# Rank-related contrasts in longevity arise from extra-group excursions not delayed senescence in a cooperative mammal

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**SUMMARY**

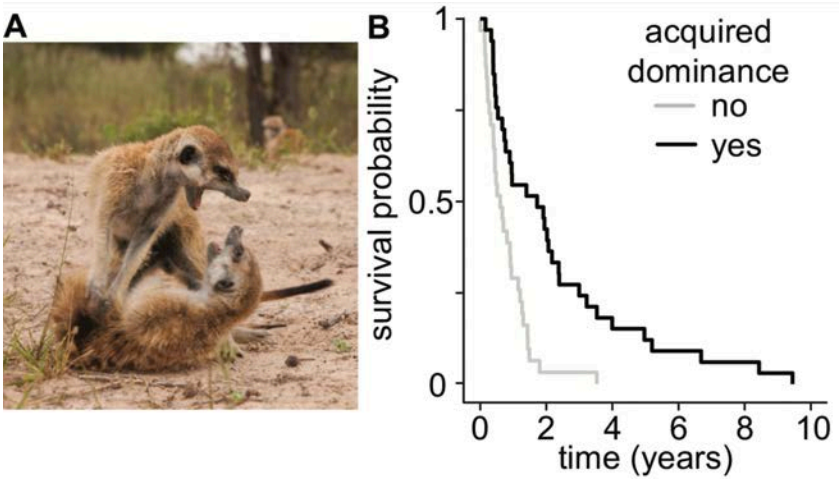
In many cooperatively breeding animal societies, breeders outlive non-breeding subordinates, despite investing heavily in reproduction [1-3]. In eusocial insects, the extended lifespans of breeders arise from specialised slowed ageing profiles [1], prompting suggestions that reproduction and dominance similarly defer ageing in cooperatively breeding vertebrates, too [4-6]. Although lacking the permanent castes of eusocial insects, breeders of vertebrate societies could delay ageing via phenotypic plasticity (similar rank-related changes occur in growth, neuroendocrinology and behaviour [7-10]), and such plastic deferment of ageing may reveal novel targets for preventing ageing-related diseases [11]. Here, we investigate whether breeding dominants exhibit extended longevity and delayed age-related physiological declines, in wild cooperatively breeding meerkats. We show that dominants outlive subordinates but shower *faster* telomere attrition (a marker of cellular senescence and hallmark of ageing [12]), and that in dominants (but not subordinates), rapid telomere attrition is associated with mortality. Our findings further suggest that, rather than resulting from specialised ageing profiles, differences in longevity between dominants and subordinates are driven by subordinate dispersal forays, which become exponentially more frequent with age and increase subordinate mortality. These results highlight the need to critically examine the causes of rank-related longevity contrasts in other cooperatively breeding vertebrates, including social mole-rats, where they are currently attributed to specialised ageing profiles in dominants [4].

## RESULTS

We combined survival, behaviour and leukocyte telomere data from wild meerkats to investigate whether contrasts in lifespan between dominants and subordinates are better explained by divergent ageing profiles or different dispersal propensities. Meerkats live in cooperatively breeding groups of up to 50 individuals (median 17) [13], in which a single dominant male and female monopolise reproduction (producing 86% and 93% of all pups, respectively). Subordinates provide care for the dominants' pups, but typically forego breeding due to reproductive suppression by the same-sex dominant (Figure 1A) [13]. At our study population in South Africa, we followed individuals from birth to death, observing behaviour and group composition three times per week and collecting blood samples at regular intervals [13]. Our study area covers over 80km$^2$ and dispersal distances are short (mean: 2.2km [14]), allowing us to detect dispersal with unusual resolution for a wild mammal.

*Dominant individuals live longer than those that never acquire dominance*

To investigate whether lifespan is extended by dominance acquisition, we compared the longevities of focal dominant individuals from the point at which they achieved dominance with those of one or more of their littermates who survived at least until the focal littermate acquired dominance, but did not become dominant themselves. This approach avoids confounds between cause and effect, such as the increasing likelihood of dominance acquisition as an individual grows older [13]. Our results showed that dominants of both sexes live consistently longer than their subordinate littermates (Figure 1b, $\chi^2_1 = 6.44$, $p = 0.01$, $n = 217$ individuals from 91 litters), and male and female survival probabilities did not differ ($\chi^2_1 = 0.35$, $p = 0.55$).

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77 **Figure 1**: **Dominant meerkats live longer than their littermates that remain subordinate**
78 **A**. A struggle for dominance between two meerkats (photo: Dominic Cram) **B.** Meerkats that
79 acquire a dominance position (black line; 2.27 ± 0.41 years, mean ± S.E.) survive almost
80 three times higher than their littermates that do not (grey line; 0.79 ± 0.13 years). Time refers
81 to the period after the focal littermate acquired dominance. Figure shows a dataset including
82 only individuals of known lifespan; inclusion of right-censored individuals for whom age at
83 death could not accurately be confirmed did not qualitatively alter the results.

