Effects of early-life competition and maternal nutrition on telomere lengths in wild meerkats

Dominic L. Cram[1,2]*, Pat Monaghan[3], Robert Gillespie[3] & Tim Clutton-Brock[1,2]

[1] Department of Zoology, University of Cambridge, Downing Street, Cambridge CB2 3EJ, United Kingdom

[2] Kalahari Meerkat Project, Kuruman River Reserve, P.O. Box 64, Van Zylsrus, Northern Cape 8467, South Africa

[3] Institute of Biodiversity, Animal Health and Comparative Medicine, College of Medical, Veterinary and Life Sciences, University of Glasgow, Graham Kerr Building, Glasgow G12 8QQ, United Kingdom

*Author for correspondence: dom.cram@gmail.com

Abstract

Early-life adversity can affect health, survival and fitness later in life, and recent evidence suggests that telomere attrition may link early conditions with their delayed consequences. Here, we investigate the link between early-life competition and telomere length in wild meerkats. Our results show that, when multiple females breed concurrently, increases in the number of pups in the group are associated with shorter telomeres in pups. Given that pups from different litters compete for access to milk, we tested whether this effect is due to nutritional constraints on maternal milk production, by experimentally supplementing females' diets during gestation and lactation. While control pups facing high competition had shorter telomeres, the negative effects of pup number on telomere lengths were absent when maternal nutrition was experimentally improved. Shortened pup telomeres were associated with reduced survival to adulthood, suggesting that early-life competition for nutrition has detrimental fitness consequences that are reflected in telomere lengths. Dominant females commonly kill pups born to subordinates, thereby reducing competition and increasing growth rates of their own pups. Our work suggests an additional benefit of infanticide may be that it also reduces telomere shortening caused by competition for resources, with associated benefits for offspring ageing profiles and longevity.

KEY WORDS

telomeres, early-life adversity, early-life stress, *Suricata suricatta,* meerkats, infanticide

Introduction

The early period of an animal's life can have a disproportionately influential role in determining health, survival and reproductive success later in life, even though it accounts for a relatively minor proportion of total lifespan [1]. Despite the importance of the early-life environment, our understanding of the physiological mechanisms underpinning its lasting and delayed consequences remains poor [2].

Telomere loss has recently been proposed as a potential molecular mechanism linking early-life adversity with later-life performance and ageing [3]. Telomeres are non-coding sequences at the ends of eukaryotic chromosomes that play a critical role in protecting genome integrity [4]. Telomeres shorten with each cell division, and this shortening is accelerated during early development and by stressors including oxidative damage and stress hormone exposure [5-7 but see 8]. When telomeres shorten beyond a critical point, the cell enters replicative senescence, and accumulation of senescent cells can impair tissue function in later life when cell renewal capacity is reduced [9]. A number of studies have shown that telomere length or rate of loss predicts survival and longevity in vertebrates [10-12] including humans [reviewed in 13], and short telomeres are associated with the systemic loss of function frequently observed in ageing individuals [14]. Accelerated telomere loss early in life may therefore advance the onset of senescence, thereby linking early-life conditions with later-life health and survival.

Early-life adversity promotes telomere shortening in a range of species, including salmon, humans and several birds [15-19]. In birds, offspring competing with more rivals, or rivals higher in the competitive hierarchy, exhibit accelerated telomere loss [20-25]. Studies of the consequences of offspring competition on telomere dynamics have thus far focussed almost exclusively on biparental species; the importance of early-life competition in species with other social systems therefore remains unclear.

In animal societies where multiple females breed the effects of early-life competition on telomere lengths are likely to be particularly pronounced, because the number of competing offspring is expected to be higher and competition therefore more intense. Where females breed asynchronously, greater age asymmetries between offspring will likely further exacerbate telomere loss for offspring that are younger or lower in the competitive hierarchy [23, 25]. Alternatively, sharing offspring care between multiple females may buffer offspring against unpredictable environments [26] and improve growth and health [27], thus relaxing competition and slowing telomere attrition. Whether the effects of early-life competition on the rate of telomere attrition in animal societies are exacerbated by increased offspring number, or mitigated by cooperative care of young, remains unknown.

