Oikos 126: 1329–1338, 2017
doi: 10.1111/oik.04421

Subject Editor: Yngvild Vindenes. Editor-in-Chief: Dries Bonte. Accepted 20 March 2017

The cost of growing large: costs of post-weaning growth on body mass senescence in a wild mammal

Frédéric Douhard, Jean-Michel Gaillard, Maryline Pellerin, Laurent Jacob and Jean-François Lemaître

F. Douhard (http://orcid.org/0000-0002-8503-1089) (fdouhard@hotmail.fr), J.-M. Gaillard, L. Jacob and J.-F. Lemaître, Université Lyon 1, CNRS, UMR5558, Laboratoire de Biométrie et Biologie Evolutive, FR-69622 Villeurbanne, France. FD also at: UMR Modélisation Systémique Appliquée aux Ruminants, INRA, AgroParisTech, Univ. Paris-Saclay, FR-75005 Paris, France. – M. Pellerin, Office National de la Chasse et de la Faune Sauvage, DER, UR Cervidés-Sanglier, Paris, France.

Individual body mass often positively correlates with survival and reproductive success, whereas fitness costs of growing large are rarely detected in vertebrates in the wild. Evidence that adult body mass progressively declines with increasing age is accumulating across mammalian populations. Growing fast to a large body can increase the cellular damage accumulated throughout life, leading body growth in early life to be negatively associated with the rate of body mass senescence. Moreover, the onset of mass senescence may strongly depend on both sex-specific reproductive tactics and environmental conditions. Assessing the timing and the rate of body mass decline with increasing age thus offers an opportunity to look for costs of having grown fast, especially after a poor start during early life, in both sexes and in different environments. Using a unique dataset including 30 years of longitudinal data on age-specific body mass collected in two roe deer *Capreolus capreolus* populations subjected to contrasted environmental conditions, we looked for potential costs of high post-weaning growth rate in terms of steeper rate of body mass senescence. Our analyses of body mass senescence accounted for the potential variation in the onset of senescence and allowed explicit comparisons of this variable between sexes and populations. Higher growth rates late in the growing period (after weaning) were associated with a steeper rate of body mass senescence, regardless of early mass (gained before weaning), but at different extents depending on sex and environmental conditions. Body mass senescence occurred earlier in males than in females, especially in the population facing limiting resources. In the wild, although heavy individuals generally survive better than small ones, the costs of growing large late in the growing period only became apparent late in life through mass senescence.

Similarly to humans (Forbes and Reina 1970), many mammals suffer from a progressive body mass loss throughout adult life in the wild (Table 1). This loss appears as a cue for individual age-related declines in survival and reproductive success that are often reported (Nussey et al. 2013). Indeed, various physiological mechanisms that likely underpin senescence in body mass − including tooth wear (Carranza et al. 2004), sarcopenia (Janssen et al. 2000), or osteoarthritis (Peterson et al. 2010) − presumably weaken the individual capacity to reproduce and survive (Gaillard et al. 2017). However, the evolutionary causes of body mass senescence remain poorly understood (Nussey et al. 2011). Molecular and cellular damage accumulating throughout life and leading to senescence have mostly been considered as costs of reproductive effort (Kirkwood 2005). In theory, fitness benefits of a high reproductive effort early in life are balanced with costs in terms of more rapid senescence in individual phenotypic traits (Kirkwood and Rose 1991) – especially survival and reproductive success as reported in several free-ranging populations of vertebrates (reviewed by Lemaître et al. 2015). Currently, growth costs are much less studied than reproductive costs in the context of early-late life tradeoffs (Lemaître et al. 2015), although rapid growth is likely to be associated with physiological costs. In order to attain a large body size, individuals must grow fast in early life and so must prioritise growth over other requirements (Clutton-Brock 1988) although these individuals commonly grow slower than they could. Most current evolutionary explanations for limited body growth relate to short-term (i.e. immediate) survival costs, especially under poor environmental conditions (Arendt 1997, Blanckenhorn 2000, Metcalfe and Monaghan 2003, Dmitriew 2011). However, long-term (i.e. delayed) costs could also occur during adulthood in terms of more rapid (i.e. steeper rate of decline) senescence. Faster growth has notably been linked to cellular damage in the form of greater levels of oxidative stress (Rollo 2002). A rapidly-grown body should be more prone to developmental errors and weaknesses (Arendt 1997, Blanckenhorn 2000, Metcalfe and Monaghan 2003), and may thereby display faster degradation (i.e. steeper rate of

Table 1. Evidence of senescence in female (F) and male (M) body mass among wild mammals. Only studies disentangling individual age-related decline (i.e. true senescence) and between-individual heterogeneity in the age-related patterns of body mass are reported. Symbols indicate whether support was found (●) or not (–).

Species	Sex	Age-related decline	Reference
Grey mouse lemur *Microcebus murinus*	F	–	Hämäläinen et al. 2014
	M	–	
Red squirrel *Sciurus vulgaris*	F	–	Descamps et al. 2008
	M	–	
Alpine marmot *Marmota marmot*	F	–	Tafani et al. 2013
	M	●	
European badger *Meles meles*	F	●	Beirne et al. 2015
	M	●	
Weddell seal *Leptonychotes weddellii*	F	●	Paterson et al. 2016
Roe deer *Capreolus capreolus*	F	●	Nussey et al. 2011
Soay sheep *Ovis aries*	M	●	Hayward et al. 2015
	F	●	Nussey et al. 2011
Bighorn sheep *Ovis canadensis*	F	●	Nussey et al. 2011
Reindeer *Rangifer tarandus*	F	●	Weladji et al. 2010

body mass senescence). Birds and mammals with higher embryo growth rate generally suffer from higher rates of age-related mortality (Ricklefs 2006). From a resource allocation perspective, fast growing individuals are also expected to allocate fewer resources to repairing cellular damage (Kirkwood and Rose 1991) in particular when they do not depend anymore on parental provisioning. For instance, weaned young mammals have to allocate most of their time to foraging. Concomitantly with changes in individual body composition, the energy requirement to produce a given gain in body mass increases with age (Eisen 1975). Thus, a high growth rate should increase damage accumulation (Hou 2014), and thereby lead to a steeper rate of body mass senescence, especially when fast growth occurs late in the growing period. To date, most evidence of (postnatal) growth costs in terms of more rapid senescence in phenotypic traits come from studies in the laboratory, and the only direct evidence so far reported in vertebrates involved fishes in controlled experimental conditions (Lee et al. 2012). In the wild, individuals of vertebrate populations usually display highly variable growth rates, including accelerated growth after a period of food shortage (compensatory growth sensu Hector and Nakagawa 2012). Moreover positive links between growth rates and susceptibility to oxidative stress (e.g. in the yellow-legged gull, *Larus michahellis*; Kim et al. 2011), and telomere erosion (e.g. in the king penguin, *Aptenodytes patagonicus*; Geiger et al. 2012) have been established. However, direct evidence that faster growth leads to a steeper rate of senescence under natural conditions is currently lacking.

