Received: 3 June 2020 | Accepted: 10 February 2021

DOI: 10.1111/1365-2435.13785

RESEARCH ARTICLE

Functional Ecology | BRITISH ECOLOGICAL SOCIETY

Mosaic metabolic ageing: Basal and standard metabolic rates age in opposite directions and independent of environmental quality, sex and life span in a passerine

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Handling Editor: Steven Portugal

Abstract

1. Crucial to our understanding of the ageing process is identifying how traits change with age, which variables alter their ageing process and how these traits associate with fitness.
2. Here we investigated metabolic ageing in outdoor-living captive zebra finches experiencing foraging costs. We longitudinally monitored 407 individuals over 6 years and collected 3,213 measurements of two independent mass-adjusted metabolic traits: basal metabolic rate (BMR_m) at thermoneutral temperatures and standard metabolic rate (SMR_m), measured as BMR_m but at ambient temperatures below thermoneutrality.
3. We define mosaic or asynchronous ageing as the difference in standardized absolute ageing rates between traits, and we estimate the degree of asynchrony using the within-individual correlation of change in trait values with age.
4. BMR_m *decreased* linearly with age, consistent with earlier reports. In contrast, SMR_m *increased* linearly with age. The absolute standardized change with age was significantly faster for BMR_m compared to $SMR_{m,}$ and the within-individual correlation of age related change was negligible. To the best of our knowledge, this is the first quantification of SMR_m ageing, and the finding that SMR_m and BMR_m age in opposite directions.
5. Neither metabolic rate nor metabolic ageing rate were associated with variation in life span between individuals. Moreover, experimental manipulations of environmental quality that decreased BMR_m and SMR_m and shortened life span by 6 months (12%) did not affect the ageing of either metabolic trait. Females lived 2 months (4%) shorter than males, but none of the metabolic traits showed sex-specific differences at any age.
6. Our findings indicate, in contrast to the current view, that baseline energy requirements increase with age, because animals do not generally live in thermoneutral conditions, and illustrate the importance of studying the ageing phenotype in an ecologically realistic setting.

KEYWORDS
ageing, environmental quality, foraging, life span, metabolism, thermoregulation

1 | INTRODUCTION

Ageing or senescence, the decline in organismal functioning with age, is ubiquitous in humans, model organisms and in the wild (Belsky et al., 2015; Fontana & Partridge, 2015; Nussey et al., 2013). Technically, senescence refers to a decline in organismal functioning that negatively affects fitness, whereas for ageing this association is not required. In this manuscript, for the sake of simplicity and because in general for many traits we do not know the association between changes in trait value and fitness, we use ageing and senescence interchangeably.

Major challenges in ageing research are (a) to identify how traits and organisms change with age, (b) the factors affecting those changes (e.g. food intake, environmental quality, developmental conditions) and (c) to what extent those changes affect life span and reproductive success (Christensen et al., 2009; Gaillard & Lemaitre, 2017; Nussey et al., 2013; Partridge & Deelen, 2018; Rando & Wyss-Coray, 2021; Williams, 1999). Here we address those three challenges studying energy metabolism. Energy metabolism is an essential component of organismal functioning, reproduction and survival and is therefore a key variable in physiology, ecology and the study of ageing (Burton et al., 2011; Drent & Daan, 1980; Norin & Metcalfe, 2019). Between individuals, there can be a consistent two or even threefold difference in minimal energy consumption and such a difference can influence behaviour, reproduction and survival (Biro & Stamps, 2010; Burton et al., 2011).

Energy metabolism can be characterized in several ways, and a tractable component that is often quantified is basal metabolic rate (BMR), that is, the minimum energy expenditure of a post-absorptive adult animal measured during the rest phase at a thermoneutral temperature (IUPS Thermal Commission, 2001; McNab, 1997). While BMR is measured in the thermoneutral zone, most endotherms spend much of their lives at ambient temperatures that are below thermoneutrality. Standard metabolic rate (SMR), which is measured in the same way as BMR except that the ambient temperature at measurement (T_a) is below thermoneutrality (Bartholomew, 1968; Robinson et al., 1983), may therefore be ecologically more relevant than BMR as it reflects metabolic rate at ambient temperatures experienced more naturally.

Basal metabolic rate is often used a measure of minimal energy consumption for endotherms commonly living at ambient temperatures below thermoneutrality, thereby implicitly assuming that changes in BMR and SMR correlate with each other within and between individuals, that is, a factor that affects BMR in one direction will affect SMR in the same direction (e.g. Biro & Stamps, 2010; Burton et al., 2011 and references therein). In birds, both BMR and SMR decrease in response to environmental and life-history challenges associated with high work load such as breeding, cold winters or migration (Andreasson et al., 2019; Briga & Verhulst, 2017; Brodin, 2007; Piersma & van Gils, 2011). However, BMR and SMR capture very distinct physiological processes. For example, BMR is essentially driven by the mass of central organs (Piersma & van Gils, 2011). In contrast, SMR is to a large extent driven by thermoregulation and insulation, in addition to the aforementioned factors (Piersma & van Gils, 2011), and these different drivers can cause BMR and SMR to change differently in response to ageing. Indeed, we previously showed that the within-individual correlation between mass-adjusted BMR (BMR_m) and mass-adjusted SMR (SMR_m) in the zebra finch *Taeniopygia guttata* is limited (between 0.04 and 0.20), despite both traits themselves having a life-long repeatability of at least 0.30 (Briga & Verhulst, 2017). How BMR_m changes with age within individuals has been quantified in a variety of species and environments, including in humans, and all studies consistently reported a decline with age (review: Elliott et al., 2015). To the best of our knowledge, how SMR_m changes with age in endotherms has not been investigated, but the low within-individual correlations indicate we cannot assume that both age traits have the same age trajectories.

In this study, we had three aims. First, we identified the age trajectories of BMR_m and SMR_m in the same individuals and whether these age trajectories are correlated. Given results in other studies (review: Elliott et al., 2015), we expected BMR_m to decline with age, but for SMR_m, it is unclear what to expect. On the one hand, because BMR_m is part of SMR_m, both traits might have similar age trajectories. On the other hand, their weak within-individual correlation leaves room for the age trajectories to be different. We are not aware of a general definition or procedure to distinguish asynchronous from synchronous ageing, or to quantify the degree of asynchrony between traits. Hence, to identify whether the ageing of both traits is correlated, we developed a statistical approach explained in detail in the methods (Section 2.3.3). In brief, the approach consists of (a) identifying the trait's age trajectories, (b) comparing their standardized absolute ageing rates and, (c) using a bivariate analysis, quantifying the within-individual correlation in the random slopes of change in trait value with age. In the second aim of the study we address the implicit assumption in ageing research that ageing and life span are associated. However, to what extent this the case remains to be shown (Rando & Wyss-Coray, 2021; Williams, 1999). Hence, we tested the hypothesis that factors causing shorter life spans also cause an earlier onset or higher rate of BMR_m and SMR_m ageing (see below). To this end, we used an experimental manipulation (foraging costs) and sex, which both affect life span, and tested whether the shortest living categories show the earliest and/or highest rate of metabolic ageing. The third aim of the study is to test directly whether individuals with higher metabolic rates have shorter life spans by quantifying the

