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The tarnished silver spoon? Trade-off between prenatal growth and telomere length in wild boar

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Abstract

Life-history theory predicts a trade-off between growth rates and lifespan, which is reflected by telomere length, a biomarker of somatic state. We investigated the correlation between telomere length and early-life growth of wild boar piglets, *Sus scrofa*, kept under semi-natural conditions with high food availability to examine our hypothesis that increased pre- and postnatal growth will lead to telomere length attrition, but that a high supply of nutrient may provide the possibility to compensate telomere loss via telomere repair mechanisms. As predicted, our data showed a clear negative correlation between birth body mass and initial telomere length: heavier neonates had shorter telomeres at birth, and we did not find an influence of the mother on initial telomere length. Body mass at birth correlated with body mass later in life and postnatal growth rate did not affect telomere length. We observed an increase in telomere length during postnatal development, suggesting that high food availability allowed piglets to invest into both, growth and telomere restoration. The increase in telomere length over the duration of the study was not accompanied by telomerase activity; thus, telomere elongation was caused either by alternative mechanisms or by short pulses of telomerase activity that we missed. Taken together, this study suggests a trade-off between investment into growth and telomere maintenance even before birth and the possibility to compensate telomere attrition during growth under high amounts of available energy.

KEYWORDS
early life, life history, prenatal growth, somatic maintenance, *Sus scrofa*, telomere attrition, trade-off

1 | INTRODUCTION

High-quality conditions experienced during early life, such as high nutrient availability, can have marked effects on the optimization of life histories and fitness of individuals, often referred to as the 'silver spoon' effect (eg Grafen, 1988; Lindström, 1999; Reichert et al., 2015). An increased body mass at birth caused by favourable environmental conditions during prenatal development is positively related to subsequent performance (eg survival, health, development and reproductive success) of juvenile animals in many species (eg

Gaillard et al., 2000; Maniscalco, 2014; Quiniou et al., 2002; Yuan et al., 2015), and this effect can persist throughout an animal´s lifetime (eg Van de Pol et al., 2006). However, individuals often face trade-offs for resource investment in fitness-related traits, such as growth and somatic maintenance (Lee et al., 2013; Vedder et al., 2018; Monaghan & Ozanne, 2018; Young, 2018; Geiger et al., 2012; but see Vedder et al., 2017). Growing evidence suggests that fast growth rates are linked to accelerated rates of telomere shortening (eg Stier et al., 2020; Vedder et al., 2018), although the mechanistic basis of these long-term effects is still not fully understood (Tarry-Adkins & Ozanne, 2011).

Telomeres, the endcaps of the chromosomal DNA, are a marker of somatic maintenance due to their role in genome protection (Kirkwood, 2002). Due to the 'end replication problem', telomeres are shortened with each somatic cell division because of the inability of the DNA polymerase to successfully replicate the ends (Olovnikov, 1971; Watson, 1972; eg Corey, 2009; Soudet et al., 2014). Telomere length, and in particular the degree of attrition, has been linked to the process of ageing, residual lifespan and senescence in many species (eg Barrett et al., 2013; Boonekamp et al., 2013; Cawthon et al., 2003; Cram et al., 2017; Eastwood et al., 2019; Heidinger et al., 2012; Wilbourn et al., 2018; Zhang et al., 2018). Telomeres can be elongated via telomere repair mechanisms, such as telomere extension by the enzyme telomerase (Greider & Blackburn, 1989). Additionally, telomeres can be elongated via alternative lengthening of telomeres (ALT), a homologous DNA-recombination mechanisms, which is not yet fully understood (eg Henson et al., 2002; Londoño-Vallejo et al., 2004; Sobinoff & Pickett, 2017). Short telomeres, as well as high amounts of telomere attrition, are often associated with a poor biological state (eg Boonekamp et al., 2013; Epel et al., 2004).

Previous studies have mainly focused on the effect of accelerated growth on telomere dynamics under limited resources. The negative correlation between growth rate and lifespan could therefore be based on the harsh environmental conditions rather than growth itself (Boonekamp et al., 2014). Higher growth rates could lead to more telomere attrition unless accompanied by an increased supply of nutrition, which allows animals to invest into antioxidative defence (to buffer damage caused by reactive oxygen species ie ROS) or repair mechanisms (Vedder et al., 2017). To understand the effects of early-life conditions on the performance of juveniles, it is necessary to investigate the trade-offs between growth and somatic maintenance under less restricted early-life conditions. Importantly, telomeres are not only shortened during early life (ie postnatal), but enhanced embryonic growth also leads to increased telomere attrition (eg Vedder et al., 2018). This, together with the strong positive correlation between high body mass at birth and subsequent performance of juvenile animals (eg Gaillard et al., 2000; Hales et al., 2013; Maniscalco, 2014; Quiniou et al., 2002; Yuan et al., 2015), suggests that there is a trade-off between prenatal growth and telomere length, which can have consequences for the individual (eg Foote et al., 2011; Gorenjak et al., 2019; McLennan et al., 2018; Vedder et al., 2018).