84

85 *Dominants' rates of telomere attrition are faster, not slower, than those of subordinates*

86

87 Next, we investigated whether the extended longevities of dominant individuals could be
88 driven by slowed rates of age-related physiological decline, by comparing rates of leukocyte
89 telomere attrition between dominants and subordinates. Telomeres are protective sequences at
90 the ends of eukaryotic chromosomes, which shorten with each cell division [12]. Unless
91 repaired [15], excessive telomere loss causes cell senescence, apoptosis, and tissue dysfunction
92 that exacerbates over time [12, 16]. As such, while the causal role played by telomeres in
93 organismal senescence remains unclear [17], rapid telomere attrition is widely interpreted as

an integrative biomarker of age-related declines in physiological state [18], and has been identified as a 'hallmark of ageing' [19].

We used a within-subject centring approach to statistically distinguish within-individual changes in telomere lengths (which are our primary interest) from between-individual effects (which reflect population-level processes such as selective disappearance [20, 21]). We hereafter use 'within-individual time' to refer to individual changes in telomere length over an animal's life, and 'between-individual age' to refer to changes in telomere length with age at the population level (see STAR Methods). Dominants and subordinates did not differ in age or telomere lengths at the start of the sampling period (age: $t_{29.938} = 1.24$, $p = 0.27$; telomere length: $t_{29.99} = 0.18$, $p = 0.86$). Moreover, within-individual telomere attrition was more rapid in dominants than in subordinates (Figure 2a, dominance status × within-individual time: $\chi_1^2 = 4.1$, $p = 0.04$, n = 99 samples from 35 individuals). Separate post-hoc models for each dominance status confirmed that telomere length declines were evident in dominants (within-individual time: $\chi_1^2 = 12.12$, $p < 0.001$, n = 54 samples from 17 individuals) but not subordinates ($\chi_1^2 < 0.01$, $p = 0.98$, n = 45 samples from 18 individuals). Telomere lengths were not significantly predicted by sex or between-individual age, either as single terms or as an interaction (all $\chi^2 < 3.01$, $p > 0.08$). Males and females showed similar within-individual declines in telomere lengths (sex × within-individual time: $\chi_1^2 = 1.15$, $p = 0.28$).