Although evolutionary theories of senescence have primarily focused on the rate of senescence, growing evidence suggests that variation across and within species in the age at the onset of senescence needs to be accounted for in the analysis of senescence (Jones et al. 2008, Péron et al. 2010), especially in relation to sex-specific reproductive tactics (Gamelon et al. 2014, Tidière et al. 2015). This seems to be particularly true for mammal body mass because mammals typically grow up to a determinate adult body mass maintained for some years until senescence starts (Nussey et al. 2011). Moreover, while consistent sex-differences in mass senescence rates have been reported to occur within species (Table 1), no explicit comparison involving the onset of mass senescence has yet been performed. Compared to females, males of polygynous and sexually size dimorphic species generally grow faster, and suffer from both higher mortality risks (Clutton-Brock et al. 1985) and earlier actuarial senescence (Tidière et al. 2015). They should thus exhibit an earlier onset of body mass senescence. However, the extent of such sex-differences may critically depend on environmental conditions. For instance, the positive relationship between sexual size dimorphism and male-biased mortality often reported across species can be masked under favourable environmental conditions (Toïgo and Gaillard 2003). Likewise, we may expect reduced sex-differences in terms of body mass senescence due to reduced growth costs when food resources are abundant. To date, sex-differences in body mass senescence have only been assessed from single populations in the wild (Table 1), so their consistency across contrasting environments remains unknown.

We used longitudinal data collected over 30 years in two roe deer populations subjected to contrasting environmental conditions to test for potential effects of early mass and subsequent late growth on body mass senescence. Roe deer are weakly polygynous ungulates, slightly dimorphic in adult body mass (males are about 10% heavier than females, Andersen et al. 1998) but displaying marked sex-differences in favour of females both in terms of survival (Gaillard et al. 1993a) and actuarial senescence (Loison et al. 1999). Adult body mass is reached at around four years of age (Hewison et al. 2011) and, as in other large mammals, females display similar mass for some years during adulthood before a progressive decline occurs in late life (Nussey et al. 2011). We first estimated the mean age at onset of body mass senescence of each one of the four 'sex-population' groups (two sexes × two populations) using the complete dataset of 454 individuals. To do this, we used a linear mixed-effect model (van de Pol and Verhulst 2006) to disentangle three main processes: a progressive decline with increasing age (i.e. chronological senescence), a terminal decline prior to death and independent of age (i.e. last year effect), and a selective disappearance (i.e. change in phenotypic composition between age-classes due to the disappearance of 'low-quality' individuals). Secondly, the four obtained senescence patterns were compared pairwise by sex and population. Thirdly, we tested for an effect of early mass (measured as fawn mass reached in their first mid-winter) and late growth (measured as residual post-weaning mass gain until adulthood) and their interaction, on the rate of body mass senescence within each sex-population group.

Material and methods

Study populations

The roe deer populations have been intensively monitored for over 35 years and are described in detail elsewhere (Gaillard

et al. 1993a, 1997). The study sites are two enclosed areas in contrasting forest habitats in France. The 2614-ha Réserve Biologique Intégrale of Chizé (CH) (46°05′N, 0°25′W) combines oceanic climate with frequent summer droughts, relatively poor soils, and low primary productivity. In contrast, the 1360-ha Territoire d'Etude et d'Expérimentation of Trois Fontaines (TF) (48°43′N, 54°10′E), under continental climatic influences, provides a relatively homogeneous and productive forest habitat (see Pettorelli et al. 2006 for a comparison of plant productivity between sites). Environmental differences between sites are also well reflected in roe deer performance, especially fawn survival, adult body mass, and female reproductive success that are all lower in CH than in TF (Gaillard et al. 1997). In both sites, about 50% of present roe deer are captured using net-driving every winter (mid-December–early March) and weighed using an electronic balance. Individuals are ear-tagged with a unique number allowing their recognition shortly after birth in spring or at their capture in winter, about eight months later. At their first winter capture most known aged roe deer are also marked with a numbered collar. Roe deer females are income breeders in which body condition is weakly linked to reproductive status (Andersen et al. 2000). Moreover, females are weighed right after the end of the embryonic diapause (in late December–early January; Aitken 1974), leading pregnancy to have no impact on female body mass.

Data

We only analysed animals captured and weighed at least once beyond six years of age, assuming that this was the minimum age at which physiological senescence could start (Jégo et al. 2014). In total, 454 individuals (97 males and 140 females in CH; 82 males and 135 females in TF) born from 1975 to 2005 and not caught in the last three years of the study (i.e. 2013–2015) were included. Thereby, virtually all individuals could be considered as dead as the probability of being not captured during three successive years is less than 10%. As the exact age of death – necessary to control for changes in phenotypic composition across age-classes due to condition-dependent mortality (i.e. selective disappearance) – was unknown for most animals, 'age at last capture' was considered instead (Nussey et al. 2008). 'Early mass' and 'late growth' could be obtained for a subset of 332 out of 454 individuals. 'Early mass' was defined as the body mass reached on 27 January (i.e. the median date of roe deer captures in our study sites), which essentially depends on birth date and maternal provisioning during pregnancy and lactation. 'Early mass' is mostly accumulated during lactation because fawns weigh between 0.6 and 2.5 kg at birth (Plard et al. 2014) and rapidly develop afterwards (Gaillard et al. 1993b, Plard et al. 2015). Although roe deer births are highly synchronized around 15 May (i.e. 80% born within less than 25 days, Gaillard et al. 1993c), birth date is negatively associated with early body growth (Plard et al. 2015), meaning that early-born fawns grow faster than late-born fawns. We could not measure early growth per se because birth date was unknown for most fawns. As the winter captures took place from mid-December to early March, we adjusted their observed body mass to 27 January in both populations, which corresponds to about eight months of age (Hewison et al. 2011). To do this we first determined the average daily gain of body mass over the capture period by using a simple linear model with fawn mass as the dependent variable and the Julian date of capture as the independent variable. For each fawn we then calculated the predicted body mass on 27 January (i.e. 'early mass') as the sum of the observed body mass and the average daily mass gain multiplied by the difference between 27 January and the date of capture. Throughout the capture period, fawns gained an average of 12 g $day^{-1} \pm 0.005$ SE (linear regression; $t = 2.65$, $p = 0.009$) and 24 g $day^{-1} \pm 0.008$ SE ($t = 2.88$, $p = 0.005$) at CH and TF, respectively, without detectable sex-differences. 'Late growth' was defined as the residual mass gain between the first winter in life and adulthood. After their first winter, fawns grow independently from maternal care as most of them are weaned in early October (Andersen et al. 1998). Adult body mass was calculated as the median mass during the prime age stage between four (after body growth has ceased) and six years of age (when senescence in body mass can begin) (Nussey et al. 2011). In all sex-population groups, post-weaning mass gain between the first winter and four years of age was negatively related to fawn mass during the first winter, which attenuated mass variation in adulthood compared to the juvenile stage (Fig. 1, grey gradient). However, regardless of fawn mass in their first winter, post-weaning mass gain varied substantially. Only this variation corresponded to 'late growth' (i.e. the residuals of the regression between fawn mass in their first winter (i.e. early mass) and post-weaning mass gain between the first winter and four years of age, Fig. 1).

