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Additive genetic variance and developmental plasticity in growth trajectories in a wild cooperative mammal

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Abstract

Individual variation in growth is high in cooperative breeders and may reflect plastic divergence in developmental trajectories leading to breeding vs. helping phenotypes. However, the relative importance of additive genetic variance and developmental plasticity in shaping growth trajectories is largely unknown in cooperative vertebrates. This study exploits weekly sequences of body mass from birth to adulthood to investigate sources of variance in, and covariance between, early and later growth in wild meerkats (*Suricata suricatta*), a cooperative mongoose. Our results indicate that (i) the correlation between early growth (prior to nutritional independence) and adult mass is positive but weak, and there are frequent changes (compensatory growth) in post-independence growth trajectories; (ii) among parameters describing growth trajectories, those describing growth rate (prior to and at nutritional independence) show undetectable heritability while associated size parameters (mass at nutritional independence and asymptotic mass) are moderately heritable ($0.09 \leq h^2 < 0.3$); and (iii) additive genetic effects, rather than early environmental effects, mediate the covariance between early growth and adult mass. These results reveal that meerkat growth trajectories remain plastic throughout development, rather than showing early and irreversible divergence, and that the weak effects of early growth on adult mass, an important determinant of breeding success, are partly genetic. In contrast to most cooperative invertebrates, the acquisition of breeding status is often determined after sexual maturity and strongly impacted by chance in many cooperative vertebrates, who may therefore retain the ability to adjust their morphology to environmental changes and social opportunities arising throughout their development, rather than specializing early.

Introduction

Individual variation in growth is unusually high in cooperative breeding societies that are characterized by morphological divergence in adults, where breeders are larger and heavier than helpers (Wilson, 1971; O'Riain & Jarvis, 1998; O'Riain *et al.*, 2000; Clutton-Brock, 2009). In eusocial insects with castes, workers are sterile and provision extraordinarily fecund queens that are much larger and live much longer than them. Divergent morphological development of queens and workers starts shortly after hatching and is often irreversible and triggered by early nutritional differences (Wheeler, 1986; Beekman *et al.*, 2006). Such divergence is thought to reflect developmental plasticity, here defined as environmentally induced variability during early development within a single genotype (Piersma & Drent, 2003), with long-lasting phenotypic effects.

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Less is known regarding mechanisms responsible for the emergence of rank-related size differences commonly observed in cooperative vertebrates (O'Riain *et al.*, 2000; Heg *et al.*, 2004; Russell *et al.*, 2004; Wong *et al.*, 2008; Young & Bennett, 2010). The best example of alternative morphotypes in cooperative vertebrates comes from the naked mole-rat *Heterocephalus glaber* (O'Riain *et al.*, 2000; Dengler-Crish & Catania, 2007), a subterranean rodent where the queen monopolizes reproduction in a large colony over many years (Jarvis, 1981; Bennett & Faulkes, 2000) and is much larger and heavier than same-sex subordinates (O'Riain *et al.*, 2000). Fast growth may facilitate dominance acquisition, as females fight fiercely over dominance and success may be size dependent (Clarke & Faulkes, 1997). Similarly, field studies of meerkats, a cooperative carnivore, suggest that individual variation in growth may reflect wider life-history trajectories: heavier females have better chances of reaching dominance and of breeding successfully (Clutton-Brock *et al.*, 2006; Hodge *et al.*, 2008), and early growth predicts future chances of acquiring breeding status (Russell *et al.*, 2007; English *et al.*, 2013b). Consequently, individuals may, as in cooperative insects, specialize early in life as a result of developmental plasticity and follow different developmental pathways leading to breeding vs. helping phenotypes.

However, it is also possible that cooperative vertebrates may avoid early specialization and retain the ability to adjust their developmental trajectories to short-term variation in their ecological or social environment. First, they live longer than invertebrates and may often face a greater range of environmental conditions over their lifetime. Second, breeding positions are generally acquired late, often after sexual maturity, and their inheritance is often largely impacted by chance. For example, in meerkats, breeding vacancies often go to the oldest subordinate female of the group (Hodge *et al.*, 2008). Finally, recent evidence indicates that there is no early behavioural specialization in meerkats, as investment in cooperative behaviour in early life does not predict ultimate breeding status (Carter *et al.*, 2014), suggesting that there is no general syndrome of early phenotypic specialization encompassing behaviour, morphology and life history. As a result, the development of phenotypic specialization in cooperative vertebrates and invertebrates may profoundly differ, and the role of developmental plasticity may be weaker in more 'totipotent' vertebrates.

Understanding the evolution of morphological divergence in cooperative breeding vertebrates requires identifying the factors underlying variation in early growth, as well as the mechanisms connecting early growth performance with later fitness-related traits such as body mass. Early growth is highly sensitive to environmental variation and particularly to maternal environment (e.g. Maestripieri, 2011). For example, studies in wild red squirrels (*Tamiasciurus hudsonicus*) suggest that maternal environment can account for up to 80% of the variance in early growth (McAdam *et al.*, 2002). Although maternal effects on growth may be weaker in cooperatively breeding societies where they may be offset by helper care (Holekamp & Dloniak, 2009; Russell & Lummaa, 2009), studies of wild cooperative carnivores indicate that other sources of variations in the early social environment, like the size of the helping cohort, may play a crucial role in regulating growth, especially during the provisioning period (reviewed by Holekamp & Dloniak, 2009). The quality of the abiotic environment may also exert a large influence on early growth variation. In Damaraland mole-rats (*Cryptomis damarensis*), seasonal fluctuations in resource availability may mediate divergent developmental trajectories, as pups from early born litters grow faster, have higher asymptotic weight and contribute less to cooperative activities than pups from later litters (Bennett & Faulkes, 2000; Bennett & Navarro, 2009).