Where early-life adversity promotes the accumulation of ageing-related damage and poor telomere integrity, we would predict that selection would favour parental strategies that protect offspring, either by improving the environment or enhancing offspring resilience to adversity. Despite extensive evidence that early-life adversity is reflected in enduring deleterious effects on telomere lengths [15-20], and that short telomeres are linked with poor health and curtailed survival [12, 14, 28, 29], little is known about parental strategies associated with slowed offspring telomere attrition, and how effective they are [30].

Here, we investigate whether early-life adversity, in the form of intense pup competition, is associated with shortened telomeres in wild Kalahari meerkat pups at emergence from the natal burrow. Meerkats (*Suricata suricatta*) live in stable cooperatively breeding groups of up to 50 individuals. Reproduction is largely monopolised by a single dominant female, but older subordinate females also attempt to breed at a lower frequency [31]. Mean litter size is 4.1 pups (range 1-8) [32]. Mixed litters are suckled indiscriminately by all lactating females [32], and pups therefore compete both with their littermates and with pups from other litters. Previous research suggests that pups compete for access to milk before emerging from the natal burrow, as experimental contraception of subordinate females leads to increased growth of the dominant's pups at emergence from the birth burrow [33]. Pups are also frequently observed aggressively competing for access to provisioning helpers after emergence [34]. After investigating whether variation in the number of competing pups affects their telomere lengths, we test whether supplementing the mother's food intake during gestation and lactation mitigates the effects of competition on pup telomeres. We then investigate whether early-life telomere lengths predict survival into adulthood. Finally, we explore the extent to which mothers reduce pup competition by killing litters born to other females, and discuss how this strategy might impact telomere dynamics in her own pups. Such infanticide is common in meerkat groups, and is almost always perpetrated by heavily pregnant females [35].

Methods

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 2015. All meerkats were habituated to close observation (<1m) and individually recognizable using small dye-marks (ca. 2cm^2, for adults and older pups) or trimming small patches of fur (ca. 0.5cm^2, for newly-emerged pups) [36]. Virtually all (>95%) meerkats could be voluntarily weighed on electronic scales (± 0.1g, Durascale, UK) before they commenced foraging in the morning, at midday and after sunset. Groups were visited 2-3 times per week to collect behavioural, life-history and body weight data. Observations of pregnancy, birth, infanticide, dominance, group size and rainfall were made using protocols detailed elsewhere [36, 37]. Mother and father identity were assigned genetically [38, 39].

Pup tail tip sampling

Meerkats are born in an underground burrow, emerging for the first time at age 3-4 weeks. Shortly after the litter’s first emergence, a small biopsy of skin from the tail-tip was collected from each pup (age 28.3 ± 3.4 days) for the determination of telomere length and parentage [39]. Skin samples were immediately transferred to 96% ethanol and stored at -20°C until DNA extraction.

Supplementary feeding experiment

To investigate the effects of early nutritional environment on telomere lengths, we fed pregnant females during gestation and lactation. In order to minimize inter-individual differences in body condition, our experimental procedure was limited to dominant females. The supplementary feeding protocol consisted of one hard-boiled egg per day (divided equally between the morning and afternoon observation sessions) commencing six weeks after the end of a dominant female’s pregnancy, and continuing until the next parturition [40]. Thereafter, fed dominant females received four eggs per week, until the pups were weaned. This feeding protocol occurred between August and November in 2011 and 2012. Control females were pregnant during the same period, and did not receive supplemental food.

Observations of Infanticide

We investigated how infanticide by dominant females affects the number of competing pups, and the likely consequences for telomere lengths in her own litter. While previous analyses of the distribution of infanticide have focussed on consequences for the victim mother (i.e. whether her litter survives or is killed [35, 37]), we quantified the *benefits* of infanticide for the perpetrator (i.e. how many competitor pups she removes). We identified periods when the dominant female is most likely to kill pups born to other females (the 30 days prior to her own parturition, hereafter termed 'high infanticide period') and least likely (the 30 days immediately after giving birth, hereafter termed 'low infanticide period') [27]. We then assessed subordinate litter survival probabilities and the total number of subordinate pups surviving to emergence during these two periods. Parturition for all females could be identified by sudden weight loss and change in body shape [36], and pup production for each period was measured as the number of pups born that survived to emergence from the birth burrow.

qPCR determination of telomere lengths

We used quantitative PCR (qPCR) analysis to measure telomere length in skin samples, based on published protocols with some modifications [41, 42]. This measure represents the average telomere length across cells in a sample, and is reported as the level of telomeric sequence abundance relative to a reference non-variable copy number gene (T/S ratio). Further details of DNA extraction and qPCR analysis can be found in the supplementary methods.

