mammals gathered from published life tables or extracted from graphs (using
163 WebPlotDigitizer (<https://automeris.io/WebPlotDigitizer/>)) over the past few years. We
164 focused mainly on reporting the m_x series, defined as number of daughters alive at birth that
165 are produced by a female of age x . For each study we collected the mean m_x value at each
166 age, as well as corresponding sample size. In some studies, the sample size was not directly
167 reported but the information on the age distribution of females was provided. In those cases,
168 we extracted the age distribution and used the number of females expected to be alive at age x
169 as the sample size of the m_x series. When m_x series were not available, we reported either the
170 age-specific litter size or the age-specific pregnancy (or birth) rates with the sample sizes (i.e.
171 in monotocous species, the m_x is equivalent to the birth rates divided by 2 when assuming a
172 balanced sex ratio at birth). Only one population per species was considered in this study.
173 When age-specific reproductive parameters were available for several populations of the same
174 species, we selected the one based on the best data quality and with the largest sample size
175 (see *Supplementary datasets* for the full list of populations included in our comparative
176 analysis).

177 Based on the accuracy of the methods used to assign the age of the individuals, we
178 distinguished two main categories of studies. The first type of study (i.e. longitudinal data)
179 corresponds to age-specific reproductive estimates obtained from the long-term monitoring of
180 individuals marked at birth or early in life when age can be accurately identified. In the
181 second type of studies (i.e. cross-sectional data), age is generally estimated through indirect

182 methods (e.g. tooth wear) and subject to uncertainty. These cross-sectional data correspond to
183 age-specific reproductive rates obtained from a single assessment of individual reproductive
184 status using individuals either controlled alive in the population or recovered as dead in the
185 field. In cross-sectional studies performed from dead recoveries, the count of embryos or
186 placental scars often provide estimates of the litter size (e.g. Lieury et al., 2017). Sampled
187 populations were also classified as hunted vs. non-hunted according to the information
188 reported in the original publication. Finally, we used the age-specific reproductive data
189 provided in the life tables to recover the age at first reproduction. We limited our research to
190 wild or semi-captive (i.e. where individuals can freely reproduce) mammalian populations.
191 However, we observed that this selection did not allow including small (and fast) mammalian
192 species, which is easily explained by the lack of long-term field studies for such species.
193 Therefore, to avoid any bias in our interpretation of the diversity of reproductive senescence
194 patterns across mammals, we included in our dataset age-specific reproductive rates gathered
195 in three captive populations of rodents, all belonging to the Cricetidae family (Field vole,
196 *Microtus agrestis*, Orkney vole, *Microtus arvalis* and Tundra vole, *Microtus oeconomus*). At
197 the end of our literature survey, we gathered age-specific reproductive data for 101
198 mammalian species (including 52 longitudinal studies and 49 cross-sectional studies)
199 encompassing a wide diversity of mammalian Orders (Figure 1). Carnivora and
200 Cetartiodactyla were the most represented Orders (35.6% and 27.7% of the species,
201 respectively) followed by Rodentia and the Primates (16.8% and 11.8% of the species,
202 respectively).

203 When possible, we extracted data on female body mass from the population where we
204 retrieved age-specific reproductive data. When this information was lacking, we looked for
205 female adult body mass from other populations. In mammals, there is widespread evidence
206 that testes mass (relative to body mass) is a reliable indicator of the level of multiple mating
207 that is experienced by females during a given reproductive event (Harcourt et al., 1995;
208 Lemaître et al., 2009; Ramm et al., 2005; Soulsbury, 2010). Indeed, multiple mating promotes
209 sperm competition among males, which in turn selects for large testes (Parker, 2016). We thus
210 used published reviews to compile data on combined adult testes mass and the corresponding
211 adult male body mass (e.g. Lüpold, 2013). Data on ovulation mode were retrieved from
212 Soulsbury (2010) and Soulsbury and Iossa (2010). All data and associated references are
213 provided in the *Supplementary datasets*.

214

215 **2.2. Measures of reproductive senescence**

216 In living organisms, age-specific fertility is predicted to “rise with age to a peak, which may
217 occur at almost any age depending on the sort of organism considered, and then fall” (Emlen
218 1970). When looking at age-specific variation in reproductive rates across mammals, this
219 prediction is most likely verified but also associated with substantial variation across species
220 in the strength of the initial increase, the form of the reproductive pattern near the peak of
221 reproduction and the strength of the decrease. For instance, in the bighorn sheep (*Ovis*
222 *canadensis*), three distinct phases are observed (Figure 2). First, the reproduction rate steadily
223 increases with age from 2 to 4 years of age and then slightly increases during the prime-age
224 stage between 4 and 12 years of age when the reproduction rate peaks and then reproductive
225 senescence begins to occur (i.e. fertility is decreasing with increasing age from 12 years of
226 age onwards). Although many mammalian species display this three-phase pattern, there is
227 still a lot of variation. For instance, the prime-age stage associated with high and constant
228 reproductive rates is not consistently observed. Reproductive rates can slightly but
229 consistently increase (such as in Bighorn sheep, Figure 2) or decrease during this prime-age
230 stage or this plateau phase can simply not exist when reproductive senescence occurs for
231 instance right after the age at first reproduction (e.g. in Short-finned pilot whale,
232 *Globicephala macrorhynchus*, Kasuya and Marsh, 1984). Lastly, reproductive senescence has
233 not been detected in all mammal species studied so far (e.g. Northern elephant seal, *Mirounga*
234 *angustirostris*, Le Boeuf and Laws, 1994).

235 To identify the different phases of age-specific variation in reproductive rates and
236 estimate when possible both the age at the onset of reproductive senescence and the rate of
237 reproductive senescence, we modeled age-specific changes of reproductive rates using
238 segmented models from the R-package *segmented* (Muggeo, 2017). We acknowledge that this
239 approach is not necessarily the most accurate to model age-specific variation in a reproductive
240 trait for each species included in our dataset. However, our goal was to assess at the large
241 scale of mammalian species the existence of reproductive senescence using a standardized
242 method, and when present to estimate both the age at the onset of reproductive senescence
243 and the rate of reproductive senescence. To account for the diversity of age-specific
244 trajectories of reproductive rates, we fitted four models of variation in reproductive rates: a
245 constant model with age, a linear model with age, a model with one threshold age including
246 two linear segments (i.e. one before and one after the threshold age) and a model with 2
247 threshold ages including 3 linear segments (i.e. one before the first age threshold, one
248 between the two age thresholds, and one after the second age threshold).

