Received: 21 September 2017 | Revised: 25 September 2018 | Accepted: 30 September 2018

DOI: 10.1111/1365-2435.13228

RESEARCH ARTICLE

Functional Ecology | BRITISH ECOLOGICAL SOCIETY

Longevity and life history coevolve with oxidative stress in birds

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Funding information
Hungarian Eötvös Scholarship, Grant/Award Number: MÁEÖ2017_16/156845; Hungarian Research Foundation, Grant/Award Number: K 113108; Postdoctoral grant, Grant/Award Number: PD 121166; János Bolyai Research Scholarship; Exploratory Research, Grant/Award Number: PN-III-P4-ID-PCE-2016-0404; Hungarian National Excellence Program; Higher Education Institutional Excellence Programme

Handling Editor: David Costantini

Abstract

1. The mechanisms that underpin the evolution of ageing and life histories remain elusive. Oxidative stress, which results in accumulated cellular damages, is one of the mechanisms suggested to play a role.
2. In this paper, we set out to test the "oxidative stress theory of ageing" and the "oxidative stress hypothesis of life histories" using a comprehensive phylogenetic comparison based on an unprecedented dataset of oxidative physiology in 88 free-living bird species.
3. We show for the first time that bird species with longer lifespan have higher non-enzymatic antioxidant capacity and suffer less oxidative damage to their lipids. We also found that bird species featuring a faster pace-of-life either have lower non-enzymatic antioxidant capacity or are exposed to higher levels of oxidative damage, while adult annual mortality does not relate to oxidative state.
4. These results reinforce the role of oxidative stress in the evolution of lifespan and also corroborate the role of oxidative state in the evolution of life histories among free-living birds.

KEYWORDS
ageing, antioxidants, comparative biology, life history, lifespan, lipid peroxidation, mortality, oxidative damage

1 | INTRODUCTION

Gerontology is concerned with the evolution of ageing or senescence, that is, the progressive loss of physiological functions with advanced age, which demographically manifests as decreased reproductive and survival rates (Ricklefs, 2008). Life-history theory is concerned with the evolution of reproductive and survival rates observed at both the individual and species level (Stearns, 1989). In general, species that feature a slower life-history strategy are hypothesized to invest more into self-maintenance at the expense of reproduction and hence show delayed ageing. On the other hand, reproduction is suggested to take precedence over self-maintenance in those species that exhibit a faster pace-of-life (Promislow & Harvey, 1990). Consequently, life-history theory is intertwined with ageing theories. Indeed, life-history pace and ageing rate appear to have coevolved among free-living species (Lemaître et al., 2015). However, what physiological mechanisms govern variation in lifespan (gerontology perspective) and underpin the inverse relationship between reproduction and survival rates (life-history theory perspective) remain central questions both on cross-individual and cross-species levels (Flatt & Schmidt, 2009).

The "disposable soma theory" is a general theory, which argues that increased investment into growth and reproduction precludes the proper maintenance of the soma and this ultimately manifests as increased mortality and/or accelerated ageing (Haussmann & Treidel, 2015; Kirkwood & Austad, 2000; Lemaître et al., 2015). However, the underlying mechanisms that cause this somatic failure remain elusive. Two mutually non-exclusive hypotheses that are mechanistic specifications of the disposable soma theory are the "oxidative stress theory of ageing" (OSTA) (Finkel & Holbrook, 2000; Sohal & Weindruch, 1996) and "oxidative stress hypothesis of life histories" (OSLH) (Costantini, 2008; Monaghan, Metcalfe, & Torres, 2009). Both theories nominate oxidative stress as a prominent candidate mechanism for the evolution of ageing in particular (OSTA) and the evolution of life-history strategies in general (OSLH). Below, we detail these two hypotheses.