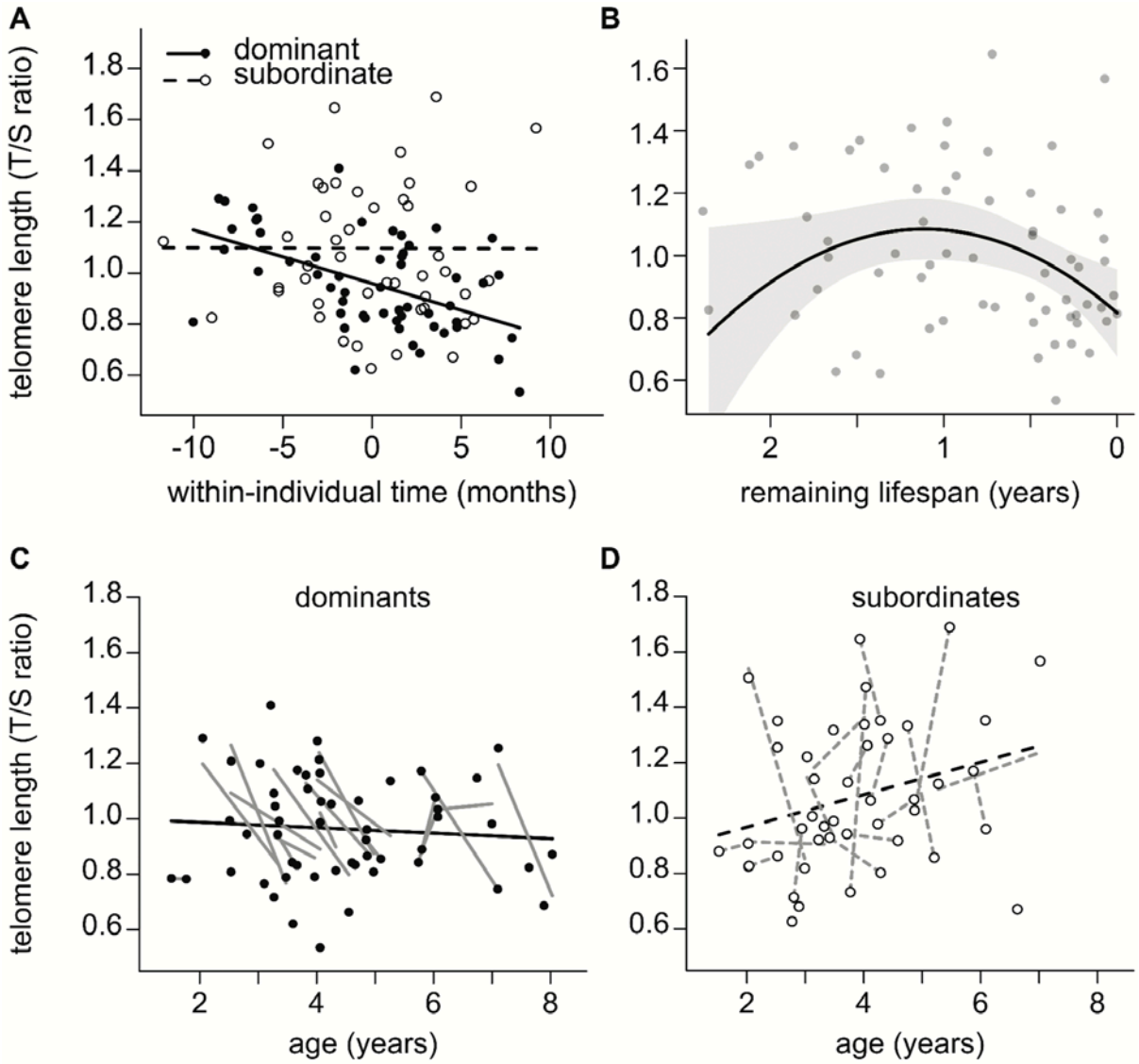


**Figure 2: Differences in telomere dynamics between dominant and subordinate meerkats are associated with mortality A.** Telomeres shortened rapidly in dominant meerkats (black points, solid line), but not in subordinates (open points, dotted line). Lines represent predictions from the dominance × within-individual time interaction in a GLMM. **B.** Telomere lengths were stable until individuals entered their final year of life, at which point they declined rapidly until death. The solid line shows the predictions from the quadratic remaining lifespan term in a GLMM. Shaded areas represent 95% confidence intervals of the fixed effects. **C.** In dominants, the within-individual telomere declines (solid grey lines) were significantly more rapid than those evident at the between-individual (population) level (solid black line), which is evidence of selective disappearance of dominants with short telomeres. **D.** By contrast in

subordinates, the within- and between-individual changes did not differ (dotted grey and dotted black lines, respectively), suggesting no selective disappearance of subordinates based on telomeres. In **C.** and **D.**, black lines are the GLMM predictions of the effect of between-individual age on telomere lengths. Grey lines represent within-individual change in telomere lengths from separate linear models for each individual. In all panels, points represent telomere lengths.

*Rapid telomere attrition is associated with impending mortality*

To investigate whether telomere shortening is associated with near-term mortality, we examined telomere dynamics in a restricted dataset of dominant and subordinate individuals with a confirmed date of death. Our results provided evidence of terminal telomere declines at the end of life: telomere lengths were stable until the final year of life, whereafter telomere attrition was rapid until death (Figure 2b, remaining lifespan$^2$, $\chi^2_1 = 5.94$, $p = 0.015$, 53 samples from 16 individuals). Telomere lengths were not significantly predicted by between-individual age in this dataset ($\chi^2_1 = 0.15$, $p = 0.70$).

We then tested for evidence of selective disappearance of dominants and subordinates with short telomeres, by comparing the slopes of within- and between-individual changes in telomere lengths [20]. In a restricted dataset of dominant individuals, the within-individual change in telomere lengths was significantly more rapid than the between-individual (population) change (Figure 2c, $\chi^2_1 = 5.12$, $p = 0.02$, see STAR Methods). Dominants' telomeres were shortening, but this was not evident at the population level because those with short telomeres were disproportionately likely to die, yielding a biased surviving cohort of older individuals with long telomeres. The disparity in slopes of within- and between-

individual effects on telomere lengths thus provides evidence of selective disappearance of dominants with short telomeres [20]. In subordinates, by contrast, we found no evidence selective disappearance based on telomere lengths (Figure 2d, $\chi^2_1 = 0.32$, $p = 0.57$).

*Subordinates spend more time on high-mortality extra-group excursions than dominants*

Given that dominants' extended longevities were unlikely to be driven by slowed age-related physiological declines (as telomere attrition was accelerated in dominants compared to subordinates), we tested whether the shorter lives of subordinates may instead be explained by patterns of dispersal. We investigated dispersal propensity in subordinates and dominants, using a within-individual comparison in a dataset of individuals who spent at least one full year as a subordinate *and* as a dominant. We divided each individual's life into years either side of its dominance acquisition, and counted the days the individual spent away from all social groups during each year (n = 245 subordinate years and 252 dominant years from 73 individuals). Males spent significantly more time per year outside of social groups than females ($\chi^2_1 = 156.42$, $p < 0.001$, n = 42 males: 19.13 ± 2.53 days/year (mean ± S.E.); n = 31 females: 15.84 ± 2.67 days/year). Controlling for the effect of sex, individuals spent significantly more time away from social groups as subordinates compared to after they acquired a dominance position (Figure 3a, $\chi^2_1 = 22.29$, $p < 0.001$).