Modelling senescence of mass in each sex-population group

The linear mixed-effect model included individual measures of annual body mass beyond six years of age as the response variable (in kg). It included the factors 'individual identity' and 'cohort' as random effects to account for the non-independence between measurements (van de Pol and Verhulst 2006), and three covariates (i.e. 'age' (transformed), 'last year of capture', and 'age at last capture'). To distinguish between the two adult life stages commonly observed in studies of age-specific body mass (i.e. stability during prime age stage and decline late in adulthood, Nussey et al. 2011), the effect of chronological age on mass was fitted using a piecewise function prior to its inclusion in the model. Indeed, an age effect was only considered beyond the age at onset of mass senescence and assumed to be absent before. Thus the transformed age was equal to the difference between the age at onset of senescence and the age at measurement if this difference was positive or null, and to zero otherwise. Terminal decline at the end of an individual's life was included with a two-level factor 'last year of capture' indicating whether or not the individual body mass measure was the last one during an individual's life (Tafani et al. 2013). Selective disappearance was accounted for by adding an effect of 'age at last capture' in the model. Linear mixed-effect models were fitted using restricted maximum likelihood ('lmer' function in the R package lme4; Bates et al. 2015) to estimate model parameters. The goodness-of-fit of linear mixed models was based on the calculation

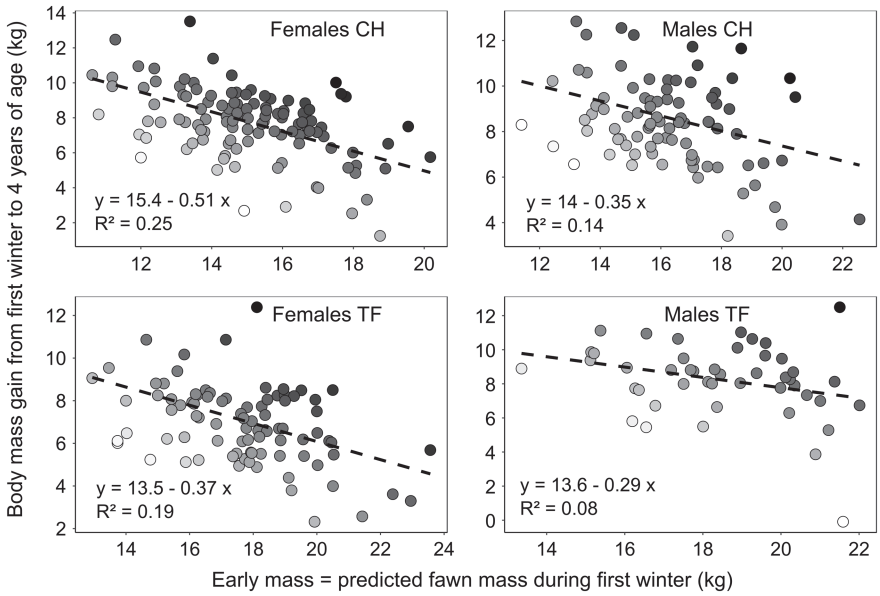

Figure 1. Relationships between individual fawn body mass in their first winter and subsequent mass gain until four years of age in male and female roe deer at Chizé (CH) and Trois-Fontaines (TF). Density of shading increases with adult body mass within each group; dashed line shows the fitted values of the linear model.

of a marginal R^2 and a conditional R^2 (based on maximum likelihood) giving the variance explained by fixed effects, and both fixed and random effects, respectively, as proposed by Nakagawa and Schielzeth (2013).

The age at onset of senescence was determined through maximization of the likelihood with the 'optimize' function of R. The corresponding likelihood profile was drawn and a 95% confidence interval (CI) could be derived from inverted likelihood ratio statistics following the procedure described by Ulm and Cox (1989) (see Supplementary material Appendix 1 for further details).

Comparison of senescence patterns between sex-population groups

We tested pairwise whether senescence in body mass started at the same age, and had the same rate with increasing age on average among individuals of different sex and population. The difference between ages at onset of mass senescence of two sex-population groups g_1 and g_2 was tested with likelihood-ratio-tests. Under the null hypothesis (H_0), a common age at onset x_0 was defined as the age resulting in maximum overall log-likelihood L_0 (i.e. $L_0(x_0) = L_{g1}(x_0) + L_{g2}(x_0)$, where L_{g1} and L_{g2} are within-group model log-likelihoods of groups g_1 and g_2, respectively). When different ages at onset x_{g1} and x_{g2} (in groups g_1 and g_2, respectively) were assumed (H_1), they were determined independently and separately as the respective maximizers of L_{g1} and L_{g2} and log-likelihoods $L_{g1}(x_{g1})$ and $L_{g2}(x_{g2})$ were summed afterwards to calculate the overall log-likelihood L_1. The test was based on the statistic $2\cdot(L_1 - L_0)$ that follows asymptotically a χ^2-distribution with one degree of freedom (i.e. the difference in the number of free parameters under H_0 and H_1) under H_0. The age at onset of mass senescence of each sex-population group was used to transform individual age at measurement with a piecewise function as previously described. Differences in the rate of mass decline with increasing age were then assessed by fitting the interaction between 'age' (transformed) and population or sex.

Effects of early mass and late growth on body mass senescence

Within each sex-population group we tested potential effects of 'early mass' and 'late growth' in interaction with 'age' (transformed) in the linear mixed-effect model of body mass senescence previously described (i.e. base model). To do this, we implemented model selection based on the Akaike information criterion corrected for small sample size (AICc) and used ΔAICc (i.e. difference in AICc between a given model and the model with the lowest AICc in the set of candidate models) (Burnham and Anderson 2002) in the R package MuMIn (Barton 2016). The whole set of candidate models included 14 models ranging from the base model (seven parameters) to the full model including a second-order interaction between 'age', 'early mass' and 'late growth' (13 parameters). To assess the potential improvement in model quality due to the interaction of 'early mass' or 'late growth', or both, with 'age', we reported results of model selection both for the whole set of candidate models and for the subset of models without the interaction terms with 'age'. A detectable negative first-order interaction between 'age' and 'early mass' would only suggest early growth costs, as 'early mass' cannot be considered as a measure of early growth per se (as previously discussed). A detectable negative first-order interaction between 'age' and 'late growth' would support the growth cost hypothesis late in the growing period. Finally, a detectable second-order interaction between 'age', 'early mass' and 'late growth' would support a scenario in which late growth costs on mass senescence rate vary according to early mass.

Data deposition

Data available from the Dryad Digital Repository: <http://dx.doi.org/10.5061/dryad.gh28q> (Douhard et al. 2017).