Oxidative stress occurs when the production of reactive oxygen species (ROS) exceeds the levels that is countered by antioxidative defence and repair mechanisms (Cohen, de Magalhães, & Gohil, 2010; Haussmann & Treidel, 2015). During phases of oxidative stress, lipid, protein and DNA damage can occur. OSTA posits that the build-up of damage to cellular structural elements or defence/repair systems, as well as the disruption of normal redox signalling plays a major role in loss of bodily functions and the aetiology of age-related diseases (Barja, 2013; Finkel & Holbrook, 2000; Kirkwood & Kowald, 2012; Sohal & Orr, 2012; Sohal & Weindruch, 1996). From a comparative perspective, the balance between production and neutralization of ROS is thought to have coevolved with the expected longevity of the species (Barja, 2013; Cohen et al., 2010; Kirkwood & Austad, 2000; Ricklefs, 2008). However, OSTA was criticized over the past two decades mainly based on studies performed on model organisms with either knockout or overexpressed antioxidant genes that do not show the predicted effect on lifespan (reviewed by Bokov, Chaudhuri, & Richardson, 2004; de Magalhães & Church, 2006; Gems & Doonan, 2009; Salmon, Richardson, & Pérez, 2010; Sohal & Orr, 2012). Nonetheless, these studies are insufficient to definitively disprove the OSTA for several reasons (Kirkwood & Kowald, 2012). First, the oxidative physiological system is a complex network in which antioxidant enzymes do not work in isolation (Kirkwood & Kowald, 2012), potentially explaining why the genetic enhancement of a single antioxidant enzyme has even resulted in harmful effects (Bokov et al., 2004; Kirkwood & Kowald, 2012). Second, the most vital currency of the OSTA is the amount of oxidative damage (Barja, 2013; Sedensky & Morgan, 2006; Sohal & Weindruch, 1996) because inference from the level of antioxidants alone is ambiguous (Costantini & Verhulst, 2009; Monaghan et al., 2009). However, oxidative damage is seldom examined (Bokov et al., 2004). Third, model organisms are often inbred, short-lived and housed under benign conditions; therefore, results obtained using these organisms might not be applicable to free-living ones that face environmental challenges, feature diverse ecologies, ageing patterns and lifespans and show anti-ageing adaptations that evolved in their natural environment (Flatt & Schmidt, 2009; Salmon et al., 2010; Vleck, Haussmann, & Vleck, 2007).

The OSTA concerns the disruption of the redox homeostasis and argues that the resultant unrepaired oxidative damage affects ageing (Kirkwood & Kowald, 2012). Two key adaptations appear to be relevant for slower ageing and/or longer lifespan: lower rate of ROS production and lower polyunsaturated fatty acid (PUFA) content of membranes (reviewed by Barja, 2013; Pamplona, Barja, & Portero-Otín, 2002; Pamplona & Barja, 2011; Sanz, Pamplona, & Barja, 2006). A lower ROS level has obvious physiological advantages, while lower PUFA content renders membranes higher resistance against peroxidative damage (Hulbert, Pamplona, Buffenstein, & Buttemer, 2007; Pamplona et al., 2002). Interspecific comparative studies demonstrated that cell cultures originating from long-lived species are more resistant against oxidative challenges than those of short-lived ones (Harper et al., 2011; Miller, Williams, & Kiklevich, 2011) and that longer lifespan coevolves with a lower rate of ROS generation (Delhaye et al., 2016; Lambert et al., 2007; Pamplona & Barja, 2011; Shi, Buffenstein, Pulliam, & Remmen, 2010) and lower membrane PUFA content (Barja, 2013; Buttemer, Battam, & Hulbert, 2008; Galván et al., 2015; Hulbert et al., 2007; Pamplona & Barja, 2011; Pamplona et al., 2002). A supposed consequence of lower ROS generation and lower membrane PUFA content of long-lived species is their lower level of oxidative lipid damage. However, the phylogenetic covariation between lifespan and oxidative lipid damage was not hitherto demonstrated, despite being a centrepiece to our understanding of lifespan variation among animals (Blount, Vitikainen, Stott, & Cant, 2016; Buttemer, Abele, & Costantini, 2010; Costantini, Rowe, Butler, & McGraw, 2010; Monaghan et al., 2009; Shi et al., 2010).

The OSLH differs from the OSTA by suggesting that oxidative stress is the mechanism that governs the covariation among life-history traits along the slow–fast continuum of life-history pace. OSLH postulates that increased fecundity causes an inevitable oxidative stress and that survival is impaired by oxidative stress; therefore, oxidative stress is thought to mediate the trade-off between investment into reproduction and self-maintenance (reviewed by Blount et al., 2016; Speakman, 2008; Costantini, 2008, 2014 ; Isaksson, Sheldon, & Uller, 2011; Monaghan et al., 2009; Metcalfe & Alonso-Alvarez, 2010; Metcalfe & Monaghan, 2013; Selman, Blount, Nussey, & Speakman, 2012). Evidence for the OSLH is controversial (Blount et al., 2016; Isaksson et al., 2011; Metcalfe & Monaghan, 2013; Monaghan et al., 2009; Selman et al., 2012), which has led to scepticism as to whether the OSLH can be seen as a unifying theory across multiple species. Nevertheless, a recent meta-analysis found that oxidative damage in different tissues is higher in breeders that have high reproductive effort as compared with breeders that have lower reproductive effort (Blount et al., 2016). However, we currently lack phylogenetic comparative studies that measure oxidative damage and assess its association with reproductive effort, survival and life-history pace (Cohen et al., 2010; Costantini, 2008; Monaghan et al., 2009; Selman et al., 2012). Regarding life-history pace, Calhoon, Jimenez, Harper, Jurkowitz, and Williams (2014) compared slow-lived tropical bird species with their fast-lived temperate sister taxa and found that tropical ones have mitochondria with less cardiolipin, the most common membrane PUFA. This finding suggests that