We then examined how an individual's propensity to leave the social group changes over time, both as a subordinate and as a dominant, to examine whether mortality risks related to dispersal are stable or increasing in the two dominance classes. For each individual from the above dataset, we assigned each year a value in reference to its dominance acquisition (e.g. -1 for the year prior to acquisition, 0 for the year of acquisition etc., hereafter termed 'years after

dominance acquisition'). Within subordinates, time outside the group increased exponentially with age (Figure 3b dotted line, years after dominance acquisition$^2$: $\chi^2_1$= 127.83, p < 0.001). Male and female subordinates spent similar time away from social groups ($\chi^2_1$= 0.57, p = 0.49). By contrast, once these same individuals acquired a dominance position, time outside the group was low and did not change with age (Figure 3b solid line, years after dominance acquisition: $\chi^2_1$= 0.97, p = 0.32). Dominant males spent longer out of the group than dominant females (sex × dominance status: $\chi^2_1$= 26.48, p < 0.001, males: 10.19 ± 2.22 days/year, females: 2.10 ± 1.42 days/year). This sex-difference likely arises due to differences in replacement after the death of male and female dominants. 'Widowers' (dominant males whose mate has died) are forced to prospect outside of the group to avoid inbreeding, while 'widows' (dominant females whose mate has died) remain in their group and pair with an immigrant male [22, 23].

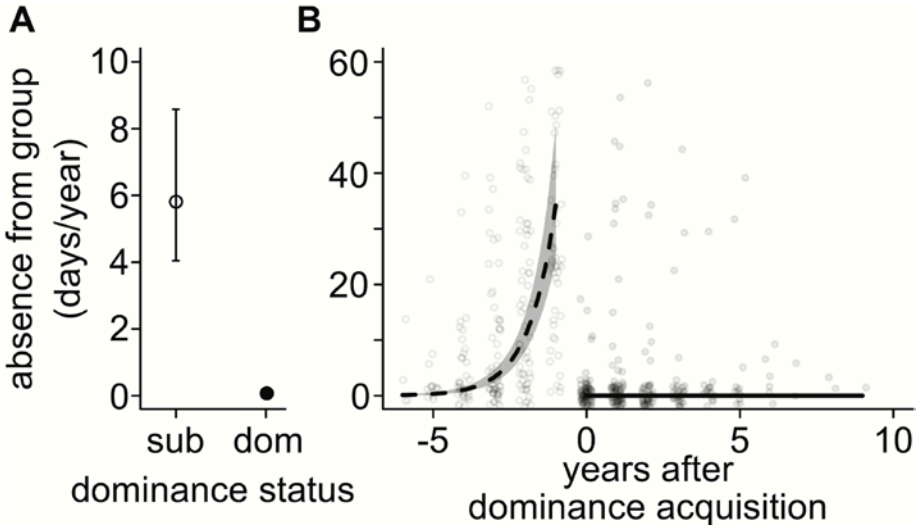


**Figure 3**: **Dominance- and age-related changes in dispersal propensities. A.** Subordinates spend significantly more time away from a stable social group compared to dominants. Points and error bars are predicted means and S.E. from a GLMM. **B.** While subordinate, individuals spend increasing periods of time away from their group as they grow older. After they acquire dominance, the same individuals spend consistently less time away from the group. Points show the number of days/year each individual spent absent from a social group, jittered for

clarity. The line shows the GLMM predictions for subordinates (dotted) and dominants (solid). The dominants' model predictions are for a female. Shaded areas are 95% confidence intervals of the fixed effects (these are imperceptible for dominants).

Finally, we investigated whether prolonged extra-group periods are associated with elevated mortality risk. We divided subordinates' lives into three-month periods, and for each period calculated how many days they spent away from stable social groups and scored whether the individual died during that period. A Cox's proportional hazard model revealed that extra-group periods conferred a substantially increased risk of mortality (Figure 4, $p = 0.04$, $n = 406$ periods from 97 individuals).

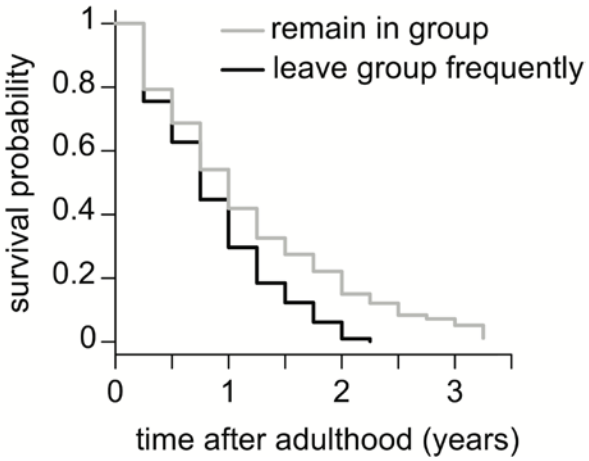


**Figure 4: Survival probability is reduced in subordinates that regularly leave their social group**. Lines represent predictions from a Cox's proportional hazard model. The grey line represents a subordinate that never leaves their home group. The black line represents a subordinate that initially spends 14 days per three-month period away from their social group, with this value increasing by 34% in each subsequent period. This matches the rate of increase in time away from the group exhibited by subordinates (see Figure 3B).

## DISCUSSION

Our findings show that dominants outlive subordinates in wild meerkat societies, yet exhibit signs of *accelerated* age-related declines in physiological state [12, 18]. Dominants showed faster rates of telomere loss than subordinates and, among dominants (but not among subordinates) individual differences in rates of telomere shortening were correlated with variation in longevity. Our analysis of age- and rank-related dispersal propensities revealed that subordinates left their natal group frequently and the probability that they would do so increased with age, while dominants spent consistently little time away from their groups and only 12% ever left it before their death (Duncan *et al.* in review). Time outside the group carries significant mortality risks, which likely account for the curtailed lifespans of subordinates relative to dominants.

