Sex-independent senescence in a cooperatively breeding mammal

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ABSTRACT

1. Researchers studying mammals have frequently interpreted earlier or faster rates of ageing in males as resulting from polygyny and the associated higher costs of reproductive competition.

2. Yet few studies conducted on wild populations have compared sex-specific senescence trajectories outside of polygynous species, making it difficult to make generalised inferences on the role of reproductive competition in driving senescence, particularly when other differences between males and females might also contribute to sex-specific changes in performance across lifespan.

3. Here, we examine age-related variation in body mass, reproductive output and survival in dominant male and female meerkats, *Suricata suricatta*. Meerkats are socially monogamous cooperative breeders where a single dominant pair virtually monopolize reproduction in each group and subordinate group members help to rear offspring produced by breeders.

4. In contrast to many polygynous societies, we find that neither the onset nor the rate of senescence in body mass or reproductive output show clear differences between males and females. Both sexes also display similar patterns of age-related survival across lifespan, but unlike most wild vertebrates, survival senescence (increases in annual mortality with rising age) was absent in dominants of both sexes, and as a result, the fitness costs of senescence were entirely attributable to declines in reproductive output from mid- to late-life.

5. We suggest that the potential for intrasexual competition to increase rates of senescence in females – who are hormonally masculinised and frequently aggressive – is offset by their ability to maintain longer tenures of dominance than males, and that these processes combined lead to similar patterns of senescence in both sexes.

6. Our results stress the need to consider the form and intensity of sexual competition as well as other sex-specific features of life history when investigating the operation of senescence in wild populations.

Key words: actuarial senescence, ageing, cooperative breeding, fitness costs, gerontology, life history, reproductive value, terminal declines

INTRODUCTION

Across vertebrate and invertebrate taxa there is striking variation in the extent to which rates of senescence differ between males and females (Austad, 2011; Barford & Dorling, 2006; Carroll & Sherratt, 2017). One of the more common arguments put forward to explain this diversity relates to variation in the intensity of reproductive competition. Life history theories of senescence predict that any increase in the allocation of resources to reproduction in early life occurs to the detriment of reproductive performance and somatic maintenance in later life, translating into an earlier onset, and faster rate of senescence in the sex experiencing increased reproductive competition (as per the life history theories of senescence- the antagonistic pleiotropy and disposable soma theories; Austad & Hoffman, 2018; Kirkwood, 2017; Williams, 1957). Because sex differences in reproductive competition are often related to the mating system of a species, the comparison of senesence trajectories across mating systems has provided a useful framework for exploring some of the key predictions of the life history theories of senescence. For example, in polygynous, sexually dimorphic mammals, where males fight frequently and display energetically costly traits that improve fighting success or help to monopolise access to females, it is often males that have shorter lifespans and show higher rates of survival ('actuarial') senescence than females (Clutton-Brock & Isvaran, 2007; Loison, Festa-Bianchet, Jullien, Jorgenson, & Gaillard, 1999; Promislow, 1992; Toïgo &

75 Gaillard, 2003). In contrast, in monogamous taxa, where levels of intrasexual competition are
76 closer to parity, sexual dimorphism in longevity and rates of senescence is often absent, or
77 much reduced (Allman, Rosin, Kumar, & Hasenstuab, 1998; Brownikowski et al., 2011;
78 Clutton-Brock & Isvaran, 2007).

79 Yet not all studies that have looked for sex differences in polygynous species have
80 found them (Tidière et al., 2014, 2015; Toïgo & Gaillard, 2003), and more generally, the
81 evidence for a direct role of sexual selection on rates of ageing is equivocal (Bonduriansky,
82 Maklakov, Zajitschek, & Brooks, 2008; Graves, 2007; Maklakov, Bonduriansky, & Brooks,
83 2009). This is likely because males and females can compete in different ways, the traits
84 involved can entail different costs, and might also be expressed at different stages of lifespan
85 (Clutton-Brock, 1983; Ralls & Mesnick, 2009; Stockley & Bro-Jørgensen, 2011; Tompkins &
86 Anderson, 2019). In this context, it is important to appreciate that sex differences in survival
87 rates and senescence depend on the characteristics of the two sexes which, in turn, depend on
88 the specific selection pressures generated by intraspecific competition in the two sexes rather
89 than on sex differences in reproductive variance (Clutton-Brock, 1983). As a result, polygynous
90 breeding systems where reproductive variance is higher in males than females will not
91 necessarily generate higher rates of mortality and senescence in males in all species.
92 Conversely, there are likely to be monogamous species where contrasts in the life histories of
93 males and females generate sex differences in survival and rates of senescence even if there are
94 no sex differences in variance in breeding success. Furthermore, there are other differences
95 between the sexes that are not directly connected to the intensity of reproductive competition,
96 such as sex differences in parental care (Allman et al, 1998), heterogamy (Marais et al., 2018),
97 or maternal transmission of the mitochondrial genome (Beekman, 2014; Zeh & Zeh, 2005),
98 which may contribute to sex differences in senescence and further obscure patterns related to
99 the mating system.

Existing data from alpine marmots *Marmota marmota* are particularly useful for highlighting the importance of life history features for sex-specific patterns of senescence in the wild. Alpine marmots are socially monogamous, but males nonetheless face high costs of territoriality (Arnold, 1990) and are frequently challenged by out-of-group males, whereas females can largely suppress challenges from females within the group (Cohas, Yoccoz, Da Silva, Goossens, & Allainé, 2006). Consequently, male alpine marmots encounter greater costs of reproductive competition and this has been used to explain why male body mass deteriorates in later life whereas female body mass shows no sign of senescing (Tafani et al., 2013). The fact that males also experience greater energetic costs of hibernation could also be a factor (Arnold, 1988). However, despite senescence in male body mass there is no clear sex difference in the intensity of survival senescence in this species, raising the possibility that male body mass declines influence fitness by acting through reproduction rather than survival (Berger et al., 2016). How commonly other non-polygynous mammals in the wild display similar sex-specific patterns of senescence is unknown as most information on monogamous taxa comes from birds or uses data from captive populations. In addition, most studies of sex differences in senescence have focussed on mortality data (Gaillard, Garratt, & Lemaître, 2017), but to understand the mechanisms underlying age-related changes in fitness in naturally regulated populations, it is important to examine age-related changes in other biological parameters that are associated with individual performance and indicate why the sexes differ. This might include age-specific changes in reproductive effort (Lemaître & Gaillard, 2017), body mass and condition (Hämäläinen et al., 2014; Tafani et al., 2013), immune function (Beirne, Waring, Mcdonald, Delahay, & Young, 2016), and haematological parameters (Jégo et al., 2014), amongst others (Nussey, Froy, Lemaître, Gaillard, & Austad, 2013). From such work, it has become increasingly clear that different fitness-related traits can display divergent age-related trajectories within individuals (Evans, Gustafsson, & Sheldon, 2011; Hayward et al., 2015;

125 Nussey et al., 2009), and are not necessarily closely related to each other (Bouwhuis, Choquet,
126 Sheldon, & Verhulst, 2012). These observations contradict Williams' prediction that
127 senescence "should always be a generalized deterioration" (1957; see Gaillard & Lemaître,
128 2017) and highlight the need to consider a wider range of life histories in order to better
129 understand the operation of senescence in natural populations.

130 In this study, we examine sex-specific patterns of senescence across a variety of traits
131 in a wild, naturally regulated population of meerkats, *Suricata suricatta,* a socially
132 monogamous, cooperatively breeding mongoose. In meerkats, reproduction is monopolised by
133 a dominant male and a dominant female in each group (Clutton-Brock & Manser, 2016) with
134 subordinate individuals of both sexes helping to protect and feed juveniles born in the group.
135 Only a small proportion of individuals ever acquire dominance (Duncan, Gaynor, & Clutton-
136 Brock, 2018; Spong, Hodge, Young, & Clutton-Brock, 2008), with those that are unsuccessful
137 in doing so experiencing an increasing mortality risk that is associated with extended periods
138 of time spent away from the natal group beyond 1.5 years of age (Cram et al., 2018). As a
139 result, subordinate individuals have shorter longevities than dominants so that the potential to
140 detect senescence in subordinate individuals is limited (Sharp & Clutton-Brock, 2010).

141 While both sexes display similarly high levels of reproductive monopoly, differences
142 in the nature of intrasexual competition in meerkats generates a larger reproductive skew in
143 females than in males (Clutton-Brock et al., 2006). Dominant females display higher
144 circulating levels of testosterone (Davies et al., 2016; a trait associated with increased
145 senescence rates elsewhere, Brooks & Garratt, 2017), and also show high levels of female-
146 female aggression, often prompting dominant females to evict subordinates and commit
147 infanticide of non-descendant kin (Clutton-Brock & Manser, 2016). Dominant females are also
148 highly fecund and it is not unusual for them to produce three litters in a single calendar year
149 (Clutton-Brock & Manser, 2016). Together, these characteristics are predicted to be associated

with higher rates of senescence in females (see Sharp & Clutton-Brock, 2011a), reversing the sexual dimorphism typically observed in polygynous mammals. However, while vacant positions of dominance prompt intense female-female aggression, once dominance has been acquired, dominant females are unlikely to be displaced because they can evict subordinate females before they have reached an age and mass at which they become serious competitors (Duncan et al., 2018), and because the costs of challenging are prohibitively high (Sharp & Clutton-Brock, 2011b). By contrast, dominant males experience a consistent risk of displacement across their tenure from immigrant males (Spong et al, 2008) and disperse from their natal group should the incumbent dominant female die. Consequently, the duration of effective breeding (tenure length) is, on average, shorter in males and than females. In other mammals, sex differences in the duration of effective breeding is correlated with sex differences in life expectancy (Clutton-Brock & Isvaran, 2007), and so the the reduced duration of effective breeding in male meerkats could partly offset the costs of intrasexual competition on females and cause sex differences in senescence to be closer to parity.