association between BMR_m or SMR_m and life span using survival analyses. We do this for several reasons. First, the knowledge of the association between trait value and life span is a key question in ageing research from a functional perspective, to identify the physiological mechanisms determining life span. For example, in this context, it has been hypothesized that individuals with high energy metabolism have shorter life spans due to high production of damaging reactive oxygen species (Harman, 1956), although the results regarding the association between energy metabolism and life span seem mixed (reviewed in: Burton et al., 2011). Second, identifying the association between trait values and life span is of interest from an evolutionary perspective because of the trait's possible association with fitness and hence to what extent traits, their age trajectories and ageing rates can be under selection (Maynard-Smith, 1962; Monaghan et al., 2008; Williams, 1957, 1999).

We tested these hypotheses in a captive population of a small passerine, the zebra finch. Zebra finches are gregarious birds endemic to Australia, where they live in environments ranging from tropical to arid regions and breed opportunistically in response to rainfall and favourable food resources (Zann, 1996). While their natural life span is not well known, 4-year-old birds have been observed in wild populations (Zann, 1996). The birds used in this study lived in captivity, which may alter metabolism and possibly its association with age compared to that of free-living animals (Auer et al., 2016; Briga & Verhulst, 2017; Wiersma & Verhulst, 2005). Indeed, an essential difference between captive and free-living individuals is that free-living animals generally face foraging costs, which can be a key parameter in metabolism and ageing (Briga et al., 2017; Briga & Verhulst, 2017; Speakman et al., 2015). Foraging costs could drive or reinforce many age-associated declines in organismal performance in the wild, although this has rarely been examined (review: Clay et al., 2018). Hence, to broaden the range of environments and increase the ecological relevance of our study, we housed the birds in outdoor aviaries, and permanently exposed half of our population to high foraging costs using a manipulation of flight cost per food reward as in Koetsier and Verhulst (2011). We applied a 2 × 2 design, independently manipulating foraging costs (benign B vs. harsh H) both during development (i.e. from birth to fledging) and in adulthood, thereby creating four groups (BB, BH, HB, HH, Figure 1). The combination of both manipulations shortened life span: individuals experiencing high foraging costs both during development and in adulthood lived on average 6 months (12%) shorter than all other groups. The foraging cost manipulation during adulthood also decreased BMR_m and SMR_m and both manipulations decreased body mass additively (Briga & Verhulst, 2017; Briga et al., 2019). Secondly, females lived 2 months shorter than males (4%; Briga et al., 2017). Hence, for the second hypothesis, that is, whether factors that shorten life span also accelerate ageing, we predicted that females and the shortest living experimental group (HH) will show the earliest and/or highest rate of metabolic ageing.

2 | MATERIALS AND METHODS

2.1 | Experimental setup

Birds were reared in either experimentally small broods (with two or three chicks) or large broods (between five and eight chicks). These brood sizes were within the range observed in the wild (Zann, 1996). Growing up in large broods increased costly begging for chicks and as a result, they received less food and had impaired growth (at age 15 days: 12%; at adulthood: 4%; Briga, 2016; Briga et al., 2017). Birds were reared with their parents until day 35. After this, birds were housed until they were approximately 4 months old in indoor cages (L × H × W: 110 × 110 × 90 cm) with up to 40 other young of the same sex, two adult males and two adult females. After this, birds were kept in outdoor aviaries until the start of the long-term foraging cost manipulation experiment (Koetsier & Verhulst, 2011), which occurred between the age of 4 months and 2 years. Birds that started the long-term foraging cost manipulation experiment at 4 months went directly from the indoor cages to the experimental aviaries, while birds that started later than 4 months were kept in the outdoor aviaries under standard housing conditions without foraging costs. For the long-term foraging cost manipulation, birds were housed in eight single sex outdoor aviaries (L × H × W: 310 × 210 × 150 cm) located in Groningen, the Netherlands (53°13′0″N/6°33′0″E). The essence of the foraging cost experiment is that in the high foraging cost treatment, birds were forced to fly from a perch to a food box without an opportunity to perch or rest en route. Birds were forced to hover at the feeder to get seeds and fly back to the perch to eat. In the low foraging cost treatment, we provided the same food box but with perches and hence birds could sit and eat without having to fly (Koetsier & Verhulst, 2011). Food (tropical seed mixture), water, grit and cuttlebone were provided ad libitum and the birds received fortified canary food (''egg food'', by Bogena, Hedel, the Netherlands) in weighed portions, which is consistent

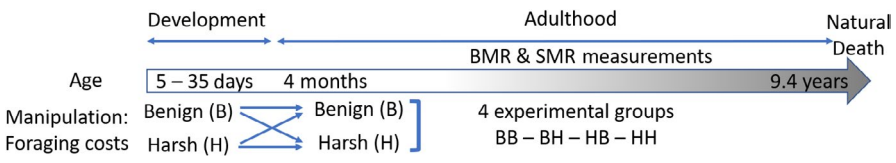

FIGURE 1 Overview of the study setup with measurements of BMR and SMR being done during adulthood. Birds were monitored from birth until natural death and belonged to one of four experimental groups determined by the foraging costs during development (benign B and harsh H) and during adulthood (benign B and harsh H) creating four groups BB-BH-HB-HH

with the conditions in other facilities (Griffith et al., 2017). At the start of the experiment each aviary contained on average 33 birds (*SD* = 1.9) with an age range of 4–24 months. While individuals of the oldest age class were not present when the experiment started, during the 8-year experiment we annually bred birds in the aforementioned experimentally manipulated small and large broods and we replaced the birds that had died with these young birds (4 months old) to keep group size, density, age structure and social environment constant across years.

2.2 | Data collection

We conducted respirometry measurements between December 2007 and April 2013. These are the same measurements as in Briga and Verhulst (2017). In brief, we collected 3,213 respirometry measurements on 407 birds. We obtained 1,233 BMR measurements from 386 individuals (Table 1; Figure S1). In the thermoneutral zone, an endotherm does not increase its metabolic rate in order to maintain body temperature. In our previous work using the same dataset we showed that the thermoneutral zone for the zebra finch is between 32 and 39°C (Briga & Verhulst, 2017): in brief, we found a quadratic association between BMR_m and T_a with a minimum BMR_m at 34.8°C (Briga & Verhulst, 2017). Within the range 32 to 39°C there was a difference of 0.027 W (or 1 *SD* BMR_m) between the maximum and minimum metabolic rate and we considered this as the thermoneutral zone. This range in thermoneutral temperatures is consistent with, but narrower than, the previously identified range between 29.5 and 40°C based on 72 measurements (Calder, 1964).