These results suggest that the extended lifespans of dominants relative to subordinates occur in spite of, and not due to, differences in rates of age-related physiological decline. Telomere attrition, an important causal factor in the accumulation of senescent cells [16], was more rapid in dominants than subordinates by two orders of magnitude, and was associated with elevated mortality. As increases in senescent cells are a major contributor to age-related declines in tissue function associated with an ageing phenotype [24], our results suggest that dominants do not show delayed ageing profiles, as has been suggested in some cooperatively breeding mammals [4-6]. Evidence of slower rates of telomere attrition in dominants would have been surprising given their greater investment in reproduction, which typically diverts resources away from self-maintenance and accelerates declines in physiological state with age [25]. Our finding that dominant male and female meerkats (who monopolise reproduction [13]) exhibit telomeres that deteriorate more rapidly than their reproductively-suppressed subordinates is

241 thus consistent with life-history and evolutionary senescence theory [26, 27]. Investment in
242 reproduction, as well as defence of their territory and dominance position, leaves dominant
243 members of animal societies with impaired antioxidant [28] and immune defences [29, 30],
244 and leads to elevated exposure to stress hormones [31]. Accelerated telomere attrition in
245 dominant meerkats is therefore likely reflective of the physiological toll of social dominance
246 and reproduction, which leaves dominants with declining health [32, 33].
247
248 Differences in longevity between dominants and subordinates are not consistent with patterns
249 of age-related increases in cellular senescence, and more likely arise due to high risks
250 associated with time spent away from the social group. Our results reveal substantial survival
251 costs of leaving the natal group [34], which arise from risks including elevated predation, fatal
252 encounters with neighbouring groups and reduced foraging success [35-38]. The longer
253 lifespans of dominants may therefore be explained by our finding that they almost never leave
254 their group after acquiring dominance, thus benefitting from the well-documented survival
255 advantages of group-living [39]. Not only do subordinates of both sexes spend more time away
256 from the group than dominants, but the frequency of their extra-group excursions rises
257 exponentially with age: older subordinate males more frequently leave the group in pursuit of
258 outbreeding opportunities, while older subordinate females are more likely to be aggressively
259 evicted by their same-sex dominant [13]. As such, the curtailed lifespans of subordinates are
260 likely not due to accelerated rates of cellular senescence relative to dominants, but arise as a
261 result of age-related phenotypic changes in subordinates, which exponentially increase their
262 frequency of high-risk extra-group excursions.
263
264 Our results emphasise that large differences in longevity, even between members of the same
265 social group, do not necessarily arise from divergent rates of senescence. These findings raise

questions about rank-related longevity differences in other mammalian cooperative breeders, especially the social mole-rats. Delayed ageing has been proposed as an explanation for dominants' longevity in several mole-rat species [4-6], but to our knowledge there is no evidence such ageing profiles exist in dominants but not subordinates. While specialised ageing physiology is evident in some eusocial insect queens [1], this arises from permanent developmental castes not found in vertebrates. In cooperatively breeding mammals, contrasts in longevity are more likely to arise from consistent differences in aggression and dispersal, which expose some group members to differentially high extrinsic mortality. As such, our understanding of the determinants of both longevity and rates of senescence in cooperative vertebrates relies on long-term individual-based studies conducted in the environment in which they evolved.

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## AUTHOR CONTRIBUTIONS

DC devised the study, carried out statistical analyses and wrote the manuscript with support from TC-B and PM. BD designed sampling protocols, CD and HS-P collected/extracted data, RG conducted laboratory analyses with advice from PM.

## DECLARATION OF INTERESTS

The authors declare no competing interests.

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## STAR METHODS

### CONTACT FOR REAGENT AND RESOURCE SHARING

Further information and requests for resources should be directed to and will be fulfilled by the Lead Contact, Dominic Cram (dom.cram@gmail.com)

### EXPERIMENTAL MODEL AND SUBJECT DETAILS

#### Ethical Note

Our work was approved by the Animal Ethics Committee of the University of Pretoria, South Africa (no. EC010-13) and the Northern Cape Department of Environment and Nature Conservation, South Africa (FAUNA 1020/2016), and adhered to the ASAB/ABS Guidelines for the Treatment of Animals in Behavioural Research and Teaching.

#### Study Population

Data collection was conducted in the context of a long-term study, monitoring a naturally regulated population of wild meerkats at the Kuruman River Reserve, South Africa (26˚ 58'S, 21˚ 49'E), between 1994 and 2016. All study individuals were individually tagged (Five Star ID, Johannesburg, South Africa), habituated to close observation (<1m), and visually recognizable using small dye-marks [13]. Groups were visited 2-3 times per week for 4-8 hours, to collect behavioural, life-history and group composition data. Dominance status and transitions in dominance were conspicuous and determined using protocols detailed elsewhere [40].