249 Models were fitted by weighting the age-specific reproductive trait by sample size.
250 Standard deviation of the reproductive trait was not used because it was not reported in most
251 cases contrary to sample size. Aggregated mean values per age were used for the model fitting
252 and, as we did not have access to the raw individual data in most cases, we had to make an
253 assumption about the distribution of the mean reproductive trait. Following the central limit
254 theorem, we considered each mean trait value to be normally distributed because individual
255 reproductive traits studied here were either binomial or Poisson distributed. All models were
256 fitted between the age at first reproduction and the last age reported in the study. For three
257 species (Crabeater seal, *Lobodon carcinophagus*; Leopard, *Panthera pardus*; Western
258 Jumping Mouse, *Zapus princeps*) we set the reproductive trait to 0 one year before the age at
259 first reproduction to anchor the models and help reach numeric convergence. For each
260 species, the most parsimonious model was selected using the Akaike Information Criterion
261 (AIC). We calculated AIC weights (AICw) to assess the relative likelihood that a given model
262 was the best among all the three fitted models (Burnham and Anderson, 2002). We selected
263 the model with the lowest AIC. To account for the propensity of AIC to retain a too complex
264 model (Link and Barker, 2006), when the difference in AIC (denoted ΔAIC) of two
265 competing models was less than two units, we retained the simplest model in accordance with
266 parsimony rules, except in two species (Alpine marmot, *Marmota marmota* and Wolf, *Canis*
267 *lupus*) for which the original studies reported statistical evidence of reproductive senescence
268 using threshold models (i.e. Berger et al., 2015, Stahler et al., 2013). In both cases, we
269 retained the model including senescence and checked that our estimates of the age at the onset
270 of reproductive senescence matched those provided in the original sources. Depending on the
271 model selected, we used a pre-defined procedure to assess the occurrence of reproductive
272 senescence (see *Appendix B* for detailed procedure).

273

274 **2.3. Statistical analyses of the factors influencing reproductive senescence**

275 To avoid any statistical issue due to phylogenetic inertia (Felsenstein, 1985), all the following
276 analyses involved phylogenetically controlled models. We used Phylogenetic Generalized
277 Least-Squares models (PGLS), with a variance-covariance matrix extracted using the R-
278 package *ape* (Paradis et al., 2004). The strength of the phylogenetic signal on the error
279 structure of each model was assessed with the Pagel's λ (Pagel, 1999, henceforth called ' λ '),
280 which is incorporated into the analysis to control for the phylogenetic dependence among
281 species (Symonds and Blomberg, 2014). Here, λ was estimated with maximum likelihood by
282 using the PGLS command from the R-package *caper* (Orme et al., 2013). In most cases, λ

283 varies between 0 (no phylogenetic signal) and 1 (the observed pattern is predicted by the
284 phylogeny; similarity among species scales proportionally to their shared evolutionary time
285 following a Brownian motion model; Pagel, 1999). As it is not possible to estimate λ for
286 binary data, we used the D statistic for this specific analysis (Fritz and Purvis, 2010), which
287 we estimated with the R-package *caper* (Orme et al., 2013). The D statistic is equal to 1 if the
288 trait of interest has a phylogenetically random distribution across the species included in the
289 phylogeny and to 0 if its evolution follows a Brownian motion model (Fritz and Purvis,
290 2010). For all the phylogenetically-controlled analyses, data were linked to a super-tree of
291 extant mammals that provides information on both topology and branch length (Bininda-
292 Emonds et al., 2008, 2007). To identify factors shaping the interspecific variation in both the
293 onset and the rate of reproductive senescence, our statistical analyses were run in the
294 following four steps.

295 First, we tested whether the probability to detect reproductive senescence was
296 influenced by the sample size (i.e. total number of reproductive records in the population; log-
297 transformed in the analysis), the hunting status of the population, the type of data (i.e.
298 longitudinal vs. cross-sectional) or the age at first reproduction (to control for the pace of
299 life). For this, we ran phylogenetically controlled logistic regressions with the presence or
300 absence of reproductive senescence using the R-package *phylolm* (Ives and Garland, 2010).
301 To identify the best models of the onset and of the rate of reproductive senescence, we
302 performed a model selection procedure using AIC (see above for a description of the
303 procedure). Second, we estimated the phylogenetic signal in both the onset and the rate of
304 reproductive senescence (see Kamilar and Cooper, 2013 for a similar approach). Third, we
305 focused on all the candidate factors for the whole set of species displaying reproductive
306 senescence ($N = 69$ species). We thus tested whether the onset of reproductive senescence and
307 the rate of reproductive senescence were influenced by both the age at first reproduction and
308 the female body mass (entered as covariates). We also included potential confounding factors
309 such as data quality (entered as a two-level categorical data: longitudinal vs. cross-sectional
310 data) and the hunting status of the population (entered as a two-level categorical data: hunted
311 vs. non-hunted population). As expected, the age at first reproduction was positively
312 associated with the female body mass (slope of 0.20 ± 0.04 , $R^2 = 0.31$, $\lambda = 0.83$, on a log-log
313 scale, *Appendix C* - Figure S1). However, we kept both variables in our model selection
314 procedure because the Pearson coefficient of correlation between the age at first reproduction
315 and female body mass ($r = 0.56$) was below the threshold of 0.7 indicating statistical issues
316 due to a collinearity among predictor variables (Dormann et al., 2013). In addition, to get

317 estimates of the rate of reproductive senescence independently of the onset of reproductive
318 senescence (slope between rate and onset of reproductive senescence of -0.81 ± 0.22 , $R^2 =$
319 0.17 , $\lambda = 0.87$, Figure 3), we included the age at the onset of reproductive senescence among
320 our set of covariates in the pool of models seeking to explain interspecific variation in the rate
321 of reproductive senescence. To identify the best models of the onset and of the rate of
322 reproductive senescence, we performed a model selection procedure using the AIC (see
323 above). Fourth, we tested on a subset of species ($N = 54$) for an effect of relative testes mass
324 on both the onset and the rate of reproductive senescence. For these analyses, we added as
325 covariates both testes mass and male body mass (Freckleton et al., 2002; Gage and
326 Freckleton, 2003) to the factors found to influence reproductive senescence traits in the
327 previous analyses, and we then updated the model selection. Fifth, we analyzed the influence
328 of the ovulation mode (i.e. spontaneous vs. induced ovulation) using another subset of species
329 ($N = 40$). We followed the exact same procedure by adding the ovulation mode (as a
330 categorical variable) to the previously identified set of factors influencing reproductive
331 senescence and by running model selection, for the onset and the rate of reproductive
332 senescence, respectively. For all the statistical analyses, the onset of reproductive senescence,
333 the rate of reproductive senescence, the age at first reproduction, testes mass and both female
334 and male body mass were log-transformed. All PGLS models were performed with R 4.0.0 (R
335 Core Development Team) using the packages *ape* (Paradis et al., 2004) and *caper* (Orme et
336 al., 2013). Unless otherwise stated, parameter estimates are given as \pm one standard error.

337

338 **3. Results**

339

340 **3.1. Occurrence of female reproductive senescence across mammalian species**

341 Reproductive senescence was detected in 68.31 % of the species (69 out of 101 species). The
342 probability to detect senescence was moderately influenced by the phylogeny ($D = 0.61$).
343 When looking at the different traits that putatively had an influence on the probability to
344 detect senescence, the selected model contains the data quality, the hunting status and the
345 sample size (*Appendix C*, Table S1; Table 1a). As we could expect, reproductive senescence
346 was more often detected in longitudinal studies (80.77 %, 42 out of 52 studies) than in cross-
347 sectional studies (55.10 %, 27 out of 49 studies) and the probability to detect reproductive
348 senescence increased with the sample size (slope of 0.36 ± 0.15 , Table 1a, *Appendix C* -
349 Figure S2). When the sample size was large (> 80 individuals), the probability to detect
350 reproductive senescence was above 0.5 (computed on the entire dataset). Reproductive

351 senescence was also more often detected in non-hunted populations (73.41 %, 58 out of 79
352 populations) than in hunted populations (50 %, 11 out of 22 populations).