We also obtained 1980 SMR measurements from 372 individuals (Table 1; Figure S1), which we measured in the same way as BMR, except that the ambient temperature at which birds were measured was below thermoneutrality, between 5 and 32°C. Among these measurements, 90% were between 12 and 29°C (Figure S2) which is within the temperature range that free-living zebra finches commonly encounter in their natural environment (Zann, 1996). In all statistical models below, we included temperature at measurement as a covariate when analysing SMR (range 5–32 C; AICc = −3125.5 relative to the best fitting model in Table S1B). For BMR models we did not include temperature at measurement given that these are thermoneutral (see above and Briga & Verhulst, 2017, Figure S2 for details).

BMR and SMR measurements were collected over an age range from 4 months to 7.2 years (Table 1; Figure S2), with a median age of 3 years and 75% of the measurements occurring below the age of 4 years, which is an age observed in several wild populations (Zann, 1996). BMR and SMR measurements were collected in the same seasons, that is, mostly in either March and April or in September and October (Table 1; Figure S3). Measurements were randomized across sex and experimental treatments.

Metabolic rate was measured overnight using an open flow respirometer situated in a dark room acclimatized to the T_a at measurement. Metabolic rate measurements started in the evening (mean = 18:10 hr; *SD* = 01:17 hr) shortly before sunset (mean = 15 min; *SD* = 1:24 hr) and continued until sunrise after which birds were moved back to their aviaries. Up to 16 individuals were taken from the aviaries and randomly transferred to one of sixteen 1.5 L metabolic chambers. Neither food nor water was available for birds during the metabolic measurements. Birds were weighed to the nearest 0.1 g before and after each measurement. We used the mean body mass value as a fixed effect in all analyses to adjust for mass (see below). Technical details about the equipment can be found in Bouwhuis et al., (2011). In brief, the air-flow through the metabolic chambers was controlled at 25 L/hr by mass-flow controllers (5850S; Brooks) calibrated with a bubble flow meter. Air was dried using a molecular sieve (3 Å Merck) and analysed by a paramagnetic oxygen analyser (Servomex Xentra 4100). During measurements each metabolic chamber or reference outdoor air was sampled every 8 min for 60 s to stabilize measurement levels (Bouwhuis et al., 2011). At each measurement, we quantified the concentrations of O_2 and CO_2 and we calculated the oxygen consumption using Eq. (6) of Hill (1972). An energy equivalent of 19.7 kJ/l oxygen consumed was used to calculate energy expenditure in watts (W). Metabolic rate was taken to be the minimum value of a 30-min running average, which included three to six measurements per individual. The first measurement hour was excluded to minimize potential effects of handling stress and incomplete mixture of air in the metabolic chamber. The BMR and SMR values used here were registered on average 9.6 hr (*SD*: = 2.8 hr) after the start of the measurements.

TABLE 1 Description of the data used. Data distributions are shown in Figures S1–S3. *N* = Number

Description	BMR	SMR
Measurement temperature (°C; range)	32–39	5–32
N measurements	1,233	1,980
N birds	386	372
N birds with >1 measurement	276	311
Median N measurements per bird (95 CI)	3 (1–8)	4 (1–14)
Median N measurements for birds with > 1 measurement (95 CI)	4 (2–8)	6 (2–15)
Mean age at measurement (95 CI, years)	2.8 (0.5–6.5)	3.4 (0.6–6.6)
Date of first measurement	16-Dec-07	30-Mar-08
Date of last measurement	15-Apr-13	14-Apr-13
N measurements in March and April	565 (46%)	796 (40%)
N measurements in September and October	383 (31%)	837 (42%)
Mean metabolic rate [W]	0.22	0.40
S.D. metabolic rate [W]	0.03	0.10
C.V. metabolic rate [W]	0.12	0.26

2.3 | Statistical analyses

2.3.1 | General approach

All analyses were done using general linear mixed models with the function 'lmer' of the package LME4 (Bates et al., 2015) in R version 3.5.3 (R Core Team, 2019). All analyses included individual as a random intercept. For all ageing analyses (Tables S4–S8), we included the age terms that differed between models (see below) as fixed effects and as random slopes nested within individuals. The random slope quantifies the within-individual variation in ageing rate and is required for the correct estimation of confidence intervals when investigating within-individual changes (Schielzeth & Forstmeier, 2009). Such models require considerable sample sizes to accurately estimate fixed and random effects, that is, at least 40 individuals should be sampled with at least 1,000 total measurements (van de Pol, 2012) and our data fulfilled those requirements (Figure S1). We did not have sufficient power to add other random terms to the model (e.g. nesting individual identity in aviary). In all analyses, we found the model best supported by the data, and hence the 'significant' predictor variables, using Burnham and Anderson's model selection approach (Burnham & Anderson, 2002; Burnham et al., 2011) based on the second order Akaike information criterion (AICc) with the function 'dredge' of the package MuMIn (Barton, 2019). In brief, this is a hypothesis-based approach that generates, given a global model, subset models that best fit the data. Model fitting should be considered as a continuum for which alternative models within 4 ΔAICc are plausible and become increasingly equivocal up to 14 ΔAICc, after which they become implausible (Burnham & Anderson, 2002; Burnham et al., 2011). To test for the 'significance' of individual terms, we compared the best fitting model and took the conservative approach to follow the ranking until we encountered the first model with or without the term of interest. In all our tables, models are ranked according to their fit on the data. For the sake of simplicity, we often limited the tables to the first 15 or 20 best fitting models and other models provided a worse model fit well above 14 ΔAICc. For models within 4 ΔAICc, we reported coefficients which are the result of model averaging from the function 'model.avg' of the package MuMIn (Barton, 2019). Confidence intervals of model parameters were estimated with the Wald approximation in the function 'confint'. Residuals of all final models were normally distributed and without influential data points or outliers.

2.3.2 | Adjusting for mass, daily and seasonal variation

To avoid confounding changes in metabolic rate with changes in mass (Briga et al., 2019), we used mass-adjusted metabolic rates in all analyses by adding mass as a covariate, which improved the model fit (BMR ΔAICc = −306.8; SMR ΔAICc = −420.0 relative to their respective best fitting models in Table S1A,B), hence using BMR_m and SMR_m throughout the manuscript.