We investigated the relationship between birth body mass and initial telomere length as well as the link between growth rate and telomere length during juvenile development of wild boar piglets, *Sus scrofa*, kept under semi-natural conditions. Wild boar sows can give birth to up to 14 piglets per litter, leading to considerable competition between siblings, especially during the first days of life (eg Andersen et al., 2011; Arnold et al., 2019). Moreover, piglets show variation in birth body mass, with a higher birth mass related to a higher survival probability compared with lighter siblings (eg Andersen et al., 2011; Drake et al., 2008).

We hypothesized that, based on findings in previous studies, (a) individuals with high birth mass will have shorter initial telomere length, due to faster intrauterine growth, (b) higher body mass later in life, ie increased growth rate, will correlate with increased telomere length attrition and therefore shorter telomere length (because growth is linked to cell division and therefore telomere attrition), (c) telomere loss will be the highest during the first days after birth due to high competition, and (d) high food availability will allow for mechanisms that prevent or restore telomere loss (ie telomerase activity), leading to longer telomeres in piglets in good body condition.

2 | MATERIAL AND METHODS

2.1 | Ethical note

All procedures have been approved by the institutional ethics and animal welfare committee and the national authority according to §§29 of Animal Experiments Act, Tierversuchsgesetz 2012 - TVG 2012 (GZ 68.205/0012-V/3b/2018).

2.2 | Animals

Six sows and one male wild boar, *Sus scrofa*, were kept and bred in an outside enclosure (2.5 ha) at the Research Institute of Wildlife Ecology, University of Veterinary Medicine, Vienna. The pigs were bought from a breeder in Hungary (Kerfa Vad Kft., 8492 Kerta Kossuth 8, Hungary; certified according to EU standards) and transported to Vienna two years before the project took place. The sows were trained to retreat into feeding boxes to gain access to the piglets. Mating occurred in November 2017, and the male was separated from the sows in early March 2018. Females were kept in pairs in farrowing boxes (160 m^2) containing two wooden hideouts (for details see Arnold et al., 2019). Four of the six sows gave birth to 26 piglets within two days, the 20th and the 21th of March 2018; no crushing of piglets by their mothers was observed. During gestation and lactation, animals were kept under high food availability. Food consisted of a mixture of approximately 1.4 kg/animal of pig feed pellets (Herbert Lugitsch und Söhne Ges.mbH, Austria) and corn, supplemented with some apples, banana and carrots; water was available *ad libitum*. Piglets started to take up solid food in addition to suckling approximately three weeks after birth.

2.3 | Data collection

Data were collected from March to July 2018. Piglets were weighed, measured and sampled for DNA and telomerase on days 1, 5, 14, 49, 76–79, 90–94, and 125–127 after birth. On days 1 and 5 after birth, piglets were temporarily removed from their mothers and transported to a laboratory where they were weighed to an accuracy of 1 g using an electronic scale (Sartorius, Göttingen, Germany). Furthermore, body length was determined with a tape measure (from the tip of the nose to the base of the tail) and tissue was sampled (see below). For identification, individuals were marked with a coloured and numbered ear tag. As the piglets were part of another study on days 1 and 5, they were returned to their mothers within 3–5 h, as soon as all measures were recorded, and samples were taken.

Not all animals were sampled/measured on all sampling points for logistical reasons because we never removed all piglets of a mother at once to limit stress. Therefore, on day 1 within the first 8–12 h after birth, samples were taken from 18 of the 26 piglets (see Table 1 for a breakdown of sample size). Two more were sampled on day 2 after birth; these samples were treated as the initial data points for the respective individuals. Similarly, data collection on days 14 and 49 was carried out on 2 consecutive days. On day 5 after birth one piglet had died, one was euthanized prior to sampling, and one was euthanized after sampling; all due to poor body condition. All three piglets were included in the analysis of day 1 after birth, and one was also included in the analysis of day 5 after birth. Telomere length values of these animals ranged between 1 and 1.2 and were well within the range of the other samples (mean day 1: 0.94; mean day 5: 0.83, Table 2). Animals sampled on days 90–94 were sacrificed for other experimental purposes and samples/measurements were taken within 30 min after death, before the cells started to degenerate. Individuals were sampled in random order during each sampling event.