species with slower life histories might suffer less oxidative damage to lipids. However, this prediction remains to be tested.

The comparative approach is thought to be a lucrative tool to detect robust relationships between physiological mechanisms and traits that are indicative of ageing and life-history pace over a wide range of free-living organisms (Barja, 2013; Blount et al., 2016; Cohen et al., 2010; Ricklefs, 2008; Shi et al., 2010; Vleck et al., 2007). Nonetheless, comparative tests of the oxidative damage prediction of OSTA are scarce and the existing ones are frequently based on pairs of sister taxa or a handful of distantly related species (Buttemer et al., 2010; Vleck et al., 2007) or do not control for phylogeny and body mass (Shi et al., 2010; Speakman, 2005). The most comprehensive comparative test of the OSLH investigated antioxidants without quantifying oxidative damage (Cohen et al., 2008), though the latter is highly desirable (Buttemer et al., 2010; Monaghan et al., 2009; Selman et al., 2012).

Here, we set out to test OSTA and OSLH using a comprehensive comparative study based on 88 free-living European bird species. This study is the first to measure lipid damage and non-enzymatic antioxidants for such a large number of bird species. We tested two important predictions of the OSTA, namely that long-lived species (a) suffer less oxidative damage (Bokov et al., 2004; Buttemer et al., 2010; Kirkwood & Austad, 2000; Sohal & Weindruch, 1996) and (b) feature higher antioxidant capacity (Bokov et al., 2004) (see prediction 1.1 in Supporting information Appendix S1: Table S1). We also tested key predictions of the OSLH, namely that (a) species with higher reproductive effort and faster pace-of-life inevitably suffer more oxidative damage and cannot invest heavily into antioxidant defence (see predictions 2.1 and 2.3 in Supporting information Appendix S1: Table S1), and (b) this oxidative cost of reproduction will contribute to their lower annual survival rate (Blount et al., 2016; Metcalfe & Monaghan, 2013; Monaghan et al., 2009; Selman et al., 2012) (see prediction 2.2 in Supporting information Appendix S1: Table S1).

2 | MATERIALS AND METHODS

2.1 | Fieldwork

Fieldwork was carried out between 2011 and 2013. We captured a total of 601 individual birds with mist-nets at various sites across Romania during their breeding season (Vágási et al., 2016). Detailed description of fieldwork can be found in the Supporting information Appendix S1 and elsewhere (Vágási et al., 2016).

2.2 | Biochemical assays

We measured three non-enzymatic antioxidant markers (total antioxidant status, TAS; uric acid, UA; and total glutathione, tGSH) and a marker of peroxidative damage to membrane lipids (malondialdehyde, MDA; detailed protocols can be found in the Supporting Information Appendix S1). We also computed residual TAS, TASua, i.e. TAS corrected for UA (Supporting information Appendix S1) and used as the fifth redox state variable in the analyses. None of the markers were altered by handling time or sample storage duration (Supporting information Appendix S1: Table S2). All the markers we measured have previously been shown to be associated with fitness parameters in wild-living organisms. Decreased non-enzymatic antioxidant levels could indicate the cost of increased reproductive effort (see, e.g., Alonso-Alvarez et al., 2004; Wiersma, Selman, Speakman, & Verhulst, 2004). Similarly, oxidative damage to lipids (e.g., reactive oxygen metabolites and MDA) might indicate the cost of fast early-life growth (Metcalfe & Alonso-Alvarez, 2010) or high reproductive effort in both birds and mammals (Blount et al., 2016; Stier, Reichert, Massemin, Bize, & Criscuolo, 2012; Xu, Yang, Speakman, & Wang, 2014).