Statistical analysis

Statistical analyses were carried out in R version 3.2.3, using a step-wise model simplification approach [43, 44]. Initially all fixed 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 maximum log-likelihood estimation), until the minimal adequate model (MAM) was obtained, in which only significant terms remained. Dropped terms were then added back in to the MAM to confirm their non-significance. The homoscedasticity and normality of residuals were confirmed by visual inspection, and all continuous predictors were scaled to a mean of 0 and standard deviation of 1. 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). Analysis using Akaike's information criterion correcting for small sample size (AICc) and inspection of the top model set (for which AICc differed by < 2) yielded qualitatively identical results [45]. We ran three sets of statistical models, first to investigate the determinants of pup telomere lengths in the large correlative dataset, second to investigate how experimental supplementary feeding of mothers impacted pup telomere lengths, and third to investigate the consequences of infanticide for pup competition.

1) What are the determinants of pup telomere lengths?

Our primary interest was the effect of the number of competing pups on telomere lengths at emergence from the natal burrow. For each sampled pup, we assessed the number of rival pups (aged under 90 days) present in the group, every day between the focal pup's birth and day of sampling for telomere length. The average of these daily rival counts represents our measure of overall competition experienced by the focal pup prior to sampling, hereafter termed 'pup number'. This estimate of pup competition includes littermates and pups from older and younger litters born to the dominant female and subordinate females.

We controlled for maternal factors that may influence offspring quality, including weight at conception, age (mean 4.9 years, range 1.2 - 8.0) and dominance status (dominant or subordinate) [46]. Social group size (average number of adult group members calculated as above for pup number) and rainfall (mm) in the month before birth can also both influence offspring quality [47]. Pup sex (male, female or unknown) and age at capture were also controlled for. We included these individual, maternal, environmental and social predictors, with our estimate of pup number, in a general linear mixed effects model (GLMM), with pup telomere length as the response. Cohort year, group ID, mother ID and litter ID were included as random terms, to account for the non-independence of pups within years, groups, mothers, and litters. Telomere lengths were available for 230 pups from 63 litters in 13 groups, born between 2009 and 2012. We also tested the effect of paternal age (mean 4.1 years, range 1.4 - 6.1) on pup telomere lengths in a reduced dataset for which the father's date of birth could be accurately determined (78 pups from 23 litters in 7 groups).

2) Does an experimentally improved nutritional environment mitigate the effects of pup number?

To test the effect of supplementary feeding of the pregnant and lactating mother on pup telomere lengths, we included experimental treatment (fed/control) as a two-level factor in a GLMM, with pup telomere length as the response and litter ID as the random term. Given our smaller sample size for the experimental dataset, only terms found to be significant in the larger correlative model were included, and two-way interactions between these and treatment. Telomere lengths were available for 25 pups from 8 litters in each treatment.

3) Do pup telomere lengths predict survival to adulthood?

We investigated whether pup telomere lengths predicted survival to adulthood (1 year old). Sub-adult meerkats do not disperse [31, 48], and any disappearance from the group before reaching adulthood is therefore likely to reflect mortality. We removed any individuals dying before reaching nutritional independence (90 days), as death at this early stage typically occurs due to starvation, predation, or becoming separated from the group, these sources of mortality are unlikely to reflect variation in telomere lengths. We used a binary term for survival to adulthood as the response in a binomial mixed effects model. We included pup telomere length as a predictor. We also controlled for other predictors known to influence telomere lengths and survival in young meerkats: sex, group size, rainfall, maternal dominance status, maternal age [47]. We controlled for the effects of pup body weight on survival, by including their bodyweight at age 40 days in the model. Group ID, mother ID and litter ID were included as random terms. This model was fitted to a dataset of 178 individuals: 161 pups from 51 litters born to dominant females, and 17 pups from 7 litters born to subordinates. The maximum confirmed lifespan for meerkats in our population is 12.2 and 12.4 years, for males and females respectively.