2.4 | Tissue sampling, relative telomere length and telomerase activity

Cell samples for telomere length and telomerase activity were taken from the buccal mucosa of the inner cheeks by twisting a brush (Gynobrush, Heinz Herenz Medizinalbedarf, Hamburg, Germany) for 20–30 s to collect mucosa cells (Hoelzl et al., 2016). For the telomere length analysis, samples were temporally stored at −20°C for not more than 5 h before they were moved to −80°C. DNA extraction was performed within one month after sampling to prevent DNA degradation. Concentration and quality (260/280 ratio) of the extracted DNA were determined using a NanoDrop 2000c (Peqlab Biotechnologie GmbH, Erlangen, Germany) and then stored at −20°C for later analyses (Cawthon, 2002). The brushes for the telomerase activity analysis were placed into 1 ml culture medium to maintain cell activity. The samples were stored at 4°C for subsequent cell preparation. Cell preparation was done within 24 h after sampling.

Relative telomere length (RTL) was assessed using the real-time quantitative polymerase chain reaction method (qPCR). In brief, this method works via the quantification of the relative copy number of telomeres (T), normalized to the relative copy number of a single copy gene (S), generating a T/S ratio which is then normalized to a standard sample included in every run to create the RTL of the unknown sample compared with the standard sample (Cawthon, 2002). Telomeric primer sequences were 5′-CGG TTT GTT TGG GTT TGG GTT TGG GTT TGG GTT TGG GTT-3′ (tel 1b) and 5′-GGC TTG CCT TAC CCT TAC CCT TAC CCT TAC CCT TAC CCT-3′ (tel 2b) as in Hoelzl et al. (2016). Primer sequences for the non-VCN gene were 5′-CCC AAG GGT TAC TTT GTC CAG AA-3′ (GusB_F2) and 5′-CGA TGT AGG CGG TAG GCG TG-3′ (GusB_R); non-variability in copy number of GusB was shown by Charron et al. (2015). All samples from one individual were always run together for the non-VCN gene and the telomere portions of the assay to reduce the effect of inter-assay variability. In total, 20 runs were needed to analyse all samples. All qPCR runs were performed within two weeks. Due to the different sampling and DNA extraction times, the storage time of extracted DNA varied between one day and up to two months. Melt curve analysis was performed to verify a single peak for all samples and no secondary amplification. Each sample was run in triplicate.

Two reference standard samples were included in every run for comparison with telomere ratios to the non-VCN gene. Heart tissue was used for standard sample one (S1) (RTL = 1) and kidney tissue for standard sample two (S2). A negative control was included in every run to control for possible contaminants in the reagents.

The intraclass correlation coefficient (ICC) was calculated as a measure of reliability within and between the runs, reflecting degree of correlation and agreement, as suggested by Koo and Li (2016). ICC estimates and their 95% confident intervals for sample triplicates were calculated in R Version 3.5.1. (R Core Team, 2018). Intra-rater ICC was calculated on all included data points based on a single-rating, absolute-agreement, 2-way mixed-effects model (ICC in library 'irr', Gamer et al., 2019). Intra-assay ICC for C_t values for telomere assay was 0.996 ($p < 0.0001$, 95% (CI 0.995–0.997)) and for GusB 0.994 ($p < 0.0001$, 95% (CI 0.993–0.996)) showing an excellent degree of reliability. The ICC for inter-assay was calculated for the

TABLE 1 Number of sampled wild boar piglets (*Sus scrofa*) per variable (RTL, ie relative telomere length; telomerase; body mass; body length) per age (day after birth)

Age (days)	1/2	5	14/15	49/50	76–79	90–94	125–127
RTL	20	24	23	23	11	11	11
Telomerase	20	24	23	23	0	11	11
Body mass [g]	26	24	23	23	11	12	11
Body length [cm]	20	24	23	23	10	12	11

TABLE 2 Range and mean (±SEM) RTL (ie relative telomere length) of all wild boar piglets (*Sus scrofa*) on sampling days 1 to 125–127

Day	1/2	5	14/15	49/50	76–79	90–94	125–127
RTL range	0.32–1.49	0.12–1.20	0.09–1.39	0.46–1.05	0.61–1.21	0.53–1.16	0.69–1.35
RTL mean ± SEM	0.94 ± 0.06	0.83 ± 0.05	0.83 ± 0.06	0.82 ± 0.04	0.90 ± 0.07	0.92 ± 0.07	1.01 ± 0.06

standard sample S1 based on a mean rating ($k = 3$), consistency, 2-way mixed-effects model. Interrater ICC for C_t values for telomere assay was 0.74 ($p = 0.03$, 95% (CI −0.108 to 0.993)) and for GusB 0.68 ($p = 0.05$, 95% (CI −0.35 to 0.992)) showing a moderate-to-good degree of reliability; as all samples per individual were run on the same plate, inter-assay variability should have minimal effect on our longitudinal results.