Data for all traits were collected throughout the year. To avoid confounding age patterns with seasonal effects (Swanson, 2010), we corrected for daily and seasonal variation in mass-adjusted metabolic rates. To this end, we first investigated for each trait which variables best characterized the effect of seasonal variation in trait values (SI 2). We quantified the effects of daily and seasonal variation by testing the effects of: (a) day length, (b) photoperiod, quantified as a dichotomous variable for increasing versus decreasing day length and (c) the minimum ambient temperature (MinT). Temperature data were collected at the weather station of Eelde, approximately 7 km from the aviaries (http://www.knmi.nl/klimatologie/), where temperature was recorded 1.5 m above-ground, every hour with accuracy of 0.1°C. Temperature data at the weather station reflect the temperature at the aviaries well as shown from data collected at the aviaries over the course of the 8 years of observation (N = 1,196, r = 0.96, Briga & Verhulst, 2015). For BMR_m, the model selection approach identified that the photoperiod was the most important covariate (ΔAICc = −54 relative to a model without photoperiod, Table S1A). The next best fitting covariate was day length, which worsened the model fit within the reasonable range of 4 AICc (ΔAICc = +3.1, Table S1A). There was no statistical support for the other tested covariates or for the interaction between photoperiod and day length (ΔAICc ≥ +8.9, Table S1A). For SMR_m, we found that both photoperiod and MinT were important (ΔAICc = −32 and ΔAICc = −8.7 respectively, Table S1B). The effect of MinT on trait values can last over a range of timescales. One approach to identify this timescale uses weighted sliding time windows, which in brief identifies, through model fitting, the time window and weighing function within this window that best fit the data (van de Pol et al., 2016; van de Pol & Cockburn, 2011). We previously used this approach to show that lower MinT increased zebra finch mortality over a weighted time window. In this time window, the MinT value within 24 hr prior to the measurement explained 77% of the effect of MinT on survival while the latter 23% of the MinT values asymptotically weighed over the 5 days prior to measurement (Briga & Verhulst, 2015). Here we followed this same weighed time window to identify whether MinT also affected metabolic rate. A weighted approach provided a slightly better model fit than relative to the MinT in the 24 hr before measurement (ΔAICc = −1.5). Overall, our results show that BMR_m and SMR_m increase in winter and spring, a result consistent with previous studies on winter acclimatized small birds (Swanson, 2010) and this dynamic is best captured by variables quantifying day length and photoperiod (Table S1A) and for SMR_m an additional variable quantifying MinT (Table S1B). In the models below we always used these variables to correct for daily and seasonal variation in both metabolic traits.

2.3.3 | Aims 1 and 2: Metabolic ageing

Population level associations between trait values and age can be composed of two processes: (a) a within-individual change in trait value with age and (b) a between individual change due to

selective disappearance of individuals with certain trait values. We distinguished the contributions of these two processes using a within subjects centring approach (van de Pol & Verhulst, 2006; van de Pol & Wright, 2009). In this approach the within-individual changes are captured in a Δage term, which is the age at measurement mean centred per individual. Models with age (instead of Δage) gave conclusions consistent with those presented using Δage (results not shown), but using Δage always improved the model fit (BMR_m: ΔAICc = −9.8; SMR_m: ΔAICc = −13.5 for the best fitting models identified below) and hence we used Δage in all models. The between individual change is captured by the term life span, mean-centred across our population and we used this mean centred value in all analyses, except for the Cox Proportional Hazard analyses (see below). At the end of the experiment, 71 of the 407 birds were still alive. These had a mean age of 5.0 years (95% CI: 2.2–8.2). For these birds, the age at death is unknown and hence we gave them a life span of zero so that they contribute only to the intercept and not to the slope of the between individual change term life span. In this formulation, selective disappearance occurs when the coefficients of change within (Δage) and between individuals (life span) differ (van de Pol & Verhulst, 2006; van de Pol & Wright, 2009). Selective disappearance can also be tested for using survival analyses, which provides a straightforward way to analyse whether selective disappearance varies between the experimental groups or sexes and we hence used this approach here (see below Section 2.3.4).

It has previously been shown that different traits can display different age trajectories, a phenomenon known as asynchronous or mosaic ageing (Cevenini et al., 2008; Hayward et al., 2015; Rando & Wyss-Coray, 2021; Walker & Herndon, 2010). However, we are not aware of a general definition or procedure to distinguish asynchronous from synchronous ageing, or to quantify the degree of asynchrony. To identify whether age-related changes in BMR_m and SMR_m are correlated, we performed our analysis in three steps. In the first step, we identified the age trajectories of BMR_m and SMR_m using univariate general linear models. When traits show different age trajectories that differ in shape, ageing is asynchronous. Second, for traits that have the same age trajectory, we compared their ageing rates. We define the ageing rates of traits to be asynchronous when the absolute values of the standardized coefficients of the age trajectories are significantly different. Standardization here is relative to the trait-specific within-individual variance, to account for the difference in within-individual variance between traits. We used absolute values because traits can age in opposite directions, but still be strongly correlated when ageing is due to a shared underlying mechanism. Note that this approach is consistent with the different age trajectories criterion: when traits show age trajectories that differ in shape, for example, quadratic versus linear, this will be asynchronous, because the absolute standardized coefficient of age squared differs 'significantly' between the traits. In the third step, we quantified the correlation between traits directly using a bivariate analyses. In this model, we used the age trajectories identified in the aforementioned univariate models and we quantified the amount of within-individual correlation in trait value with age directly through the correlation of random slopes.

We tested a series of age trajectories, including no change with age or a change that is linear, quadratic, terminal (i.e. before death), the combination of linear and terminal and threshold models. We tested terminal changes by adding a terminal term, coded as a binomial factor for whether or not an individual died within the year following the measurement. Threshold models, are a more complex but regularly found trajectory in ageing studies (Briga et al., 2019; Douhard et al., 2017). In threshold models, change in a trait value starts from a specific age onwards (but not a fixed number of years before death as for terminal models), resulting in two (1 threshold) or three (2 thresholds) linear changes in trait values with age (Figure S4). Here we tested threshold models, but lacked the power to distinguish between one and two threshold models or to identify biologically relevant confidence intervals around the threshold ages. Therefore, we fitted threshold models only to test if they supported the conclusions based on the linear models, which they did (Tables S10–S12).

We tested whether (a) sex and (b) the environmental manipulations affected metabolic ageing by including the interaction between the age terms (Δage, Δage^2 or terminal year) and the sex or experimental manipulation terms. Sex was always coded as a two-level factor. To test whether the environmental manipulations affected the age trajectories of BMR_m or SMR_m we (a) used three-way interactions (e.g. Δage × development × adult; in which 'development' and 'adult' refer to the experimental manipulations during development and adulthood respectively), (b) grouped experimental manipulations into one factor with four levels and (c) because the HH group is the only group that showed reduced life span relative to all other groups, we also coded experimental group as a two-level factor, that is, with HH as one level relative to the three other groups (BB, BH, HB) pooled together. We here present the results of approaches (a) and (c), but note that the three approaches gave consistent conclusions. Interactions between sex and the environmental manipulations and higher order interactions between sex and the environmental manipulation age terms were never significant and we do not show them here.