Results

Body mass senescence in relation to sex and population

Heavier adult roe deer, either because of sex (male), or population habitat (TF), or both, had an earlier onset of body mass senescence (Fig. 2). However, as expected, our model including individual annual body mass beyond six years of age as the response variable (in kg) in each sex-population group revealed a positive effect of selective disappearance. Therefore heavier animals lived longer, even though this effect was not statistically significant, except for females at TF (Table 2, 'Age at last capture'). As indicated in Table 2 ('Age at onset'), males had an earlier onset of body mass senescence than females at CH ($\chi^2_{(1)} = 7.8$, $p = 0.005$), but not at TF ($\chi^2_{(1)} = 1.8$, $p = 0.179$) where a common age at onset of 7.7 years was the most likely (see Supplementary material Appendix 2 for likelihood profiles). The population-difference in the onset of mass senescence was statistically significant for females (earlier at TF than at CH; $\chi^2_{(1)} = 6.3$, $p = 0.012$) but not for males ($\chi^2_{(1)} = 2.5$, $p = 0.1$). No difference in the rate of body mass decline with increasing age was detected, neither between sexes ($\chi^2_{(1)} = 0.26$, $p = 0.61$ in CH; $\chi^2_{(1)} = 0.55$, $p = 0.46$ in TF), nor between populations ($\chi^2_{(1)} = 1.00$, $p = 0.32$ for females; $\chi^2_{(1)} = 2.29$, $p = 0.13$ for males). A substantial terminal decline effect ('last year of capture') on body mass was found only in the lightest and the heaviest sex-population groups (i.e. CH females and TF males, respectively; Table 2). Differences in body mass senescence between sexes and populations could not simply be attributed to differences in prime-age adult body mass as repeating the analysis using relative body mass as the response variable (i.e. the ratio between body mass and prime-age adult body mass) yielded comparable results: the age at onset (with 95% CI, in years) was 12.0 (10.5, 14.3) for CH females, 8.5 (7.7, 10) for CH males, 7.6 (6.4, 8.9) for TF females, and 8.5 (< 6, 9.6) – so still very uncertain – for TF males, whereas the subsequent rate of body mass decline was around 1.5% of prime-age adult body mass per year for all these groups.

Early and late growth effects on body mass senescence in relation to sex and population

The base models of body mass senescence previously presented were substantially improved by including 'early mass' (i.e. fawn mass during their first winter) and 'late growth' (i.e. post-weaning mass gain between the first winter and adulthood) as additional covariates (Table 3; ΔAICc between the base model and the best model among the set of candidate models considering the inclusion of 'early mass', 'late growth', and their interaction (set_1)). These effects captured most of the between-individual and cohort variation previously reported (i.e. random effects in Table 2), as also shown by the increase in marginal R^2 between models of set_1 and base models. In addition, except for males at CH, models were further improved with the first-order interaction between 'age' and 'late growth' (Table 3; ΔAICc between the best models of set_1 and the best model among the set of candidate models considering the inclusion of 'early mass', 'late growth', and their interaction, as well as the interactions of these terms with body mass decline with age (set_2)). In these groups, 'late growth' negatively interacted with 'age' to shape senescence in mass (Table 4), which supports the growth costs hypothesis late in the growing period. In contrast, 'early mass' had no detectable influence on mass senescence rates (Table 3) and its interaction with 'age' was thus not included in the selected models (see Supplementary material Appendix 4 for parameter estimates of the

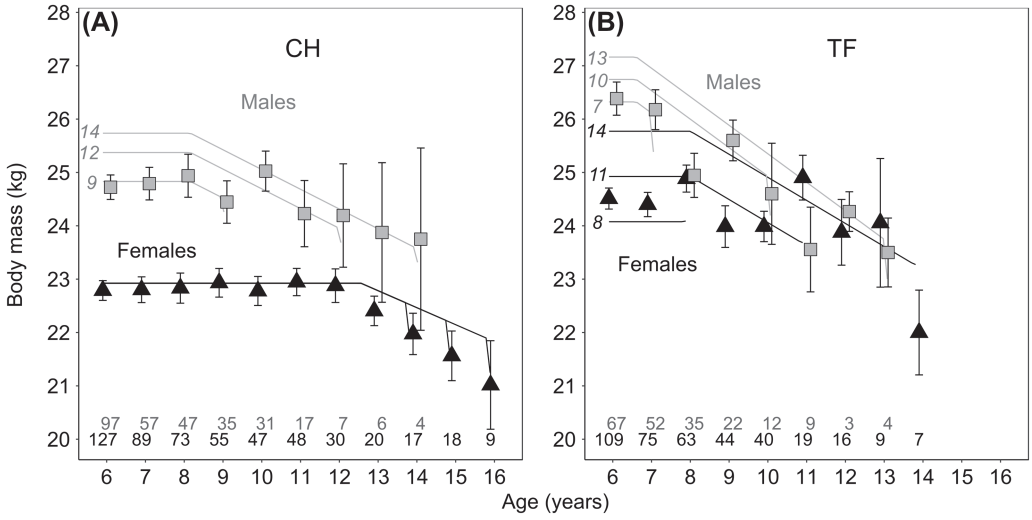

Figure 2. Relationships between age and body mass of adult roe deer of two populations: (A) Chizé (CH) and (B) Trois-Fontaines (TF). Points are means with SE bars (only means calculated with $n \geq 3$ were plotted, n per age class and per sex are provided above the *x*-axis). Lines are model predictions for different ages at last capture (indicated in italic on the left hand side of the lines, in years). Lines are superimposed on each other for CH females as the selective disappearance effect was almost null in this group (Table 2).

Table 2. Parameter estimates of linear mixed-effect model including individual annual body mass beyond 6 years of age as the response variable (in kg) for female and male roe deer at Chizé (CH) and Trois Fontaines (TF). In fixed effects, 'Age at last capture' refers to the selective disappearance of low-quality individuals between age-classes, 'Age' to the decline in body mass with increasing age at measurement after the onset of body mass senescence, and 'Last year of capture' to a terminal additional decline occurring during the last year of life. Marginal and conditional R^2 give the variance explained by fixed effects, and both fixed and random effects, respectively.