The intra-assay coefficient of variation (CV), an estimate of system precision, for example the random variation between triplicates, was further used to assess reproducibility. Mean inter-assay CV of S1 for C_t values of the non-VCN gene was 0.33% and for the telomere assay 0.30%.

Telomerase activity was measured with a telomeric repeat amplification protocol (TRAP), first described by Kim et al. (1994), but following the procedure adapted for droplet digital PCR described by Ludlow et al. (2014). More details on qPCR and TRAP assay can be found in the supplement.

2.5 | Data analyses

Data are presented as mean ± standard error of mean (SEM); *N* denotes the number of individuals. Statistical analyses were carried out with R Version 3.5.1. (R Core Team, 2018). Sample sizes varied between 26 and 19 individuals (Table 1).

As the sows gave birth within 2 days, the piglets' age differed by a maximum of 1 day. Data were pooled to one sampling point, respectively, for calculating the mean RTL and RTL ranges. The same was done when samples were taken on consecutive days, and therefore, samples from piglets at the age of 76–79 days, 90–94 days and 125–127 days were treated as one sampling point for mean RTL and RTL ranges (Bekaert et al., 2005; Dantzer & Fletcher, 2015). One RTL result was excluded from the statistical analyses because of an error during sample preparation.

For statistical analyses, 'mother', 'ID' and 'sex' were defined as factors; all non-factorial variables ('birth body mass', ie body mass at first sampling; 'body mass', ie measurements on corresponding sampling days; 'telomeres initial', ie RTL at first sampling; 'telomeres later', ie RTL on corresponding sampling days, 'body length', 'growth rate', 'day', ie the age of piglets, 'litter size') were scaled to ease comparisons (mean centred and divided by standard deviation). As it was not possible to get reliable measures for the piglet´s body length as they grew older, because of the increasing difficulties in handling of the animals, we could not use BMI to calculate growth rate and thus calculated growth rate as the difference in body mass between two sampling points ('day').

Initial RTL was tested for differences in the variance between the litters using F test (var.test in library 'stats', R Core Team, 2018). Paired samples *t* test was used to compare initial RTL and RTL on day five after birth. Linear models were used to analyse the effect of sex and litter size on birth body mass and on initial RTL (lm in library 'stats', R Core Team, 2018), followed by type II sum-of-squared ANOVA (ANOVA in library 'car', Fox & Weisberg, 2019).

We ran a linear mixed-effect model to separate between- and within-subject effects of age on RTL (lme in library 'nlme', Pinheiro et al., 2020) as recommended by Van de Pol and Wright (2009) and included the mean individual age (for between-subject effects) and the relative individual age (difference between age at sampling and the mean individual age; for within-subject effects) as fixed effect predictors, followed by type II sum-of-squared ANOVA. Marginal and conditional model coefficients of determination (R^2) were computed using package MuMIn (Barton, 2019).

Linear mixed-effect models were used to analyse the effect of birth body mass on initial RTL, to test whether body mass at birth influenced RTL later in life, to analyse the effect of birth body mass on body mass at later sampling points, the effect of growth rate on RTL and the effect of sex on RTL. All models included 'mother' as random effect intercept to correct for differences between the litters, 'ID' as a nested random effect intercept to correct for repeated measures and 'day' as a covariate to correct for age. Models with 'growth rate' included 'birth body mass' as a covariate to correct for initial body mass. All models looking at RTL later in life included initial RTL as a covariate to correct for the 'regression to the mean' phenomenon (Hoelzl et al., 2016). All linear mixed-effect models were followed by type II sum-of-squared ANOVA (as there was no significant interaction between predictors) using Wald Chi-squared tests, and model residuals were tested for normal distribution with Shapiro–Wilk tests (shapiro.test in library 'stats', R Core Team, 2018).

3 | RESULTS

3.1 | Body mass and sex ratio

Litter size ranged from six to seven piglets. The birth sex ratio was 11 females to 15 males (0.73:1). Birth body mass of the piglets ranged from 1120 g to 1612 g with an overall mean of 1390 ± 24 g ($N = 26$; range of litter mean body mass: 1245–1498 g). Litter size and sex had an effect on the birth body mass of piglets, with piglets of smaller litters having a higher birth mass than piglets of larger litters ($F_{1, 23} = 26.82$, $p < 0.001$) and males being heavier than females ($F_{1, 23} = 11.91$, $p = 0.002$).