### METHOD DETAILS

455 Capture and blood sampling

456 Adult meerkats were captured and anaesthetized using established protocols [41], and a blood
457 sample (0.2 - 2.6ml, depending on body mass) was drawn from the jugular vein using a 25G
458 needle and syringe. Whole blood samples were stored in EDTA tubes (Lasec, Johannesburg,
459 South Africa) and frozen at -80°C. Samples were later transported on dry ice from the field site
460 to our laboratory in Glasgow, UK, where they were stored at -80°C until analysis. In our
461 standard capture schedule, we aimed to capture individuals within every six-month window,
462 beginning at age six months. In the current study, we used samples collected after 18 months,
463 according to the availability of individuals or equipment.

464

465 qPCR determination of telomere lengths in leukocytes

466 We used quantitative PCR (qPCR) analysis to measure leukocyte telomere lengths in whole
467 blood samples, based on published protocols with some modifications [42, 43]. This measure
468 represents the average telomere length across cells in a sample and is reported as the level of
469 telomeric sequence abundance relative to a reference non-variable copy number gene (T/S
470 ratio).

471

472 DNA was extracted using Gentra Puregene Blood Kits (QIAGEN Ltd, Manchester, United
473 Kingdom), broadly following standard protocols for extraction of genomic DNA from 300µl
474 whole blood, with the following volume modifications: 600µl of Cell Lysis buffer and 5µl
475 Proteinase K (20mg/ml) were used for lysis, 230µl of Protein Precipitation Solution was added
476 to lysate, 1.5µl glycogen solution (QIAGEN Ltd, Manchester, United Kingdom) was added to
477 supernatant before DNA precipitation step, 700µl isopropanol was used for DNA precipitation,
478 700µl of 70% ethanol was used for pellet washing, and DNA was rehydrated with 25µl DNA

Hydration Solution. Samples were incubated at 56°C for 1-3 hours until completely lysed, and DNA was left to rehydrate at 4°C overnight before being mixed with 175µl PBS to be purified using MACHEREY-NAGEL NucleoMag® Blood 200µL kits (MACHEREY-NAGEL GmbH & Co. KG, Düren, Germany) in tandem with KingFisher™ Flex Purification System (Thermo Scientific, Wilmington DE, USA), following kit protocols. DNA was eluted in kit elution buffer MBL5 (5 mM Tris, pH 8.5) and stored at -20°C until further use. DNA concentration and purity was assessed using a Nanodrop-8000 Spectrophotometer (Thermo Scientific, Wilmington DE, USA). Average DNA concentration was 45.7 ± 19.4 ng/ul (mean ± SD) and average 260/280 and 260/230 ratios were 1.93 ± 0.07(mean ± SD) and 1.98 ± 0.12 (mean ± SD), respectively. DNA integrity was assessed by running 30ng of DNA in a 0.8% agarose gel at 120V for 30 minutes and was deemed to be acceptable for telomere measurement.

We used quantitative PCR (qPCR) to measure telomere length, based on published methods [42, 43] with some modifications. This measure represents the average telomere length across cells in a sample, and is reported as the abundance of telomeric sequence relative to a non-variable copy number gene. We used RAG1 as a reference gene, on account of its known status as a single copy gene in vertebrates [44] and used a primer pair designed from Accession JQ073171.1 selected for their good performance, lack of non-specific binding and lack of primer-dimer (confirmed by melt curve analysis and gel electrophoresis) during optimisation. HPLC purified primers were synthesised by IDT® (Integrated DNA Technologies, Leuven, Belgium), re-suspended, diluted and stored at -20°C until assays were run. DNA samples (1.25ng) were assayed in triplicate and on separate plates for telomere and single-copy targets. Reactions were conducted using 1X Absolute blue qPCR SYBR green Low Rox master mix (Thermo Scientific, Wilmington DE, USA) with RAG1 forward (5’-CAT TGA GAC AGT CCC TTC CAT AG-3’) and reverse (5’-GGA GGC ATT GGG ATT CTT GTA-3’) primers at 500nM and telomere primers Tel1b (5’-CGG TTT GTT TGG GTT TGG GTT TGG GTT TGG

GTT TGG GTT-3') and Tel2b (5'-GGC TTG CCT TAC CCT TAC CCT TAC CCT TAC CCT TAC CCT-3') [45] at 900nM, bringing reaction volumes up to 25µl with water. Mx3000P 96-well skirted plates (Agilent, Santa Clara, United States) were manually loaded, sealed with 8x strip optical caps (Agilent, Santa Clara, United States) and run in an Agilent Technologies Stratagene Mx3005P real-time PCR machine. RAG1 thermal profile was 15 min at 95°C, followed by 40 cycles of 15 s at 95°C, 30 s at 60°C, 30 s at 72°C. Telomere thermal profile was 15 min at 95°C, followed by 30 cycles of 15 s at 95°C and 30 s at 58°C. Both assays were followed by melt curve analysis of (58–95°C 1°C/5 s ramp). Dissociation curves showed a single peak for both assays in all reactions. The telomere assay had very late amplification in the no template control (NTcalC) (Ct > 26.95). This was deemed to be late formation of primer-dimer and acceptable considering the highest Ct of our DNA samples (17.37). There was no amplification in NTC for the RAG1 assay. A pooled aliquot of DNA samples was serially diluted (5ng to 0.31ng) to generate a 5- point standard curve for each plate, which was used to calculate plate efficiencies (1.945-1.995 for RAG1 plates; 1.983-2.179 for telomere plates). The $r^2$ for all plates was >0.985. Efficiency controlled relative telomere lengths (T/S ratios) were calculated for each sample using the 1.25ng point of the standard curve as the "gold" control sample for each plate [46] using the following equation:

$$T/S = (E_{TELO}\text{^}(Ct_{TELO[GOLD]} - Ct_{TELO[SAMPLE]}))/ (E_{RAG1}\text{^}(Ct_{RAG1[GOLD]} - Ct_{RAG1[SAMPLE]}))$$