4) How does infanticide affect the number of competing pups?

We contrasted the fates of subordinate litters born in periods of high and low dominant female infanticide. First, for each dominant female parturition (n = 158), we counted subordinate parturitions during the two periods (30 days before and after dominant parturition). Infanticide typically takes places shortly after birth, so we classed each subordinate parturition as a ‘success’ or ‘infanticide’ according to whether the litter survived its first two days (litter loss after this point is more likely to be due to starvation or predation [35, 37]). Although new-born litters remained in the burrow for up to four weeks, their survival could be recorded daily by observing whether the group continued to leave baby-sitters during foraging trips [35]. The number of successes and infanticides were then used as the response term in a binomial mixed effects model, with the high/low infanticide period fitted as a two-level predictor. The random terms were dominant female pregnancy ID, dominant female ID and group ID. Second, for each dominant female parturition, we calculated the *total number* of emerging subordinate pups born during the two infanticide periods, and fitted this as the response term in a GLMM with a Poisson distribution. The main predictor of interest was the two-level high/low dominant female infanticide period, and we controlled for the number of subordinate females giving birth.

Results

1) What are the determinants of pup telomere lengths?

Male and female pups had similar telomere lengths at four weeks (χ^2_2= 1.14, p = 0.47, supplementary table 1). Pup telomere lengths were not associated with their mother's dominance status, the number of helpers in the group, the pup's age or the amount of rainfall in the month before their birth (all χ^2_1< 1.53, p > 0.23). Older mothers produced pups with significantly longer telomeres (χ^2_1= 9.57, p = 0.002, Fig 1 a). There was a trend for lighter mothers to produce pups with slightly longer telomeres, but this was not statistically significant (χ^2_1= 3.56, p = 0.06). In contrast to the positive effect of maternal age, in a reduced dataset for which father age was known, older fathers tended to produce pups with shorter telomeres, although this association was not significant (χ^2_1 = 3.28, p = 0.07).

Controlling for the effect of mother's age, pup telomeres were significantly shorter when the number of competing pups was high (χ^2_1= 5.55, p = 0.018, Fig 1b): telomeres were 13.3% longer when pup number was lowest compared to highest.