Body mass at birth correlated with body mass at later sampling points (day 5–day 127): individuals with a higher birth body mass also had a higher body mass later in life ($\chi^2 = 5.54$, $df = 1$, $p = 0.02$, $R^2m = 0.92$, $R^2c = 0.95$, $N = 24$, $n = 104$; Figure 1).

3.2 | Relative telomere length

Initial RTL ranged from 0.32 to 1.49 with a mean initial RTL of 0.94 ± 0.06 ($N = 20$). There was a significant effect of body mass at birth on initial RTL: individuals with a higher birth body mass had shorter initial RTL than individuals with low birth body mass ($\chi^2 = 5.02$, $df = 1$, $p = 0.03$, $R^2m = 0.21$, $R^2c = 0.21$, $N = 20$; Figure 2). Despite the small difference in piglet numbers per litter, litter size also had an effect on initial RTL of piglets and piglets of smaller litters had shorter initial RTL than piglets of larger litters ($F_{1, 18} = 4.8$, $p = 0.04$). Sex had no significant effect on initial RTL ($F_{1, 18} = 2.84$, $p = 0.12$).

The variance of initial RTL within litters (mean SD = 0.21) was twice as large as between litters (SD = 0.09). Accordingly, the increase in variance explained by the model including the random effect 'mother' ($R^2 = 0.2090$) was negligible compared with the variance explained by body mass alone ($R^2 = 0.2088$).

There was no significant difference in mean RTL between the first 5 days after birth ($t = 1.84$, $df = 17$, $p = 0.08$; Table 2), but we did find a significant positive effect of age on RTL over the whole sampling period, indicating an increase in RTL over time ($\chi^2 = 9.85$, $df = 1$, $p = 0.002$, $R^2m = 0.12$, $R^2c = 0.38$, $N = 19$, $n = 81$; Table 2; see supplement for individual trajectories). We did not detect any between-subject effects ($\chi^2 = 2.44$, $df = 1$, $p = 0.12$, $N = 25$, $n = 123$), and thus, within-subject differences, ie the relative age of each individual, were the better predictor for change in RTL ($\chi^2 = 4.64$, $df = 1$, $p = 0.03$, $N = 25$, $n = 123$).

Although initial RTL did not predict later life RTL ($\chi^2 = 2.52$, $df = 1$, $p = 0.11$, $R^2m = 0.12$, $R^2c = 0.38$, $N = 19$, $n = 81$), high body mass at birth had a negative effect on RTL later in life ($\chi^2 = 14.81$, $df = 1$, $p < 0.001$, $R^2m = 0.33$, $R^2c = 0.4$, $N = 19$, $n = 81$; Figure 3). Postnatal growth rate had no effect on RTL ($\chi^2 = 1.43$, $df = 1$, $p = 0.23$, $R^2m = 0.34$, $R^2c = 0.42$, $N = 19$, $n = 81$), and there was no significant effect of sex on RTL later in life ($\chi^2 = 0.34$, $df = 1$, $p = 0.56$, $R^2m = 0.13$, $R^2c = 0.38$, $N = 19$, $n = 81$).

Despite detecting an increase in RTL over time, we detected no telomerase activity via ddTRAP. This is unlikely to be due to assay failure as both positive control cell lines produced the expected signals of telomerase activity.

4 | DISCUSSION

This study shows a clear negative correlation between body mass at birth and initial RTL in wild boar piglets, suggesting a trade-off between embryogenic growth and telomere maintenance. Although this negative effect of high birth body mass on RTL was still apparent in later life, we did not find a trade-off between postnatal growth and RTL. Telomere length increased on average over the sampling period and, because postnatal growth rate did not affect RTL, RTL in later life was independent of the initial RTL. That is, telomeres of individuals born with a high body mass remained shorter than those of their lighter-born siblings, whereas individuals born with longer telomeres and a lighter body mass were able to increase body mass without an apparent trade-off on telomere length. Although we did not measure fitness effects, increased telomere attrition is known to be associated with reduced lifespan and reproductive success (eg Eastwood et al., 2019; Heidinger et al., 2012; Ilska-Warner et al., 2019); thus, our data suggest a 'tarnished silver spoon' effect of accelerated prenatal growth on telomere length. The positive effect of high resource availability during pre- and postnatal development (the 'silver spoon') is balanced by a physiological component leading to increased prenatal

