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LETTER

Reproductive effort accelerates actuarial senescence in wild birds: an experimental study

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Abstract

Optimality theories of ageing predict that the balance between reproductive effort and somatic maintenance determines the rate of ageing. Laboratory studies find that increased reproductive effort shortens lifespan, but through increased short-term mortality rather than ageing. In contrast, high fecundity in early life is associated with accelerated senescence in free-living vertebrates, but these studies are non-experimental. We performed lifelong brood size manipulation in free-living jackdaws. Actuarial senescence – the increase in mortality rate with age – was threefold higher in birds rearing enlarged- compared to reduced broods, confirming a key prediction of the optimality theory of ageing. Our findings contrast with the results of single-year brood size manipulation studies carried out in many species, in which there was no overall discernible manipulation effect on mortality. We suggest that our and previous findings are in agreement with predictions based on the reliability theory of ageing and propose further tests of this proposition.

Keywords

Age structure, force of mortality, Gompertz, jackdaw, life history, survival, trade-off.

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INTRODUCTION

Actuarial senescence – the increase in mortality rate with age – is thought to arise through the accumulation of somatic damage, making organisms more vulnerable to physiological and environmental challenges (Kirkwood & Rose 1991). The rate of ageing, or the rate of actuarial senescence, is generally assumed to depend on resource allocation towards reproduction, at the expense of somatic repair (Kirkwood 1977; Kirkwood & Rose 1991; Partridge & Barton 1993; McNamara *et al*. 2009). However, empirical tests of the effect of reproductive effort on the rate of senescence have yielded mixed results.

Variation in mortality trajectories and lifespan can not only arise through changes in the rate of ageing but also through age-independent changes in baseline mortality (Pletcher *et al*. 2000; Partridge *et al*. 2005). For instance, human mortality risk may be reduced at old age by the use of a walker, but removal of the walker reverses the effect, which shows that ageing is not affected by this intervention. Both kind of effects have been found in response to interventions that affect lifespan (Pletcher *et al*. 2000; Mair *et al*. 2003; Simons *et al*. 2013). Establishing the relative contribution of actuarial senescence and baseline mortality to changes in lifespan is of interest because they are likely to represent different ecological and/or physiological mechanisms. Moreover, life history evolution of iteroparous species will depend on whether fitness costs of reproduction are due to increased baseline mortality, and hence no longer play a role when a reproductive bout is completed, or due to actuarial senescence, in which case they are carried through to later bouts of reproduction.

Studies in which reproductive effort was manipulated in captive invertebrates generally found that increased reproductive effort shortened lifespan, but this effect was due to increased baseline mortality rate, without affecting actuarial senescence (Partridge & Andrews 1985; Tatar *et al*. 1993; Hsin & Kenyon 1999; Flatt *et al*. 2008). This may be due to the fact that the invertebrate studies were carried out in captivity, because actuarial senescence is the outcome of an increase in susceptibility to environmental and physiological challenges with age (Medawar 1952) and it seems plausible that removal of most environmental challenges will affect the pattern that is observed. Comparative studies do find that fecundity correlates positively with actuarial senescence between species (Jones *et al*. 2008; Ricklefs 2010). Likewise, some long-term studies of free-living vertebrates report that high reproductive output early in life is associated with accelerated reproductive senescence within species (McCleery *et al*. 1996; Reid *et al*. 2003; Reed *et al*. 2008; Bouwhuis *et al*. 2010; Robinson *et al*. 2012). However, these studies used natural variation in reproductive output and it is difficult to ascertain that the accelerated senescence can be attributed to increased reproductive effort, as opposed to some unidentified confounding variable. Such confounds may be unexpected and have large effects. For example, reproductive senescence in great tits distinctly differed between locally born and immigrant birds (Bouwhuis *et al*. 2010). Manipulating reproductive effort largely resolves this issue (Gustafsson & Sutherland 1988) and in the wild such experiments have frequently been carried out, mainly in birds. Although this work has yielded some convincing examples of survival costs of reproduction, (Reid 1987; Daan *et al*. 1996; Verhulst 1998), a recent meta-analysis showed no over-

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all effect across bird studies (Santos & Nakagawa 2012). Similar experiments in rodents have also yielded mixed results (Mappes *et al*. 1995; Koskela 1998; Koivula *et al*. 2003; Skibiel *et al*. 2013). More importantly, with respect to actuarial senescence, experimental effects on survival were studied for only 1 year after the manipulation (but see Reid 1987; Wheelwright *et al*. 1991; Erikstad *et al*. 2009), and without considering age, precluding estimates of the manipulation on actuarial senescence. Hence, whether investment in reproduction accelerates actuarial senescence in free-living vertebrates is still an open question.