To investigate sex-specific patterns of senescence in meerkats we examined age-related changes in body mass, reproductive output and annual survival in individuals that acquired dominance within their lifetime. We then used the estimates for age-related changes in reproduction and survival to calculate the fitness costs of senescence for dominant meerkats (i.e. the difference between observed reproductive value and the hypothetical reproductive value if senescence were not occurring). In so doing, we add to the small number of mammal species in which such metrics have been calculated (Bouwhuis et al., 2012; Kowald & Kirkwood, 2015), and provide the first information from a cooperative breeder. Lastly, we examined whether the reproductive declines we detected in one sex contributed to the reproductive declines shown by the other sex (Fay, Barbraud, Delord, & Weimerskirch, 2016; Lemaître & Gaillard, 2017). Such contributions of partner age are likely to be particularly

strong in monogamous species like meerkats where multiple mating is limited and where partners can be paired for long periods, potentially leading to correlated senescence in males and females. If, for example, males and females tend to pair up and then remain together into late life, then the poorer quality of both males and females in late life might amplify declines in reproductive performance. If, on the other hand, there is no underlying relationship between male and female age in later life, this would imply that the processes underlying reproductive senescence relate to intrinsic features of the individuals in question.

METHODS

Study Population

Data were collected from a wild population of meerkats in the Kalahari region of South Africa (26°58'S, 21°49'E) between December 1996 and September 2018. Approximately 50 social groups were followed during this time, comprising over 2500 individuals habituated to be observed at short distance (< 2m). Individuals were tagged with a subcutaneous transponder chip at emergence and given a unique fur dye mark to allow identification. Groups were visited 3-4 days a week, enabling intensive sampling of life history information (births, deaths, evictions, babysitters, changes in pregnancy or dominance status), behaviour and body mass. Body mass records were obtained early in the morning prior to foraging by enticing individuals onto an electronic balance with crumbs of boiled egg as a reward. As stated above, subordinates that never acquire a dominance position but remain in the study population until their death have usually died before they reach three years of age. Moreover, for a further sizeable proportion of subordinate individuals we do not know their fate (i.e. death or emigration), meaning that we cannot control for possible effects of selective disappearance, an important source of between-individual variation that could lead to biased estimates of within-individual trait changes across lifespan (Van de Pol & Verhulst 2006). The same bias is not present in

dominant individuals, whose fate is usually known. For these reasons, we limited our analyses to individuals that acquired a position of dominance during their lifetime, and of these individuals, we only considered those with a confirmable death. Confirmable deaths included individuals that were last seen in a state of terminal decline or were euthanised on site because they had developed clear outward signs of morbidity linked to advanced-stage tuberculosis.

Statistical Analyses

Statistical analyses consisted of three steps. In the first step, we examined age-related variation in body mass, reproductive output and mortality, with models parameterised so that we could directly compare sex differences in ageing trajectories. In the second step, we extended the best supported models from step one to incorporate effects of partner age and thereby examine whether senescence declines were conflated by partner effects. In the third step, we used information on age-related changes in reproduction and survival to calculate the fitness costs of senescence (Bouwhuis et al., 2012; Kowald & Kirkwood 2015). All analyses were undertaken in the R statistical environment v3.6.0 (R Core Team, 2019). Estimates present the mean ± 1 standard error of the mean unless otherwise stated.

Age-related variation in body mass and reproductive output

The body mass dataset partitioned individual lifespans into 4-month periods, with body mass then calculated as the mean daily morning mass within each period. This allowed us the highest possible resolution of sampling without losing full periods of weights due to pregnancy, which we excluded. Specifically, we excluded any pregnancy weights by back-casting 70 days from the day of birth or litter loss, thus removing any weight increases due to gestational growth (Fig. S1). On average, this resulted in 24.4 ± 0.6 mass records/female/period and 43.1 ± 0.7 mass records/male/period. In total, the body mass dataset comprised 83 females and 53 males.

For the reproductive output dataset, we instead partitioned individual lifespans into 6-month periods. Preliminary analyses suggested that doing so reduced the number of zeroes in the dataset and therefore improved the fit of models compared to shorter time intervals. Reproductive output was defined as the number of offspring that were produced by a male or female within each 6-month period that survived to nutritional independence at 3 months of age. 78.0% of pups that survive to nutritional independence go on to reach adulthood at one year of age (n = 2040 pups between January 1994 and July 1998). Parentage was assigned through genetic analysis of 18 microsatellites derived from tissue samples taken from the tip of individuals tails (Nielsen, 2012), and where genetic data were missing, maternity could be inferred from field observations where we were certain only a single female had given birth. The reproductive output dataset comprised 95 females and 67 males that produced an average of 1.67 ± 0.11 and 0.721 ± 0.10 pups/6-month period, respectively.

Age-related variation in many vertebrate traits often takes the form an initial early-life increase, a mid-life plateau, and a later-life decline. To capture this pattern for body mass and reproductive output in meerkats, we fitted a series of mixed effects models that included chronological age either as a quadratic function (a linear and quadratic age term) or as a threshold function (usually where linear slopes are estimated on either side of each fitted threshold age). Threshold functions are generally better equipped to reliably recover the full age-dependence of trait change but do so at the expense of additional parameters. We implemented our models in a Bayesian framework using the *brms* package (Bürkner, 2018). This offers a distinct advantage over a frequentist treatment, for while the former generates a posterior distribution for threshold parameters upon which other model terms are conditioned, frequentist analyses must fit multiple models and secondarily estimate the position of any thresholds (with associated confidence) through likelihood profiling (Ulm 1989). We modelled body mass using a Gaussian error distribution, and reproductive output using a zero-inflated

negative binomial distribution with a single zero-inflation parameter applied to all observations (zi ~ 1).

In order to test for sex differences in ageing patterns we adopted a similar approach to Tompkins and Anderson (2019) and fitted six models for each trait (Table S1). In model 1, age was included as a quadratic function, and males and females were assumed to follow the same age trajectory. In model 2 the linear and quadratic age terms of model 1 were each interacted with a covariate for sex to allow for male and female age trajectories to differ. We then specified four forms of threshold model. For body mass, this included two thresholds for each sex, one in early life (first threshold age = $T_{SEX,1}$) and one in mid to late life (second threshold = $T_{SEX,2}$). For reproductive output we only fit a single threshold in mid to late life as preliminary model fitting found no evidence for an additional threshold in early life. Threshold models then differed in the extent to which they forced males and females to have sex-specific slopes on age across lifespan, and/or sex-specific thresholds (Table S1). In the most advanced threshold model for body mass, model 6 (from which other models were derived), different threshold ages and different slopes across age were parameterised for males and females, such that the body mass of meerkat individual *i* at age *j* was parameterised as:

$$\mu_{ij} = \alpha + \beta_1 Age_{ij} + \beta_2 Sex_i + \beta_3 Age_{ij} Sex_i + \beta_4 (1 - Sex_i)(Age_{ij} - T_{F,1})_+ + \beta_5 Sex_i (Age_{ij} - T_{M,1})_+ + \beta_6 (1 - Sex_i)(Age_{ij} - T_{F,2})_+ + \beta_7 (1 - Sex_i)(Age_{ij} - T_{M,2})_+ + \beta_k X_k \ldots + u_i + u_{group} + \varepsilon_{ij}$$

where for thresholds $T_{SEX,t}$, $(Age_{ij} - T_{SEX,t})_+ = (Age_{ij} - T_{SEX,t}) * I(Age_{ij} \geq T_{SEX,t})$. $I(Age_{ij} \geq T_{SEX,t})$ is an indicator function equalling 1 when $Age_{ij} \geq T_{SEX,t}$, and 0 otherwise. Thus, β_4 and β_5 are the difference in the slope of each response variable on age *after* the first threshold age relative to the slopes *before* the first threshold age (for females and males, respectively). The

step function 'switches' β_4 and β_5 terms off for ages $\leq T_{SEX,1}$ and on for ages > than $T_{SEX,1}$ in each sex, where sex is a dummy variable with females coded as "0" and males as "1". Additional population-level "fixed" effects ($\beta_k X_k \ldots$) included the age at first dominance (AFD), AFD:sex, longevity, longevity:sex, total rainfall, group size, season, and dominance status. Total rainfall during each period was calculated from onsite rain gauge data; on days with missing information (10.3%), we imputed rainfall values from a remote-sensing dataset provided by the NASA GES DISC (Goddard Earth Sciences Data and Information Services Centre). Group size was taken as the average daily number of group members > 6 months old in each time period. Season was coded as a two-level factor (first = Oct-Mar, second = Apr-Sep). Dominance status indicated whether an individual was dominant within the period in question (recall that all individuals in the dataset do become dominant at some point in their lifetime). In the body mass models, we also included two predictors to examine possible sex differences in terminal decline (TD, TD:sex), where TD was a two-level factor noting whether it was the last period of an individual's life. Finally, u_i and u_{group} represent group-level ("random") effects of individual identity and group identity, and ε_{ij} is the residual error.

All Bayesian mixed effects models were fitted with four chains of 3000 iterations, of which 2000 were dedicated to the warm-up. We chose normal priors for all population-level and group-level effects. We also set upper and lower bounds on the prior for the threshold ages to more efficiently sample the posterior ($T_{SEX,1}$: lower bound = 1, upper bound = 4; $T_{SEX,2}$: lower bound = 4, upper bound = 8). Model diagnostics and posterior predictive checks highlighted adequate mixing of chains and appropriate choice of priors and error distributions. All continuous parameters were z-score transformed prior to model fitting, apart from age. Within each model, we used 95% Bayesian credible intervals (BCI) drawn from the posterior distribution as a measure of uncertainty, deeming as biologically important any effects where the credible intervals did not overlap zero. The predictive ability of candidate models was

compared using k-fold cross-validation ('k-Fold IC') using subset number k = 10. This method divides the data into ten subsets and validates the results of the nine subsets for each missing dataset. For each model, we also calculated the Bayesian equivalent of the R^2 using *bayes_R2* function in *brms* (Gelman, Goodrich, Gabry, & Ali, 2017).