Group 'sex-population' (n)	Females CH (140)			Males CH (97)			Females TF (135)			Males TF (82)		
Age at onset (years) 95% CI	12.6 (10.3, 14.7)			8.2 (7.6, 9.4)			8.0 (7.0, 9.8)			6.6 (<6, 8.9)		
Random effects	V	SD		V	SD		V	SD		V	SD	
Individual identity	2.65	1.63		3.69	1.92		2.02	1.42		5.03	2.24	
Cohort year	0.79	0.89		0.94	0.97		0.46	0.68		0.15	0.39	
Residual	1.34	1.16		0.75	0.87		1.90	1.38		1.57	1.25	
Fixed effects	Estimate	SE	p	Estimate	SE	p	Estimate	SE	p	Estimate	SE	p
Intercept	23.0	0.66	***	23.2	1.17	***	21.8	0.79	***	25.4	1.44	***
Age at last capture	0.00	0.058		0.18	0.121		0.28	0.078	***	0.12	0.162	
Age	–0.32	0.084	***	–0.37	0.058	***	–0.43	0.070	***	–0.56	0.109	***
Last year of capture	–0.62	0.131	***	–0.25	0.136	†	0.01	0.199		–0.87	0.238	**
Marginal R^2	0.03			0.05			0.09			0.12		
Conditional R^2	0.73			0.86			0.60			0.80		

†p < 0.1, *p < 0.05, **p < 0.01, ***p < 0.001.

full models). For a same early mass roe deer that gained 1 kg more than others during late growth suffered from an additional body mass loss of 100–110 g per year (females at CH and TF, respectively) to 180 g (males at TF) after the onset of senescence (Table 4). Within each group of individuals that we successively split according to 'early mass' and 'late growth' ('Low' = below the median; 'High' = equal or above the median, Supplementary material Appendix 3), subgroups with a high late growth (except those for males at CH) generally had a body mass senescence rate about 2–3 times steeper than subgroups with a low late growth, regardless of early mass (Fig. 3).

Discussion

We found clear evidence of an age-related decline in body mass for both sexes in both populations, demonstrating that body mass senescence is the rule in free-ranging roe deer populations. This finding supports recent analyses performed in single populations of mammals (Table 1). Patterns of body mass decline with increasing age in male and female roe deer match those of actuarial senescence previously reported in the same populations (Loison et al. 1999), although their exact synchrony should be specifically tested. Males – which grow about 10% larger than females (Andersen et al. 1998) – also start to senesce earlier in life, but only in the population facing the more limited conditions (CH). There is no evidence that males and females senesce at different rates. More importantly, regardless of early mass reached during the first winter (at about 8 months of age), weaned individuals growing at a high rate afterwards until adulthood generally show a steeper rate of mass senescence than those growing slower during the same stage. This brings support to the growth costs hypothesis. Until now growth costs on mass senescence had never been reported in a vertebrate population in the wild.

Table 3. Best fitting models among the set of candidate models considering the inclusion of 'early mass' (EM), 'late growth' (LG), and their interaction (set_1), as well as the interactions of these terms with body mass decline with age (set_2), as additional fixed effects in the linear mixed-effect model including individual annual body mass beyond six years of age as the response variable (in kg) (i.e. base model described in Table 2; seven estimated parameters) for female and male roe deer at Chizé (CH) and Trois Fontaines (TF). In each set the best model is reported (terms of candidate models in grey with symbols indicating included (●) and excluded (−) terms) based on ΔAICc (i.e. difference in AICc between the model with lowest AICc in the wider set (set_2) and the model considered); marginal and conditional R^2 give the variance explained by fixed effects, and both fixed and random effects, respectively.

Group 'sex-population'	Females CH			Males CH			Females TF			Males TF		
Terms	set_2	set_1	base	set_2	set_1	base	set_2	set_1	base	set_2	set_1	base
Terms of base model	●	●	●	●	●	●	●	●	●	●	●	●
Early mass (EM)	●	●		●	●		●	●		●	●	
Late growth (LG)	●	●		●	●		●	●		●	●	
EM × LG	●	●		−	−		●	−		●	●	
Age × EM	−			−			−			−		
Age × LG	●			−			●			●		
Age × EM × LG	−			−			−			−		
n estimated parameters	11	10	7	9	9	7	11	9	7	11	10	7
ΔAICc	0.0	4.0	248	0.0	0.0	246	0.0	8.8	112	0.0	5.7	96
Marginal R^2	0.70	0.69	0.04	0.87	0.87	0.06	0.58	0.56	0.11	0.78	0.76	0.17
Conditional R^2	0.71	0.71	0.73	0.87	0.87	0.85	0.65	0.63	0.61	0.78	0.76	0.78

Table 4. Parameter estimates of the selected linear mixed-effect model including individual annual body mass beyond six years of age as the response variable (in kg) in female and male roe deer at Chizé (CH) and Trois Fontaines (TF), with effects of 'early mass' (EM) and 'late growth' (LG). Symbols (−) indicate non-included terms (see Table 3).

Group 'sex-population' (n)	Females CH (113)			Males CH (85)			Females TF (88)			Males TF (46)		
Random effects	V	SD		V	SD		V	SD		V	SD	
Individual identity	0.05	0.23		0.00	0.00		0.36	0.60		0.00	0.00	
Cohort year	0.00	0.00		0.03	0.16		0.00	0.00		0.00	0.00	
Residual	1.20	1.10		0.71	0.84		1.73	1.32		1.76	1.33	
Fixed effects	Estimate	SE	p	Estimate	SE	p	Estimate	SE	p	Estimate	SE	p
Intercept	22.9	0.278	***	24.5	0.36	***	24.0	0.609	***	26.7	0.939	***
Age at last capture	–0.01	0.023		0.03	0.036		0.06	0.059		–0.01	0.107	
Early mass (EM)	0.44	0.029	***	0.66	0.028	***	0.60	0.050	***	0.66	0.064	***
Late growth (LG)	0.91	0.036	***	0.97	0.032	***	0.62	0.070	***	1.02	0.085	***
EM × LG	–0.03	0.017	*	–			0.05	0.030		–0.07	0.031	*
Last year of capture	–0.49	0.134	***	–0.19	0.139		–0.36	0.221	†	–1.20	0.032	**
Age	–0.43	0.082	***	–0.38	0.056	***	–0.29	0.076	***	–0.40	0.154	*
Age × LG	–0.10	0.041	*	–			–0.11	0.031	***	–0.18	0.071	*

†p < 0.1, *p < 0.05, **p < 0.01, ***p < 0.001.

Growing large generally increases survival prospects within wild populations (e.g. Gaillard et al. 2000 in roe deer and bighorn sheep) likely because large individuals often acquire more resources and can therefore allocate greater amounts of it to both repair and growth than small individuals (van Noordwijk and de Jong 1986). However, within our sex-population groups the magnitude of differences in individual quality was probably low. Indeed, in these two roe deer populations, viability selection effects peak early in life, before weaning (Garratt et al. 2015). Thus, as only successfully weaned individuals were analysed here, they were of relatively high phenotypic quality compared to their