Once we identified the best fitting age trajectories, we tested for differences in the rate of ageing between BMR_m and SMR_m. To this end, we compared the ratio of the absolute value of the coefficient of the Δage term in the aforementioned final models (Δage models in Table S4) relative to the within-individual variance for each trait. We standardized relative to within-individual variance because ageing is a within-individual change and hence we wanted to exclude variance due to between-individual differences. We estimated the within-individual variances in two ways. First, by subtracting the between-individual variance from the total variance, which somewhat overestimated the within-individual variance because it also includes the residual variance. Second, we used the fraction of the within-individual variance capturing changes in trait values with Δage, that is, the variance

explained by the random slopes. Note that for both approaches we maximized the estimates of within-individual variance by using the final models without the Δage term as fixed effect. We estimated the 95% confidence intervals (95% CI) around the trait's ageing rate by multiplying the error around the Δage coefficient of the aforementioned final models with a factor 1.96. We considered the differences in the rate of ageing between traits as statistically significant when these 95% CI did not overlap. We estimated these variance components using three approaches: (a) a frequentist approach with the aforementioned 'lmer' models, (b) a Bayesian approach with the 'mcmcglmm' function of the MCMCGLMM package (Hadfield, 2010) and (c) using the Bayesian approach with the 'brm' function of the BRMS package (Bürkner, 2017). Here we show the results of approach (a) and (c), but note that the three approaches gave consistent results (results not shown; see below for Bayesian specifications).

An approach to quantify the degree of asynchronous ageing is to estimate the within-individual correlation in ageing rate between BMR_m and SMR_m in a bivariate analysis. In this analysis, we also used the aforementioned final models (with the Δage term) and the amount of within-individual correlation in trait value with Δage was captured by the correlation of the random slopes. We performed this analysis using the final models (Δage models in Table S4) with the functions 'bf' and 'brm' of the package BRMS (Bürkner, 2017). Note that in this approach the sample size was somewhat reduced because the package required the data to be structured into matched BMR_m and SMR_m measurements. Here, we matched measurements within the same season within a year (March and April vs. September and October, Figure S3) with a mean difference between matched measurements of 22 days (4< 95% CI <80). This resulted in 1682 matches for 309 individuals. In all Bayesian analyses, we used flat or weakly informative priors and ran four chains with 1.5×10^6 iterations, a 10,000 iterations burn-in and a thinning interval of 100, yielding effective sample sizes of >10,000, a Rhat of 1.00 and low levels of autocorrelation (mean $r = -0.002$ with all $r < 0.1$).

2.3.4 | Aim 3: Associations with life span

To investigate the association between BMR_m or SMR_m and life span we performed Cox Proportional Hazard analyses (CPH) using the function 'coxme' of the package COXME (Therneau, 2019). To avoid pseudo-replication by repeated measurements, we used the first measurement of each individual. These BMR and SMR values were corrected for mass, temporal and seasonal covariates and we used these values for the CPH analyses. In the CPH models we included sex and the four experimental groups as covariates, as these have been shown to affect life span (Briga et al., 2017, 2019). In these analyses, aviary was included as a random intercept to account for the joint housing of birds. Of the 407 individuals for which we have a metabolic rate measurement, we monitored 336 until natural death and right censored (a) 48 individuals that were still alive after 8 years of monitoring and (b) 23 individuals that died by accident or were euthanized for welfare considerations. These 8 years of monitoring are consistent with the study on body mass ageing in Briga et al., (2019). CPH analyses require predictors to be proportional which was the case as indicated by the 'cox.zph' function ($\chi^2 = 2.49$, $p > 0.11$).

3 | RESULTS

3.1 | Basal metabolic rate

We first investigated the age trajectory of BMR_m within individuals. We tested for changes with age that were either linear, quadratic, terminal or a combination of linear and terminal. The best fitting shape was a linear decline with age (ΔAICc < −15.7; Figure 2a; Table S4A). Individuals lost on average 0.0024 W/year (−0.0034 <95% CI <−0.0014).

We then investigated whether the environmental manipulations that shortened life span, also accelerated BMR_m decline. High foraging costs during adulthood, but not during development (i.e. brood size

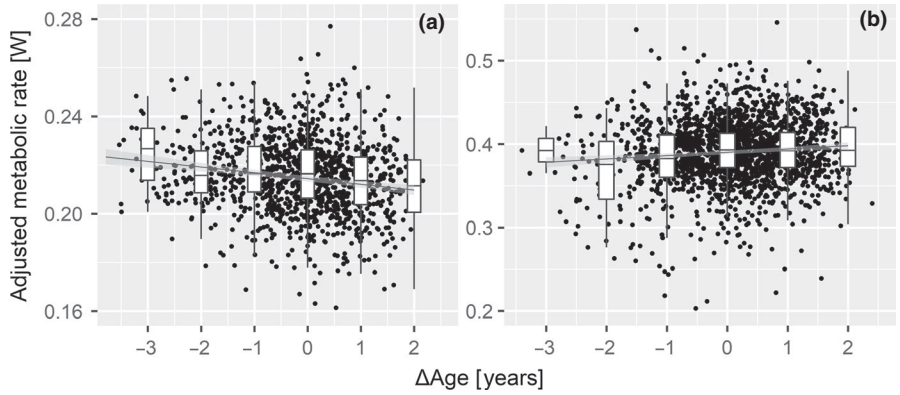

FIGURE 2 Within individuals (a) BMR_m decreased with age, while (b) SMR_m increased. Values are adjusted for mass, seasonal variation, experimental groups, sex and for SMR_m also the ambient temperature at measurement following the best fitting (Δage) models in Table S4. Δage is the mean centred within-individual age over all its measurements. Boxplots show medians, quartiles and 95% CI per year. Horizontal lines illustrate model fit and grey shaded area represents the 95% CI around the fit. Note the *Y*-axes on different scales between (a) and (b)

manipulation) decreased BMR_m (development: ΔAICc = +9.7; adulthood: ΔAICc = −12.6; Table S2). There was no evidence for different age trajectories (Table S4A) or for a difference in the magnitude of the linear decline between experimental groups (Figure 3a–d; Δage × manipulation: ΔAICc > +13.8; terminal year × manipulation: ΔAICc > +21.4; Table S5A,B). Testing the HH group against all other experimental groups supported the same conclusion (ΔAICc > +13.5). Females lived on average 2 months shorter than males (4%; Briga et al., 2019) and had a somewhat higher BMR_m, but this difference was not 'statistically significant' (ΔAICc = +4.7; Table S2). We also found no statistical support for a sex-specific BMR_m response to the environmental manipulations (ΔAICc > +11.6; Table S2) or for sex-specific BMR_m ageing (Figure 3e,f; ΔAICc > +19.2; Table S7). Thus, BMR_m decreased linearly with age and this decrease was independent of factors that affected life span.

We then investigated the association between BMR_m and life span. In the best fitting general linear model, the linear decline in BMR_m with age was indistinguishable between and within individuals, with their respective slopes of −0.0018 W/year (−0.0027 < 95% CI < −0.0010; Δage term in model 1 in Table S5A,B) and −0.0024 W/year (−0.0034 < 95% CI < −0.0014; life span term in model 1 in Table S5A,B), indicating no selective disappearance. This was confirmed with CPH models that showed neither a linear nor a quadratic association with life span (e.g. when individuals with intermediate BMR_m live longest; Figure 4; ΔAICc > +1.3; Table S9A). Thus, there was no association between BMR_m and life span.