353

354 **3.2. Factors modulating female reproductive senescence**

355 The age at the onset of senescence was strongly influenced by the phylogeny ($\lambda=0.96$, 95% CI
356 [0.88, 0.99]). Species that displayed a late age at the onset of reproductive senescence were
357 typically long-lived mammals (e.g. Proboscidae), which could confound the effect of the
358 phylogeny (Figure 1). To disentangle these effects, we scaled the age at the onset of
359 reproductive senescence by the age of the oldest individual sampled in the population. The
360 influence of the phylogeny on the standardized age at the onset of reproductive senescence
361 was still statistically significant but lower than the one previously observed for the *absolute*
362 age at the onset of reproductive senescence ($\lambda=0.66$, 95% CI [0.22, 0.88]; Figure 1). Three
363 cetaceans known to display extended periods of post-reproductive lifespan (see Péron et al.,
364 2019a) showed very early *relative* ages at the onset of reproductive senescence (i.e. onset
365 located at 11.5%, 24.5% and 35.4% of the lifespan for *G. macrorhynchus*, *O. orca* and *P.*
366 *crassidens*, respectively). Interestingly, when applying our statistical procedure to study
367 reproductive senescence in human populations (see *Appendix D* for an illustration), which
368 display the longest post-reproductive life ever reported in mammals, we noted a possible
369 association between an early onset of reproductive senescence and the existence of an
370 extended period of post-reproductive lifespan.

371 The selected model of among-species variation in the age at the onset of reproductive
372 senescence included both age at first reproduction (slope of 0.45 ± 0.12) and body mass
373 (slope of 0.10 ± 0.04) (Table 1b, *Appendix C* - Table S2A). As expected from the existence of
374 both time and allometric constraints on overall life histories, the onset of reproductive
375 senescence increased with both age at first reproduction and female body mass (Figure 4a,
376 4b). Once scaled, the effect of the age at first reproduction was stronger than the effect of
377 body mass (slope of 0.40 ± 0.11 vs. 0.26 ± 0.12), with an effect 1.54 times stronger. Although
378 not retained in the selected model, data quality and hunting status were also included in the
379 model with the lowest AIC value (*Appendix C* - Table S2A) suggesting that, for a given age at
380 first reproduction and a given body mass, the age at the onset of reproductive senescence
381 tends to be later in populations monitored longitudinally and hunted (Table 1c, *Appendix C* -
382 Figure S3).

383 We then analyzed the effect of testes mass (relative to body mass) on the onset of
384 reproductive senescence. The selected model only included the age at first reproduction

385 (*Appendix C* - Table S2B) but the model with the lowest AIC value included an additional
386 negative effect of relative testes mass on the onset of reproductive senescence (slope of -0.12
387 ± 0.07 , Table 1d). For a given age at first reproduction, females with high levels of multiple
388 mating (i.e. species where males carry large testes relative to body mass) tend to show an
389 earlier age at the onset of reproductive senescence (Figure 5). Finally, the onset of
390 reproductive senescence was earlier in species with induced ovulation than in species with
391 spontaneous ovulation (Figure 6). Indeed, the selected model included both the age at first
392 reproduction and the ovulation mode (*Appendix C* - Table S2C, Table 1e). Moreover, the
393 effect of the ovulation mode remained statistically significant when the relative testes mass
394 was added to the model (Table 1e). Interestingly, when considering this model, the effect of
395 testes mass (controlled for body mass) was also statistically significant (Table 1f).

396 Similar to the onset of reproductive senescence, the rate of senescence was strongly
397 influenced by the phylogeny ($\lambda=0.94$, 95% CI [0.84, 0.99], *Appendix C* - Figure S4). The
398 retained model explaining variation in the rate of reproductive senescence across mammalian
399 species included both the age at first reproduction and female body mass (Table S3A), as
400 expected from both time and allometric constraints. Thus, the rate of senescence decreased
401 with increasing age at first reproduction and female body mass (Table 1g, Figure 2c, 2d).
402 However, contrary to what was observed for the age at the onset of reproductive senescence,
403 the effect of body mass on the rate of reproductive senescence was 1.48 times stronger than
404 the effect of the age at first reproduction once both traits were scaled (slope of -0.83 ± 0.22
405 vs. -0.56 ± 0.20). Here, we can notice that the onset of reproductive senescence was not
406 retained in the selected model (*Appendix C* Table S3A), which highlights that the three
407 biological times included in the analysis positively covaried, with the age at first reproduction
408 being more closely associated to the rate of reproductive senescence than the age at the onset
409 of reproductive senescence. Subsequent analyses did not reveal any detectable influence of
410 either relative testes mass or ovulation mode on the rate of reproductive senescence (*Appendix*
411 *C* - Table S3B, S3C). Importantly, when we restricted our analyses to species for which age-
412 specific reproductive data are based on m_x series, all results were qualitatively unchanged
413 (*Appendix C* - Table S3D).

414

415 **4. Discussion**

416 *The widespread occurrence of reproductive senescence in mammalian females*

417 Our comparative analysis provides firm evidence that reproductive senescence is widespread
418 across mammals in the wild. More specifically, we found support for an age-specific decline

419 in reproductive performance in more than two-third (68.3%) of the species included in our
420 analysis, and at least four lines of evidence suggest that we currently underestimate its
421 occurrence.

422 First, the ability to detect reproductive senescence was much higher when analyzing
423 age-specific reproductive traits from longitudinal trajectories rather than cross-sectional data.
424 A higher accuracy in age assessment of longitudinal data compared to cross-sectional data is
425 most likely to be involved. For instance, while longitudinal studies are performed from
426 known-aged individuals (generally marked near birth or within the early life period), cross-
427 sectional data rely on age estimates most often based on tooth wear, which varies in reliability
428 among species (Hamlin et al., 2000). This difference in the quality of the age estimation thus
429 likely explains our results.

430 Second, the probability to detect reproductive senescence increased with sample size
431 (i.e. the number of reproductive records per species). Therefore, the absence of reproductive
432 senescence in e.g. Sea Otter (*Enhydra lutris*) or Orangutan (*Pongo pygmaeus*) might simply
433 be a direct consequence of low sample size ($N = 36$ and $N = 58$ for which the probability to
434 detect senescence was only 0.40 and 0.46, respectively; see *Appendix C* - Figure S2). This
435 shows that we faced a lack of statistical power to detect a statistically significant decline in
436 reproductive performance in late life in some hunted populations (e.g. racoon dog,
437 *Nyctereutes procyonydes*, Helle and Kauhala, 1993). It is also noteworthy that sample size
438 also influenced the shape we identified for the reproductive senescence patterns because the
439 number of segments (from 1 to 3) increased with the sample size (*Appendix C* - Table S5).

440 Third, we might have failed to detect reproductive senescence in some of the species
441 due to discrepancies between our approach and the approaches used in the original studies. To
442 compare reproductive senescence across mammalian species, we had to apply a standardized
443 approach to estimate parameters of reproductive senescence across all species, which has led
444 to some differences in the data themselves (aggregated data per age in our study vs. individual
445 data in the original study) and also in the way data were analyzed. For instance, in Columbian
446 ground squirrels (*Spermophilus columbianus*), Broussard and colleagues (Broussard et al.,
447 2003) detected reproductive senescence by comparing two groups of females (2-5 years old
448 vs. 6-9 years old), whereas we did not detect reproductive senescence by analyzing the full
449 age-dependent reproductive trajectory. Despite these constraints the species-specific estimates
450 we reported for the onset of reproductive senescence closely matched the information
451 provided in the original source (*Appendix A*, Table S2).