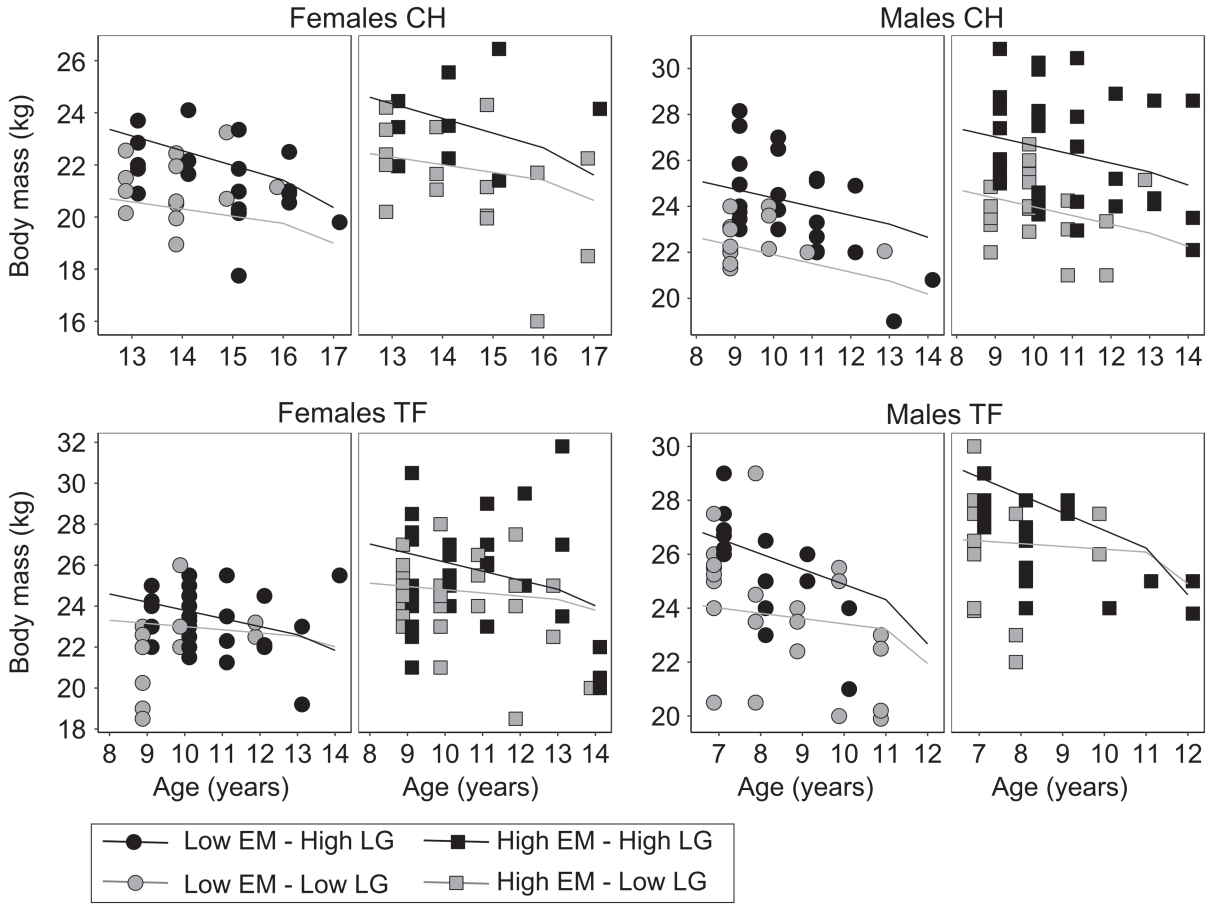

Figure 3. Effects of 'early mass' (EM) and 'late growth' (LG) on individual body mass decline from the age of onset of senescence onwards within each sex-population group. Subgroups include individuals whose growth trait (EM or LG) is below ('Low') and equal or above ('High') the median value of the group. Lines are predictions from the selected models, assuming a constant age at last capture in each group (maximum age of the *x*-axis in each group). CH = Chizé; TF = Trois-Fontaines.

counterparts selectively removed at a younger age. This is especially true for females at CH that only showed mass senescence at old age. Under these conditions, we demonstrated that growing large for a given fawn mass in the first winter actually entails a cost in terms of mass senescence among older individuals. This may be consistent with survival costs of growing large or tall typically indicated by a size-lifespan tradeoff among breeds of laboratory rodents (Rollo 2002) or dogs (Kraus et al. 2013) and in some human populations (Samaras 2009). Yet, as we did not control for individual variation in the intensity of reproductive effort during the prime age stage, we cannot tease apart whether steeper rates of mass senescence directly resulted from physiological costs associated with high growth rates or indirectly from the negative effects of high reproductive effort displayed by heavy prime-aged adults (e.g. higher fecundity in heavy females; Gaillard et al. 2000, larger territory to defend by heavy males; Vanpé et al. 2010). As our measure of late growth was also strongly correlated to prime-age adult body mass (a correlation coefficient around 0.8 in the different sex-population groups), being a large adult per se may be a more important cause of the steeper rates of mass senescence than late growth. In particular, it may be difficult for large individuals to fully meet their high energy requirements (i.e. including those of cellular damage repair) under prolonged periods of food scarcity (Millar and Hickling 1990). A high late growth following a relatively low early mass may also lead to a mismatch between the absolute metabolic rate in adulthood and the relatively small size and cell numbers of certain organs fixed during early life (Metcalfe and Monaghan 2003). Although it would be extremely valuable to control for the potential influence of adult body mass and reproductive effort in the analysis of growths effects on senescence, this seems hardly feasible when only relying on natural variation in growth patterns (Metcalfe and Monaghan 2003). Controlled studies using growth manipulation then become essential (Lee et al. 2012).

Surprisingly, our results indicate that although male roe deer grow larger than females they do not systematically pay higher growth costs in terms of mass senescence. Usually, roe deer males start to defend their territory from three years old onwards (Vanpé et al. 2010), whereas most females first give birth at two years old (Gaillard et al. 1992). Thus females may be more constrained than males by having a shorter time window to grow large enough to start reproducing (Hewison et al. 2011). Females may then experience greater physiological damage during their shorter growing period, which would counterbalance the cost of reaching and maintaining a heavier adult body mass in males. Alternatively, as multiple processes likely underpin mass senescence (Nussey et al. 2011), we cannot exclude that an uncontrolled process overrides size or growth effects on mass senescence, which would make them apparently absent, as in males at CH.