Early determination of growth trajectories can also reflect genetic variation. Additive genetic variance in growth is substantial in many vertebrates, as growth rates have been successfully targeted by animal breeding programmes (e.g. Hernández *et al.*, 2004; Nkrumah *et al.*, 2007; Elzo *et al.*, 2012) and biomedical studies have revealed substantial additive genetic variance in children's growth trajectories (Choh *et al.*, 2011; Johnson *et al.*, 2011). Studies on wild vertebrates, including on some cooperative breeders, have shown that the additive genetic variance of morphometric traits measured at various developmental stages is often significant and substantial (e.g. Réale *et al.*, 1999; Wilson *et al.*, 2005, 2006; Nielsen, 2013), suggesting that growth may retain a genetic component even in natural populations where environmental variation is extensive.

Detecting genetic variance consistently across life stages in the developmental sequence of a trait does not, however, imply that genetic variance is primarily responsible for the phenotypic covariance observed in this trait across life stages. Instead, measuring the genetic and environmental components of the covariance between early growth and later growth may help to determine whether an early growth effect on adult size is primarily a consequence of genetic variation or plasticity, respectively. Whereas no study has examined genetic and environmental sources of covariance between early and later growth in wild vertebrates, some studies have examined genetic sources of covariance in body mass or size at various developmental stages and indicate that genetic correlations across ages are generally strong and positive (Cheverud *et al.*, 1983; Kirkpatrick & Losvold, 1992; Wilson *et al.*, 2006), suggesting that genetic effects contribute to mediate the covariance between early and later mass or size.

However, sources of covariance between early and later growth may differ from sources of covariance among size or mass at different ages, as growth during a given developmental window may be less affected by carry-over effects from previous stages than age-related size. Recent studies of eusocial insects suggest that the quality of early environment may determine alternative growth trajectories corresponding to different social castes through developmental plasticity and epigenetic effects (Drewell *et al.*, 2012; Weiner & Toth, 2012). In cooperative vertebrates, we may expect that under a similar scenario, any phenotypic covariance observed between early growth and parameters characterizing subsequent growth trajectories, especially adult size, would be mediated by early environmental (rather than additive genetic) effects.

This study aims at quantifying the relative importance of genetic and environmental sources of variance in, and covariance between, early growth rate (EGR) and later growth trajectories of wild cooperative meerkats inhabiting the Kalahari Desert. Despite living in a highly seasonal environment, meerkats breed throughout the year. Reproduction is largely monopolized by the alpha pair, and group size ranges from 3 to 50 individuals (Clutton-Brock, 2009). Pups are provisioned by helpers for approximately 2 months following their emergence from the natal burrow, about 1 month after birth (Clutton-Brock *et al.*, 2001; Clutton-Brock, 2009), and recent studies have shown that abiotic (rain and season), maternal (age and rank) and social (number of helpers and littermates) factors affect individual growth trajectories (Russell *et al.*, 2002; English *et al.*, 2012, 2013a). Finally, morphological traits (body mass and skeletal size) show moderately heritable variation that increases with age (Nielsen, 2013).

We examine several genetic and environmental (group, maternal, litter identity and birth year) sources of variance in and covariance between EGR (during the provisioning period) and each of three parameters describing the growth curve between nutritional independence and adulthood (body mass at nutritional independence, growth rate at nutritional independence and asymptotic body mass). We use a high-resolution longitudinal and multigenerational phenotypic data set based on weekly weight measures together with quantitative genetic models to ask three questions. First, do individual growth trajectories diverge early in life? If so, we expect a positive phenotypic covariance between EGRs and subsequent growth parameters. Second, is individual variation in growth trajectories heritable? If so, we expect a significant additive genetic variance in growth parameters. Finally, are additive genetic effects responsible for any early divergence in growth trajectories? If so, we expect the phenotypic covariance between early growth and subsequent growth trajectories to be primarily driven by genetic, rather than by early environmental effects.

Materials and methods

Study species and population

Individual data were collected between 1998 and 2011 as part of a long-term study of a wild meerkat population at the Kuruman River Reserve in the Northern Cape, South Africa (26°580S, 21°490E). Further details on the study site and population are described elsewhere (Clutton-Brock *et al.*, 1998, 1999a; Russell *et al.*, 2002). Meerkats live in groups comprising 3–50 individuals, including a dominant breeding pair and subordinate individuals of both sexes (Clutton-Brock, 2009). Groups were visited on average three times per week in order to record life-history events (birth, death, immigration, emigration) as well as group composition. All individuals in the population were tagged with unique subcutaneous transponder chips and were identifiable in the field through dye marks on their fur. When they first emerge from the natal burrow (at approximately 3 weeks of age), a 2- to 5-mm tissue biopsy from the tail tip of each pup is taken for genetic analysis (Spong *et al.*, 2008). All sampled individuals have been genotyped at up to 18 variable microsatellite loci so that genetic data were available for 86% of the total recorded population (Nielsen *et al.*, 2012). Meerkats were fully habituated to human observers and were regularly (about twice per week) weighed to the nearest gram in the field using a portable balance, on which they were trained to climb in return for a small reward of water or crumbs of hard-boiled egg. Data used here were all collected in the morning, soon after meerkats emerge from their sleeping burrow, to avoid short-term fluctuations in mass owing to variation in foraging success. Growth trajectories presented here are exclusively based on longitudinal weight records, as additional developmental measures such as skeletal size were only collected opportunistically during captures (requiring anaesthesia), generating no or few repeated records per individual at heterogeneous points in time.