452 Lastly, there is now widespread evidence that the senescence process is asynchronous
453 among traits (Gaillard and Lemaître, 2017; Promislow et al., 2006). For instance, some
454 immune traits drop sharply with increasing age, while others remain quite constant throughout
455 life (Cheynel et al., 2017). Interestingly, the asynchrony in senescence patterns can also occur
456 among traits within the single reproductive function (Lemaître and Gaillard, 2017). Let
457 compare for instance reproductive senescence in Alpine marmot (*Marmota marmota*) and
458 Soay sheep (*Ovis aries*). In marmots, female litter size decreases with increasing age but
459 offspring mass stays constant with maternal age (Berger et al., 2015b), whereas the opposite
460 is observed in Soay sheep (Hayward et al., 2013). Our analysis mostly focused on the m_x
461 values but we cannot exclude that species in which we did not detect reproductive senescence
462 display age-specific decline in at least another reproductive trait. For instance, a recent meta-
463 analysis demonstrated that a decreasing offspring survival with increasing maternal age is
464 widespread in mammals (Ivimey-Cook and Moorad, 2020).

465 Overall, current evidence suggests that reproductive senescence is - similarly to
466 actuarial senescence - the rule rather than the exception in mammals. Nevertheless, the shape
467 of reproductive senescence (here depicted by the variable number of segments included in the
468 selected model, *Appendix A*, Table S1) as well as its timing (i.e. age at the onset of
469 reproductive senescence) and intensity (i.e. rate of reproductive senescence) display a high
470 diversity across mammalian species.

471

472 *The influence of life history, mating behavior and ovulation mode on reproductive senescence*

473 As predicted, we found that both the onset and the rate of reproductive senescence were
474 tightly associated with the age at first reproduction. In absence of data required to estimate
475 generation time, the age at first reproduction constitutes the most reliable proxy of the species
476 position along the slow-fast continuum (Gaillard et al., 2005). As previously acknowledged
477 by Jones et al. (2008), slow species show a later age at reproductive senescence and a lower
478 rate of reproductive senescence compared to fast species. The species ranking according to
479 the pattern of covariation in biological times is the most recognized axis of variation across
480 mammalian species (Gaillard et al., 2016; Stearns, 1983). Since both the onset and the rate of
481 reproductive senescence can be expressed in time units (years and years⁻¹, respectively), these
482 two metrics are logically aligned with the other biological times (e.g. generation time, age at
483 first reproduction, lifespan or gestation length). Moreover, as several life history traits
484 measured in time units are known to be strongly influenced by phylogeny (e.g. Capellini et
485 al., 2008 for the sleep duration; Wootton, 1987 for the age at first reproduction, Lemaître et

486 al. 2020b for adult lifespan), it is not so surprising that both the onset and the rate of
487 reproductive senescence also depend on the species taxonomic position. When the onsets of
488 reproductive senescence are scaled by the maximum lifespan recorded in the focal
489 populations, (i.e. standardized for the species pace of life), the influence of the phylogeny was
490 unsurprisingly strongly reduced.

491 Once controlled for the species-specific pace of life, we found that traits associated
492 with female mating behavior and physiology influenced the timing of reproductive
493 senescence. Interestingly, the rate of reproductive senescence was not detectably influenced
494 by mating patterns, which adds to the growing evidence that both the onset and the rate of
495 senescence have to be investigated simultaneously when it comes to identify factors
496 influencing senescence patterns. Contrary, to our expectations, the onset of reproductive
497 senescence was earlier in species with induced ovulation than in those with spontaneous
498 ovulation. This might seem surprising at first glance because the intensity of sperm
499 competition (and its possible deleterious consequences for females) are known to be higher in
500 presence of spontaneous ovulation (Iossa et al., 2008; Soulsbury and Iossa, 2010). Yet, two
501 hypotheses can be proposed to explain this unexpected result. First, in many species with
502 induced ovulation, males bear keratinized spines on the surface of the penis (e.g. Dixson,
503 1991 common marmoset *Callithrix jacchus*; Lemaître et al., 2012 in bank voles, *Myodes*
504 *glareolus*), which could be involved in the induction of ovulation during the mating (Parag et
505 al., 2006; Stoddart and Milligan, 1979). The density of spines on the male genitalia can in
506 some species be high, with spines being particularly sharp (Orr and Brennan, 2016; Parag et
507 al., 2006; Stockley, 2002). On the long run, repeated injuries caused by spines to the female
508 reproductive tract likely impair female's reproductive condition and might be responsible for
509 the earlier age at the onset of reproductive senescence we reported for these species. While
510 this hypothesis clearly deserves further consideration from studies performed both within and
511 across species, it might not be the whole story, as some species with spontaneous ovulation
512 can also bear spines (e.g. in some primates, see Stockley, 2002). Another (non-mutually
513 exclusive) explanation is that the efficiency of the neuro-endocrinological pathway regulating
514 the relationship between mating and ovulation (reviewed in Bakker and Baum, 2000)
515 decreases with increasing age. So far, the ageing of many physiological functions governing
516 female reproduction has been described in human and laboratory rodents (vom Saal et al.,
517 1994). However, whether neuronal mechanisms controlling the cascade of physiological
518 responses (e.g. release of reproductive hormones by the hypothalamic–pituitary–gonadal axis)

519 following mating in species with induced ovulation are particularly prone to senescence is
520 currently unknown.

521 We also found that - independently of the ovulation mode - species where females
522 mate multiply with different males during a given reproductive bout (i.e. when male's relative
523 testes mass is high) also suffer from an earlier onset of reproductive senescence. This
524 contrasts with a previous comparative analysis that revealed that females displaying such
525 behavior do not pay any costs in terms of reduced lifespan or strengthened actuarial
526 senescence (Lemaître and Gaillard, 2013). Overall, this suggests that the physiological costs
527 associated with multiple mating in females are paid in terms of reproductive senescence rather
528 than actuarial senescence. A few hypotheses can be elaborated to explain this link. For
529 instance, evidence that infectious diseases negatively impact reproductive success is
530 increasing (e.g. Nguyen et al., 2015; Pioz et al., 2008). The increased risk of contracting
531 sexually transmitted diseases throughout life in promiscuous females (Nunn et al., 2014)
532 might compromise reproductive performance in late life. In addition, mammalian females
533 might also suffer from the repeated exposure to the male seminal fluid. Such deleterious
534 consequences have been reported in *Drosophila melanogaster*, for which some specific
535 seminal fluid proteins influence various aspects of female reproductive physiology, notably
536 receptivity (see Chapman, 2001 for a review). The composition of the seminal fluid is also
537 complex in mammals (Poiani, 2006) and can impact female reproductive physiology (e.g. by
538 inducing ovulation in some camelids, see Ratto et al., 2010). However, whether male seminal
539 fluid proteins are associated with long-term deleterious effects on female reproductive success
540 is yet to be investigated and constitutes one promising avenue of research.

541

542 *Future directions*

543 We provide a first evidence of the high diversity of reproductive senescence patterns across
544 mammals in the wild and identify key biological and behavioral factors that shape this
545 diversity. Yet, we are only scratching the surface of the reproductive senescence conundrum
546 and we point out below some of the most promising avenues for future research in this area.