The finding of a sex-difference in senescence under poor environmental conditions is usually interpreted as male viability costs of growing or maintaining a larger size than females when food is scarce (Toigo and Gaillard 2003). For instance, in highly dimorphic and polygynous species such as red deer, mortality is male-biased from the juvenile stage onwards, especially under food shortage (Clutton-Brock et al. 1987). In contrast juvenile male and female roe deer survive equally well (Gaillard et al. 1997). Moreover, the overall mortality experienced during early life widens the extent to which females outlive males later in life (Garratt et al. 2015). Presumably in females juvenile mortality is strongly condition-dependent, meaning that in cohorts facing high rates of juvenile mortality 'low-quality' young females are selectively removed so at the adult stage these cohorts are mainly made up of high quality, long-lived females. In contrast average lifespan of adult males is relatively insensitive to the rate of juvenile mortality (Garratt et al. 2015). This hypothesis is in line with our interpretation that sex difference in the onset of mass senescence is mainly driven by differential female responses to environmental conditions. Prime-aged females of long-lived iteroparous species should favour their own survival over their current reproductive effort when facing under-nutrition (Hirshfield and Tinkle 1975). Accordingly, the probability of successfully weaning two fawns in a given year is lower in the poor conditions of CH than in the good conditions of TF (Douhard et al. 2014) and between-year variation in fawn survival is also higher at CH than at TF (Gaillard et al. 1997). Thus females at TF may provide a higher reproductive effort and consistently exhibit an earlier onset of mass senescence compared to those at CH. In contrast, it is more doubtful that adult males should be able to trade their reproductive effort for a better survival (Festa-Bianchet 2012). Adult roe deer males face a long energy-demanding period (5–6 months) to defend their territory every year, both at TF and at CH, which may consistently lead to a high accumulation of physiological damage with increasing age, whatever the output in terms of reproductive success. Accordingly, the population-difference in the onset of mass senescence was much lower for males than for females. Male and female roe deer may have evolved different life histories, and thereby a different strength of body mass senescence. At first sight, this seems consistent with a role of sexual selection (Bonduriansky et al. 2008). However, roe deer are only subjected to a weak intensity of sexual selection (Andersen et al. 1998) and the similarity in mass senescence rate we observe between sexes is consistent with the weak degree of polygyny in roe deer. Therefore sex-difference in the onset of mass senescence may not ultimately be caused by the intensity of sexual competition per se, but by costs linked to species-specific mating tactics, in particular male physiological and behavioural attributes for territory defence in roe deer (e.g. glandular secretion, rutting behaviour) (Tidière et al. 2015).

Why animals do not grow at their maximum rate and what determines the optimal body size in a population are long-standing questions that are challenging to address in the wild owing to the large number of ecological and environmental factors possibly affecting growth tactics (Dmitriew 2011). Although individual variation in fast growth rates should imply both fitness benefits and costs, evidence for costs of growing or being large remains scarce in wild vertebrates compared to the widely reported size benefits in terms of longevity (Blanckenhorn 2000). Our study thus provides rare, yet circumstantial, evidence that growing large through high growth rate during the late growing period can actually entail a cost in terms of mass senescence among old individuals, but only in some specific sex and environmental conditions. Although the proximal mechanisms

underpinning this steeper rate of mass senescence in response to high post-weaning growth rate remain to be assessed, our study provides the first evidence that growth ultimately contributes to mass senescence patterns, which may impact age-specific survival and reproductive success.

Acknowledgements – We thank all the ONCFS staff volunteers who organized the roe deer captures. We are extremely thankful to Mathieu Douhard, Neil Metcalfe and Subject Editor Yngvild Vindenes for insightful comments on earlier versions of this work.
Funding – This work was funded by Agence Nationale de la Recherche (ANR-15-CE32-0002-01).

References

Aitken, R. J. 1974. Delayed implantation in roe deer (*Capreolus capreolus*). – J. Reprod. Fertil. 39: 225–233.
Andersen, R. et al. 1998. The European roe deer: the biology of success. – Scand. Univ. Press.
Andersen, R. et al. 2000. Factors affecting maternal care in an income breeder, the European roe deer. – J. Anim. Ecol. 69: 672–682.
Arendt, J. D. 1997. Adaptive intrinsic growth rates: an integration across taxa. – Q. Rev. Biol. 72: 149–177.
Bates, D. et al. 2015. lme4: Linear mixed-effects models using Eigen and S4. R package ver. 1.1-7. – J. Stat. Softw. 67: 1–48.
Barton, K. 2016. Package 'MuMIn': Multi-model inference for R, ver. 1.15.6. – <https://cran.r-project.org/web/packages/MuMIn/MuMIn.pdf>, accessed 7 February 2017.
Beirne, C. et al. 2015. Sex differences in senescence: the role of intra-sexual competition in early adulthood. – Proc. R. Soc. B 282: 20151086.
Blanckenhorn, W. U. 2000. The evolution of body size: what keeps organisms small? – Q. Rev. Biol. 75: 385–407.
Bonduriansky, R. et al. 2008. Sexual selection, sexual conflict and the evolution of ageing and life span. – Funct. Ecol. 22: 443–453.
Burnham, K. P. and Anderson, D. R. 2002. Model selection and multimodel inference. a practical information-theoretic approach. – Springer.
Carranza, J. et al. 2004. Disposable-soma senescence mediated by sexual selection in an ungulate. – Nature 432: 215–218.
Clutton-Brock, T. 1988. Reproductive success: studies of individual variation in contrasting breeding systems. – Univ. Chicago Press.
Clutton-Brock, T. H. et al. 1985. Parental investment and sex differences in juvenile mortality in birds and mammals. – Nature 313: 131–133.
Clutton-Brock, T. H. et al. 1987. Early development and population dynamics in red deer. I. Density-dependent effects on juvenile survival. – J. Anim. Ecol. 56: 53–67.
Descamps, S. et al. 2008. Age-specific variation in survival, reproductive success and offspring quality in red squirrels: evidence of senescence. – Oikos 117: 1406–1416.
Dmitriew, C. M. 2011. The evolution of growth trajectories: what limits growth rate? – Biol. Rev. Camb. Phil. Soc. 86: 97–116.
Douhard, M. et al. 2014. Fitness consequences of environmental conditions at different life stages in a long-lived vertebrate. – Proc. R. Soc. B 281: 20140276.
Douhard, F. et al. 2017. Data from: The cost of growing large: costs of post-weaning growth on body mass senescence in a wild mammal. – Dryad Digital Repository, <http://dx.doi.org/10.5061/dryad.gh28q>.
Eisen, E. J. 1975. Results of growth analysis in mice and rats. – J. Anim. Sci. 42: 1008–1023.
Festa-Bianchet, M. 2012. The cost of trying: weak interspecific correlations among life-history components in male ungulates. – Can. J. Zool. 90: 1072–1085.
Forbes, G. B. and Reina, J. C. 1970. Adult lean body mass declines with age: some longitudinal observations. – Metabolism 19: 653–663.
Gaillard, J.-M. et al. 1992. Effects of age and body weight on the proportion of females breeding in a population of roe deer (*Capreolus capreolus*). – Can. J. Zool. 70: 1541–1545.
Gaillard, J.-M. et al. 1993a. Roe deer survival patterns: a comparative analysis of contrasting populations. – J. Anim. Ecol. 62: 778–791.
Gaillard, J.-M. et al. 1993b. Effects of cohort, sex, and birth date on body development of roe deer (*Capreolus Capreolus*) fawns. – Oecologia 94: 57–61.
Gaillard, J.-M. et al. 1993c. Timing and synchrony of births in roe deer. – J. Mammal. 74: 738–744.
Gaillard, J.-M. et al. 1997. Early survival in roe deer : causes and consequences of cohort variation in two contrasted populations. – Oecologia 112: 502–513.
Gaillard, J.-M. et al. 2000. Body mass and individual fitness in female ungulates: bigger is not always better. – Proc. Biol. Sci. 267: 471–477.
Gaillard, J.-M. et al. 2017. Senescence in mammalian life-history traits. – In: Shefferson, R. P. et al. (eds), The evolution of senescence in the tree of life. Cambridge Univ. Press, pp. 126–155.
Gamelon, M. et al. 2014. Do age-specific survival patterns of wild boar fit current evolutionary theories of senescence? – Evolution 68: 3636–3643.
Garratt, M. et al. 2015. High juvenile mortality is associated with sex-specific adult survival and lifespan in wild roe deer. – Curr. Biol. 25: 759–763.
Geiger, S. et al. 2012. Catching-up but telomere loss: half-opening the black box of growth and ageing trade-off in wild king penguin chicks. – Mol. Ecol. 21: 1500–1510.
Hämäläinen, A. et al. 2014. Senescence or selective disappearance? Age trajectories of body mass in wild and captive populations of a small-bodied primate. – Proc. R. Soc. B 281: 20140830.
Hayward, A. D. et al. 2015. Asynchrony of senescence among phenotypic traits in a wild mammal population. – Exp. Gerontol. 71: 56–68.
Hector, K. L. and Nakagawa, S. 2012. Quantitative analysis of compensatory and catch-up growth in diverse taxa. – J. Anim. Ecol. 81: 583–593.
Hewison, A. J. M. et al. 2011. Reproductive constraints, not environmental conditions, shape the ontogeny of sex-specific mass-size allometry in roe deer. – Oikos 120: 1217–1226.
Hirshfield, M. F. and Tinkle, D. W. 1975. Natural selection and the evolution of reproductive effort. – Proc. Natl Acad. Sci. USA 72: 2227–2231.
Hou, C. 2014. Increasing energetic cost of biosynthesis during growth makes refeeding deleterious. – Am. Nat. 184: 233–247.
Janssen, I. et al. 2000. Skeletal muscle mass and distribution in 468 men and women aged 18–88 yr. – J. Appl. Physiol. 89: 81–88.
Jégo, M. et al. 2014. Haematological parameters do senesce in the wild: evidence from different populations of a long-lived mammal. – J. Evol. Biol. 27: 2745–2752.
Jones, O. R. et al. 2008. Senescence rates are determined by ranking on the fast-slow life-history continuum. – Ecol. Lett. 11: 664–673.
Kim, S.-Y. et al. 2011. Quantitative genetic evidence for tradeoff between growth and resistance to oxidative stress in a wild bird. – Evol. Ecol. 25: 461–472.