Partner age effects

To assess the extent to which reproductive declines in one sex might contribute to or partly explain reproductive declines in the other sex, we modelled the relationship between the age of a dominant female and the age of her male partner across her period of tenure, and vice versa. Partner age was fitted as the response variable in general additive models (gam), with the age of the focal individual included as a sex-specific smoother function in each (6 knots). As this preliminary analysis hinted at a linear increase in partner age with the age of the focal dominant (Fig. S2; female model edf = 1, F_1=31.93, $p < 0.001$; male model edf = 1, F_1=2.54, $p = 0.114$), we re-fitted the best supported reproductive output model from the above analysis (but this time excluding information from subordinates), and included a linear covariate for partner age, and an interaction between partner age and sex. To allow for a non-linear effect of partner age on reproductive output, we also fitted one further model with a quadratic partner age effect, and an interaction between the quadratic term and sex. We examined the influence of including partner age terms on the estimates of reproductive decline in male and female meerkats.

Age-related changes in mortality

To test for sex differences in longevity and survival across age we performed both semi-parametric and parametric survival modelling with the *survival* and *flexsurv* packages (Jackson 2016; Therneau, 2015). Our sample consisted of 98 females and 71 males with confirmed final

fates, 63 females and 92 males who disappeared during the study with their fate being unknown, and 9 females and 7 males who were still alive at the end of the study. Individuals of unknown fate, who either disappeared during the study or were still alive at the end of study sampling period, can still be incorporated into the analysis through censoring. However, two key assumptions of censoring are that it is random with respect the individuals affected, and independent of the process of mortality such that individuals do not experience a change in mortality risk due to being censored. This is unlikely to be the case in meerkats, where censorship represents either unobserved mortalities or individual dispersal events, both of which will introduce positive bias and lead to overestimation of survival unless accounted for. To investigate the effect of censoring bias on our estimates of longevity and survival in males and females we performed a sensitivity analysis using the *InformativeCensoring* package (Ruau et al., 2016) by re-running semi-parametric cox proportional hazard models whilst either increasing or decreasing the hazard that censored individuals are exposed to after censorship via the gamma imputation method (Jackson et al., 2014).

As the length of our study (21 years) is considerably longer than the oldest individual of known fate within our population (12.4 years), the possible bias introduced by excluding individuals censored during our study is expected to be negligible (and far less than the bias introduced by their inclusion). Therefore, when performing parametric survival models to characterise the pattern of survival senescence our dataset only included individuals of known fate and individuals alive at the end of the study (who were censored). Null models were fitted with various error distributions (Gompertz, exponential, log-logistic, log-normal, gamma and Weibull) and model selection was guided by AIC and visual inspection of predicted survival and hazard plotted against the raw data. Males and females were first modelled independently to confirm their survival patterns could be best modelled with the same error distribution; then a model including both sexes was fitted, with a sex term fitted to all parameters of the error

distribution. For comparison these analyses were repeated with datasets where no truncation of censored individuals was undertaken and where all individuals censored prior to study end were considered as unobserved mortalities and thus modelled as known deaths.

Age-related reproductive value and the fitness costs of senescence

We calculated age-related variation in reproductive value (RV) using predicted changes in reproductive output and survival across lifespan. Reproductive value was calculated according to Stearns (1992):

$$RV_a = \sum_{x=a}^{x=w} \frac{l_x}{l_a} m_x$$

where a is the age for which reproductive value is being calculated, w is the age at last reproduction, l_x is survival at age x and m_x is reproductive output at age x. Further details are provided in the Supporting Information.

Finally, we used information on age-related changes in reproduction and survival to quantify the fitness costs of senescence (Bouwhuis et al., 2012; Kowald & Kirkwood 2015). Because annual survival probability in meerkats was constant beyond the age of peak reproductive output (at age 5.4 for both males and females), the fitness costs of senescence could be entirely attributed to reproductive senescence. We calculated the costs of reproductive senescence, C_{RS}, as the difference between the estimated reproductive value at age 1 (to be consistent with Bouwhuis et al., 2012), and the hypothetical reproductive value at this age if reproductive declines were absent. For the latter, reproductive output was held constant from the age of peak reproductive output and RV was estimated as above. C_{RS} is then $[(RV_{observed} - RV_{no\ RS})]/ RV_{no\ RS}$ x 100%.

RESULTS

373 *Age-related variation in body mass*

374 Both males and females displayed a significant decline in body mass in later life (Fig.
375 1). Model comparisons highlighted that age-related variation in body mass was best described
376 by threshold models that partitioned lifespan into three stages: an early-life increase, a mid-life
377 plateau, and later-life senescence (Table 1). In the best fitting model (model 3), males and
378 females were parameterised to share common slopes and common threshold ages, suggesting
379 that both the onset and rate of senescence were independent of sex (Fig. 1, Table 2).
380 Specifically, the onset of senescence in body mass was estimated at 5.56 years for both sexes
381 (95% BCI = 5.10 - 6.13), after which point males and females lost 19.35 grams per year (95%
382 BCI = -26.36 – -12.35). The absence of sex differences in body mass senescence was reinforced
383 by the most parameterised threshold model (model 6, Δk-Fold IC = 17.1), where early-life and
384 late-life thresholds, and the slopes on age, all displayed similar estimates in males and females
385 (Table S4). Aside from their age-related changes in body mass, both sexes displayed a terminal
386 decline in their final period of life equating to 32.07g in females (95% BCI = -40.95 – -23.08)
387 and 30.85g decline in males (95% BCI = -41.53 – -17.75). At the between-individual level, in
388 neither sex was there strong statistical support for the selective disappearance of lighter
389 individuals (female estimate = 7.47, 95% BCI = -3.19 – 18.01; male estimate = 3.84, 95% BCI
390 = -14.51 – 14.76), nor was there a clear influence of the age of dominance acquisition (Table
391 S2).

392

393 *Age-related variation in reproductive output*

394 Males and females both experienced age-related declines in reproductive output (Table 3,
395 Fig. 2a, 2b). In the best supported model (model 5), males and females shared a common
396 threshold age term, but were given separate slopes on age which took the form of a linear
397 increase from mid-life to late life, followed a subsequent period of reproductive decline.

Further examination of the model output highlighted that it was the initial mid-life increase in reproductive output that drove this trend, with estimates for the later-life slope showing no apparent difference between males and females (Table 2). As for body mass then, both the onset and rate of senescence in reproductive output were independent of sex (Fig. 2). Based upon the best supported model, females experienced a 72.1% reduction in reproductive output between the ages of 5 and 9, and males a 74.3% decline in reproductive output over the same age period. The best supported model also highlighted the selective disappearance of females with lower reproductive output, with a 1 standard deviation increase in the longevity term (2.45 years) being associated with 1.55 more pups per 6 month period (mean estimate on log-scale = 0.44, 95% BCI = 0.11 – 0.77, Table S3). A comparable trend was not present in males (mean estimate on log-scale = 0.29, 95% BCI = -0.22 – 0.84)

Although analyses of the raw data provided some suggestion that older dominant individuals were more likely to be paired with an older partner (Fig. S2), the effect was weak and the inclusion of partner age in a re-fitted reproductive output model (model 5) did not affect the estimated onset or rate of senescence when compared to a model where partner age terms were absent (Fig. S3). Nor were the partner age terms themselves significant in the updated models (additional model one: partner age estimate = 0.03, 95% BCI = -0.16 – 0.26, partner age:sex estimate = -0.20, 95% BCI = -0.63 – 0.25; additional model two: partner age^2 estimate = -0.02, 95% BCI = -0.17 – 0.14, partner age^2:sex estimate = -0.28, 95% BCI = -0.61 – 0.05).

Age-related changes in mortality

While a cox proportional hazard model assuming independent censoring suggests that males have marginally longer life spans than females (estimate = -0.342, SE = 0.158, p = 0.030), this result is not robust to expected censoring bias. Our sensitivity analysis revealed that even under the conservative assumption that censored individuals are exposed to only a small increase in mortality risk compared to non-censored individuals, the sex difference in

lifespan no longer held (Fig. 3). Moreover, when we excise individuals that were censored before the end of the study, or if we treat them as having immediately died, no sex difference was apparent (Fig. 3). In the scenario where censoring is associated with reduced risk the effect of sex remains stable.

Parametric modelling of our survival data revealed that the pattern of survival in meerkats was best described by a log-normal distribution (Fig. S4). The log-normal distribution model was in the top cohort of candidate models for both sexes and represented the model of best fit with both sexes modelled together (Table S5, Fig. S4). The log-normal distribution allows mortality risk to initially increase with the risk reaching an asymptote later in life (Fig. 4C). This suggests the absence of survival senescence in meerkats with the log-normal model providing a better fit than models with distributions that can capture survival senescence should it be present, such as the Gompertz and Weibull distributions (Table S5). However, as sample sizes decrease later in life the power with which to detect senescence declines. Annual mortality derived from our log-normal survival model tracks mortality probabilities well with reasonable sample sizes up to around 8 years (Fig. 4A, B), after which we are unlikely to be able to detect senescent trends. While the parametric models revealed no difference in the log mean parameter between the sexes (estimate = 0.015, 95% CI = -0.137 – 0.167), indicating no difference in mean longevity, there was a marginally significant difference between the sexes in the log standard deviation parameter (estimate = -0.264, CI = -0.481 – -0.047), reflecting the reduced variance in male lifespan.