Rainfall was measured daily (in mm) using a standard rain gauge. When daily records were missing (< 1% of days), we used rainfall data from a remote-sensing data set provided by the NASA GES DISC (Goddard Earth Sciences Data and Information Services Center) Giovanni online data system (described in Acker & Leptoukh, 2007). Comparing the KMP records with the remote-sensing records on days where both were available indicated a strong positive correlation (Pearson's correlation, $r_p = 0.71$, d.f. = 1481, $P < 10^{-4}$).

Modelling growth

Our modelling approach followed previous work (English *et al.*, 2012) in this population, which compared the fits of several standard growth curves (monomolecular, von Bertalanffy, Gompertz, logistic, and Richards)

described by Gaillard *et al.* (1997) and Zullinger *et al.* (1984) to long-term changes in meerkat body mass, identifying the monomolecular model as the best description of meerkat growth trajectories. The extent to which environmental parameters, such as rainfall and season, may affect variation in meerkat body mass was investigated, revealing that incorporation of accumulated rainfall over 9 months, as well as seasonality, substantially improved the growth curve fits. Finally, this previous study also revealed that fitting a biphasic monomolecular model, accounting for differential growth before and after nutritional independence, substantially improved the model fit, thereby suggesting that processes influencing growth differ across these periods. Based on these findings, we chose to quantify growth before and after nutritional independence using distinct models, to ensure that parameter estimates for growth before and after nutritional independence may not directly constrain or influence each other during model fitting, allowing us to examine their biological covariance.

Early growth (1–3 months)

We calculated growth rates between 1 and 3 months for 1329 individuals born between 1998 and 2011, including 702 males and 627 females. The mean number of weight measures available per individual between 0 and 2 months was 13.3 ± 7.0, and between 2 and 4 months was 32.9 ± 15.5. To limit error due to missing data or variation in sampling effort, we calculated an interpolated monthly mass measure for each individual at 1 and 3 months of age (see Ozgul *et al.*, 2010 for a similar approach). As weight is a simple quadratic function of age between 1 and 3 months of age (see Fig. S1), this measure was calculated by fitting linear mixed-effect models, for all individuals, to mass measurements for 1 month before and after each monthly age, with age and age^2 as fixed effect terms, and individual as a random term. These models were then used to predict each individual's weight for its exact monthly age. Early growth was then computed as the difference between weight predictions obtained at one and at 3 months of age, providing a single-parameter measure that was recently shown to predict a key fitness component in this population, the probability to acquire breeding status (English *et al.*, 2013b).

Lifetime growth (3–24 months)

To quantify lifetime growth in meerkats, we analysed mass data on individuals who survived at least 2 years (730 days) and modelled mass from 3 months until 2 years, the age by which most individuals have reached adult weight (English *et al.*, 2012). Individual mass time series were truncated at 2 years to ensure that individual differences in longevity would not affect the shape of the trajectories, although this does not ensure a random sample of individuals. We also excluded data for (i) pregnant females from their conception dates [approximately 70 days prior to parturition or 40 days prior to the first day of detectable pregnancy in cases where abortions occurred (Clutton-Brock *et al.*, 1998)] until birth or abortion (removing 2796 out of 145 673 mass records for 169 females: < 2% of data) and (ii) individuals with fewer than 80 data points within the considered time frame (n = 24), to ensure that growth parameters were estimated with accuracy. In total, we analysed mass data for 531 individuals (270 males, 261 females) with an average of 271 body mass records per individual (range 81–417).

We fitted a monomolecular model to body mass records of each individual in our data set in order to calculate nonlinear least square estimates of the parameters of a monomolecular model (Fig. 1) using the 'nls' function implemented in R 2.15.0. Parameter estimation was therefore independent from one individual to the next. We made several decisions in the implementation of this analysis, mainly based on the empirical questions addressed. We re-parameterized the monomolecular model as well as incorporated rainfall and season effects as formulated by English *et al.* (2012) when calculating individual growth parameters (see Appendix S1 for details). This procedure served, in effect, to correct the fitted mass for total rainfall over the preceding 9 months, as well as for the day of the year, in order to, first, increase the precision of individual parameter estimation by increasing the overall model fit and, second, to limit noise when examining genetic determinants of between-individual variation in growth by correcting for basic seasonal fluctuations affecting individual growth trajectories.