$E_{TELO}$ and $E_{RAG1}$ are the reaction efficiencies of each target plate on which a sample was run, $Ct_{TELO[GOLD]}$ and $Ct_{TELO[SAMPLE]}$ are the mean Cts of the gold and experimental sample on the telomere plate, respectively and, similarly, $Ct_{RAG1[GOLD]}$ and $Ct_{RAG1[SAMPLE]}$ are the mean Cts of the gold and experimental sample on the telomere plate, respectively. Technical replicates falling outside 0.5Cts were excluded or repeated, as were samples that were beyond the limits of the standard curve. Samples were assigned to plates randomly. The average intraplate variation of the Ct values for RAG1 and telomere plates was 0.38% and 0.56%, respectively.

Intraplate variation was calculated as the coefficient of variation of the replicates of the gold sample (1.25ng standard curve point) within a given plate. Interplate variation, calculated as the coefficient of variation of the ΔCt for the gold sample on all plates, was 3.19%. An additional sample was run on every plate and the coefficient of variation for the T/S ratio for this sample was 6.18%. To confirm assay repeatability, a set of samples were assayed twice; T/S values were highly correlated (Pearson correlation, n = 19 samples, r = 0.909).

## QUANTIFICATION AND STATISTICAL ANALYSIS

Statistical analyses were carried out in R version 3.2.3, using a step-wise model simplification approach [47, 48] unless otherwise specified. Initially all terms of interest were fitted, followed by the stepwise removal of terms whose removal from the model resulted in a non-significant change in deviance (using a likelihood-ratio test for model comparison), until the minimal adequate model (MAM) was obtained, in which only significant terms remained. Dropped terms were added to the MAM to confirm their non-significance. The significance of all terms was tested either by removing the terms from the MAM (if the term was in the MAM) or adding the terms to the MAM (if the term was not included in the MAM). All analyses of telomere data refer to telomere lengths; no models contain derived calculations of changes in telomere lengths between time-points. We use telomere 'loss', 'attrition', 'shortening' or similar to refer to the slopes of telomere lengths against age or time.

*1) Do dominants and subordinates differ in longevity?*

We compared the longevities of focal individuals, from the point at which they achieved dominance, with those of one or more of their littermates who survived at least until the same time-point, but did not become dominant themselves. We used a Cox proportional hazards model [49] to test the influence of dominance status and sex on survival in a dataset of 217 individuals from 91 litters. The Cox model is a nonparametric survival analysis [50] that accounts for the influence of multiple predictors simultaneously and over time on the risk of death (i.e., the hazard rate), with no assumption of the shape of the hazard function. The model was fitted using a mixed effects Cox model ("Coxme" package [51]) in order to include litter ID as a random term. While subordinates are more likely to disperse than dominants [13], this is unlikely to bias our estimates of longevity, for three reasons. First, dispersal distances are typically short (mean: 2.2 km, interquartile range: 1.08-2.66km [14]), facilitating detection of successful dispersal in our large study area [> 80km², 13]. Second, we closely monitor our study population for the establishment of new groups by dispersing subordinates. While dispersing males are occasionally accepted into neighbouring groups, females never are [13], and as such any dispersing female that does not return or establish a new group has likely died. Third, our statistical approach is robust to disappearance of individuals: those for whom death could not be confirmed (and who may therefore have dispersed) were right-censored, and were thus not assumed to have died. In a restricted dataset of individuals for whom death could be confirmed, a mixed-effects Cox model yielded qualitatively identical results. Death was confirmed by finding a carcass or radio-collar, by observing rapid declines in health followed by disappearance from the group, or by euthanasia of individuals showing advanced stages of tuberculosis infection (such individuals would otherwise die within weeks [52]).

*2) Do dominants and subordinates differ in rates of telomere attrition?*

As samples were not available for the litter-matched dataset used above, we used a dataset of 99 samples collected between April 2014 and January 2016. While our primary interest was within-individual longitudinal changes in telomere lengths over time, a standard mixed modelling approach fails to discriminate within-subject effects and between-subject effects (e.g. selective disappearance [21]). Differentiating between these effects is important, as population-level patterns frequently mask or even operate in the opposite direction to within-subject change over time [53]. To statistically distinguish these effects, we used within-subject centring [20], which replaces the age parameter with two new variables, "between-individual age" and "within-individual time." The former is the average age from all telomere samples for a given individual, and the latter is the deviation from this for each sample, such that for each sample, age = between-individual age + within-individual time. Replacing age with these two terms in the model provides distinct estimates for the between-subject effect (between-individual age) and the within-subject effect (within-individual time). For a dataset of 99 samples from 35 individuals (range 2-6 samples per individual, mean 2.9, 17 dominants, 18 subordinates), we fitted telomere length as the response in a general linear mixed model (GLMM) with individual ID, group ID and telomere assay plate as random terms. We fitted dominance status, sex, within-individual time and between-individual age as fixed effects, as well as the interactions: dominance × within-individual time, sex × within-individual time and sex × between-individual age.