Age-related reproductive value and the fitness costs of senescence

Prior to the acquisition of dominance reproductive value increased gradually, peaking at around 2.5 years in females and 4.5 years for males, before declining thereafter (Fig. 4c). With no evidence of survival senescence in dominant individuals, these declines in reproductive

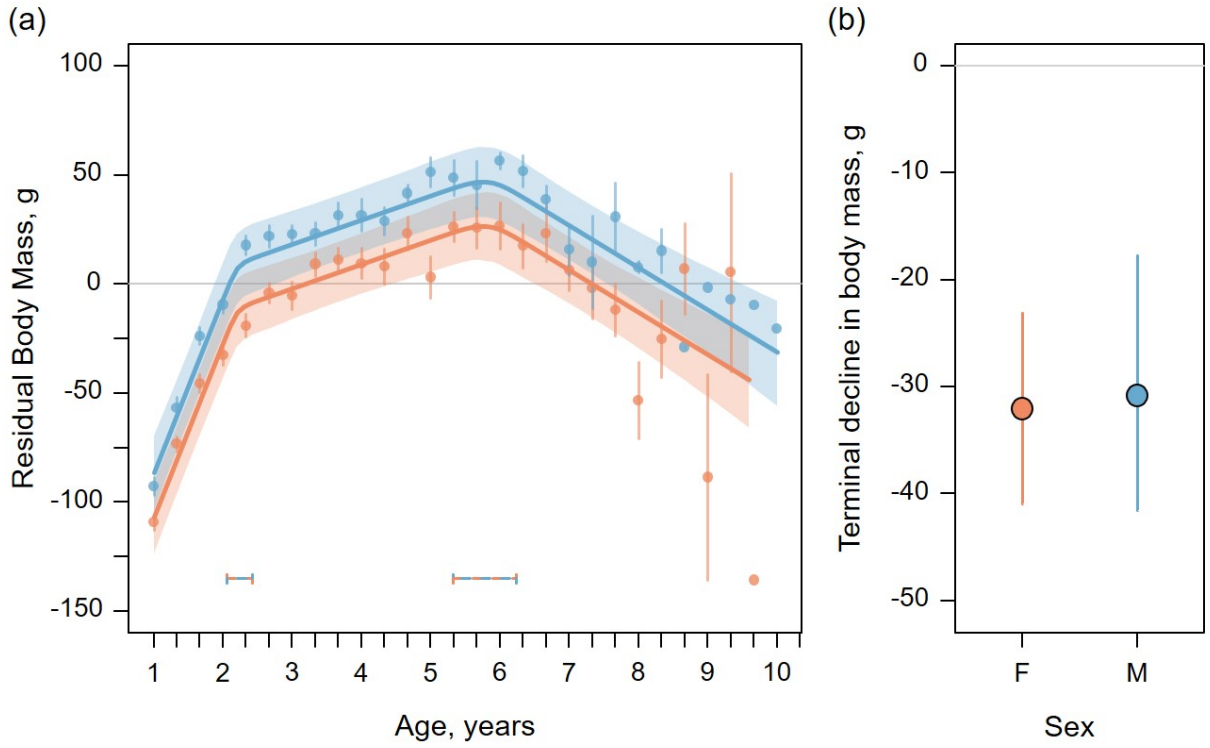
449 value – the fitness costs of senescence – can be entirely attributed to reproductive senescence.
450 Excluding reproductive senescence from the life history increases the reproductive value of
451 females at age 1 from 12.68 to 14.49, and males at age 1 from 7.22 to 8.25. This entails a fitness
452 cost of reproductive senescence, C_{RS}, of 12.5% in females, and 12.6% in males.

TABLE 1. Comparison of models investigating sex-specificity of senescence in meerkats. Models are ranked according to k-fold IC, with the lowest k-fold IC taken as the best-supported model (**bold**). Threshold models differed in the extent to which they allowed males and females to have common or distinct sex-specific estimates for threshold ages (T) and/or slopes on age (as detailed in the main text). 'params' refers to the number of parameters estimated by each model. All models included additional population-level ('fixed') and group-level ('random') terms as described in the main text.

		Body Mass				Reproductive Output			
Model	**Sex- and age-related predictors**	**params**	**k-fold IC (SE)**	**Δ k-fold IC (SE)**	**Bayesian R^2 (95% BCI)**	**params**	**k-fold IC (SE)**	**Δ k-fold IC (SE)**	**Bayesian R^2 (95% BCI)**
	Quadratic models								
1	Age + Age^2 + Sex	16	14857.4 (71.1)	93.8 (25.8)	0.766 (0.753, 0.778)	16	2351.2 (92.6)	31.2 (11.6)	0.377 (0.293, 0.460)
2	Age + Age^2 + Sex + Sex:Age + Sex:Age^2	18	14847.7 (71.3)	84.1 (25.0)	0.768 (0.755, 0.779)	18	2331.6 (92.6)	11.6 (12.0)	0.372 (0.283, 0.454)
	Threshold models								
3	Common T, common slopes on age	**19**	**14763.6 (74.5)**	**0.0**	**0.783 (0.771, 0.794)**	17	2338.3 (91.2)	18.3 (10.4)	0.378 (0.292, 0.462)
4	Sex-specific T, common slopes on age	21	14785.7 (75.0)	22.1 (13.6)	0.784 (0.772, 0.794)	18	2334.6 (90.5)	14.6 (9.0)	0.380 (0.292, 0.462)
5	Common T, sex-specific slopes on age	22	14766.6 (75.2)	3.0 (15.2)	0.784 (0.771, 0.795)	**19**	**2320.0 (90.9)**	**0.0**	**0.370 (0.284, 0.456)**
6	Sex-specific T, sex-specific slopes on age	24	14780.7 (76.0)	17.1 (14.4)	0.784 (0.772, 0.794)	20	2339.4 (92.0)	19.4 (8.2)	0.374 (0.288, 0.457)

TABLE 2. Threshold age estimates and estimated slopes of age for body mass and reproductive output from the best-supported model in each case (model 3 and 5, respectively). 95% BCI shown in parentheses. Estimates for the slopes of reproductive output are on the link-scale (log link). Supporting information provides equivalent terms from the most heavily parameterised model for comparison.

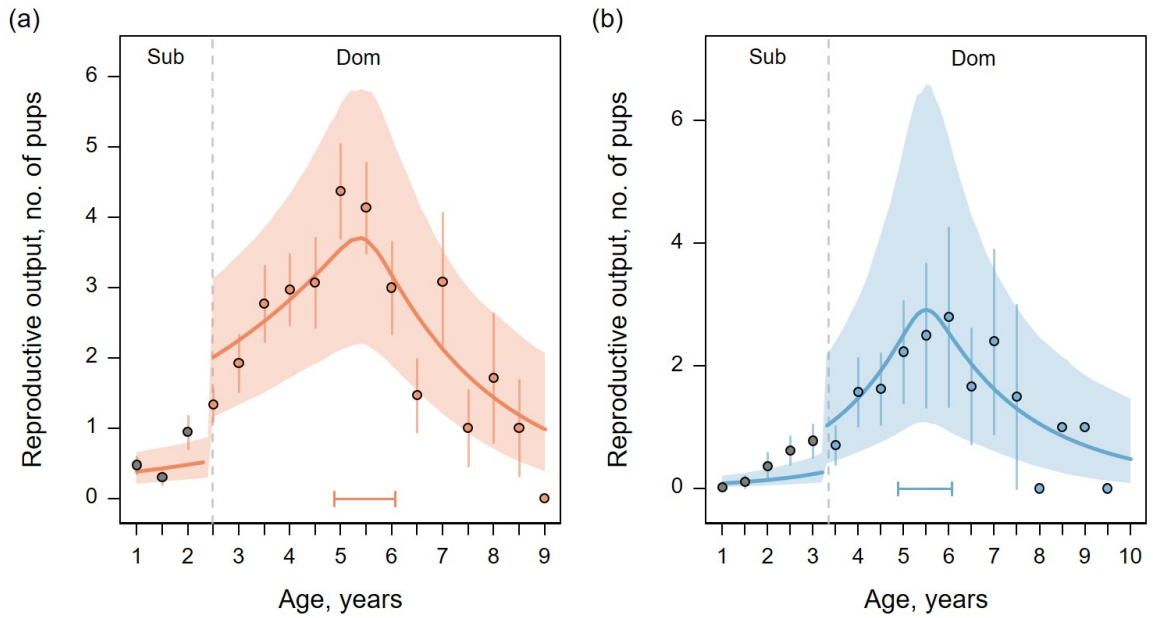
Trait	Model	Early-life slope	Threshold 1 (early-life to mid-life)	Mid-life slope	Threshold 2 (mid-life to late-life)	Late-life slope
Female and male body mass	3	80.21 (69.81, 91.26)	2.20 (2.05, 2.42)	11.11 (0.96, 21.09)	5.81 (5.32, 6.24)	-19.35 (-26.25, -12.53)
Female reproductive output	5	NA	NA	0.23 (0.10, 0.36)	5.44 (4.87, 6.06)	-0.41 (-0.68, -0.18)
Male reproductive output	5	NA	NA	0.53 (0.31, 0.75)	5.44 (4.87, 6.06)	-0.45 (-0.80, -0.15)



485

486 **FIGURE 1:** (a) Age-related variation in body mass in female (red) and male (blue) meerkats
487 after accounting for the effects of selective disappearance, terminal decline, social status, group
488 size, rainfall and season. Solid circles represent the mean residual body mass per age with their
489 associated standard error bar (with sex differences in the intercept removed from the residuals
490 to allow visualisation of sex differences in average body mass). Solid lines display predicted
491 age-related changes in body mass according to the best-supported model. The upper and lower
492 limits of the coloured shaded areas show the 95% BCI estimates of the chronological age effect
493 based upon fixed effects uncertainty. The 95% BCI of the threshold estimates are shown by the
494 horizontal error bars, with males and females sharing a single estimate for both thresholds. The
495 population-level mean body mass is 710.5g. (b) Terminal declines in body mass of female (red)
496 and male (blue) meerkats in the last three months of life. Solid points display the predicted
497 body mass decline, with the 95% BCI represented by the vertical lines.

498



499

500 **FIGURE 2:** Age-related variation in reproductive output in female (red, (a)) and male (blue,
501 (b)) meerkats. Solids lines display the predicted number of pups produced to emergence during
502 each 6-month period for individuals who acquire dominance. The upper and lower limits of the
503 coloured shaded areas show the 95% BCI estimates of the chronological age effect based upon
504 fixed effects uncertainty. The 95% BCI of the threshold estimates are shown by the horizontal
505 error bars. Predictions were made either side of the population mean age of dominance
506 acquisition period (vertical striped line), with predictions representing subordinate
507 reproductive output before this age and dominant reproductive output after. For predictions,
508 rainfall and group size were set at the mean, longevity was set at 7 years, and season was set
509 as "second" (Apr-Sep). Points display the raw data with vertical error bars indicating ± 1 SEM.
510 Points are coloured to emphasize that, on average, most of the raw data prior to the vertical line
511 comes from individuals while they are subordinate (dark grey), whereas after this point, most
512 data comes from individuals that are dominant (red/blue).