The final function used to model individual growth trajectories was thus as follows:

$$\text{Mass} = A - (A - m_0) \cdot e^{-k_0 \cdot (\text{age}-90)/(A-m_0)} + s \cdot \sin\left(\frac{2\pi(\text{date} - d)}{365.25}\right) + R \cdot \text{rain}$$

with A the asymptotic mass (Fig. 1), m_0 the initial mass (at 90 days of age), k_0 the initial growth rate (at 90 days of age), s the amplitude of the seasonal oscillation, d the time phase shift and R the rainfall effect estimate (for the last three parameters, we used estimates from English *et al.*, 2012; see Appendix S1). All these analyses were run using software R 2.15.0 (R Development Core Team 2012).

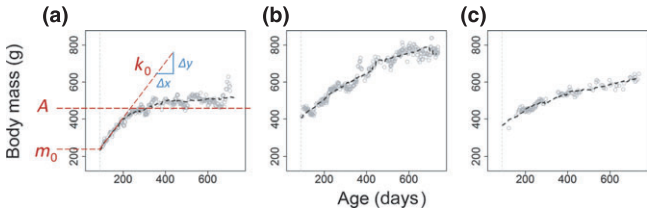

Fig. 1 Example growth trajectories for three individual meerkats (a–c). Individual mass records are indicated by grey dots and fitted growth curves by the black dotted line. An illustration of the three parameters of the re-parameterized monomolecular model is proposed in (a): m_0 represents body mass at the starting point of the curve (at the age of 90 days, as shown by the light grey vertical dotted line), k_0 represents the initial growth rate (at the age of 90 days) and is represented as a slope defined by $k_0 = \Delta y/\Delta x$, and A represents the asymptotic body mass.

Pedigree and inbreeding coefficients

The pedigree for this study population was reconstructed using a combination of microsatellite data and phenotypic descriptors and two parentage inference programs: COLONY2 (Wang, 2004; Wang & Santure, 2009) and MASTERBAYES (Hadfield *et al.*, 2006). Full details of the molecular and pedigree construction and structure can be found elsewhere (Nielsen *et al.*, 2012). In brief, the full pedigree contains 2147 individuals, spans up to eight generations, and contains 1531 individuals with both parents known and a further 411 individuals with only maternity known (see Table S1). The section of the full pedigree which is informative with respect to the analyses presented here ('pruned' pedigree) is described in Table S1. Wright's (1921) inbreeding coefficients (F) were calculated for each individual using PEDIGREEVIEWER (http://www-personal.une.edu.au/~bkinghor/pedigree.htm) (Kinghorn, 1994), under the assumption that all founders and immigrants were unrelated (Nielsen *et al.*, 2012).

Partitioning phenotypic variance in growth

Two growth parameters, k_0 and A, were log-transformed to satisfy normality assumptions. The phenotypic variance was then partitioned for each growth parameter by running a univariate animal model, a mixed-effect model allowing us to estimate additive genetic variance and heritability, the proportion of phenotypic variance explained by additive genetic variance (Kruuk, 2004), and fitted using restricted maximum likelihood with the R package ASReml-R. The univariate model investigating sources of variance on early growth included 1329 individuals for which early life data were available, whereas the three univariate models investigating sources of variance in later growth included 531 individuals for which lifetime growth trajectories were available. We minimized the number of fixed effects in the model in order to partition the variance of the raw phenotypes on which natural selection operates. We nevertheless included two fixed effects: sex and inbreeding coefficients. Sex was fitted as a fixed effect to account for potential sexual dimorphism in growth rates, whereas inbreeding coefficient was included as a fixed effect since some studies have suggested that these nonadditive genetic effects may occasionally interfere with estimates of additive genetic variance in traits suffering from substantial inbreeding depression (Reid *et al.*, 2006), which is the case for meerkat growth (Nielsen *et al.*, 2012). Variance components (random effects) were initially estimated from univariate animal models built as follows:

$$\boldsymbol{y} = \boldsymbol{Xb} + \boldsymbol{Z}_1\boldsymbol{a} + \boldsymbol{Z}_2\mathbf{bl} + \boldsymbol{Z}_3\boldsymbol{m} + \boldsymbol{Z}_4\mathbf{bg} + \boldsymbol{Z}_5\mathbf{by} + \boldsymbol{e}$$

where $\boldsymbol{y}$ = vector of observed phenotypic values; $\boldsymbol{b}$ = vector of fixed effects; $\boldsymbol{a}$ = vector of additive genetic effects; $\mathbf{bl}$ = vector of birth litter effects; $\boldsymbol{m}$ = vector of maternal effects; $\mathbf{bg}$ = vector of birth group effects; $\mathbf{by}$ = vector of birth year effects; $\boldsymbol{e}$ = vector of residual effects; $\boldsymbol{X}$ and $\boldsymbol{Z}_{1-5}$ = corresponding data-based design matrices.

For each trait, the phenotypic variance (V_P) that was unexplained by the fixed effects was thus partitioned as follows in the full model:

$$V_\mathrm{P} = V_\mathrm{A} + V_\mathrm{BL} + V_\mathrm{M} + V_\mathrm{BG} + V_\mathrm{BY} + V_\mathrm{R}$$

where V_P = total phenotypic variance; V_A = additive genetic effect variance; V_BL = birth litter effect variance; V_M = maternal effect variance; V_BG = birth group effect variance; V_BY = birth year effect variance; V_R = residual effect variance.

The proportion of variance attributed to each source of variance was then calculated by dividing the relevant variance component by the total phenotypic variance conditional on the fixed effects (e.g. heritability $h^2 = V_\mathrm{A}/V_\mathrm{P}$).