Here, we manipulated brood size in a population of free-living jackdaws *Corvus monedula* to test the hypothesis that investment in reproduction increases actuarial senescence. This manipulation successfully modifies the reproductive effort by the parents (Lessells 1993; Santos & Nakagawa 2012), and hence their remaining resources for somatic maintenance and repair. Conceptually, we see our manipulation as an investigation of the mortality consequences of a hypothetical mutation that results in females producing either more or fewer offspring than they would otherwise do. Mutations are carried for life and in agreement with this perspective we subjected individuals to the same manipulation for life, and investigated the cumulative effect of the repeated manipulations. We show that experimentally increasing reproductive effort increased actuarial senescence in a wild bird population without affecting baseline mortality.

MATERIAL AND METHODS

Study system and manipulation of reproductive effort

Jackdaws are sexually monogamous small semi-colonial corvids with bi-parental care and very low divorce rate that produce on average 4.5 eggs per year in a single clutch (Röell 1978). We studied jackdaws in seven nest box colonies in the vicinity of Groningen, the Netherlands (53.1708° N, 6.6064° E) in the period 2004–2012. Laying date of the first egg, clutch size and hatch date were established by regular nest checks (see Salomons *et al*. 2008 for details). We caught birds in their nest box during the breeding season using remote-controlled trapdoors. Upon first capture, birds were marked with a unique combination of colour rings and a metal numbered ring, and in subsequent years individuals were identified either by re-trapping or by reading the colour rings.

We manipulated brood size by net +2 or −2 nestlings (we had no control group to increase statistical power). Whenever possible, three nestlings were moved to the brood that was designated to be enlarged, and one nestling from this enlarged brood was relocated to the matched reduced brood. Manipulated broods were equally distributed over the colonies (Table S2) and matched by hatch date. Nestlings were relocated when the oldest nestling was 4 days old. Relocated nestlings were randomly chosen using first a laptop and later on a smart phone app that simulated dice with a numeric range that was set to be equal to the number of nestlings. When a brood that was designated to be reduced contained two nestlings, we reduced brood size by one nestling, and broods containing one nestling (0.3% of broods; excluded from analysis) were not further reduced to avoid nest desertion. Likewise, some broods were enlarged with one nestling in case the matched reduced brood contained only two nestlings. Broods that could not be enlarged were excluded from analysis. Once an individual parent had been assigned to an experimental category (reduced or enlarged brood) it remained in that category for the duration of the study, and received the same manipulation each year that it returned. In newly formed pairs it occasionally happened that the two pair-members had received different manipulations in a previous year (< 5%), and in these cases we assigned the pair to the manipulation category of the female. Survival of the corresponding males was censored at the moment of switching treatments, i.e. all survival data after switch of experimental treatment were omitted from the analysis and the fate of these individuals was designated to be 'unknown'.

Experimental data

In total, 186 individual parents were manipulated since 2005. Of these individuals, year of birth was known for 30 individuals (18 ringed as nestling and 12 as yearling, which can be distinguished by their brown plumage coloration). The exact year of death was known for only two individuals. Of the 186 individuals that were manipulated at least once, 101 individuals were manipulated two times or more, and 36 three times or more (maximum = 5).

Nestlings were weighed at manipulation and subsequently when 10, 20 and 30 days old (they fledge when ±35 days old). Cumulative brood mass gain after manipulation was calculated by summing the last mass measurement of nestlings that died before fledging with the mass of surviving nestlings at fledging, subtracting the brood mass immediately after the brood size manipulation. We take cumulative brood mass gain to be a proxy for the amount of food provisioned by the parents. Cumulative brood mass gain (gram) was substantially higher in enlarged broods (513.69 ± SE 28.05) when compared to reduced broods (398.34 ± 32.03; $P < 0.001$), and we take this effect as evidence that our manipulation successfully altered reproductive effort. This agrees with what is generally found in response to brood size manipulation when parental care is observed directly (Lessells 1993; Santos & Nakagawa 2012).