513

514

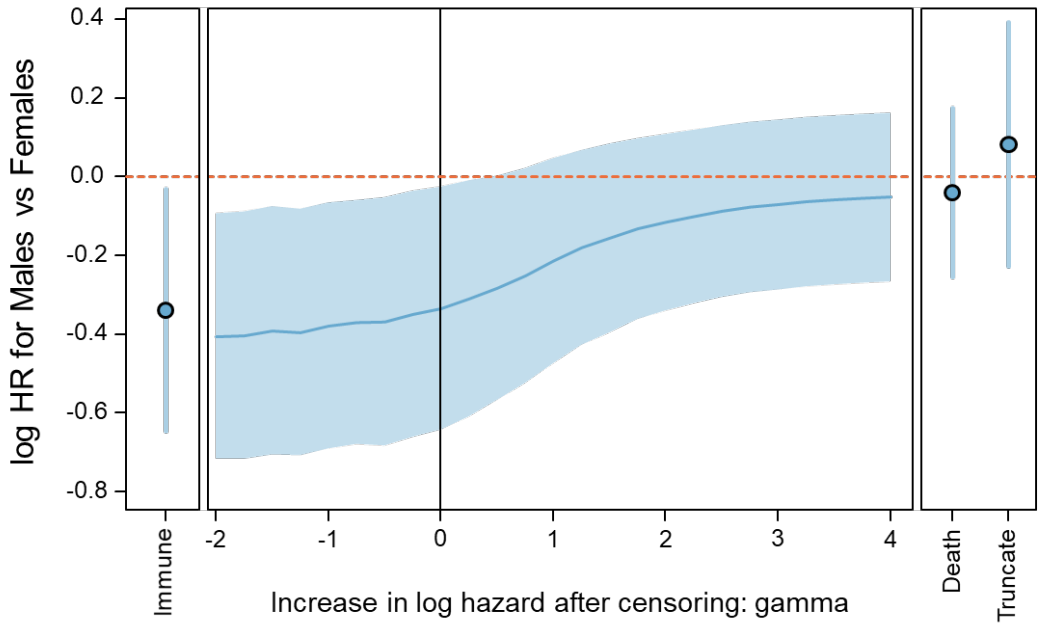
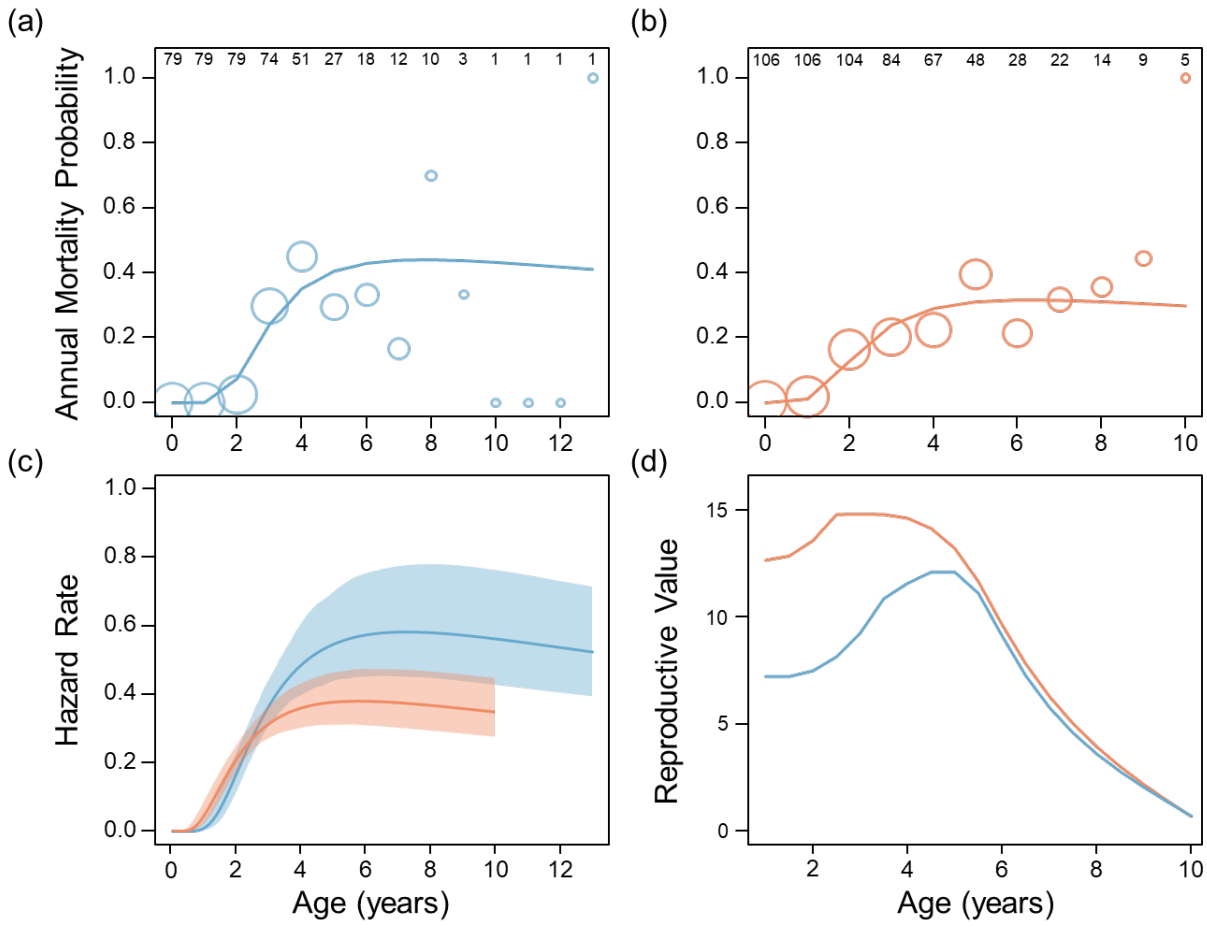


FIGURE 3: The change in the estimated effect size of the sex term (solid blue line give the mean, blue shading the 95% confidence intervals) in relation to the hazard adjustment applied to censored individuals, as estimated by cox proportional hazard survival models. An increased log hazard of zero (vertical solid line) represents the standard model where censorship is assumed to be independent of mortality and individuals that are censored are expected to experience no change in mortality risk. Where confidence intervals cross zero (orange dashed line) the effect of sex is not significant. Point estimates and accompanying confidence intervals are plotted for the extreme scenarios where individuals that are censored become immune to mortality (Immune) and where censorship leads to instantaneous mortality (Death). Additionally, the point estimate and confidence intervals are plotted for a data set where individuals that disappear during the study are truncated and individuals still alive at the end of study are censored with no adjustment to their log hazard (Truncate); this is the data set used for down-stream parametric survival modelling.



532

533 **FIGURE 4:** (a, b) Annual mortality curves, (c) hazard rates and (d) reproductive value across
534 life span for male (blue) and female (red) meerkats that acquired dominance at some point in
535 their lives. (a, b) Solid lines represent annual mortality probabilities calculated for males (a)
536 and females (b) using survival probabilities predicted from the parametric survival model of
537 best fit (Fig. S4), with points representing raw values for annual survival probability with the
538 size of point representing the total number of individuals observed across the year log scaled
539 with exact values reported at the top of the figures. (c) Solid lines represent the estimated hazard
540 rate from our model of best fit with the 95% confidence intervals plotted as shaded areas.
541 Estimates are derived from a parametric survival model with a log-normal distribution and sex
542 fitted as a covariate to the ancillary log standard deviation parameter but not the log mean
543 parameter. (d) Reproductive values are calculated using predicted estimates of survival (Fig.
544 S4) and reproductive output (Fig. 2).

DISCUSSION

Our study finds that in meerkats, the form and rate of senescence across three components of life history are similar in males and females. We show that the onset and rate of senescence in body mass and reproductive output were largely independent of sex, with trait values peaking between 4 and 6 years of age and declining at similar rates thereafter. Age-related survival probability was also unaffected by sex, but unlike the former two traits, we found no evidence of survival senescence in dominant individuals as annual survival probability remained constant beyond the age of peak reproductive output. Consequently, the fitness costs of senescence in meerkats could be entirely attributed to reproductive deterioration in later life, contributing to an estimated reduction in reproductive value of approximately 12.5% when compared to the hypothetical reproductive value at 1 year of age if no senescence were to occur. These results agree with a wider body of literature which has highlighted that senescence in wild populations is often asynchronous across traits (Evans et al, 2011; Hayward et al., 2015; Nussey et al., 2009), though the extent to which this is the case varies widely across taxa and even among species with apparently similar ecologies (Bouwhuis et al., 2012).

Alongside research on grey mouse lemurs *Microcebus murinus* (Hämäläinen et al., 2014), alpine marmots (Tafani et al., 2013), and red *Canis rufus* and gray wolves *Canis lupus* (MacNulty, Smith, Mech, & Eberly, 2009; Sparkman et al., 2017), our study is one of only a handful to provide quantitative information on sex-specific patterns of senescence in a non-polygynous mammal in the wild, and is the first to combine information from multiple traits concurrently. Such a treatment is timely, as the predominance of long-term studies focussed on polygynous mammals has led to the conclusion that divergent ageing rates between the sexes are typically driven by sex differences in the intensity of intrasexual competition (Beirne, Delahay, & Young, 2015; Lemaitre, Gaillard, Pemberton, Clutton-Brock, & Nussey, 2014, Nussey et al., 2009). However, while this assertion might hold generally, we should not expect

570 this to be the case in all species (Lemaître and Gaillard, 2012; Tidière et al., 2014) as there are
571 likely to be other aspects of demography and life history that are also important in affecting
572 how males and females allocate resources to somatic maintenance, survival and reproduction
573 across the lifespan.