Although the significance of the fixed effects was evaluated by a F-test using a hypothesis-testing framework, we used Akaike Information Criterion (AIC) to select the models that best described the data while maintaining the lowest number of explanatory variables (parsimony principle) in the variance component analyses, as we had no *a priori* expectation regarding the

relative importance of the sources of variance and covariance. Two models differing by less than two units of AIC were considered to receive equal statistical support (Burnham & Anderson, 1998). The full set of alternative models considered is presented in Tables S2 and S3.

Next, we ran six bivariate models, each containing two of the four growth parameters [early growth, m_0, $\log(k_0)$, $\log(A)$] as response terms, aimed at simultaneously partitioning the sources of variance and covariance among those terms, in order to test whether observed phenotypic covariance among parameters is primarily of genetic or of environmental origin. These models were fitted on the intersection data set of individuals for which both early and later growth parameters had been estimated ($n = 523$). We initially attempted to fit trivariate models but these failed to converge. We included in bivariate models all random terms found to be significant in at least one of the two univariate models analysing variation in the two response terms. The parsimony of including a source of covariance (for additive genetic variance or a given environmental effect) between parameters in bivariate models was evaluated by comparing the AIC values of a model including this covariance with a model where the considered covariance was fixed to 0.

Results

Do growth trajectories diverge early in life?

Early growth rate significantly predicted subsequent growth trajectories, through a strong positive phenotypic correlation with estimated mass at nutritional independence m_0 (Pearson's correlation, $n = 531$, $r_s = 0.72$, $P < 10^{-3}$), a weak negative phenotypic correlation with growth rate at nutritional independence k_0 (Pearson's correlation, $n = 531$, $r_s = -0.11$, $P < 10^{-3}$) and an intermediate positive phenotypic correlation with asymptotic mass A (Pearson's correlation, $n = 531$, $r_s = 0.22$, $P < 10^{-3}$; Table 1, Fig. 2). Overall, individuals with a fast early growth ended up with a higher asymptotic mass, despite a negative correlation between early growth and $\log(k_0)$ that reflected the occurrence of post-independence compensatory growth for individuals that started with a slow early growth. Growth trajectories showed little differences between the sexes: males grew slightly faster than females shortly after nutritional independence as shown by a slight sex difference in $\log(k_0)$ ($F_{1,385} = 24.70$, $P < 10^{-3}$), but there was no sex difference for EGR, m_0 and $\log(A)$ ($P > 0.05$ in all cases, see Table 1). Finally, EGR was negatively affected by inbreeding ($F_{1,240} = 4.90$, $P < 10^{-3}$), as was

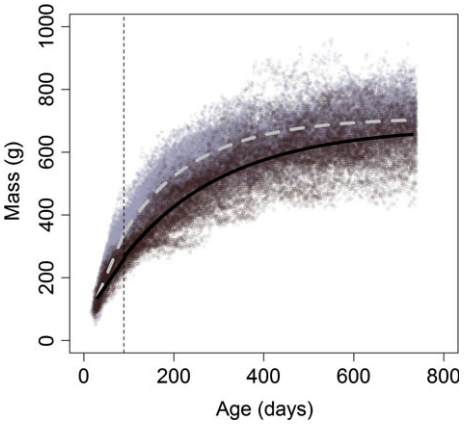

Fig. 2 Early growth rate predicts subsequent growth trajectories. Lighter grey dots correspond to weight records from individuals with fast early growth falling above the first quartile of the distribution of growth before nutritional independence. Darker grey dots correspond to weight records from individuals with slow early growth falling below the third quartile of the distribution of growth before nutritional independence. The grey dashed line represents the average growth rate before nutritional independence followed by the composite of the monomolecular mixed model after nutritional independence for the individuals with fast early growth, and the black solid line represents the average growth rate before nutritional independence followed by the composite of the monomolecular mixed model after nutritional independence for individuals with slow early growth. The vertical dashed line at 90 days denotes the approximate age of nutritional independence. Only individuals for which both early and later growth parameters were available ($n = 523$) are shown here.

Table 1 Mean values and correlations among growth parameters.

	EGR	m_0	log (k_0)	log (A)
Mean ± SD				
Males	3.12 ± 0.67	246.40 ± 57.16	0.55 ± 0.39	6.48 ± 0.15
Females	3.12 ± 0.59	251.72 ± 56.58	0.41 ± 0.36	6.48 ± 0.16
Correlations				
m_0	$r_s = 0.72$, $P < 10^{-3}$	–	–	–
$\log(k_0)$	$r_s = -0.11$, $P = 0.02$	$r_s = -0.42$, $P < 10^{-3}$	–	–
$\log(A)$	$r_s = 0.22$, $P < 10^{-3}$	$r_s = 0.39$, $P < 10^{-3}$	$r_s = -0.44$, $P < 10^{-3}$	–

EGR, early (pre-independence) growth rate (g/j); m_0, mass at independence (g); k_0, post-independence growth rate (g/j); and A, asymptotic mass (g).
Correlations were calculated using Pearson's correlations using 531 individuals.

mass at independence m_0 ($F_{1,344} = 5.48$, $P = 0.021$). Inbreeding effects were not detected later in life [$P > 0.05$ for log(k_0) and log(A)].