Bayesian survival trajectory analysis

Our capture mark recapture (CMR) data are based on the repeated sampling of individuals that we first marked and released, and at each subsequent year were either observed or missed or recovered dead. With respect to the estimation of age-specific mortality rate two challenges need to be solved; (i) because individuals may be alive while not being observed, the proportion of returning individuals underestimates the survival probability. Furthermore (ii), our data are left truncated (a number of individuals were born before the start of the study), and both left and right censored (years of birth and, or, death are unknown for a number of individuals). To solve (i) we performed CMR analysis using the 'Bayesian

Survival Trajectory Analysis' (BaSTA) package in R (Colchero *et al.* 2012), which yields survival estimates that are corrected for the probability of recapture. In our data, recapture probability was 85% (CI 79–90%). More importantly, recapture rate was similar across manipulation groups [reduced 86% (CI 80–92%), enlarged 82% (CI 71–90%)], and this finding was also supported by multistate capture–recapture analysis performed with E-SURGE (see Data S1), allowing for robust comparison of survival between groups. To solve (ii) we used BaSTA (Colchero *et al.* 2012) to perform CMR analysis within a Bayesian hierarchical framework, which allowed us to fit parametric survival functions. With this procedure, missing times of birth and death are imputed from the population means, and based on the survival function, actuarial senescence can be quantified (Colchero & Clark 2011). Parametric survival functions are optimised using a Markov chain Monte Carlo (MCMC) simulation procedure. Actuarial senescence has been described with a number of mathematical functions (Gavrilov & Gavrilova 1991), of which the Gompertz and the Weibull functions are used most frequently. The Weibull function assumes independence of extrinsic/baseline and intrinsic/ageing mortality (Ricklefs & Scheuerlein 2002), while in the Gompertz function actuarial senescence is an age-dependent multiplier of baseline mortality rate. We used the Gompertz function because it best fitted our observed survival trajectories (see model selection below). Instantaneous mortality rate $(u)x$ is determined by the equation

$$u(x) = \exp(b0 + b1 * x + c) \quad (1)$$

where $b0$ is the baseline mortality, and $b1$ the dependency of mortality on age (x). We included the age at first manipulation as proportional hazard covariate (c = coefficient*mean age at first manipulation) to control for variation in the age at which individuals were enrolled in treatment. Mean age of first manipulation was 2.04 years and, probably due to the limited range, did not significantly affect mortality (log hazard ratio = −0.257; CI −0.70, 0.10). BaSTA estimates $b0$ and $b1$ for each level of categorical covariate included in the analysis, thus we obtained separate estimates of $b0$ and $b1$ for individuals rearing reduced or enlarged broods. Remaining lifespan was calculated using the life table produced by BaSTA.

Markov chain Monte Carlo optimisation was done using four parallel simulations with 800 000 iterations, 100 000 burn in period and 2000 interval sampling each (see Fig. S2 for trace plots). Model parameters converged appropriately, serial autocorrelations where low (< 0.05), and the resulting posterior distributions of $b0$ and $b1$ (N = 1400 each) allowed for robust comparison between manipulation groups. After optimisation we described the posterior distribution divergence of $b0_{reduced}$ vs. $b0_{enlarged}$ and $b1_{reduced}$ vs. $b1_{enlarged}$ using the Kullback-Leibler divergence calibration (KLDC) (McCulloch 1989) that is included in BaSTA. KLDC values can be interpreted as the probability between 0.5 and 1 of values drawn from one distribution being from the other distribution. If the KLDC = 0.5, this signifies that the distributions are identical, if the KLDC = 1, this signifies that the distributions are completely non-overlapping. Following general convention, we determined probabilities of > 95% to indicate a significant difference.