574 In meerkats, we suggest that the form of reproductive competition is as important as its
575 intensity in affecting patterns of senescence. If intensity were more important, we would have
576 expected dominant females to display an earlier onset and faster rate of senescence than
577 dominant males, as dominant females are hormonally masculinised (Davies et al., 2016), show
578 elevated parasite burdens (Smyth & Drea, 2016), and display regular bouts of aggression with
579 subordinates to suppress their reproduction and prompt their eviction (Clutton-Brock et al.,
580 2006). On the other hand, females can better manage the risk or usurpation and can maintain
581 long tenures of dominance, whereas dominant males are exposed to the periodic threat from
582 intruding males seeking to challenge their paternity share and their dominance. As a result,
583 although the frequency with which males face reproductive competition is much lower than
584 that faced by females, the implications for their tenure maintenance, and thus their continued
585 survival are more severe, as reflected in their shortened tenures. Taken together, we suggest
586 that the potential for intrasexual competition to increase rates of senescence in females is offset
587 by their ability to maintain longer tenures of dominance than males, and that these processes
588 combined lead to similar patterns of senescence in both sexes. Or, put differently, the realised
589 costs of competition on fitness are not divergent enough to have led to the evolution of sex
590 differences in senescence trajectories in meerkats.

591 We found that body mass and reproductive output senesced in parallel. For female
592 meerkats, the fitness consequences of reduced body mass have already been well described
593 (Ozgul, Bateman, English, Coulson, & Clutton-Brock, 2014), making it likely that the
594 downturns in body mass are causally related to decreases in reproductive output through

595 reductions in litter size and in the frequency of breeding (Sharp & Clutton-Brock, 2010). For
596 males, the consequences of reduced body mass on fitness are less clear. We did not find any
597 support for the selective disappearance of lighter males (that had acquired dominance), but it
598 is possible that males in poorer condition in later life are less able to monopolise the paternity
599 of dominant females and maintain their position of dominance. In making this suggestion, it
600 must also be remembered that the reproductive output of any male is in part influenced by the
601 quality of his female partner, and vice versa (Fay et al, 2016; Lemaître & Gaillard, 2017), such
602 that the fitness declines in one sex could contribute heavily to fitness declines in the other sex.
603 However, we do not find convincing support for partner age effects in our study, and by
604 implication it is likely that intrinsic physiological declines in males and females are mostly
605 responsible for the observed reproductive declines in either sex.
606 Our study also detected a strong age-independent contribution to body mass variation in
607 the form of terminal declines. Terminal declines in condition are often thought to reflect age-
608 independent deterioration due to disease (Coulson & Fairweather, 2001). In meerkats,
609 tuberculosis represents a possible mechanism by which terminal declines might be mediated,
610 and anecdotally, individuals reaching an advanced stage of morbidity through tuberculosis
611 exposure display dramatic reductions in body mass, dying shortly afterwards. The telomeres of
612 meerkats also shorten rapidly in the period immediately prior to death (Cram et al. 2018). A
613 broader examination of physiological changes across the lifespan would no doubt identify
614 further markers of bodily decline associated with age-dependent and age-independent mass
615 variation (e.g. muscle wasting, Sierra et al., 2013), but it would be particularly useful to know
616 whether reductions in body mass compromise foraging efficiency. A large proportion of the
617 daily activity budget of meerkats is spent digging for subterranean invertebrates so any
618 downturn in foraging ability is likely to be particularly damaging for individual condition.

Despite undergoing body mass senescence, neither male nor female meerkats that acquired showed evidence for increasing rates of mortality in later life. The absence of survival senescence contrasts with the general pattern seen in mammals and birds (Gaillard et al., 2017; Jones et al., 2014), though our result should be taken with the caveat that survival analyses were restricted to individuals that acquired dominance. That said, several recent studies of unusually long-lived species such as bats and seabirds have suggested that survival senescence in wild vertebrate populations is sometimes negligible (Coulson & Fairweather, 2001; Fleischer, Gampe, Scheuerlein, & Kerth, 2017). These species are also characterised by low fecundities imposed by energetic constraints (e.g. through flight: Jones & MacLarnon, 2001; or chick development: Lack, 1968), and with these constraints on reproductive output, lifespan extension might provide the main evolutionary route to maximising fitness. Any survival senescence is therefore likely to carry heavy fitness costs in long-lived species, and this could provide strong selection against senescence. By comparison, meerkats are not particularly long-lived for their size, and their fecundity is high, so similar arguments are unlikely to explain the absence of survival senescence in dominant meerkats (or naked mole-rats *Heterocephalus glaber*: Ruby, Smith, & Buffenstein, 2018). Why then does mortality rate not increase in old age in meerkats? One possible explanation relates to group living. Incumbent dominants can maintain long tenures sheltered from extrinsic mortality in large groups with dedicated sentinels (Cram et al. 2018), and this buffering effect of group living might be enough to prevent age-dependent increases in mortality risk in dominants irrespective of individual declines in condition. Alternatively, the presence and intensity of survival senescence might vary over time as environmental and demographic conditions modify the likelihood of different forms of intrinsic and extrinsic mortality (Berger et al., 2018; Hämäläinen et al., 2014; Ronget, Garratt, Lemaître, & Gaillard, 2017), and might go undetected when these sources of variation are not accounted for in survival analyses. A failure to detect survival senescence could also

644 reflect sampling effort when longitudinal studies have not been run for long enough to capture
645 its onset (Péron, Gimenez, Charmantier, Gaillard, & Crochet, 2010). With only modest
646 amounts of data from very long-lived individuals, it is not currently feasible to investigate
647 whether this is the case in meerkats with a high degree of confidence- but our data does provide
648 reasonable evidence that general increases in mortality rate in the reproductive cohort of
649 meerkats are absent several years after senescence has already begun in reproduction and body
650 mass (see also Sharp & Clutton-Brock, 2010, 2011a).

651 The realisation that males and females differed little in their annual survival probably was
652 only arrived at through an appreciation of censoring. The independence of censoring is a critical
653 assumption of most standard survival models but is often violated in studies of wild animals
654 (Murray, 2006). Meerkats provide a case in point where censoring is not independent and is
655 instead associated with missed or increased mortality, and models not taking this into account
656 are subject to positive biases that tend to overestimate longevity. The level of bias produced by
657 non-random censoring is related to the proportion of individuals censored (Campigotto &
658 Weller, 2014), and as proportionally more males than females were censored in our dataset, we
659 pick up a spuriously significant sex difference in longevity when censoring bias was not
660 accounted for. While it is generally impossible to assess the extent to which the baseline
661 hazards of censored individuals change, by using sensitivity analyses one can examine how
662 variation in baseline hazard influences specific covariates of interest (Jackson et al., 2014) and
663 use this to guide interpretation of any results. In our case, even a minor increase in hazard for
664 censored individuals (a reasonably conservative assumption) lead to a loss of significance and
665 a reduction in the effect size of the sex term on longevity, a result that was also found when
666 individuals that disappeared prior to study end were truncated rather than censored. Thus, with
667 censoring-induced bias considered, we do not find strong support for differences in longevity
668 between the sexes.

669 Irrespective of the causes of reproductive declines in dominant meerkats, direct fitness
670 represents only one avenue of reproductive success in those individuals experiencing
671 senescence: dominants can also accrue indirect fitness benefits through the reproduction of
672 subordinates in their group. In stable groups, the costs of subordinate reproduction to dominants
673 are high (Bell et al., 2014), and dominant females consequently employ behavioural tactics to
674 limit the breeding opportunities of subordinates. Even so, these costs are likely to be lower
675 when their own reproductive output is reduced, as in old age. Older, lighter dominants are
676 presumably also less able to control subordinate reproduction should they attempt to do so.
677 Either way, the reproductive output of subordinate group members may increase as dominant
678 females age and therefore cause indirect fitness to form a greater contribution to the
679 reproductive success of dominants in later life. If indirect fitness were shown to increase in
680 old-aged individuals, there are two important consequences. Firstly, it would imply that the
681 fitness costs of reproductive senescence are overestimated when only direct fitness is
682 considered. Secondly, it might explain why dominant individuals appear to favour survival
683 over reproduction in later life, as their own reproductive declines could be partly offset by the
684 greater reproductive success of their close relatives.

685 In concert with other studies, our results emphasize that broad categorisation into
686 mating systems will likely only get us so far in understanding sex differences in ageing in wild
687 vertebrates, for within mating systems and within species, sex differences in the degree to
688 which males and females compete for reproductive opportunities, and the manner in which
689 they do so, vary widely. In order to better understand why ageing rates differ so widely within
690 and between species in the wild, and in particular, between the sexes, it will therefore be
691 necessary to generate more targeted questions that place specific aspects of species' life
692 histories at the forefront of tests of evolutionary theories of senescence.
693

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AUTHOR'S CONTRIBUTIONS

JT, CD, and TC-B conceived of the ideas for the work with earlier input from SS. JT and CD collated and analysed the data. A first draft was put together by JT and TC-B, with all authors contributing to subsequent drafts. All authors gave their approval for publication. TC-B and MBM initiated and organised the long-term data collection.

DATA ACCESSIBILITY

The data for our manuscript will be deposited in the Dryad digital repository.

REFERENCES

Allman, J., Rosin, A., Kumar, R., & Hasenstaub, A. (2002). Parenting and survival in anthropoid primates: Caretakers live longer. *Proceedings of the National Academy of Sciences*, *95*(12), 6866–6869. doi: 10.1073/pnas.95.12.6866

Arnold, W. (1988) Social thermoregulation during hibernation in alpine marmots (*Marmota marmota*). *Journal of Comparative Physiology B*, *158*, 151-156.

Arnold, W. (1990). The evolution of marmot sociality: II. Costs and benefits of joint hibernation. *Behavioral Ecology and Sociobiology*, *27*(4), 239–246. doi: 10.1007/BF00164895

Austad, S.N. (2011). Sex differences in longevity and aging. In E. Masoro & S.N. Austad (Eds.), *Handbook of the Biology of Ageing* (7th ed., pp. 479–495). San Diego: Academic Press.