Are growth trajectories heritable?

Additive genetic variance was retained in the best univariate models for mass at independence m_0 and for transformed asymptotic mass log(A), but not for pre-independence or transformed post-independence growth rates EGR and log(k_0) indicating that the latter two parameters are not heritable (Tables 2 and S2, Fig. 3). The heritability of m_0 and log(A) was 17 ± 8% and 23 ± 9%, respectively. The effect of group and maternal identity was not significant in any of the models considered. In contrast, litter identity accounted for most of the phenotypic variance in early growth and m_0, for nearly half of the variance for m_0 and log (k_0) and for about one-quarter for log(A) (Table 2, Fig. 3). Birth year was present in the final model for each parameter and accounted for 5–30% of the phenotypic variance (Table 2, Fig. 3).

Are additive genetic or early environmental effects responsible for early divergence in growth trajectories?

Additive genetic variance appeared as a significant source of positive phenotypic covariance between EGR and log(A), whereas shared early environment – as measured by a common birth litter and year – did not play any significant role in the covariance between these two parameters (Fig. 4 and Tables S3–S10). There was significant positive genetic covariance between mass at independence and asymptotic mass. Sources of phenotypic covariance between all other parameters were exclusively environmental and mostly due to birth litter identity (Fig. 4 and Tables S3–S10). Specifically, birth litter mediated the negative phenotypic covariance between EGR and log(k_0), which suggests

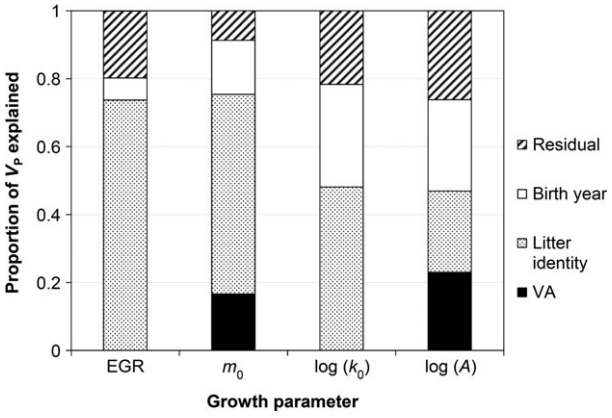

Fig. 3 Variance component analysis of the growth parameters. EGR, early (pre-independence) growth rate; m_0, mass at independence; k_0, post-independence growth rate; A, asymptotic mass. The mean and standard deviation of the proportion of the total phenotypic variance accounted by each random effect are shown for all four growth parameters examined.

Table 2 Variance component analysis summary from the most parsimonious univariate model for each growth parameter.

	Random effect	Variance	SD	CV	Ratio variance component/V_P
Early growth	VA	ns	ns	ns	ns
(n = 1329 individuals)	Litter	0.2920	0.0253	0.0940	0.737 ± 0.038
	Birth year	0.0260	0.0171	0.0084	0.065 ± 0.041
	Residual	0.0779	0.0039	0.0250	0.197 ± 0.017
Lifetime growth					
(n = 531 individuals)					
m_0	VA	540.00	254.00	1.0172	0.166 ± 0.076
	Litter	1920.00	236.00	0.9451	0.588 ± 0.067
	Birth year	517.00	285.00	1.1414	0.159 ± 0.075
	Residual	285.00	141.00	0.5647	0.087 ± 0.046
log(k_0)	VA	ns	ns	ns	ns
	Litter	0.0660	0.0079	0.1374	0.481 ± 0.077
	Birth year	0.0416	0.0198	0.0866	0.302 ± 0.102
	Residual	0.0298	0.0024	0.0620	0.217 ± 0.037
log(A)	VA	0.0054	0.0022	0.0008	0.230 ± 0.089
	Litter	0.0056	0.0011	0.0009	0.239 ± 0.051
	Birth year	0.0063	0.0030	0.0010	0.269 ± 0.094
	Residual	0.0061	0.0013	0.0009	0.262 ± 0.072

SD, standard deviation; CV, coefficient of variation (calculated as the ratio of the variance due to a given effect over the mean of the response trait); V_P, total phenotypic variance; ns, nonsignificant.
Alternative models compared in the model selection procedure are shown in the Table S2.