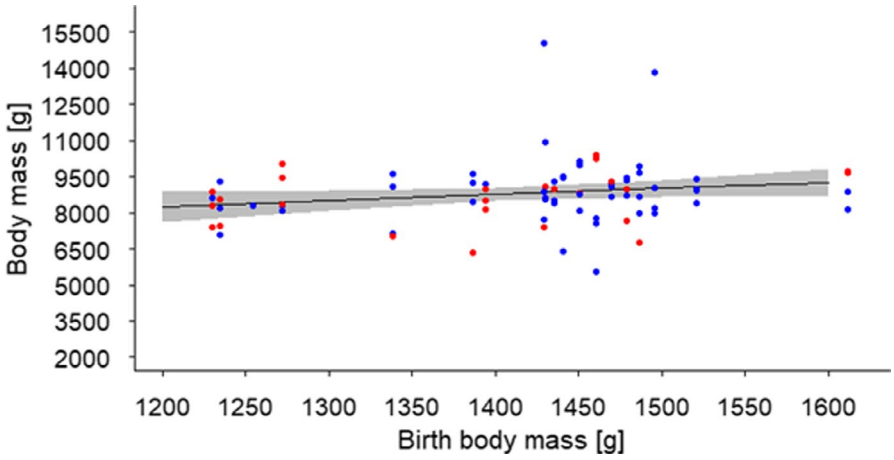

FIGURE 1 Relationship between body mass at birth and body mass in five- to 127-day-old wild boar piglets (*Sus scrofa*). Females depicted in red, males in blue. Raw data with model predicted relationship shown. Grey area indicating standard error. Statistics were corrected for repeated measurements. Piglets with a higher birth body mass also showed higher body mass at later sampling points (day 5 to day 127) ($\chi^2 = 5.54$, $df = 1$, $p = 0.02$, $R^2m = 0.92$, $R^2c = 0.95$, $N = 24$, $n = 104$)

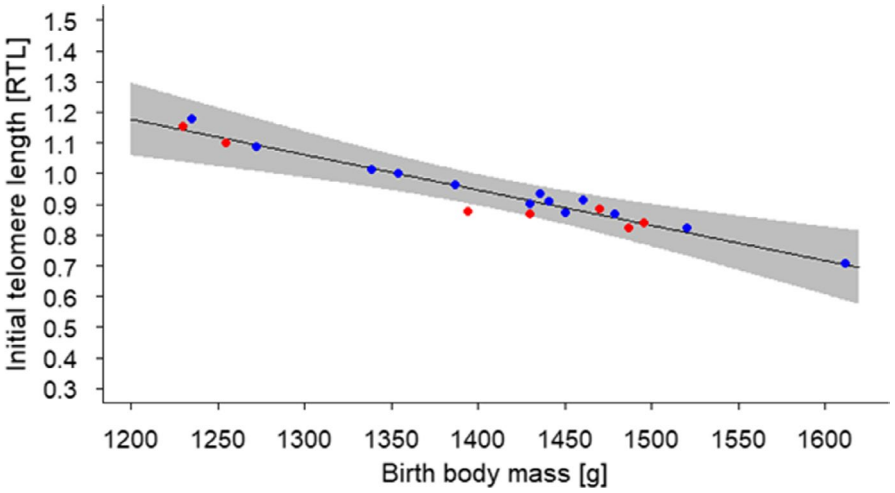

FIGURE 2 Relationship between initial RTL (ie relative telomere length) and body mass of wild boar piglets (*Sus scrofa*) at birth. Females depicted in red, males in blue. There was a negative effect of body mass at birth on initial RTL ($\chi^2 = 5.02$, $df = 1$, $p = 0.03$, $R^2m = 0.21$, $R^2c = 0.21$, $N = 20$). Raw data with model predicted relationship shown. Grey area indicating standard error