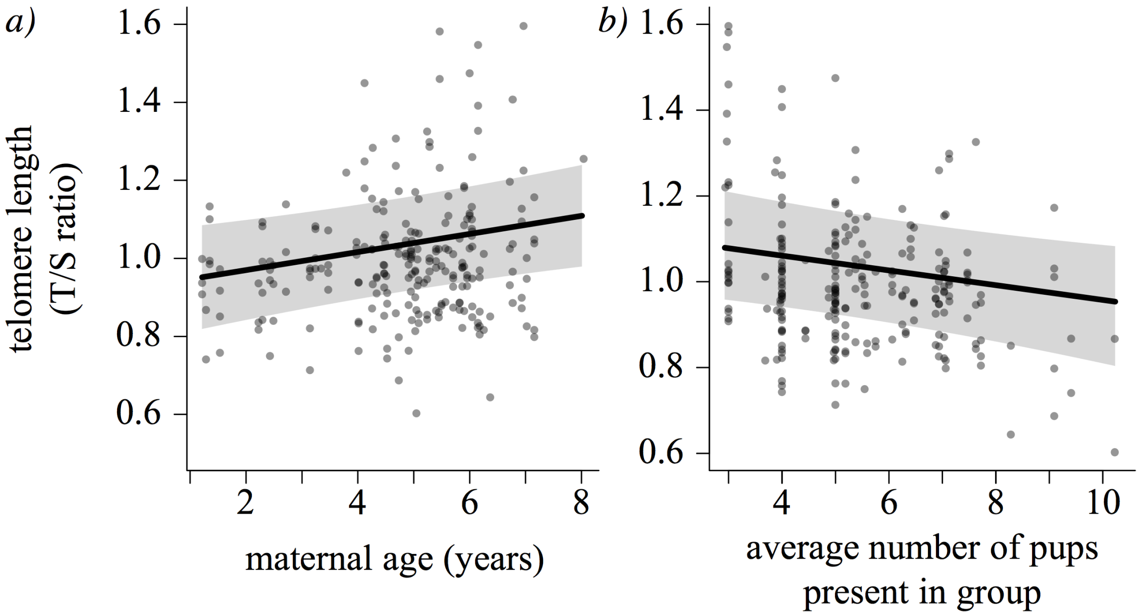

Figure 1 a) the positive association between maternal age and pup telomere length at emergence from the natal burrow. The line represents the model predictions from a GLMM, with an average pup number of 5.43. **b)** The negative association between the number of competitors a pup encounters in the first weeks of life, and its telomere length at emergence from the natal burrow. The line represents the model predictions from a GLMM, with an average maternal age of 4.86 years. In both figures, the points represent raw data, which are translucent for clarity. Shaded areas represent the 95% confidence intervals of each model prediction.

2) Does an experimental feeding of mothers mitigate the effects of pup number?

The effect of maternal supplementary feeding on pup telomere lengths was evident as a significant interaction between experimental treatment and pup number (χ^2_1= 16.47, $p < 0.001$, Fig 2, Supplementary table 2). While control pups had shorter telomeres under greater pup competition, no similar pattern was observed in pups from fed mothers. In contrast to our larger, correlative dataset, in our restricted experimental dataset, pup telomere lengths were not significantly affected by maternal age, either as a single term or in the interaction with treatment (both $\chi^2 < 2.23$, $p > 0.14$). Retention of the non-significant maternal age in the model did not qualitatively change the results.

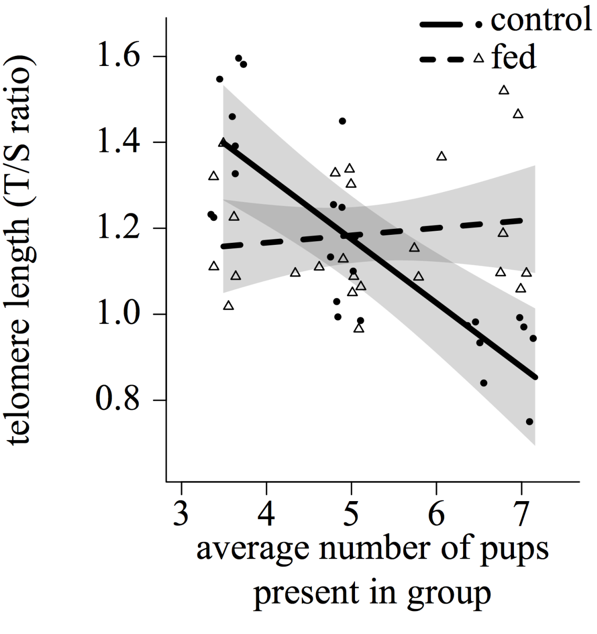

Figure 2 the effect of experimental maternal feeding (during gestation and lactation) on pup telomere lengths is dependent on the number of competitor pups. In control litters (filled points and solid line) there is a negative relationship between the number of pups and telomere lengths, while in litters from mothers receiving supplementary feeding (open triangles and dashed line) this negative association disappears. Lines represent model predictions for a mean maternal age of 4.5 years, from a GLMM with telomere length as the

response, and maternal age, experimental treatment, and the interaction between treatment and number of pups. Shaded areas represent the model's 95% confidence intervals for each model prediction line. Points represent raw data, and are jittered on the x-axis for clarity.

3) Do pup telomere lengths predict survival to adulthood?

A pup's probability of survival to adulthood was positively predicted by its weight (χ^2_1= 16.24, p < 0.001, Supplementary table 3) and its mother's age (χ^2_1= 4.88, p = 0.027). In this dataset, pups born to dominant females were less likely to survive to adulthood compared to those born to subordinates (χ^2_1= 14.03, p < 0.001), however this may be driven by poor data availability for subordinates: only 17 pups (9% of this dataset) were born to subordinates. Pups were less likely to survive in larger groups (χ^2_1= 4.15, p = 0.042). Controlling for these significant effects, pups with longer telomeres were significantly more likely to survive to adulthood (χ^2_1= 17.93, p < 0.001, Figure 3). Survival to adulthood was not significantly predicted by pup sex or rainfall (both χ^2_1 < 0.82, p > 0.36).