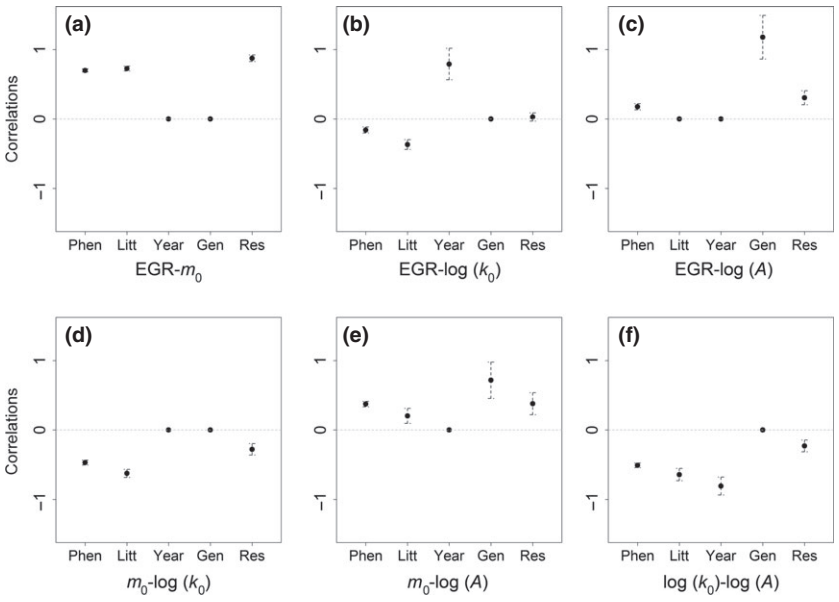

Fig. 4 Sources of covariance between growth parameters. (a) EGR and m_0; (b): EGR and log(k_0); (c): EGR and log (A); (d): m_0 and log(k_0); (e): m_0 and log (A); (f): log(k_0) and log(A). The estimated correlations (±SD) are shown for the raw growth parameters ('Phen') as well as for each possible source of covariance examined (random effects): 'Litt': litter identity, 'Year': birth year, 'Gen': genetic and 'Res': residual variance.

that litters growing slowly before nutritional independence compensated afterwards.

Discussion

We first investigated the extent to which early growth rate EGR affected subsequent growth trajectories in wild meerkats. The phenotypic covariance between pre-independence growth and two of three post-independence growth parameters (mass at independence m_0 and transformed asymptotic mass A) was positive, which suggests that fast-growing pups often maintain their advantage until adulthood. This advantage may help to understand why fast early growth predicts early breeding success in both sexes (Russell *et al.*, 2007) and chances of acquiring dominance in females (Hodge *et al.*, 2008; English *et al.*, 2013b). However, the strength of the correlation between early growth and adult mass was weak and the distribution of meerkat growth trajectories does not show any bimodal signal distinguishing slow and fast growers as may be expected for alternative developmental paths similar to those observed in the morphological castes of eusocial insects (Wheeler, 1986; Beekman *et al.*, 2006). Instead, the negative covariance between pre- and post-independence growth rate suggests the occurrence of compensatory growth at the population level and may relate to seasonal rhythm, where pups born during the dry lean season often reach independence shortly before or after the first rains, when food availability increases. Future studies may investigate the determinants and consequences of episodes of compensatory growth, as studies from a range of organisms suggest that catch-up growth may bear metabolic costs, in the form of increased oxidative stress, that may be associated with fitness costs (Metcalfe & Monaghan, 2001; De Block & Stoks, 2008).

We next investigated the extent of heritable variation in growth trajectories. Significant additive genetic variation was only detected in two growth parameters, mass at independence m_0 and transformed asymptotic mass log(A), but not in pre-independence growth rate EGR or transformed post-independence growth rate log (k_0), suggesting that growth rates may be extremely plastic phenotypes. Our estimates of the heritability of mass parameters are consistent with findings from a recent analysis of genetic and environmental sources of variation in morphometric measures around 3, 6 and 18 months of age in the same population (Nielsen, 2013). These values lie in the range reported for the heritability of body mass for age in other natural populations of mammals (e.g. Réale *et al.*, 1999; Wilson *et al.*, 2005, 2006).

Empirical investigations of the heritability of growth curve parameters in domestic vertebrates have sometimes reported high heritability for all growth parameters (h^2 = 20–50%: (Mignon-Grasteau *et al.*, 1999; Grossman & Bohren, 2008; Lewis *et al.*, 2002), whereas others have reported higher heritability for asymptotic mass than for growth rate (Le Rouzic *et al.*, 2008; Aslam *et al.*, 2011). The extent of environmental influence on growth rates by comparison to adult mass in wild birds (Ricklefs & Peters, 1981; Gebhardt-Henrich & van Noordwijk, 1991, 1994) has led some authors to hypothesize that genetic variation would be more important on the asymptotic part of the growth curve than during development (Gebhardt-Henrich, 1992), supporting our observations. Studies of the heritability of growth parameters in wild mammals are rare, making it difficult to place our results in a broad comparative framework. The heritability of prenutritional

independence growth is low ($h^2 = 0.10 \pm 0.05$) in wild red squirrels (McAdam *et al.*, 2002), suggesting that early growth may often be very plastic. Growth parameters may be less heritable than age-dependent mass or size measures as growth, in contrast to size, is not affected by carry-over effects from previous life stages.

In line with previous studies on the same meerkat population (Nielsen *et al.*, 2012), we detected inbreeding effects in early life traits (EGR and m_0), which disappeared later in development. This decline in the intensity of inbreeding effects throughout development is not a consequence of early postnatal selection against inbred individuals, as the same subset of individuals was used to calculate all three post-independence growth parameters. It is thus likely that early life-history stages show greater sensitivity to inbreeding depression than subsequent ones, supporting findings from other wild vertebrates (Keller, 1998; Slate *et al.*, 2000).