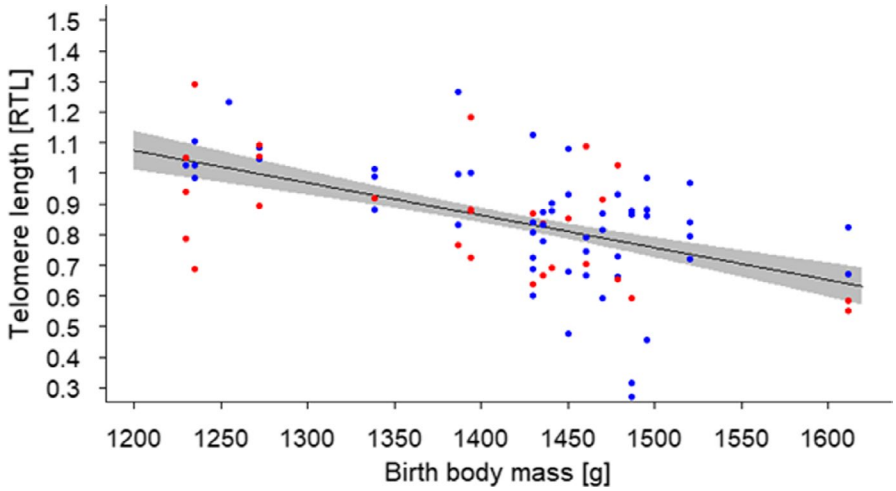

FIGURE 3 Relationship between RTL (ie relative telomere length; sampling points between day 5 and day 127) and birth body mass of wild boar piglets (*Sus scrofa*). The figure shows 81 data points from 19 individuals, females depicted in red, males in blue. There was a negative effect of high birth mass on RTL over the whole sampling period($\chi^2 = 14.81$, $df = 1$, $p < 0.001$, $R^2m = 0.33$, $R^2c = 0.4$, $N = 19$, $n = 81$). Statistics were corrected for repeated measurements. 'Mother' was included as random effect and 'ID' as nested random effect. Raw data with model predicted relationship shown. Grey area indicating standard error

telomere loss, likely related to higher ROS damage, which tarnishes the initial 'silver spoon' advantage.

Despite the small sample size, we can exclude heritability of telomere length as a contributing factor as RTL of piglets at birth were not more similar within litters than between litters, indicating the absence of maternal effects on telomere length. Moreover, as all piglets in our study stem from the same sire, paternal effects can also be dismissed. Our results are in line with a number of studies that showed that increased prenatal tissue growth leads to enhanced metabolic production of ROS and therefore telomere attrition (eg Haussmann et al., 2011; Serra et al., 2000). To our knowledge, this study is the first to show this trade-off in a mammalian species. A recent study on Atlantic salmon, *Salmo salar*, found that faster-developing embryos have shorter telomeres than less developed conspecifics at the same chronological age (McLennan et al., 2018). Furthermore, a study on hatchlings of common terns, *Sterna hirundo*, found that despite individuals having the same mass at hatching, a slower embryonic development led to hatchlings with longer telomeres than hatchlings with a faster embryonic development (Vedder et al., 2018). This suggests that the rate of cell divisions is more important in terms of telomere attrition than the total number of cell divisions (Vedder et al., 2018). Fast growth rates are linked to high metabolic rates and therefore high oxidative stress levels, which are considered to increase telomere damage (eg Monaghan & Ozanne, 2018; Von Zglinicki, 2002). Combined with the 'end replication problem', leading to loss of telomeric DNA within each cell division, this likely explains our observation of heavier piglets having shorter telomeres at birth and vice versa (Olovnikov, 1971; Watson, 1972).

In contrast to our expectation and to the observed trade-off between enhanced embryogenic growth and telomere length at birth, we did not see a decrease but rather an increase in RTL over

the first months of the piglets' life. Furthermore, we also did not observe a decrease in mean RTL during the first days after birth (although the non-significant result may be due to our limited sample size), which are considered to be the most stressful in the life of a piglet. Piglet mortality in domestic pigs, *Sus scrofa domesticus*, is the highest within the first three days after birth and most piglets die from crushing or starvation (eg Dyck & Swierstra, 1987; Edwards, 2002). The observed increase in RTL during postnatal development indicates that the piglets in our study were able to invest into both growth and telomere restoration and supports our hypothesis that high amount of available food can provide the possibility to compensate telomere loss (eg Kärkkäinen et al., 2021). Previous studies suggested that higher postnatal growth rates lead to more telomere attrition if growth is not accompanied by an increased supply of nutrition to buffer the oxidative damage (eg Vedder et al., 2018). Therefore, the absence of a negative effect of the postnatal growth rate on RTL may thus be explained by the high amount of available energy, allowing the individuals to invest into growth and at the same time buffering the oxidative damage by enhancing antioxidative defence mechanism (eg Stewart et al., 2003; Von Zglinicki, 2002). Previous studies reporting telomere lengthening often explain these findings to be a result of measurement errors (eg Chen et al., 2011; Steenstrup et al., 2013). However, several studies have now found that improved environmental conditions and lower levels of life-history stress, for example reduced climate stressors (Foley et al., 2020), parasitic load (Asghar et al., 2018), increased food supply (Hoelzl et al., 2016) or the presence of helpers at the nest (Brown et al., 2021), can lead to telomere restauration or lengthening. There is indeed increasing acceptance that telomere length may be more of a biomarker for current somatic state than a mere reflection of accumulating damage with increasing age (eg Brown et al., 2021; Hoelzl et al., 2016). We used extensive longitudinal sampling and found a clear overall increase in RTL over time. Therefore, we can rule out that our results are due to selective disappearance or measurement errors but are more likely the outcome of life-history trade-offs.

Interestingly, our results also show that the negative impact of prenatal growth could not be compensated by ample supply of nutrition.