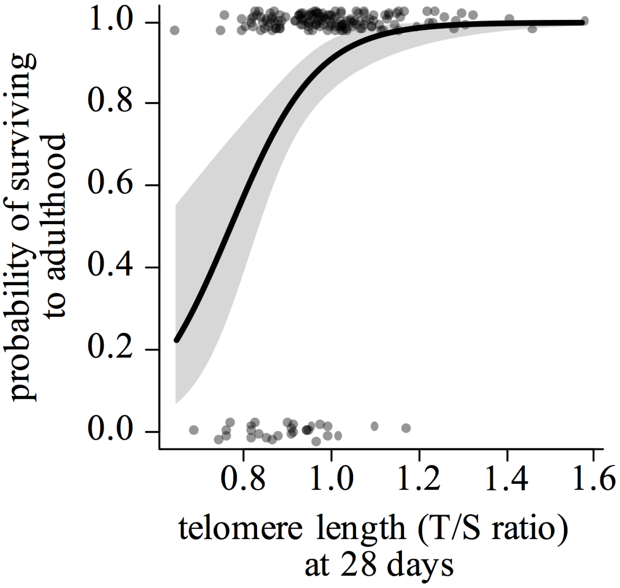

Figure 3 The positive association between pup telomere length and survival to adulthood. The line represents the model predictions from a GLMM, for a pup with a dominant mother and all other significant predictors at their mean (pup weight: 230g , maternal age: 4.8 years, group size: 19.2). The points represent raw data (jittered on the y-axis for clarity) and the shaded areas are 95% confidence intervals of the model predictions.

4) How does infanticide affect pup competition?

Pregnant dominant females commonly kill pups born to subordinate females shortly after they are born [35]. The probability of a subordinate litter surviving its first two days was 9.4% if it was born during the high dominant female infanticide period (the 30 days before dominant female parturition), compared to 71.6% during the low dominant female infanticide period (the 30 days after dominant female parturition) (χ^2_1= 118.57, $p < 0.001$, Fig. 4 a, Supplementary table 4i).

After controlling for the number of subordinate females giving birth (χ^2_1= 41.38, $p < 0.0001$, Supplementary table 4ii), infanticide by the pregnant dominant female significantly reduced the number of subordinate pups surviving to emergence (χ^2_1= 107.36, $p < 0.0001$, Fig 4 b), and the number of surviving non-littermates that pups born to the dominant female had to compete with fell from a median value of 4 to zero. This suggests that the infanticidal behaviour of dominant females leads to a substantial reduction in pup competition for her own litter. From our analysis of the effects of pup number on telomere lengths, we estimate that the removal of 4 rival pups is likely to be associated with a 7.3% increase in telomere lengths for the dominant female's pups.