Austad, S.N., & Hoffman, J.M. (2018). Is antagonistic pleiotropy ubiquitous in aging biology? *Evolution, Medicine, and Public Health 1*, 287–294. doi: 10.1093/emph/eoy033

Barford, A., & Dorling, D. (2006). Life expectancy: women now on top everywhere. *British Medical Journal*, *332*, 808.

Beekman, M., Dowling, D.K., and Aanen, D.K. (2014) The costs of being male: are there specific effects of uniparenal mitochondrial inheritance? *Philosophical Transactions of the Royal Society B*., 369, 20130440.

Beirne, C., Delahay, R., & Young, A. (2015). Sex differences in senescnece: the role of intra-sexual competition in early adulthood. *Proc. R. Soc. B.*, 282, 20151086. doi: 10.1098/rspb.2015.1086.

Beirne, C., Waring, L., Mcdonald, R.A., Delahay, R., & Young, A. (2016). Age-related declines in immune response in a wild mammal are unrelated to immune cell telomere length. *Proceedings of the Royal Society B*, *283*, 20152949. doi: 10.1098/rspb.2015.2949

Bell, M.B.V, Cant, M.A., Borgeaud, C., Thavarajah, N., Samson, J., & Clutton-Brock, T.H. (2014). Suppressing subordinate reproduction provides benefits to dominants in cooperative societies of meerkats. *Nature Communications*, *5*, 4499. doi: 10.1038/ncomms5499

Berger, V., Lemaître, J.-F., Dupont, P., Allainé, D., Gaillard, J.-M., & Cohas, A. (2016). Age-specific survival in the socially monogamous alpine marmot (Marmota marmota): evidence of senescence. *Journal of Mammalogy*, *97*(3), 992-1000. doi: 10.1093/jmammal/gyw028

Bonduriansky, R., Maklakov, A., Zajitschek, F., & Brooks, R. (2008). Sexual selection, sexual conflict and the evolution of ageing and life span. *Functional Ecology*, *22*(3), 443–453. doi: 10.1111/j.1365-2435.2008.01417.x

Bouwhuis, S., Choquet, R., Sheldon, B. C., & Verhulst, S. (2012). The Forms and Fitness Cost of Senescence: Age-Specific Recapture, Survival, Reproduction, and Reproductive Value

763 in a Wild Bird Population. *The American Naturalist*, *179*(1), E15–E27. doi:
764 10.1086/663194

765 Brooks, R.C., & Garratt, M.G. (2016) Life history evolution, reproduction, and the origins of
766 sex-dependent aging and longevity. *Ann. N. Y. Acad. Sci.*, *1389*, 92-107. doi:
767 10.1111/nyas.13302

768 Brownikowski, A.M., Altmann, J., Brockman, D.K., Cords, M., Fedigan, L.M., Pusey, A.,
769 Stoinski, T., Morris, W.F., Strier, K.B., Alberts, S.C. (2011). Aging in the natural world:
770 comparative data reveal similar mortality patterns across primates. *Science*, *331*, 1325-
771 1328.

772 Bürkner, P.-C. (2018). brms: Advanced Bayesian multilevel modeling with the R package
773 brms. *The R Journal*, *10*(1), 395–411.

774 Carroll, J., & Sherratt, T.N. (2017) Actuarial senescence in laboratory and field populations of
775 Lepidoptera. *Ecological Entomology* 42, 675-679. doi: 10.1111/een.12417

776 Campigotto, F., & Weller, E. (2014) Impact of Informative Censoring on the Kaplan-Meier
777 Estimate of Progression-Free Survival in Phase II Clinical Trails. *Journal of Clinical*
778 *Oncology, 32*(27), 3068-3074. doi: 10.1200/JCO.2014.55.6340

779 Clutton-Brock, T. (1983). Selection in relation to sex. In D. Bednall (Ed.), *Evolution from*
780 *molecules to men* (pp. 457–481). Cambridge, UK: Cambridge University Press.

781 Clutton-Brock, T.H., Hodge, S.J., Spong, G., Russell, A.F., Jordan, N.R., Bennett, N.C., …
782 Manser, M.B. (2006). Intrasexual competition and sexual selection in cooperative
783 mammals. *Nature*, *444*(7122), 1065–1068. doi: 10.1038/nature05386

784 Clutton-Brock, T.H., & Isvaran, K. (2007). Sex differences in ageing in natural populations of
785 vertebrates. *Proceedings of the Royal Society B: Biological Sciences*, *274*(1629), 3097–
786 3104. doi: 10.1098/rspb.2007.1138

787 Clutton-Brock, T.H., & Manser, M. (2016). Meerkats: Cooperative breeding in the Kalahari.
788 In W.D. Koenig & J.L. Dickinson (Eds.), *Cooperative Breeding in Vertebrates: Studies*
789 *of Ecology, Evolution, and Behavior* (pp. 294–317). doi: DOI:
790 10.1017/CBO9781107338357.018

791 Cohas, A., Yoccoz, N.G., Da Silva, A., Goossens, B., & Allainé, D. (2006). Extra-pair paternity
792 in the monogamous alpine marmot (*Marmota marmota*): The roles of social setting and
793 female mate choice. *Behavioral Ecology and Sociobiology*, *59*(5), 597–605. doi:
794 10.1007/s00265-005-0086-8

795 Coulson, J.C., & Fairweather, J.A. (2001). Reduced reproductive performance prior to death
796 in the Black-legged Kittiwake: senescence or terminal illness? *Journal of Avian Biology*,
797 *32*, 146–152.

798 Cram, D., Monaghan, P., Gillespie, R., Dantzer, B., Duncan, C., Spence-Jones, H., & Clutton-
799 Brock, T. (2018). Rank-related contrasts in longevity arise from extra-group excursions
800 not delayed senescence in a cooperative mammal. *Current Biology* 28, 2934-2939. doi:
801 10.1016/j.cub.2018.07.021

802 Davies, C.S., Smyth, K.N., Greene, L.K., Walsh, D.A., Mitchell, J., Clutton-Brock,T.H., &
803 Drea, C.M. (2016). Exceptional endocrine profiles characterise the meerkat: sex , status ,
804 and reproductive patterns. *Nature Scientific Reports*, 6, 35492. doi: 10.1038/srep35492

Duncan, C., Gaynor, D., & Clutton-Brock, T. (2018). The importance of being beta: female succession in a cooperative breeder. *Animal Behaviour*, *146*, 113–122. doi: 10.1016/j.anbehav.2018.10.013

Evans, S.R., Gustafsson, L., & Sheldon, B.C. (2011). Divergent patterns of age-dependence in ornamental and reproductive traits in the collared flycatcher. *Evolution 65*(6), 1623–1636. doi: 10.1111/j.1558-5646.2011.01253.x

Fay, R., Barbraud, C., Delord, K., & Weimerskirch, H. (2016). Paternal but not maternal age influences early-life performance of offspring in a long-lived seabird. *Proceedings of the Royal Society B: Biological Sciences*, *283*(1828). doi: 10.1098/rspb.2015.2318

Fleischer, T., Gampe, J., Scheuerlein, A., & Kerth, G. (2017). Rare catastrophic events drive population dynamics in a bat species with negligible senescence. *Scientific Reports*, *7*(1), 1–9. doi: 10.1038/s41598-017-06392-9

Gaillard, J.-M., Garratt, M., & Lemaître, J-F. (2017). Senescence in Mammalian Life History Traits. In R. P. Shefferson, O.R. Jones, & R. Salguero-Gómez (Eds.), *The Evolution of Senescence in the Tree of Life* (pp. 126–155). Cambridge, UK: Cambridge University Press.

Gaillard, J.-M., & Lemaître, J-F. (2017) The Williams' legacy: a critical reappraisal of his nine prediction about the evolution of senescence. *Evolution*, *71*(12), 2768-2785. doi:10.1111/evo.13379

Gelman, A., Goodrich, B., Gabry, J., & Ali, I. (2017). R-squared for Bayesian regression *models.* Available at: http://www.stat.columbia.edu/~gelman/research/unpublished/. Last accessed August 2019.

Graves, B.M. (2007). Sexual selection effects on the evolution of senescence. *Evolutionary Ecology*, *21*(5), 663–668. doi: 10.1007/s10682-006-9144-6

Hämäläinen, A., Dammhahn, M., Aujard, F., Eberle, M., Hardy, I., Kappeler, P.M., … Kraus, C. (2014). Senescence or selective disappearance? Age trajectories of body mass in wild and captive populations of a small-bodied primate. *Proceedings of the Royal Society B: Biological Sciences*, *281*, 20140830.

Hayward, A.D., Moorad, J., Regan, C.E., Berenos, C., Pilkington, J. G., Pemberton, J.M., & Nussey, D.H. (2015). Asynchrony of senescence among phenotypic traits in a wild mammal population. *Experimental Gerontology*, *71*, 56-68. doi: 10.1016/j.exger.2015.08.003

Jackson, C. (2016). flexsurv: A platform for parametric survival modeling in R. *Journal of Statistical Software*, *70*(8). doi: 10.18637/jss.v070.i08

Jackson, D., White, I.R, Seaman, S., Evans, H., Baisley, K., & Carpenter, J. (2014). Relaxing the independent censoring assumption in the Cox proportional hazards model using multiple imputation. *Statistics in Medicine*, *33(27)*. doi: 10.1002/sim.6274

Jégo, M., Lemaître, J.-F., Bourgoin, G., Capron, G., Warnant, C., Klein, F., … Gaillard, J.-M. (2014). Haematological parameters do senesce in the wild: evidence from different populations of a long-lived mammal. *Journal of Evolutionary Biology*, *27*(12), 2745–2752. doi: 10.1111/jeb.12535

Jones, K.E., & MacLarnon. A. (2001) Bat life histories: testing models of mammalian life-history evolution. *Evolutionary Ecology Research*, *3*, 465-476.