It is possible that embryonic growth has a higher impact on the telomere dynamics than postnatal growth, because it constitutes such an important phase in development including organogenesis and maturation of the foetus, leading to a high cell division rate (Otani et al., 2016). It may thus be easier to cope with the negative effects of postnatal growth on telomere length than during the embryonic development (Entringer et al., 2018). Supporting this, a recent study manipulating the incubation temperature of Japanese quails (*Coturnix japonica*) embryos showed a clear effect of prenatal conditions on peri- and postnatal telomere length (Stier et al., 2020).

According to the disposable-soma theory by Kirkwood (1977), individuals should invest into somatic maintenance if the probability of dying due to environmental hazards is lower than the probability of surviving until future reproduction (Kirkwood, 2002). Therefore, increased survival probability of piglets with high birth body mass should select for investment into somatic maintenance and thus telomere length (Kirkwood, 2002). Interestingly, the number of siblings (ie litter size) influenced body mass at birth and consequently also RTL. Large litter sizes can negatively affect birth body mass of piglets through limited space in the uterus and reduced nutrient supply for each foetus (eg Pere & Etienne, 2000). Piglets with a high body mass at birth had short initial RTL, likely due to enhanced embryonic growth, but initial RTL did not predict later telomere length. Body mass at birth further correlated with body mass later in life, showing that piglets with a higher birth body mass were also able to maintain their good body condition over time. This could be due to the increased competitiveness and thus access to food of piglets with higher birth body mass (eg Milligan et al., 2002). On the other hand, the negative effect of high birth body mass on RTL was visible over the whole sampling period, indicating that the ability of heavier individuals to restore is limited. Previous studies found a clear correlation between high birth body mass and survival suggesting that enhanced embryonic growth should be favoured over telomere maintenance (eg Hales et al., 2013; Maniscalco, 2014; Quiniou et al., 2002; Yuan et al., 2015). Our results highlight the importance of taking into account the effect of prenatal development on telomere length, especially because telomere length at birth is seen as the 'baseline' and seems to be carried through to adulthood (eg Gorenjak et al., 2019; Vedder et al., 2018).

Some studies suggest a sex difference in telomere maintenance in accordance with the higher mortality rate in the adult heterogametic sex (reviewed in Barrett & Richardson, 2011). We did not find a difference in initial RTL nor later RTL between the sexes. The reason could be our limited sample size or that we observed only the first four months of the piglets life and sex-specific differences in telomere maintenance may take longer to become apparent (Barrett & Richardson, 2011).

Interestingly, despite the elongation of RTL in our study, no telomerase activity was found. We can rule out assay failure for the lack of detection as the positive control samples gave a signal. Telomerase is a reverse transcriptase enzyme with the ability to elongate the G-rich strand terminus (Von Zglinicki, 2002). However, in long-lived animals, telomerase activity is usually downregulated in most somatic cells as a tumour suppressing mechanism (Monaghan, 2012). Therefore, one reason could simply be that telomerase was active at different time points than when the samples were taken or was otherwise downregulated. Therefore, the increase in RTL could have been reached by alternative lengthening of telomeres (ALT), as Neumann et al. (2013) found in normal mouse somatic cells. ALT is mostly described as based on homologous DNA-recombination, but the distinct mechanisms are not fully understood (eg Londoño-Vallejo et al., 2004; Sobinoff & Pickett, 2017). Moreover, as already mentioned, investment into antioxidative defence mechanisms to buffer oxidative damage could have helped to maintain, but not to elongate, telomere length (eg Stewart et al., 2003; Von Zglinicki, 2002).

Taken together, our results suggest a trade-off between growth and telomere maintenance in wild boar piglets even before birth.

Although a high birth body mass is associated with higher early-life survival probability and individual quality (eg Andersen et al., 2011; Drake et al., 2008), our study shows that being born with a high body mass can have a negative and long-lasting impact on telomere dynamics and therefore may potentially affect life performance throughout an animal´s life. Thus, any 'silver spoon' effects linked to high birth weights may be 'tarnished' by prenatal telomere attrition in the wild boar.

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CONFLICT OF INTEREST

The authors have no conflict of interest to declare.

AUTHOR CONTRIBUTIONS

J.N. conceived the idea for the study. M.S., J.N. and S.V. conducted the experiments and analysed the data. M.S., F.H. and S.S. conducted genetic analyses. T.R. provided statistical support. M.S. wrote the first version of the manuscript. All authors edited and approved the final version of the manuscript and agree to be held accountable for the content.

PEER REVIEW

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DATA AVAILABILITY STATEMENT

The data that support the findings of this study are openly available in Dryad at https://doi.org/10.5061/dryad.k3j9kd58c.

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SUPPORTING INFORMATION

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