3.2 | Standard metabolic rate

We first investigated the shape of the age trajectory, testing for linear, quadratic and/or terminal changes. In contrast to BMR_m, which *decreased* linearly with age, SMR_m *increased* linearly with age (ΔAICc < −4.0; Figure 2b; Table S4B). Individuals gained on average 0.0042 W/year (0.0019 < 95% CI < 0.0063). This coefficient seemed to increase with lower ambient temperature T_a at measurement, but this interaction did not improve the model fit (Δ age × T_a ΔAICc = +11 compared to the best fitting model in Table S4B).

We then investigated whether the manipulations that affected life span also affected SMR_m ageing. High foraging costs during adulthood, but not during development (i.e. brood size manipulation) decreased SMR_m (development: ΔAICc = +10.5; adulthood: ΔAICc = −67.6; Table S3). There was no evidence for different age trajectories (Table S4B) or in the increase between experimental groups (Figure 3g–j; Δage × manipulation: ΔAICc > +8.1; terminal year × manipulation: ΔAICc > +16.1; Table S6). Testing the HH group against all other experimental groups supported the same conclusion (ΔAICc > +11.8). Just as for BMR_m, sexes did not differ in their SMR_m (ΔAICc = +11.8; Table S3), in their SMR_m response to the environmental manipulations (ΔAICc > +21.9; Table S3), in their age trajectories or in their rate of SMR_m ageing (Figure 3k,l; ΔAICc > +22.1; Table S8). Thus, SMR_m increased linearly with age and this increase was independent of factors that affected life span.

Lastly, we investigated the association between SMR_m and individual variation in life span. In the best fitting model in Table S4B, the linear increase in SMR_m with age differed somewhat between and within individuals, with respective slopes of 0.0022 W/year (0.0004 < 95% CI < 0.0039; Δage term in model 1 in Table S6A,B) and 0.0042 W/year (0.0018 < 95% CI < 0.0063; life span term in model 1 in Table S6A,B), which suggests there could be some selective disappearance with respect to SMR_m, but note the overlapping

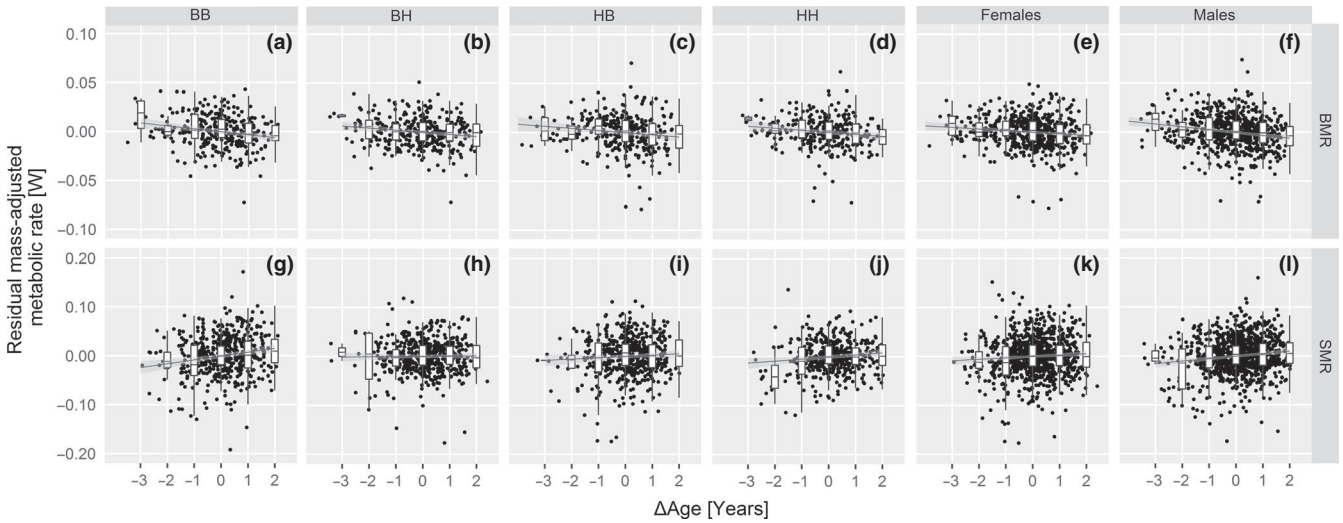

FIGURE 3 Within-individual changes in BMR_m and SMR_m are independent of environmental quality (BMR_m: a–d; SMR_m: g–j) and of sex (Females: e, k; Males: f, l). Experimental group combinations are abbreviated, in chronological order, such that the first letter stands for developmental environment (B for benign or small broods, H for harsh or large broods) and the second letter for adult environment (B for benign or low foraging costs, H for harsh or high foraging costs). BMR_m or SMR_m values are residuals corrected for mass, seasonal variation, ambient temperature at measurement (SMR_m), sex (a–d; g–j), experimental groups (e, f, k, l). Boxplots show medians, quartiles and 95% CI per year. Horizontal lines are group specific model fits (±95% CI)

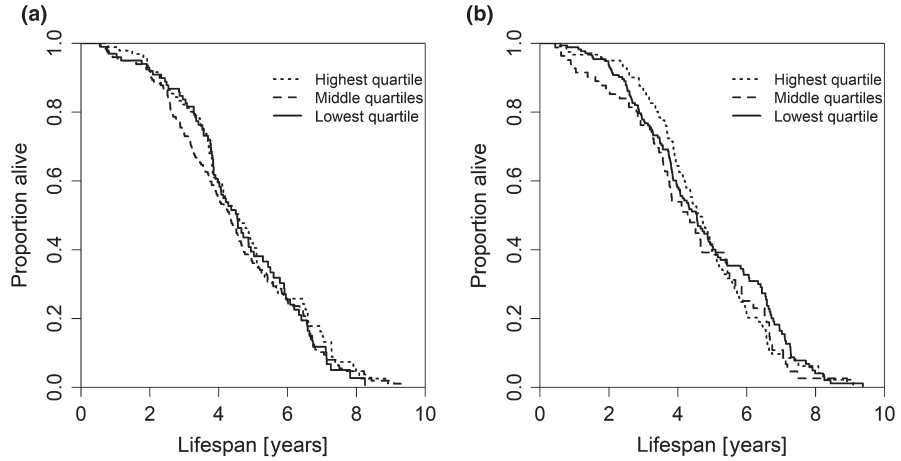

FIGURE 4 BMR_m (a) and SMR_m (b) were not associated with life span. To avoid pseudo-replication by repeated measurements, we used the first measurement of each individual. BMR_m or SMR_m values are residuals corrected for mass, seasonal variation, ambient temperature at measurement (SMR_m), experimental groups and sex, as in the survival analyses in Table S9. For graphical purposes, we show data in three groups: (i) the lowest quartile (<25%), (ii) the highest quartile (>75%) and (iii) the two middle quartiles (25%–75%) pooled as one line. Note however that we performed the analyses with BMR_m and SMR_m as continuous variables

95% CI. Indeed, in a CPH analyses we found little statistical support for selective disappearance for a linear association between SMR_m and life span (Figure 4; ΔAICc > +1.7; Table S9B) and even less for a quadratic association between SMR_m and life span (Figure 4; ΔAICc > +3.0; Table S9B). Thus, we found no evidence for an association between SMR_m and life span.