Variance component analyses further indicate that group identity does not explain any variance in growth, although meerkat growth rates increase in large groups (Russell *et al.*, 2002). Important fluctuations in group size can occur over a relatively short time scale (less than the average meerkat lifetime) due to extreme climatic events like droughts (Clutton-Brock *et al.*, 1999a; Bateman *et al.*, 2013) that are not captured using the factor 'group identity' in a multigenerational analysis. Along similar lines, maternal effects on meerkat growth are undetectable. It is possible that they may be offset by helper effects (Russell *et al.*, 2002; Russell & Lummaa, 2009), but phenotypic models in the same population have detected effects of maternal age and rank on offspring growth (English *et al.*, 2013a). Due to the long reproductive careers of dominant females, who often give birth to many litters across a variety of environmental conditions (Hodge *et al.*, 2008), most of the variance in maternal environment may occur within rather than among females as a consequence of the plasticity of maternal traits such as condition (Wilson & Festa-Bianchet, 2009) and are probably absorbed in litter identity rather than in maternal identity in our modelling structure.

Litter identity, which had a large explanatory power in our models, may encompass more than variation in the maternal environment. Littermates are closely related, and litter identity may therefore absorb some additive genetic variance. However, the correlation between the genetic matrix and the litter identity matrix is the same for all growth parameters examined, so is not expected to affect the relative importance of genetic variance between parameters. Littermates also share the same social environment including access to helpers, and previous research has shown that pup growth rate increases with the number of carers per pup, indicating that sibling competition intensifies with litter size (Russell *et al.*, 2002; English *et al.*, 2013a). Finally, extrinsic environmental variation represents a last obvious source of between-litter variance in growth (English *et al.*, 2013a). Climatic conditions and food availability around birth vary extensively from one litter to the next as meerkats breed several times a year in a highly seasonal environment (Doolan & Macdonald, 1996; Clutton-Brock *et al.*, 1999b).

We finally investigated the extent to which genes or early environment may mediate the long-lasting effects of pre-independence growth on subsequent growth trajectories and on adult mass. Bivariate models revealed that sources of covariance between growth parameters were exclusively environmental before adulthood, whereas additive genetic effects represented the only source of covariance identified between the onset (growth prenutritional independence EGR) and the endpoint [adult mass $\log(A)$] of the growth trajectory. Unexpectedly, early environmental effects due to litter identity and birth year failed to mediate early growth effects on adult mass, despite being responsible for most phenotypic variance in early growth. Although it is possible that other, nonexamined sources of early environmental variance may influence adult mass or that sensitive developmental windows occur before 1 month of age (and were thus not targeted by our phenotypic measures), our findings suggest that the role of developmental plasticity may be less important in cooperative vertebrates than it is thought to be in eusocial insects (Drewell *et al.*, 2012; Weiner & Toth, 2012).

Instead, the development of morphological and life-history divergence in cooperative vertebrates may be more contingent on environmental variation affecting later stages of development than in cooperative invertebrates. The causes underlying these profound differences may be multiple and remain to be identified. They may reflect fundamental contrasts in morphology and life history, where invertebrates are more constrained by their exoskeleton and associated fixed developmental steps (metamorphosis, moults, etc.) or a greater need for the longer-lived vertebrates, who may face a greater variety of environments throughout their life, to permanently readjust their development to recent environmental changes. They may also relate to contrasts in social organization, where the queen may exert a greater control over the inheritance of the breeding status, which is determined early, in eusocial insects. In contrast, stochastic factors often play a decisive role in the acquisition of breeding positions in vertebrates, who generally only obtain them when they are fully grown-up and may thus retain their ability to compete as long as possible, rather than specializing early (Clutton-Brock, 2009).

Our results reveal that growth rate prior to nutritional independence contributes to shape lifetime growth trajectories until adulthood, although its effect on adult mass, an important determinant of female breeding success in meerkat societies (Clutton-Brock *et al.*, 2006), is relatively weak. We found no evidence

for alternative developmental paths leading to breeders vs. helpers that would be characterized by a bimodal distribution of growth parameters, with fast and slow growers. Instead, environmental effects generate most variation in growth trajectories throughout development, which is occasionally mediated by episodes of compensatory growth. The heritability of growth parameters is undetectable to moderate, whereas the weak positive covariance between early growth and adult mass is partly genetic in origin. These results reveal that meerkat growth trajectories remain plastic throughout development, rather than showing early and irreversible environmentally induced divergence. Overall, our findings may reflect profound differences in the development of morphological and life-history variation between cooperative invertebrates and vertebrates, which may be more contingent of environmental variation affecting later stages of development than in cooperative invertebrates. Further investigation of the proximate causes and evolutionary significance of these contrasts represents an important avenue for future research.

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Supporting information

Additional Supporting Information may be found in the online version of this article:

Appendix S1 Parameterization of the monomolecular model.

Table S1 Summary statistics for the pedigrees used in the analyses.

Table S2 Model selection for univariate models of growth parameters.

Table S3 Model selection for bivariate models investigating sources of variance and covariance in early and post-independence growth.

Table S4 Model selection for bivariate models investigating sources of covariance among post-independence growth parameters.

Table S5 Sources of phenotypic variance and covariance between early growth and m_0.

Table S6 Sources of phenotypic variance and covariance between early growth and $\log(k_0)$.

Table S7 Sources of phenotypic variance and covariance between early growth and $\log(A)$.

Table S8 Sources of phenotypic variance and covariance between m_0 and $\log(k_0)$.

Table S9 Sources of phenotypic variance and covariance between m_0 and $\log(A)$.

Table S10 Sources of phenotypic variance and covariance between $\log(k_0)$ and $\log(A)$.

Figure S1 Plot of weight by age between 1 and 3 months of age.

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