547 We found a huge diversity of reproductive senescence patterns across mammalian
548 species. Nevertheless, whether this interspecific variation translates to high variation among
549 populations within a given species remains unknown. Indeed, while there is now compelling
550 evidence that environmental conditions modulate age-specific survival patterns, as individuals
551 in captivity have longer lifespan and similar or later onset of actuarial senescence than their
552 wild conspecifics (Tidière et al. 2016), similar large-scale comparative studies are lacking for

553 reproductive senescence. Empirical studies have revealed that the amount of resources
554 available in the environment positively influences the female reproductive output (e.g.
555 Persson, 2005). However, as females age, their ability to maintain large home range (e.g. Froy
556 et al., 2018 in Soay sheep, *Ovis aries* and red deer, *Cervus elaphus*), to acquire resources (e.g.
557 MacNulty et al., 2009 in wolves) or to eat and digest food (e.g. Gaillard et al. 2015 for a study
558 on tooth wear in ruminants) are impaired, which ultimately decreases the efficiency of food
559 provisioning to their progeny during the energy-demanding period of maternal care
560 (Gittleman and Thompson, 1988; Clutton-Brock et al. 1989; see also below) and thereby
561 jeopardizes their reproductive success. However, whether the quality and quantity of
562 resources available in the environment mitigate the reproductive ageing cost of these
563 functional declines remains to be investigated. Moreover, following a resource-based
564 allocation trade-off, a high reproductive effort during early life can be paid in terms of
565 increased reproductive senescence (Douhard et al., 2020; Lemaître et al., 2015; Nussey et al.,
566 2006). For instance, it has been suggested that the early onset of reproductive senescence
567 reported in domestic mammals might be due to the strong artificial selection for a high
568 reproductive effort during early life in domestic animals (Myrsterud et al., 2002 for sheep,
569 Grange et al. 2009 for horses, *Equus caballus*). Alternatively, whether these costs are rather
570 induced by a mismatch with environmental conditions remains unknown. In that context,
571 comparing mammalian populations facing contrasted environmental conditions would allow
572 assessing whether some species simply escape reproductive senescence or whether
573 particularly good environmental conditions prevent reproductive senescence to occur.

574 While we found that female mating behavior, ovulation mode, allometry and pace of
575 life all shape reproductive senescence, many other biological factors are likely to influence
576 either its onset or its rate. It is beyond the scope to list all of them but we can emphasize two
577 aspects we consider as being particularly promising: the cost of lactation and the type of
578 placentation. In mammals, the lactation period constitutes the most energy-demanding part of
579 the reproductive cycle (Clutton-Brock et al., 1989) and to produce high quality milk in
580 sufficient quantity, females need to increase their food intake. Interestingly, the magnitude of
581 this extra food intake varies substantially across species (see Douhard et al., 2016; Hayssen
582 and Orr, 2017 for reviews), and the energy intake required by lactating females can
583 sometimes be two- or threefold that required during the non-breeding period (see Speakman,
584 2008 for some striking examples in rodents and insectivores). The physiological costs
585 associated to lactation are diverse and well-documented, ranging from organ remodeling (e.g.
586 increased size of liver, pancreas and mammary glands) to a risk of hyperthermia or bone loss

587 (due to the need to transfer calcium to the offspring) (Dufour and Sautner, 2002; Jasienska,
588 2013; Speakman, 2008). On the contrary, much less is known on the long-term fitness costs of
589 lactation, notably in terms of ageing. We hypothesize that the repeated physiologically costly
590 periods of lactation should translate into an earlier/and or stronger reproductive senescence.
591 Moreover, such costs are likely to be buffered or exacerbated by the quality of the
592 environment, which in turn might constitute selective pressures towards specific reproductive
593 tactics. For instance, the harsh environmental conditions faced by female white-tailed deer
594 (*Odocoileus virginianus*) in Minnesota (i.e. severe winters and wolf predation) might explain
595 their relatively low allocation to reproduction throughout their lives, a tactic that might
596 ultimately minimize reproductive senescence as reported by DelGuidice and colleagues
597 (DelGiudice et al., 2007) in this population. Another interesting feature of the mammalian
598 reproductive machinery is the structure of the placenta that can influence the amount of
599 nutrients transferred by the mother to the fetus(es). Indeed, the morphology of the placenta is
600 highly variable across species, notably in its degree of invasiveness and interdigitation
601 (Wildman, 2011), which ultimately modulates the degree and the surface for nutrient transfer
602 during gestation (Capellini et al., 2011). Therefore, the placenta constitutes a critical organ for
603 parent-offspring conflicts (Haig, 1993). It has already been observed that the embryo growth
604 rate is approximately twice as fast in more interdigitated placentas (Capellini et al., 2011) and
605 that placenta types covary with the pace of life. Slow-living species are generally
606 characterized by an invasive placenta (i.e. hemochorial placenta) while fast species are most
607 often characterized by less invasive placenta types (i.e. endotheliochorial or epitheliochorial
608 placenta) (Garratt et al., 2013). The evolution of a less invasive placenta has allowed mothers
609 to limit fetal manipulation (i.e. when food transfer during gestation is largely under the
610 fetus(es) control) and to control better food provisioning among offspring and across
611 reproductive events (Garratt et al., 2013). Therefore - everything else being equal - such
612 placenta types might confer to females a way to buffer reproductive senescence.

613 As mentioned earlier, our work focused on female reproductive rate, as females
614 concentrate the huge majority of the data published so far. However, recent studies have
615 highlighted the evolutionary relevance of studying male reproductive senescence (Lemaître
616 and Gaillard, 2017; Monaghan and Metcalfe, 2019), as well as its potential medical
617 implications (Levine et al., 2018). As a consequence, evidence that male reproductive
618 senescence is pervasive in the wild (reviewed in Lemaître and Gaillard, 2017) is
619 accumulating. Similar to what we have discussed above for females, male reproductive
620 senescence can be assessed through a large variety of traits, notably an age-specific decline in

621 secondary sexual traits, ejaculate quality, mating rate or reproductive success. However, to be
622 directly comparable, male and female reproductive senescence need to be investigated on the
623 same trait and the potential confounding effects of the partner's age need to be accounted for
624 (Thorley et al., 2020). Surprisingly, we still lack a comprehensive theoretical framework for
625 the evolution of sex differences in reproductive senescence. However, following our current
626 life history framework, we can expect polygynous mammals where males disproportionately
627 allocate to sexual competition to display most strongly male-biased reproductive senescence.
628 Data available so far suggest that this scenario might be true. In the monogamous meerkat
629 (*Suricata suricatta*), no sex difference in reproductive senescence has been detected (Thorley
630 et al., 2020) while in polygynous red deer (*Cervus elaphus*), the rate of reproductive
631 senescence (measured through annual fecundity) is much steeper in males than in females
632 (Nussey et al., 2009; see also Muller et al., 2020 in chimpanzee, *Pan troglodytes*
633 *schweinfurthii* but see Festa-Bianchet, 2012 for some counter-examples, e.g. Bighorn sheep,
634 *Ovis canadensis*, in Ram mountain). Studies investigating these questions in a wider range of
635 species are now required.

636

637

638

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644

645 **Author contributions**

646 JFL and JMG designed the study. JFL, JMG and VR collected, analyzed and wrote the
647 manuscript.

648

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655

656 **Declaration of Competing Interest**

657 The authors declare no conflict of interest

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- 1000

1001 **Table 1:** Phylogenetically controlled comparative analyses of the probability to detect
 1002 reproductive senescence and on factors influencing both the onset and the rate of reproductive
 1003 senescence. This table provides parameter estimates from the models discussed in the text
 1004 (statistically significant effects occur in bold). For each model displayed below, the estimates
 1005 are not scaled.