Jones, O.R., Scheuerlein, A., Salguero-Gómez, R., Camarda, C.G., Schaible, R., Casper, B.B., … Vaupel, J.W. (2014). Diversity of ageing across the tree of life. *Nature*, *505*(7482), 169–173. doi: 10.1038/nature12789

Kirkwood, T. (2017). The Disposable Soma Theory Origins and Evolution. In R.P. Shefferson, O.R. Jones, & R. Salguero-Gómez (Eds.), *The Evolution of Senescence in the Tree of Life* (pp. 23–39). Cambridge, UK: Cambridge University Press.

Kowald, A., & Kirkwood, T.B.L. (2015) Evolutionary significance of ageing in the wild. *Experimental Gerontology*, 71, 89-94.

Lack, D. *Ecological adaptations for breeding in birds* (pp. 238-241). London, UK: Methuen.

Lemaître, J.-F., & Gaillard, J.-M. (2012). Male survival patterns do not depend on male allocation to sexual competition in large herbivores. *Behavioral Ecology*, *24*(2), 421–428. doi: 10.1093/beheco/ars179

Lemaître, J.-F., Gaillard, J.-M., Pemberton, J.M., Clutton-Brock, T.H., & Nussey, D.H. (2014). Early life expenditure in sexual competition is associated with increased reproductive senescence in male red deer. *Proceedings of the Royal Society B: Biological Sciences*, *281*, 20140792. doi: 10.1098/rspb.2014.0792

Lemaître, J. F., & Gaillard, J.-M. (2017). Reproductive senescence: new perspectives in the wild. *Biological Reviews*, *92*(4), 2182–2199. doi: 10.1111/brv.12328

Loison, A., Festa-Bianchet, M., Jullien, J.-M., Jorgenson, J.T., & Gaillard, J.-M. (1999). Age-Specific Survival in Five Populations of Ungulates: Evidence of Senescence. *Ecology*, *80*(8), 2539–2554. doi: 10.1890/0012-9658

MacNulty, D.R., Smith, D.W., Mech, L.D., & Eberly, L.E. (2009). Body size and predatory performance in wolves: Is bigger better? *Journal of Animal Ecology*, *78*(3), 532–539. doi: 10.1111/j.1365-2656.2008.01517.x

Maklakov, A.A, Bonduriansky, R., & Brooks, R.C. (2009). Sex differences, sexual selection, and ageing: an experimental evolution approach. *Evolution 63*(10), 2491–2503. doi: 10.1111/j.1558-5646.2009.00750.x

Marais, G.A.B., Gaillard, J.-M., Vieira, C., Plotton, I., Sanlaville, D., Gueyffier, F., & Lemaître, J.-F. (2018). Sex gap in aging and longevity: can sex chromosomes play a role? *Biology of Sex Differences*, *9*(1), 1–14. doi: 10.1186/s13293-018-0181-y

Murray, D. (2006). On Improving Telemetry-Based Survival Estimation. *Journal of Wildlife Management 70*(6), 1530-1543. doi:10.2193/0022-541X(2006)70[1530:OITSE]2.0.CO;2

Nielsen, J.F. (2012). *The evolutionary genetics of meerkats (Suricata suricatta)*. University of Edinburgh.

Nussey, D.H., Froy, H., Lemaître, J.-F., Gaillard, J.-M., & Austad, S.N. (2013). Senescence in natural populations of animals: widespread evidence and its implications for bio-gerontology. *Ageing Research Reviews*, *12*(1), 214–225. doi: 10.1016/j.arr.2012.07.004

Nussey, D.H., Kruuk, L.E.B., Morris, A., Clements, M.N., Pemberton, J.M., & Clutton-Brock, T.H. (2009). Inter- and intrasexual variation in aging patterns across reproductive traits in a wild red deer population. *The American Naturalist*, *174*(3), 342–357. doi: 10.1086/603615

Ozgul, A., Bateman, A.W., English, S., Coulson, T., & Clutton-Brock, T.H. (2014). Linking body mass and group dynamics in an obligate cooperative breeder. *The Journal of Animal Ecology*, *83*(6), 1357–1366. doi: 10.1111/1365-2656.12239

Péron, G., Gimenez, O., Charmantier, A., Gaillard, J.-M., & Crochet, P.-A. (2010). Age at the onset of senescence in birds and mammals is predicted by early-life performance. *Proceedings of the Royal Society B: Biological Sciences*, *277*(1695), 2849–2856. doi: 10.1098/rspb.2010.0530

Promislow, D. (1992). Costs of sexual selection in natural populations of mammals. *Proceedings of the Royal Society B: Biological Sciences*, *247*, 203–210.

R Core Team (2019). *R: A language and environment for statistical computing*. Vienna, Austria: R Foundation for Statistical Computing.

Ralls, K., & Mesnick, S. (2009). Sexual Dimorphism. In B. Wuersig, & J. Thewissen (Eds.), *Encyclopedia of marine mammals* (pp. 1005–1011). Amsterdam: Academic Press.

Ronget, V., Garratt, M., Lemaître, J.F., & Gaillard, J. M. (2017). The 'Evo-Demo' Implications of Condition-Dependent Mortality. *Trends in Ecology and Evolution*, *32*(12), 909–921. doi: 10.1016/j.tree.2017.09.003

Ruau, D., Burkoff, N., Bartlett, J., Jackson, D., Jones, E., Law, M., & Metcalfe, P. (2016). Multiple Imputation for Infromative Censoring. version 0.3.4, https://CRAN.R-project.org/package=InformativeCensoring

Ruby, J.G., Smith, M., & Buffenstein, R. (2018). Naked mole-rat mortality rates defy gompertzian laws by not increasing with age. *ELife*, *7*, 1–18. doi: 10.7554/eLife.31157

Sharp, S.P., & Clutton-Brock, T.H. (2010). Reproductive senescence in a cooperatively breeding mammal. *Journal of Animal Ecology*, *79*(1), 176–183. doi: 10.1111/j.1365-2656.2009.01616.x

Sharp, S.P., & Clutton-Brock, T.H. (2011a). Competition, breeding success and ageing rates in female meerkats. *Journal of Evolutionary Biology*, *24*(8), 1756–1762. doi: 10.1111/j.1420-9101.2011.02304.x

Sharp, S.P., & Clutton-Brock, T.H. (2011b). Reluctant challengers: why do subordinate female meerkats rarely displace their dominant mothers? *Behavioral Ecology*, *22*(6), 1337–1343. doi: 10.1093/beheco/arr138

Sierra, E., Fernández, A., de los Monteros, A.E., Arbelo, M., de Quirós, Y.B., & Herráez, P. (2013). Muscular senescence in cetaceans: adaptation towards a slow muscle fibre phenotype. *Scientific Reports*, *3*, 1795. doi: 10.1038/srep01795

Smyth, K., & Drea, C. M. (2016) Patterns of parasitism in the cooperatively breeding meerkat: a cost of dominance for females. *Behavioural Ecology*, *27*, 149-157. doi: 10.1093/beheco/arv132.

Sparkman, A.M., Blois, M., Adams, J., Waits, L., Miller, D.A.W., & Murray, D.L. (2017). Evidence for sex-specific reproductive senescence in monogamous cooperatively breeding red wolves. *Behavioral Ecology and Sociobiology*, *71*(1), 6. doi: 10.1007/s00265-016-2241-9

Spong, G. F., Hodge, S. J., Young, A.J., & Clutton-Brock, T.H. (2008). Factors affecting the reproductive success of dominant male meerkats. *Molecular Ecology*, *17*(9), 2287–2299.

931 doi: 10.1111/j.1365-294X.2008.03734.x

932 Stearns, S. (1992). *The Evolution of Life Histories*. Oxford, UK: Oxford University Press.

933 Stockley, P., & Bro-Jørgensen, J. (2011). Female competition and its evolutionary
934 consequences in mammals. *Biological Reviews of the Cambridge Philosophical Society*,
935 *86*(2), 341–366. doi: 10.1111/j.1469-185X.2010.00149.x

936 Tafani, M., Cohas, A., Bonenfant, C., Gaillard, J.-M., Lardy, S., & Allainé, D. (2013). Sex-
937 specific senescence in body mass of a monogamous and monomorphic mammal: the case
938 of Alpine marmots. *Oecologia*, *172*(2), 427–436. doi: 10.1007/s00442-012-2499-1

939 Therneau, T. (2015). A Package for Survival Analysis in R. version 2.44, https://CRAN.R-
940 project.org/package=survival.

941 Tidière, M., Gaillard, J.-M., Müller, D.W.H., Lackey, L.B., Gimenez, O., Clauss, M., &
942 Lemaître, J.-F. (2014). Males do not senesce faster in large herbivores with highly
943 seasonal rut. *Experimental Gerontology*, *60*, 167–172. doi: 10.1016/j.exger.2014.11.003

944 Tidière, M., Gaillard, J.-M., Müller, D.W.H., Lackey, L.B., Gimenez, O., Clauss, M., &
945 Lemaître, J.-F. (2015). Does sexual selection shape sex differences in longevity and
946 senescence patterns across vertebrates? A review and new insights from captive
947 ruminants. *Evolution*, *69*, 1–18. doi: 10.1111/evo.12801

948 Toïgo, C., & Gaillard, J.-M. (2003). Causes of sex-biased adult survival in ungulates : sexual
949 size dimorphism, mating tactic or environment harshness? *Oikos*, *101*(2), 376–384.

950 Tompkins, E.M., & Anderson, D.J. (2017) Sex-specific patterns of senescence in Nazca
951 boobies linked to mating system. *Journal of Animal Ecology* 88, 986-1000. doi:
952 10.1111/1365-2656.12944.

953 Ulm, K. (1989) On the estimation of threshold values. *Biometrics* 45, 1324-1326.

954 van de Pol, M., & Verhulst, S. (2006). Age-dependent traits: a new statistical model to separate
955 within- and between-individual effects. *The American Naturalist*, *167*(5), 766–773. doi:
956 10.1086/503331

957 Williams, G.C. (1957). Pleiotropy, Natural Selection, and the Evolution of Senescence.
958 *Evolution*, *11*(4), 398–411.

959 Zeh, J.A., & Zeh, D.W. (2005) Maternal inheritcance, sexual conflict and the maladapted male.
960 *Trends in Genetics*, 21(5), 281-286.

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