Models and model selection

To verify which function (Gompertz or Weibull) was the most appropriate to use we compared the fit of these two functions to our data. We performed model selection based on the lowest deviance information criterion (DIC) (Millar 2009). The Gompertz had the lowest DIC value relative to the Weibull function (DIC values were 1048 and 1066 respectively), but we note that the results with respect to the manipulation effect are similar for both models.

RESULTS

Individuals rearing enlarged broods (EB) had lower annual mean local survival rate (59%) compared with individuals rearing reduced broods (RB; 72%, Fig. 1). This difference in mean survival rate gradually increased when sub setting the data for individuals which received > 1 (37 vs. 67% survival) and > 2 (20 vs. 61%) manipulations (Fig. 1). Survival decreased with increasing number of consecutive manipulations, which is likely to be partly due to decreasing age-specific survival. Survival declined more in EB individuals, which is in qualitative agreement with increased actuarial senescence, but the same pattern could arise if reproductive effort increases baseline mortality rate. These differences in the raw data are supported by multistate capture–recapture analysis (see Data S1), which showed that the best fitting models included manipulation dependent survival probabilities, and

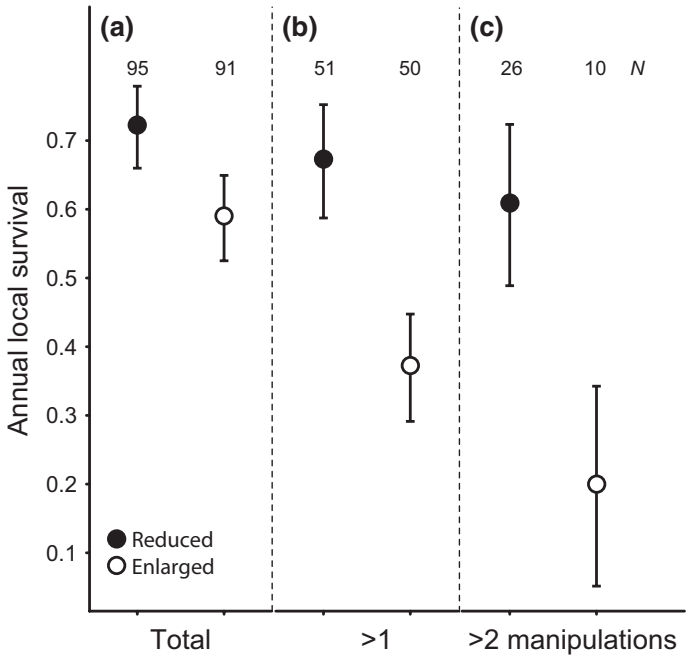

Figure 1 Mean annual local survival rate of individuals rearing reduced or enlarged broods (±SE). Values on the y-axis represent the raw data, i.e. probability to return, corrected for the probability of recapture as estimated by BaSTA (using the population mean of 85% because capture probability was independent of manipulation; see also Data S1). On the x-axis data are shown of all individuals (a), and those of subsets of individuals that received more than 1 (b), or more than 2 (c) consecutive brood size manipulations. The survival differences between manipulation groups are supported by multistate capture–recapture models (see Data S1).

furthermore, that there was a negligible (~1%) difference in recapture probability between manipulation groups (Fig. S1). Models allowing the recapture rate to vary simultaneously across time and manipulation groups were not supported (Table S1), showing that reproductive effort affected survival, but not the probability of recapture.

We used Bayesian survival trajectory analysis (Colchero *et al.* 2012) to test the hypothesis that reproductive effort significantly affects actuarial senescence. BaSTA models that included experimental treatment yielded a lower DIC (delta DIC = 32.72). EB individuals showed a threefold increase in actuarial senescence relative to RB individuals (mean posterior $b1_{EB}$ = 0.50; 95% CI 0.19, 0.83 vs. $b1_{RB}$ = 0.15; 95% CI −0.04, 0.37; KLDC = 0.96; Fig. 2a), while baseline mortality was indistinguishable between treatment groups (mean posterior $b0_{EB}$ = −1.59; 95% CI −2.34, −0.87 vs. $b0_{RB}$ = −1.25; 95% CI −1.88, −0.60; KLDC = 0.69; Fig. 2a). This resulted in 34% lower mean remaining life span of EB individuals at the age of 2 (remaining lifespan = 1.73 vs. 2.64 years). Female and male mortality patterns were indistinguishable, also within brood size manipulation categories, showing that reproductive effort affected mortality of the sexes similarly (see table S3). There was a weak trend for RB male baseline mortality to be somewhat higher than EB baseline mortality, for which we have no particular explanation.