*3) Are individual differences in rates of telomere loss related to longevity?*

We first investigated whether rates of telomere attrition were more rapid in individuals approaching the end of life, in a restricted dataset including only individuals for whom the date of death could be accurately confirmed. Unfortunately, it was not possible to contrast

dominants and subordinates in this limited dataset (n = 53 samples from 11 dominants and 6 subordinates). We fitted between-individual age and remaining lifespan (at sampling date) as predictors in a GLMM, with telomere length as the response and individual and telomere plate ID as random terms. We also fitted the quadratic polynomial of remaining lifespan as a predictor, to test for non-linear declines in telomere lengths as an individual approaches the end of its life.

We then examined evidence of selective disappearance in our full dataset (which includes all telomere samples). When the slope of telomere attrition is steeper for within-individual than between-individual comparisons, this is evidence that disproportionate disappearance of individuals with short telomeres is concealing within-individual declines in telomere lengths at the population level [54]. We created separate datasets for dominants and subordinates, to investigate the role of selective disappearance in each. We then fitted separated GLMMs for dominants and subordinates, with telomere length as the response and individual ID and telomere plate ID as random terms. Unpartitioned age at sampling and between-individual age were fitted as fixed effects. We then removed the between-individual age term to test for significant differences between the slopes of within- and between-individual changes in telomere lengths, using a likelihood-ratio test [20].

*4) Are contrasts in longevity between dominants and subordinates attributable to age-related contrasts in dispersal?*

We investigated how dominance affects the amount of time per year meerkats spend away from stable social groups, using a within-individual comparison in a dataset of individuals who spent at least one full year as a subordinate *and* as a dominant (n = 42 males, n = 31 females). We

627 defined an individual as ‘away from stable social groups’ if it was not present at its home group,
628 and not observed joining any other social group. We then assessed the length of each extra-
629 group foray, from the day the individual left its home group until the day it returned to a stable
630 group (either by re-joining its home group, joining another group, or founding a new group).
631 We divided each individual’s life into years either side of its dominance acquisition, and
632 counted how many days the individual spent away from all social groups during each year.
633 First, we contrasted the number of days per year each meerkat spent outside of its social group
634 during its period as a subordinate and as a dominant. We fitted the number of days outside the
635 group per year as the response term in a GLMM with a Poisson distribution. Individual sex and
636 dominance status (subordinate / dominant) were fitted as predictors, and individual ID, natal
637 group ID, litter ID and cohort year were fitted as random terms. We also fitted an observation-
638 level random term, to correct for overdispersion [55].
639
640 We then examined how the period individuals spent outside of a stable social group changed
641 over time. For each individual from the above dataset, we assigned each year a value in
642 reference to its dominance acquisition (hereafter ‘years after dominance acquisition’). The year
643 immediately prior to dominance acquisition was scored as -1 (with the preceding years as -2, -
644 3 etc.), and the year beginning at dominance acquisition as 0 (with subsequent years as 1, 2
645 etc.). This permitted an analysis of how an individual’s propensity to leave the social group
646 changes over time, both before and after its dominance acquisition. Preliminary inspection of
647 the data suggested markedly different patterns in individuals before and after dominance
648 acquisition. We therefore ran separate models for the two periods (pre-acquisition
649 subordinates: 245 years from 71 individuals, post-acquisition dominants: n = 252 years from
650 71 individuals, mean ± S.D. age of dominance acquisition: 34.4 ± 12.2 months). In each
651 GLMM, we used a Poisson distribution and fitted time spent outside of the group (days/year)

as the response, with sex as a factor and 'years after dominance acquisition' as a covariate. The random terms were individual ID, natal group ID, litter ID and cohort year. As above, we included an observation-level random term, to correct for overdispersion.

Finally, we investigated whether prolonged periods away from stable social groups are associated with elevated mortality risk. Given preliminary inspections of the previous datasets, which suggested dominants spend close to zero time outside of their social group, we restricted this analysis to subordinates. In a dataset of individuals with known age at death, we divided each animal's life into three-month windows, from the onset of adulthood (one year old) until their death. In each window (n = 406 windows from 97 individuals), we calculated how many days the individual spent away from stable social groups. This data was then fitted in a Cox's proportional hazards survival model using the R-package 'Survival' [56]. The response term was the individual's age at the start of a given time window, and a binary variable denoting whether the individual died during that window. The period of time spent away from the social group during each window was fitted as a time-dependent covariate. Results presented are from the full model.

## DATA AND SOFTWARE AVAILABILITY

Data used in these analyses are available via the Mendeley Data Repository (doi:10.17632/2rywb953sw.1)