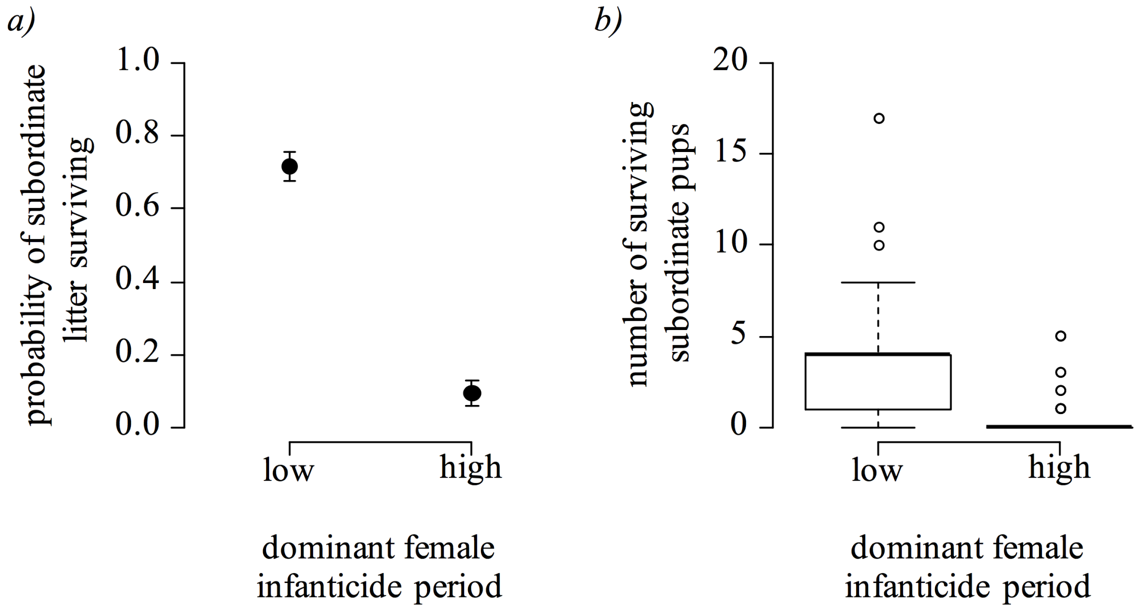

Figure 4 a) the effect of dominant female infanticidal behaviour on the probability of subordinate litters surviving their first two days of life. Points represent predicted means and standard errors from a binomial mixed model with dominant female infanticide risk as the only predictor. **b)** The effects of infanticide by dominant females on the total number of surviving pups produced by all subordinate females in their group.

Discussion

Our results show that early-life competition is associated with shortened telomeres in wild meerkats, and that this effect is evident by the time pups leave the natal burrow at approximately 28 days old. The detrimental effect of competition on pup telomere lengths disappears when mothers are given supplementary food during gestation and lactation, suggesting that the reduction in telomere lengths in pups facing more rivals is a consequence of competition for food. Pups with short telomeres show a lower probability of survival to adulthood. Dominant females can reduce pup competition through killing other pups born in their group at times while they are pregnant, with likely benefits for their own pups' telomere lengths and survival.

Our findings from a wild, social mammal extend evidence that in several biparental bird species, offspring competing with more rivals, or rivals higher in the competitive hierarchy, exhibit accelerated telomere loss [20-25]. There are several non-mutually exclusive explanations for the association between high numbers of competing offspring and shortened telomeres in meerkats. Under greater competition, offspring typically expend more energy gaining access to food (either through elevated begging, or competing to access food [49-51]). Early-life adversity can also trigger physiological stress mechanisms, which confer short-term benefits, but are costly in the long-term, as they allocate resources to immediate survival at the expense of somatic maintenance [52]. Greater competition can also promote accelerated growth profiles, whereby offspring grow faster than their optimal developmental rate in order to out-compete rivals [53]. Aggressively competing to access food, physiological stress cascades and accelerated growth can all lead to elevated oxidative damage and accelerated telomere attrition [54-58 but see 8].

Our experimental results show that high numbers of competing pups are no longer associated with reduced telomere lengths when maternal food intake is increased during gestation and lactation. Previous evidence suggests that experimental food supplementation of weaned meerkat pups reduces aggressive pup competition [59], and that a relaxed early-life competitive environment slows telomere shortening in other species [23]. Increased maternal weight during gestation is positively associated with meerkat pup weight at weaning [46], suggesting that heavier females are better able to provision their young, leading to reduced early-life competition. Pups from experimentally fed mothers may therefore exhibit longer telomeres due to a relaxed early competitive environment, arising from improved pup quality at birth (thus enhancing pup competitive ability), or elevated maternal milk yield or micronutrient content (thus reducing the pups' need to compete for food) [60].

Our finding that variation in offspring telomere lengths is associated with maternal and paternal age should be interpreted with caution. Pups born to older mothers had longer telomeres, and while a female could provide better care for offspring as she grows older, it is equally possible that early mortality of poor-quality females leads to disproportionate numbers of high-quality females in older cohorts [61, 62]. The positive effect of maternal age on pup telomere lengths may therefore be due to selective disappearance, rather than within-female change. Furthermore, maternal age at conception is unrelated to offspring telomere length in other mammals [63]; whether subsequent litters of pups have longer telomeres as the mother grows older is therefore unclear. We also find that paternal age has a weak negative effect on pup telomeres lengths. This result is surprising, given that paternal age at conception is typically positively associated with offspring telomeres (e.g. in humans [63] and chimpanzees (*Pan troglodytes)* [64]). It is possible that older male meerkats lose condition faster than humans or chimpanzees, with concomitant decreases in sperm and pup telomere lengths, but further work would be needed to clarify the role of paternal age at conception in meerkat telomere dynamics.