Our estimates refer to local survival, in that permanent dispersal cannot be distinguished from death. Thus, if our manipulation affected dispersal rate this would bias our estimate of the experimental effect on survival. However, very few jackdaws dispersed between colonies and the likelihood of

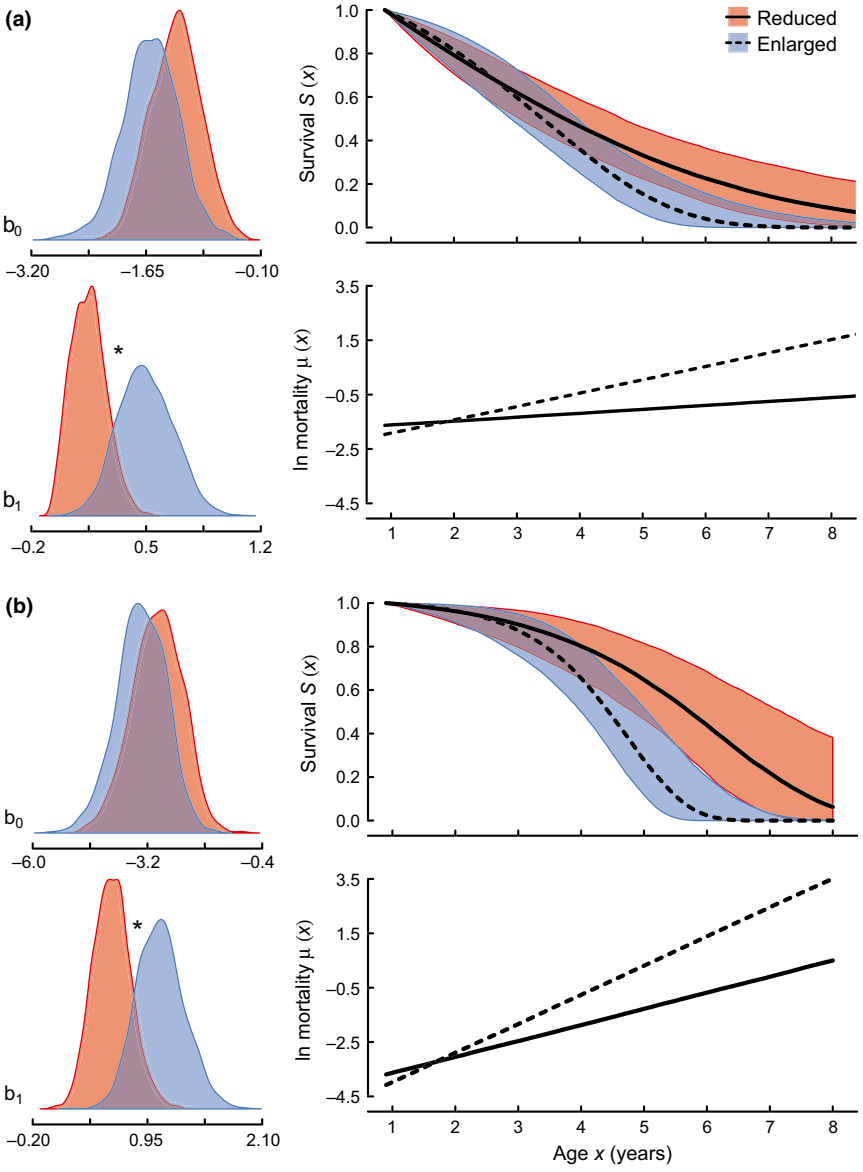

Figure 2 Estimated age-specific survival and mortality rates of (a) the total number of individuals and (b) the subset of resident birds, rearing reduced or enlarged broods. The coloured areas in the survival graphs represent the 95% confidence intervals. Posterior distributions of $b0$ and $b1$ are shown, where $b0$ determines the intercept and $b1$ the slope of the natural logarithm (ln) of age-dependent mortality rate [$(u)x$], which is plotted on the right side. Posterior distributions with a divergence of > 95% are highlighted with an asterisk (*).