	Dependent variables	Independent variables	Slope ± SE	t	λ [95% CI]	N	R²
a	Probability to detect reproductive senescence	Intercept	1.29 ± 0.40	3.22	NA	101	NA
		Sample size	0.36 ± 0.15	2.32			
		Hunting status	-1.24 ± 0.55	-1.72			
		Data quality	-0.71 ± 0.41	-1.72			
b	Onset of reproductive senescence	Intercept	0.78 ± 0.59	1.31	0.75 [NA; 0.93]	69	0.4
		Age at first reproduction	0.45 ± 0.12	3.68			
		Female body mass	0.10 ± 0.04	2.19			
c	Onset of reproductive senescence	Intercept	0.95 ± 0.51	1.85	0.63 [NA; 0.91]	69	0.5
		Age at first reproduction	0.52 ± 0.12	4.39			
		Female body mass	0.08 ± 0.04	1.92			
		Hunting status	0.26 ± 0.16	1.6			
		Data quality	-0.28 ± 0.13	-2.19			
d	Onset of reproductive senescence	Intercept	0.42 ± 0.47	0.91	0 [NA; 0.80]	54	0.704
		Age at first reproduction	0.62 ± 0.13	5.05			
		Testes mass	-0.12 ± 0.07	-1.75			
		Male body mass	0.16 ± 0.07	2.35			
e	Onset of reproductive senescence	Intercept	1.70 ± 0.13	13.04	0 [NA; 0.367]	40	0.74
		Age at first reproduction	0.71 ± 0.09	7.96			
		Ovulation mode	-0.50 ± 0.18	-2.71			
f	Onset of reproductive senescence	Intercept	0.45 ± 0.55	0.81	0 [NA; 0.45]	34	0.78
		Age at first reproduction	0.53 ± 0.13	4.11			
		Ovulation mode	-0.74 ± 0.22	-3.38			
		Testes mass	-0.25 ± 0.10	-2.44			
		Male body mass	0.23 ± 0.09	2.61			
g	Rate of reproductive senescence	Intercept	0.81 ± 0.96	0.85	0.58 [0.18; 0.86]	69	0.49
		Age at first reproduction	-0.64 ± 0.23	-2.78			
		Female body mass	-0.31 ± 0.08	-3.85			

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1008 **Captions for figures**

1009

1010 **Figure 1:** Phylogenetic tree of the species analyzed. The inner band displays the occurrence
1011 of reproductive senescence (i.e. presence vs. absence), the middle band displays the *absolute*
1012 onset of reproductive senescence (in years) and the outer band displays the *relative* onset of
1013 reproductive senescence (absolute onset of reproductive senescence divided by the age of the
1014 oldest individual sampled in the population, in proportion).

1015

1016 **Figure 2:** Age-specific m_x in Bighorn sheep, *Ovis canadensis* at Ram mountain. The fit of the
1017 selected two-threshold segmented model is presented. There are three distinct phases in the
1018 age-specific reproductive trajectory. The section (A) corresponds to the increasing phase of
1019 the reproductive output for young adult females. The section (B) corresponds to a phase for
1020 prime-age females during which m_x slightly increases and the section (C) corresponds to a
1021 senescent phase characterized by both an onset and a rate of reproductive senescence.

1022

1023 **Figure 3:** Relationship between the rate of reproductive senescence and the onset of
1024 reproductive senescence (on a log-log scale) (Slope of -0.81 ± 0.22 ; $R^2 = 0.17$; $N=69$ species).

1025

1026 **Figure 4:** Relationship between the age at the onset of reproductive senescence (a) or the rate
1027 of reproductive senescence (c), and the age at first reproduction (on a log-log scale) ($N=69$
1028 species). In panels (a) and (c) data are represented independently of female body mass while
1029 in panels (b) and (d) data are split according to the female body mass category (light, $N= 34$
1030 species, heavy, $N = 35$ species in each body mass category).

1031

1032 **Figure 5:** Relationship between the onset of reproductive senescence and the relative testes
1033 mass ($N=54$ species). The onset of reproductive senescence corresponds to the residuals of the
1034 regression of the onset of reproductive senescence against the age at first reproduction (on a
1035 log-log scale). The relative testes mass corresponds to the residuals of the regression of the
1036 testes mass against the male body mass (on a log-log scale). In this model, the influence of the
1037 phylogeny is not statistically different from 0 ($\lambda = 0$ [NA, 0.80]) and the residuals of the
1038 models were thus computed with Ordinary Least Square regressions.

1039

1040 **Figure 6:** Box-plot displaying the differences in the onset of reproductive senescence between
1041 species with spontaneous ($N= 28$) and induced ($N = 12$) ovulation. The onset of reproductive
1042 senescence corresponds to the residuals of the regression of the onset of reproductive
1043 senescence against the age at first reproduction (on a log-log scale, $N=40$ species). In this
1044 model, the influence of the phylogeny is not statistically different from 0 ($\lambda = 0$ [NA, 0.37])
1045 and the residuals of the models were thus computed with Ordinary Least Square regressions.