3.3 | Asynchronous ageing of BMR_m and SMR_m

BMR_m decreased on average −0.0024 W/year (−0.0034 < 95% CI < −0.0014), while SMR_m increased on average 0.0042 W/year (0.0019 < 95% CI < 0.0063). The between-individual variance in mean trait values (random intercept) explained 28% of the variance in BMR_m and 20% of the variance in SMR_m respectively. This was tenfold higher than the between-individual variance in within-individual change with age (random slope), which was small at 2.9% for BMR_m and 2.6% for SMR_m, indicating that individuals varied little in their metabolic rate age trajectories (relative to their differences in mean metabolic rates). Comparing the absolute value of these Δage coefficients relative to the within-individual variance indicated that BMR_m aged at a rate of 8.2 times the within-individual variance (4.9 < 95% CI < 11.5), while SMR_m aged more slowly at a rate of 2.5 times the within-individual variance (1.2 < 95% CI < 3.8). Estimates using only the fraction of within-individual variance in Δage (random slope) yields an even larger difference in ageing rates (BMR_m: 203, 122 < 95% CI < 284; SMR_m: 75, 36 < 95% CI < 115). Note that the 95% CI of these estimates did not overlap for either approach and hence the higher rate of ageing of BMR_m over SMR_m is statistically supported. Finally, using a bivariate model, we estimated a within-individual correlation in BMR_m and SMR_m with age (i.e. correlation in random slopes) of 0.09 (−0.29 < 95% CI < 0.37), showing that the age trajectories of BMR_m and SMR_m were not only different but also independent.

4 | DISCUSSION

Metabolic rate is a key physiological trait in ecology, life history and the biology of ageing. The minimum energy expenditure at thermoneutral temperatures or BMR_m has been quantified in many endothermic species and consistent with those previous studies (review: Elliott et al., 2015), including in zebra finches (Moe et al., 2009; Rønning et al., 2014), we found that BMR_m decreased with age. However, in contrast to BMR_m, SMR_m *increased* with age. In addition to a difference in direction, the traits also differed in their rate of ageing when expressed relative to their respective within-individual variation. Moreover, the age trajectories of the two traits were only weakly correlated within individuals, indicating that these metabolic traits aged independently. An experimental manipulation of increased foraging costs that decreased life span also decreased BMR_m and SMR_m, a result consistent with that of earlier studies in birds and laboratory rodents (Schubert et al., 2008, 2009; Vaanholt et al., 2007; Wiersma & Verhulst, 2005). However, metabolic ageing was independent of environmental quality and neither metabolic rate nor metabolic rate ageing were associated with life span in our study. Here we discuss four implications of our results in light of other studies on metabolism and ageing.

4.1 | Metabolic rate

The first implication focusses on the concept of metabolic rate as a trait. To the best of our knowledge, the independent asynchronous ageing of both metabolic traits is a new finding. We believe this finding is important because it indicates that these traits change with age independently, presumably because they are driven by different processes, especially thermoregulation is part of SMR but not of BMR (see below). We previously reported that despite BMR_m and SMR_m being repeatable over many years (0.30), the correlations between

them at the within-individual level were low ($0.04 < r < 0.22$; Briga & Verhulst, 2017). Our results show that these traits' independent ageing contributed to this finding. These results, together with that of others (Pettersen et al., 2018), show that 'metabolic rate', is not one trait. In this context, SMR_m is the metabolism at ambient temperatures that free-living endotherms encounter in their daily lives, indicating that SMR_m might be an ecologically more relevant trait than BMR_m.

4.2 | Metabolic ageing

Second, we discuss the possible physiological underpinnings of metabolic ageing. For BMR_m, the mechanism underlying its decrease with age is likely to involve reductions in the size of metabolically expensive organs, such as the heart, liver, kidneys and muscle, as was shown for humans and laboratory rodents (Roberts & Rosenberg, 2006). There is also evidence for a small age-associated decline in metabolism per unit of tissue (Roberts & Rosenberg, 2006) and body temperature (Blatteis, 2012; Florez-Duquet & McDonald, 1998; Weinert, 2010). To what extent these factors contributed to the decrease in BMR_m remains to be investigated.

Contrary to the decrease in BMR_m, SMR_m increased with age. This indicates that thermoregulation changed with age and we propose that this could reflect at least two underlying processes. Firstly, birds may need more energy to maintain body temperature as they age, for example, due to poorer insulation or reduced metabolic efficiency. Secondly, birds in this study had lower body temperatures at sub-thermoneutral ambient temperatures than at thermoneutral ambient temperatures (Briga & Verhulst, 2017), and tolerance for low body temperature may have declined with age. This might occur because the ability to warm up decreases with age, a phenomenon that occurs in laboratory rodents (Florez-Duquet & McDonald, 1998). A diminished tolerance for low body temperature would also explain the apparent contradictory findings that SMR_m decreased in low-quality environments but increased with age. We propose that this apparent contradiction arises because the low SMR_m in low-quality environments is a thermoregulatory energy saving mechanism, which older birds perhaps cannot afford. Thus, we propose that the mechanisms underlying the different age trajectories between BMR_m and SMR_m reflect declines in insulation and/or efficiency of heat production.

4.3 | Physiological processes mediating environmental effects on life span

Third, we evaluate the role of metabolic rate in mediating the effect of environmental and sex-specific differences in life span. Several previous studies in endotherms have revealed associations between BMR_m and life span (Burton et al., 2011; Rønning et al., 2016) and in humans, high BMR_m is associated with increased mortality risk (Ruggiero et al., 2008). We previously found that individuals experiencing high foraging costs both during development and in adulthood lived on average 6 months (12%) shorter than all other groups and that females lived 2 months (4%) shorter than males (Briga & Verhulst, 2017; Briga et al., 2019). If these differences in life span were to be mediated by high metabolic rate, we expected the HH group and females to show higher BMR_m and/or SMR_m. However, this is not the result we found. Instead, the environmental factors that decreased life span also decreased BMR_m and SMR_m. Even more, we found no consistent associations between life span and BMR_m or SMR_m, quantified either directly (Figure 4), through sex-specific differences in life span or through the experimental environmental manipulation of life span. Given the large sample size ($N > 400$ individuals), we assume that this negative result is not due to a lack of power. Therefore, it seems likely that our previously found environmental and sex-specific differences in life span are mediated by physiological pathways other than minimal energy metabolism. Some promising results indicate that glucose metabolism, insulin related pathways and the associated glucocorticoid levels are good candidates, although none of these provide a complete picture yet (Jimeno et al., 2017, 2018; Montoya et al., 2018; Regan et al., 2020) and other physiological mechanisms, such as oxidative stress and the immune system, warrant further study (De Coster et al., 2011; Froy et al., 2019; Peters et al., 2019; Simons et al., 2014; Speakman et al., 2015).