dispersal was independent of manipulation direction (5.5 vs. 6.3%). Because natural cavities suitable for breeding are scarce in our study area, there is also little opportunity for birds to move elsewhere. Furthermore, the annual probability to return is 66% on average (range = 56–81% between years), which overlaps with a survival estimate of 65–69% based on ring recovery data of dead birds (Dobson 1990), which method yields global survival estimates that are not confounded by dispersal. This also suggests that permanent dispersal from our study area is very low and unlikely to bias our estimates.

We note, however, that there is likely to be some heterogeneity with respect to dispersal propensity within our population. Jackdaw colonies consist of a majority of resident birds that return each year, and a minority of intruding non-residents that may or may not return to breed in the same colony in later years (Röell 1978). Such heterogeneity may distort in particular the estimates of the first part of the survival trajectory, because emigration of intruders will be translated into higher mortality than the actual mortality. We, therefore, repeated the MCMC optimisation using the same model, but with the subset of resident individuals, defined as individuals that were present in a colony for at least 2 years ($N = 115$). In this subset, mortality rate accelerated almost twice as fast with age in EB individuals (mean posterior $b1_{EB} = 1.08$; 95% CI 0.65, 1.56 vs. $b1_{RB} = 0.60$; 95% CI 0.25, 0.99; KLDC = 0.96; Fig. 2b), while baseline mortality was again indistinguishable between treatments (mean posterior $b0_{EB} = -3.35$; 95% CI −4.60, −2.23 vs. $b0_{RB} = -2.96$; 95% CI −4.21, −1.85; KLDC = 0.59; Fig. 2b). The difference in *b1* resulted in a 64% lower mean remaining life span of EB individuals at the age of 3 (remaining lifespan = 0.69 vs. 1.90 years). We conclude (i) that brood size manipulation increased actuarial senescence, without affecting baseline mortality and (ii) that the consequences for remaining lifespan are more pronounced when the analysis is restricted to resident birds.

DISCUSSION

We experimentally tested the prediction that investment in reproduction accelerates actuarial senescence in a wild bird population. Our main finding is that repeatedly raising enlarged broods shortens remaining life span by 34–64% on average, depending on the exact data selection. Furthermore, this effect arises through an increase in actuarial senescence (*b*1 in the Gompertz equation), with no discernible effect on baseline mortality rate (*b*0). Although this manipulation effect is considerable, it probably underestimates the effects of a natural change in clutch size. This is because we manipulated the number of young when broods were 4 days old, thereby excluding the costs associated with egg production and incubation. Investment up to hatching can be substantial (Monaghan & Nager 1997; Visser & Lessells 2001; de Heij *et al*. 2006), and we therefore predict that a natural change in the number of offspring would yield an even larger difference in actuarial senescence.

The fitness costs of reproduction and other fitness enhancing resource drains (e.g. sexual signalling) are a major component of the contemporary evolutionary ecology framework, and a large effort has gone into testing for such trade-offs using brood size manipulation in birds. It is striking, therefore, that this collective research effort has not yielded a convincing verdict on the importance of the fitness costs of reproduction (Santos & Nakagawa 2012), and we see understanding this result as a major challenge. In our view, the way forward is to on the one hand generate replicates of this study to ascertain the generality of our results. On the other hand, identifying the mechanistic basis is another major challenge, and we see a prominent role for longitudinal sampling of physiological parameters in the wild in an experimental setting. Identifying the mechanistic basis of our results may provide a key to understanding why single-year brood size manipulations have revealed little effect on parental survival.