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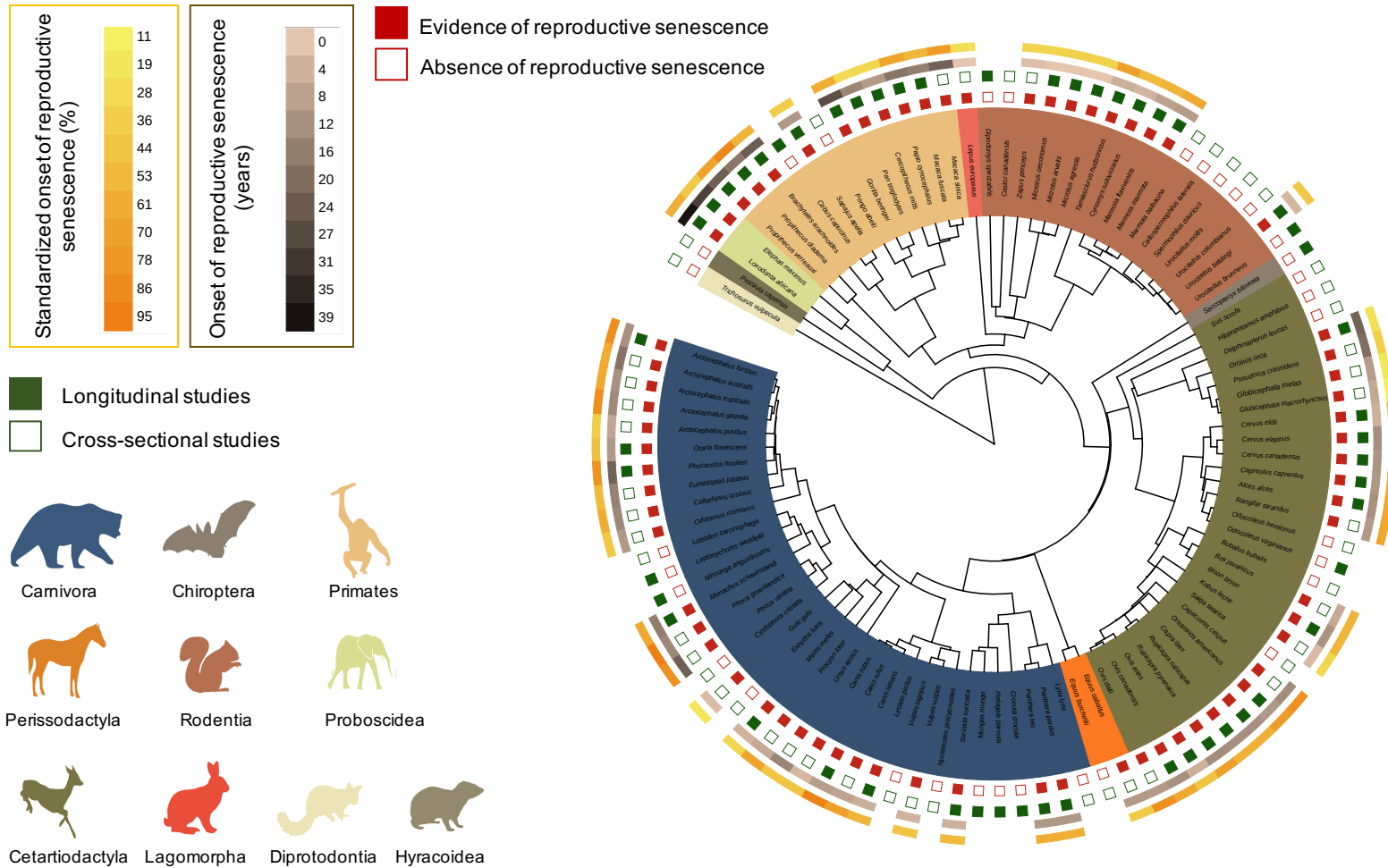
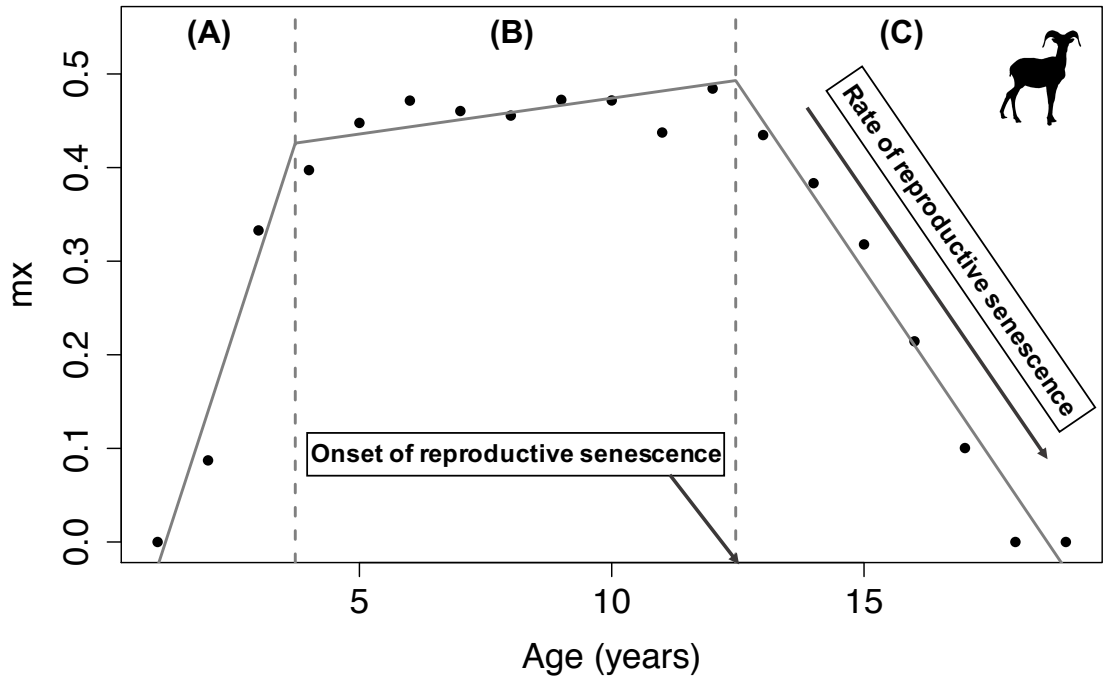


FIGURE 1

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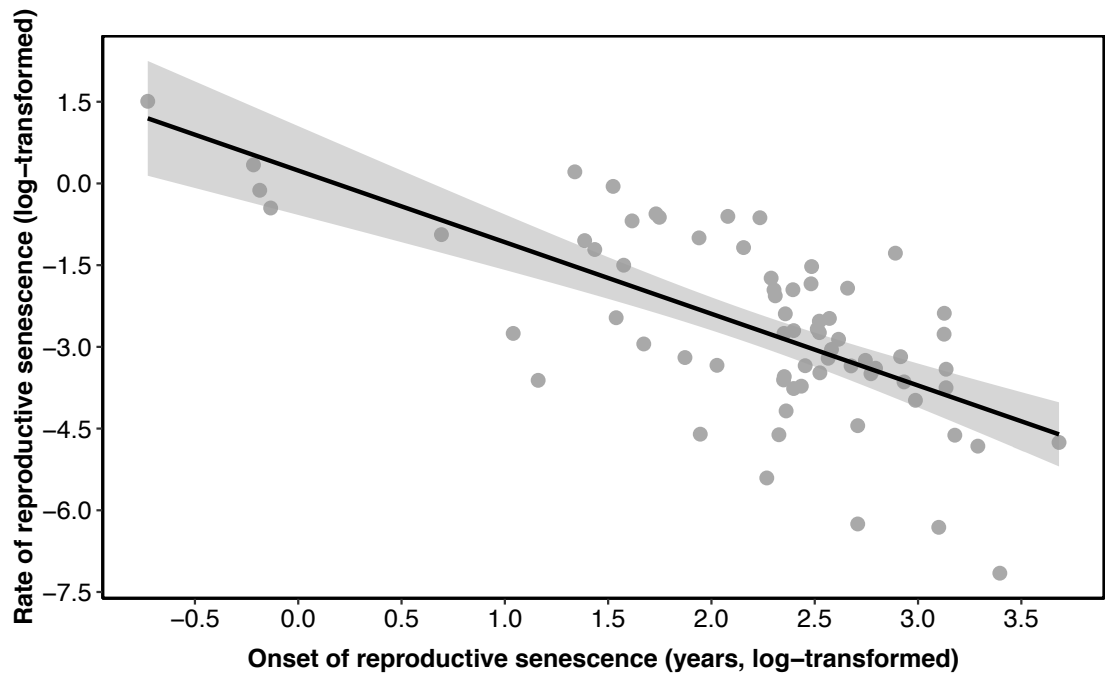
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FIGURE 2