Shortened pup telomeres following early-life competition may be associated with significant fitness costs, given our finding that reduced telomere lengths predicted low survival to adulthood. Short telomeres and rapid telomere attrition are associated with reduced survival and curtailed longevity in a number of species, both in captivity and in the wild [10-12, 28]. Given that we found short telomeres were linked to reduced survival during meerkats' first year of life, this likely does not reflect accelerated senescence, as senescence is typically only manifest after meerkats reach three years old [65]. Similarly, early-life telomere dynamics are linked with survival during the first years of life in other wild mammals [29], suggesting that telomeres are not only linked with ageing-related mortality, but provide an integrative bio-marker of somatic damage which can be associated with mortality at any age [66]. Whether telomere dynamics in adult meerkats are predictive of age-related mortality requires further investigation.

Our results suggest that infanticide by dominant females leads to marked reductions in the number of competitors faced by their own litters. Previous evidence suggests that experimental reductions of pup number, either by temporary pup removal or contraception of subordinate females, leads to increased weight gain in the remaining pups [33, 67]. Heavier pups are subsequently more likely to survive to adulthood and acquire dominance [67, 68], suggesting that this accelerated growth does not exceed the optimal growth rate, and therefore confers little costs. By eliminating rival offspring, dominant females are therefore likely to improve the condition, survival and probability of dominance acquisition of their own litters.

Our findings highlight a further potential benefit of infanticide: removal of competitor pups may be associated with a significant increase in pup telomere lengths. Longer telomeres are associated with improved early-life survival in meerkats, and later-life benefits including delayed senescence and improved longevity in a number of other species [10-12, 14]. Such later-life benefits may be particularly important in meerkats: in dominants (who monopolise reproduction), the primary determinant of lifetime reproductive success is dominance tenure length [69]. Dominants of both sexes exhibit senescence and their tenure typically ends when they are unable to repel same-sex challengers [36, 39, 65, 70]. In addition to the above benefits for offspring condition and dominance acquisition, infanticide may therefore allow dominant females to improve pup telomere lengths, thus delaying their onset of senescence, extending their dominance tenures and increasing their lifetime reproductive success. While the level and type of parental care has been shown to influence offspring telomere lengths in humans and captive rhesus monkeys [30, 71, 72], to our knowledge this is the first evidence that a specific maternal strategy (killing competitor pups) has associated benefits for offspring telomere lengths.

Conclusion

Our results suggest that in a social species, where offspring competition may be particularly pronounced, an unfavourable early-life competitive environment accelerates telomere loss under natural conditions, with potentially lifelong consequences [12]. Despite the observed enduring detrimental effects of early-life adversity on telomere dynamics [20-25, 28, 58], and the clear selection pressure this places on parents, few studies have investigated whether parents are able to protect offspring telomeres by improving the early environment. In meerkats, dominant females kill rival litters to reduce competition for their own pups, resulting in improved pup condition and likely benefits for telomere lengths and longevity. Overall, our results highlight that both the early environment and protective parental strategies may affect offspring telomere lengths, and without detailed consideration of both we are likely to underestimate the role of telomere dynamics in shaping life-histories, ageing profiles and fitness.

Ethics statement

Our work was approved by the Animal Ethics Committee of the University of Pretoria, South Africa (no. EC010-13) and by the Northern Cape Department of Environment and Nature Conservation, South Africa (FAUNA 1020/2016).

Data accessibility

All data used in analyses and figures are included in the electronic supplementary material.

Competing interests

We declare we have no competing interests.

Author contributions

This study was designed by D.L.C. and T.H.C.-B.; P.M and R.G. planned and implemented the laboratory analyses and advised on interpretation of telomere data. D.L.C. planned and implemented the statistical analyses; D.L.C. and T.H.C.-B. wrote the paper, with extensive advice from P.M. and R.G.. All authors contributed to the manuscript, approved the final version and are accountable for the work.

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