It is generally assumed that the effects of reproductive effort on actuarial senescence arise as a consequence of increased somatic damage accumulation with age (Kirkwood 1977). Alternatively, however, reproductive effort has a larger impact on mortality at old age compared to young age without direct effects on ageing (Partridge & Andrews 1985). This could for instance arise if mortality risk by predation increases with age, independent of the manipulation, and when at the same time parents rearing enlarged broods spend more time foraging, thereby being more exposed to predators. Thus, increased actuarial senescence may be observed in response to a manipulation, without accelerated physiological ageing as the underlying cause. A way to resolve whether treatment effects are caused by accelerated physiological ageing, as opposed to a change in age-dependent susceptibility to the manipulation, is switching the manipulation from enlarged to reduced effort and vice versa. In invertebrates, such switching experiments have demonstrated that reproductive effort increases short-term mortality, but not the rate of ageing (Partridge & Andrews 1985; Tatar *et al*. 1993; Hsin & Kenyon 1999; Flatt *et al*. 2008), because after the switch subjects quickly adopted the instantaneous mortality rate that matched their current treatment. When the manipulation of effort had affected physiological ageing, there would have been a lagging effect of the previous treatment on mortality rate. Unfortunately, our current data set includes too few individuals that switched treatment ($n = 9$, excluded from the analysis from the switch onward) to use this approach, and obtaining sufficient numbers of birds that were manipulated for a number of years before and after a treatment switch would be a challenge. Instead, to resolve this issue in future studies we propose to measure physiological ageing more directly in the context of our experimental design, using biomarkers such as telomere length for which we have previously identified an association with mortality in our study species (Salomons *et al*. 2009).

Previous studies that manipulated avian reproductive effort in the wild found mixed results for survival rate and, when studies were pooled in a meta-analysis, the overall effect was not significant despite a clear manipulation effect on reproductive effort (Santos & Nakagawa 2012). At first sight, this contrasts with our study, where manipulation of brood size had a strong effect on remaining lifespan. However, earlier studies estimated effects of a single brood manipulation on survival over only 1 year, and also in our data set there is no

manipulation effect on mortality at young age after the first manipulation (Fig. 2). Thus, we see our findings as being consistent with the meta-analysis results of Santos & Nakagawa (2012).

Why survival is unaffected after the first year of manipulation is a question that still needs to be resolved, because it suggests that parents could make a larger reproductive effort than they do, without paying a price in terms of reduced survival. Our findings, together with the meta-analysis results (Santos & Nakagawa 2012), suggest that birds can apparently cope with the increased effort for 1 year without paying an immediate survival cost. Restraining from maximal reproductive effort may be optimal, because this enables birds to cope with unpredictable adverse circumstances during breeding. This could be beneficial, because high reproductive output relative to the population level in a bad year likely outweighs equal effort in a good year (Fisher 1930). Apparently, this buffer is exhausted at some point after the second manipulation, leading to a large survival cost in later years.

Conceptually, a buffer as outlined above is reminiscent of the reliability theory of ageing (Gavrilov & Gavrilova 2001; Boonekamp *et al.* 2013), although we recognise that there are likely to be several models that would fit our results. In brief, reliability models of ageing assume that a system (organism) consists of multiple elements that can replace each other, and that the system collapses at the demise of the last element. The failure rate of redundancy elements is synonym to the rate of damage accumulation. Element failure rate is generally assumed to be age-independent, but such models nevertheless predict an exponential increase in mortality, much like the pattern in natural populations. High reproductive effort may be interpreted to increase the failure rate of redundancy elements resulting in accelerated ageing. When animals are young, and hence redundancy is not yet approaching critical levels, an increase in element failure rate would increase mortality rate but only in the long term, advancing the moment that redundancy reaches a critical level. Thus, such a model would explain why there is no effect of a single year of manipulation, in particular when animals are first manipulated when they are young, as in this study. According to this theory, one would predict that a brood size manipulation carried out in late life would have stronger effects on survival to the next year, compared to the effect of the same manipulation early in life. Unfortunately, we do not have the data to test this, but correlational studies indeed suggest that the costs of reproduction may vary with age (Proaktor *et al.* 2007; Descamps *et al.* 2009). We see testing this hypothesis as an important subject of future studies, to further evaluate the theoretical setting in which best to understand our results and the lack of results from single-year brood size manipulation studies.

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AUTHOR CONTRIBUTIONS

SV and HMS designed the experiment. HMS and JJB collected the data with help of SV and CD. JJB analysed the data, and wrote the first draft of the manuscript with SV. SB contributed the CMR analysis with E-surge. All authors contributed substantially to revisions.

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