Kirkwood, T. B. L. 2005. Understanding the odd science of aging. – Cell 120: 437–447.
Kirkwood, T. B. L. and Rose, M. R. 1991. Evolution of senescence: late survival sacrificed for reproduction. – Phil. Trans. R. Soc. B 332: 15–24.
Kraus, C. et al. 2013. The size–life span tradeoff decomposed: why large dogs die young. – Am. Nat. 181: 492–505.
Lee, W.-S. et al. 2012. Experimental demonstration of the growth rate – lifespan tradeoff. – Proc. R. Soc. B 280: 20122370.
Lemaître, J.-F. et al. 2015. Early-late life tradeoffs and the evolution of ageing in the wild. – Proc. R. Soc. B 282: 1–10.
Loison, A. et al. 1999. Age-specific survival in five populations of ungulates: evidence of senescence. – Ecology 80: 2539–2554.
Metcalfe, N. B. and Monaghan, P. 2003. Growth versus lifespan: perspectives from evolutionary ecology. – Exp. Gerontol. 38: 935–940.
Millar, J. S. and Hickling, G. J. 1990. Fasting endurance and the evolution of mammalian body size. – Funct. Ecol. 4: 5–12.
Nakagawa, S. and Schielzeth, H. 2013. A general and simple method for obtaining R^2 from generalized linear mixed-effects models. – Methods Ecol. Evol. 4: 133–142.
Nussey, D. H. et al. 2008. Measuring senescence in wild animal populations: towards a longitudinal approach. – Funct. Ecol. 22: 393–406.
Nussey, D. H. et al. 2011. Patterns of body mass senescence and selective disappearance differ among three species of free-living ungulates. – Ecology 92: 1936–1947.
Nussey, D. H. et al. 2013. Senescence in natural populations of animals: widespread evidence and its implications for bio-gerontology. – Ageing Res. Rev. 12: 214–225.
Paterson, J. T. et al. 2016. Patterns of age-related change in reproductive effort differ in the prenatal and postnatal periods in a long-lived mammal. – J. Anim. Ecol. 85: 1540–1551.
Péron, G. et al. 2010. Age at the onset of senescence in birds and mammals is predicted by early-life performance. – Proc. R. Soc. B 277: 2849–2856.
Peterson, R. O. et al. 2010. Ecology of arthritis. – Ecol. Lett. 13: 1124–1128.
Pettorelli, N. et al. 2006. Using a proxy of plant productivity (NDVI) to find key periods for animal performance: the case of roe deer. – Oikos 112: 565–572.
Plard, F. et al. 2014. Mismatch between birth date and vegetation phenology slows the demography of roe deer. – PloS Biol. 12: e1001828.
Plard, F. et al. 2015. Disentangling direct and growth-mediated influences on early survival: a mechanistic approach. – J. Anim. Ecol. 84: 1363–1372.
Ricklefs, R. E. 2006. Embryo development and ageing in birds and mammals. – Proc. R. Soc. B 273: 2077–2082.
Rollo, C. D. 2002. Growth negatively impacts the life span of mammals. – Evol. Dev. 4: 55–61.
Samaras, T. T. 2009. Should we be concerned over increasing body height and weight? – Exp. Gerontol. 44: 83–92.
Tafani, M. et al. 2013. Sex-specific senescence in body mass of a monogamous and monomorphic mammal: the case of Alpine marmots. – Oecologia 172: 427–436.
Tidière, M. et al. 2015. Does sexual selection shape sex differences in longevity and senescence patterns across vertebrates? A review and new insights from captive ruminants. – Evolution 69: 3123–3140.
Toïgo, C. and Gaillard, J.-M. 2003. Causes of sex-biased adult survival in ungulates: sexual size dimorphism, mating tactic or environment harshness? – Oikos 101: 376–384.
Ulm, K. and Cox, C. 1989. On the estimation of threshold values. – Biometrics 45: 1324–1328.
van de Pol, M. and Verhulst, S. 2006. Age-dependent traits: a new statistical model to separate within- and between-individual effects. – Am. Nat. 167: 766–773.
van Noordwijk, A. J. and de Jong, G. 1986. Acquisition and allocation of resources : their influence on variation in life history tactics. – Am. Nat. 128: 137–142.
Vanpé, C. et al. 2010. Assessing the intensity of sexual selection on male body mass and antler length in roe deer *Capreolus capreolus*: is bigger better in a weakly dimorphic species? – Oikos 119: 1484–1492.
Weladji, R. B. et al. 2010. Age-specific changes in different components of reproductive output in female reindeer: terminal allocation or senescence? – Oecologia 162: 261–271.

Supplementary material (available online as Appendix oik-04421 at <www.oikosjournal.org/appendix/oik-04421>). Appendix 1–4.