The birds in our study lived in captivity, which raises the question to what extent the ageing patterns observed here can be extrapolated to the wild. The decrease in BMR_m with age has been observed in a wide range of species and environments, including in free-living birds and mammals and hence there is little doubt that BMR_m ageing occurs in the wild (Elliott et al., 2015). However, it is not well-known what proportion of time animals in the wild spend in thermoneutral conditions, and hence what the ecological relevance of BMR is. Measurements of SMR are likely to have greater ecological relevance (e.g. Andreasson et al., 2019), assuming that our findings can be extrapolated to wild animals, which remains to be tested. However, if confirmed, our findings suggest that baseline energy requirements increase with age, in contrast to the current view, with repercussions for how much energy animals have available for reproduction and survival.

4.4 | The evolution of ageing

Fourth, we believe our study has some implications regarding the evolution of ageing. Evolutionary theories of ageing (Williams, 1957; Maynard-Smith, 1962), suggested that traits would evolve to age in synchrony, because '*natural selection will always be in greatest opposition to the decline of the most senescence-prone system*' (Williams, 1957). Our findings run counter to this prediction because BMR_m and SMR_m, together with our previous studies on body mass and bill colour (Briga et al., 2019; Simons et al., 2012, 2016), showed that traits have different age trajectories and ageing rates. The metabolic age trajectories are linear, the age trajectory of body mass was quadratic, and this varied with foraging costs in females, but not in males, and bill colour, a sexual signal, remained constant throughout adult life until a decline

in the terminal year. Our combined results thus show that traits age asynchronously in zebra finches, a phenomenon coined asynchronous or mosaic ageing (Cevenini et al., 2008; Rando & Wyss-Coray, 2021; Walker & Herndon, 2010) and which has also been observed in other systems such as Drosophila (*Drosophila* spp.) or Soay sheep *Ovis aries* (Bansal et al., 2015; Belsky et al., 2015; Hayward et al., 2015; Herndon et al., 2002; Nussey et al., 2009). The occurrence of mosaic ageing remains to be explained by evolutionary theory.

One of the requirements for natural selection to occur is that a trait is associated with fitness. Hence the evolution and plasticity of age trajectories and ageing rates will depend on the trait's association with fitness. In our study system, body mass and bill colour associated with life span (Briga et al., 2019; Simons et al., 2012, 2016), hence their age trajectories may be under selection. In contrast, BMR_m and SMR_m did not associate with life span and the lack of such associations raises the question whether variation in BMR_m and SMR_m can be considered of evolutionary importance. However, given that both BMR_m or SMR_m show life-long repeatability (e.g. Briga & Verhulst, 2017:0.3), have a heritable component in several species including in zebra finches (e.g. Rønning et al., 2007) and are consistently adjusted to environmental conditions (Schubert et al., 2009; Wiersma & Verhulst, 2005; Figure 3; Table S2 and S3), suggests that BMR_m and SMR_m are traits of functional importance. The lack of association between BMR_m or SMR_m, and life span can occur because an individual can adjust in many ways for changes in BMR_m or SMR_m because metabolic rate is a property emerging from all metabolic processes combined. For example, elderly birds can compensate for increases in SMR_m through decreased diurnal activity, increasing joint perching at night or other energy-saving mechanisms. Paradoxically, the lack of association between a trait and life span (or fitness) can also be the result of its functional importance, if canalization, a process in which traits will show less variation when that trait is important for fitness, is strong enough to reduce variation in a trait to low level (Boonekamp et al., 2018; Flatt, 2005; Waddington, 1942).

It is often implicitly assumed that factors changing life span will also alter ageing rate. To what extent and for what kind of traits this is the case remains to be identified (Bansal et al., 2015; Christensen et al., 2009; Rando & Wyss-Coray, 2021; Williams, 1999). Our study shows that the lifelong increase of foraging costs and sex-specific differences in life span, did not alter metabolic ageing (Figure 3). Hence, in our experiment, we found little evidence for such an association. Given the sample size (metabolic rate: $N > 3{,}000$ measurements), we assume that our negative results are not due to a lack of power. Interestingly, the (between-individual) variance in BMR_m and SMR_m ageing is a strikingly 10-fold smaller than the (between-individual) variance in mean for BMR_m and SMR_m. Hence, the lack of factors affecting BMR_m and SMR_m ageing and the small variance in BMR_m and SMR_m suggest a role for canalization (Boonekamp et al., 2018; Flatt, 2005).

We previously found environmental and sex-specific body mass ageing, but the onset or rates of ageing were not necessarily earlier or higher in shorter lived individuals (Briga et al., 2019). In contrast, the sexual signal bill coloration, through its terminal declining age trajectory, directly associates with life span (Simons et al., 2016). Apparently, factors affecting life span do not always affect ageing rate, a phenomenon which has also been suggested for genetic factors (Burger & Promislow, 2006). Predicting when or whether an environmental variable that alters life span will also affect the ageing of some traits remains a challenge to address both theoretically and empirically. The roles of the aforementioned hypotheses such as canalization, together with other hypotheses (e.g. Cohen et al., 2020), remain to be tested.

ACKNOWLEDGEMENTS

We thank many people for help with data collection, including Gerard Overkamp, Jorrick Wubs, Maite Punter, Egbert Koetsier and the late René Beamonte-Barrientos. We thank two anonymous reviewers and the editors of Functional Ecology for useful comments that improved the manuscript and Jennie Crawley for the English check. We thank the Ella and Georg Ehrnrooth Foundation for funding (MB) and the University of Turku for funding open access publishing through the FinELib agreement.

CONFLICT OF INTERESTS

The authors declare no conflict of interests.

AUTHORS' CONTRIBUTIONS

M.B. and S.V. conceived the ideas, designed the methodology, analysed the data and wrote the manuscript. M.B. collected the data.

DATA AVAILABILITY STATEMENT

Data available from the Dryad Digital Repository https://doi.org/10.5061/dryad.pzgmsbck8 (Briga & Verhulst, 2021).

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

How to cite this article: Briga M, Verhulst S. Mosaic metabolic ageing: Basal and standard metabolic rates age in opposite directions and independent of environmental quality, sex and life span in a passerine. *Funct Ecol*. 2021;00:1–14. https://doi.org/10.1111/1365-2435.13785