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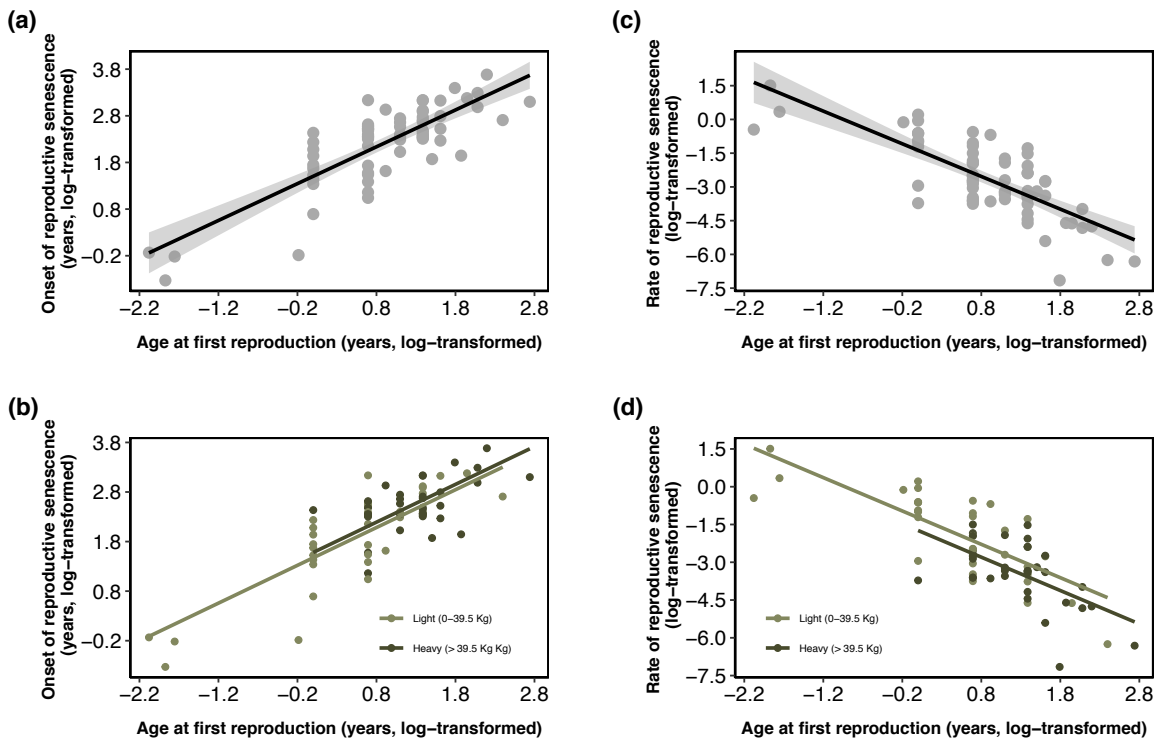


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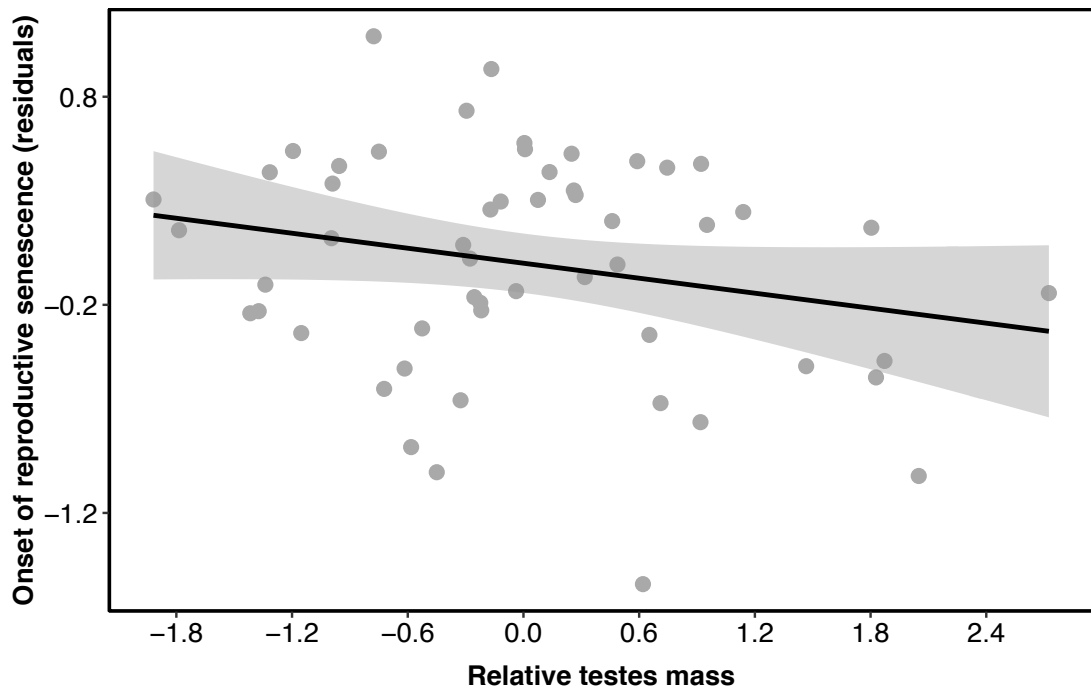
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FIGURE 3



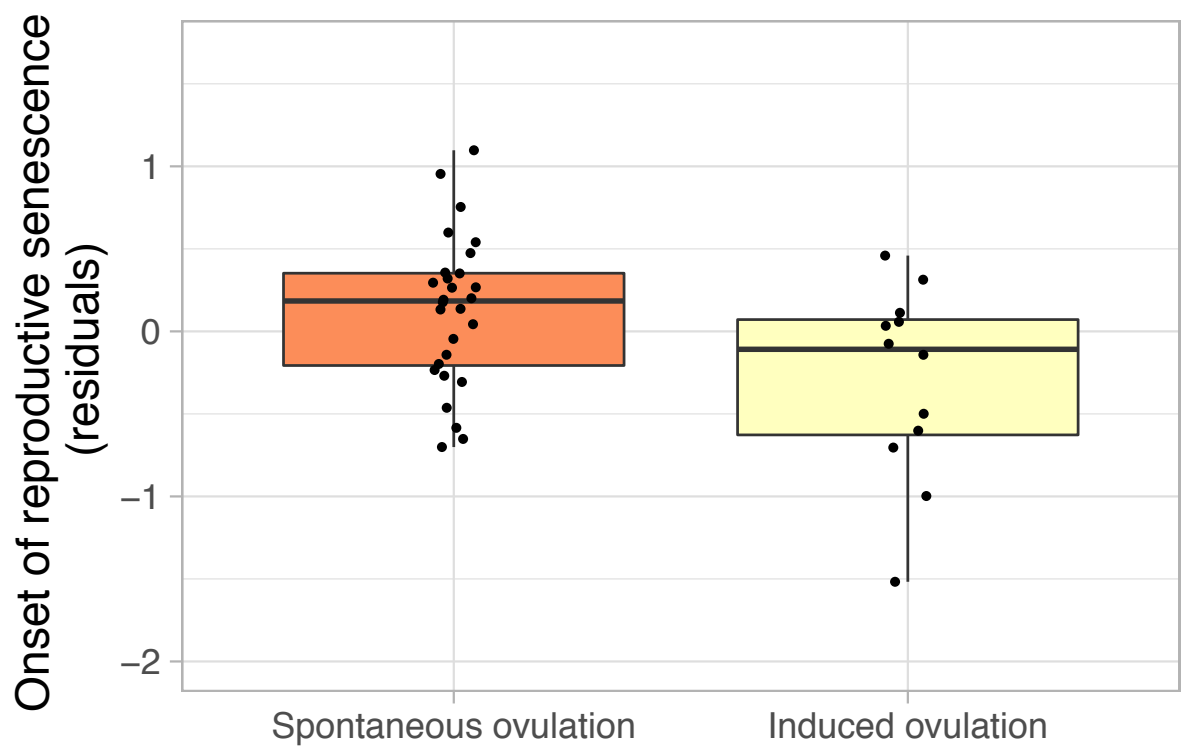
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FIGURE 4



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FIGURE 5



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FIGURE 6