2.3 | Ageing, life-history and confounding variables

It is within the scope of OSTA to answer why species differ so widely in their maximum lifespan potential (MLSP) (Barja, 2013; Cohen et al., 2010; de Magalhães & Church, 2006). Despite known limitations (de Magalhães & Costa, 2009), MLSP is an acceptable indicator of the rate of ageing (Baudisch, 2011; de Magalhães & Church, 2006). This is because MLSP is thought to be dependent largely on intrinsic conditions (i.e., physiological and cellular functioning) contrary to mean lifespan (the inverse of annual mortality rate; see Supporting Information Appendix S1), which is mostly determined by extrinsic conditions (Barja, 2013). Therefore, MLSP is viewed as an upper margin for longevity, one that is allowed by physiological deterioration. We retrieved MLSP data from the manually curated AnAge database build 13 (de Magalhães & Costa, 2009) together with the sample size of individual recoveries and whether the MLSP data derive from wild-living or captive individuals (see, e.g., Galván et al., 2015; Supporting Information Appendix S1).

Life history was characterized by brood value, annual adult mortality rate and pace-of-life. Brood value gives the quantile contribution of a single average clutch to the lifetime fecundity using the formula clutch size/(clutch size × broods per year × reproductive lifespan), where mean reproductive lifespan is 1/annual mortality (sensu Bókony et al., 2009). Brood value is higher (closer to 1) in species that have elevated current reproductive investment contrary to ones that give priority to future reproduction (i.e., have brood value closer to 0). Mean clutch size and average number of broods per year were gathered from (Snow, Perrins, & Cramp, 1998). The fact that brood value is significantly and positively related to annual adult mortality rate, but it is unrelated to MSLP (Supporting information Appendix S1: Table S4), shows that mortality rate is the proper life-history trait for testing the potential mediatory role of oxidative stress in the reproduction–survival trade-off as formulated by the OSLH. Adult annual mortality rate was obtained from two large datasets (Møller, 2006; Székely, Liker, Freckleton, Fichtel, & Kappeler, 2014) and was complemented from additional sources (see Supporting information Appendix S1: Table S5). Adult mortality rates retrieved from the three sources were strongly positively correlated (Spearman's rank correlation, all $rho > 0.69$, all $df > 23$, all $p < 0.001$);

thus, the final values used in the analysis were obtained by averaging these datasets. The covariation among six life-history traits (body mass (Dunning, 2008), egg mass, clutch size, incubation period, fledging period (Snow et al., 1998) and MLSP; all $\log_{10}$-transformed) was used to extract an axis that describes the pace-of-life (see, e.g., Cohen et al., 2008). This axis was found by means of phylogenetically controlled principal component analysis (PCA; see Supporting Information Appendix S1). The first two principal components (PC1 and PC2) explained 70.39% and 15.07%, respectively, of variation in life histories (Supporting information Appendix S1: Table S6). PC1 is positively loaded for all life-history traits but clutch size (Supporting information Appendix S1: Table S6) and is interpreted as the axis of slow–fast pace-of-life syndrome ("POLS axis") with higher values marking slow-living species. PC2, having high load only for MLSP (Supporting information Appendix S1: Table S6), produced similar results to MLSP and hence was not considered.

2.4 | Statistical analyses

We build phylogenetic generalized least squares (PGLS) models controlled for phylogeny (Supporting information Appendix S1: Figure S1) and body mass, as suggested (Buttemer et al., 2010). The statistical analyses are detailed in the Supporting Information Appendix S1. Briefly, each PGLS model was based on the entire species pool and models were weighted by within species sample size (i.e., sampling effort). We included multiple redox state markers into the models as explanatory variables because these covary (Vágási et al., 2016), and this approach allowed to assess whether any of the oxidative traits is related to the response variable after controlling for the effects of other redox parameters. All variables were $\log_{10}$-transformed to meet the normality assumption. The full models were reduced to minimal adequate models by backward stepwise elimination with the more permissive criterion of $p < 0.1$ in order to retain marginal explanatory terms as well (i.e., $0.05 < p < 0.1$). Body mass was always retained in the minimal models indifferent of its significance level (see Supporting information Appendix S1: Table S7 and related text). Given that physiological and life-history traits often intercorrelate, we verified whether there is a multicollinearity problem in the models by computing the variance inflation factors (VIFs; Supporting information Appendix S1) within each minimal adequate model and found that values were all below the more conservative VIF < 5 threshold (max VIF = 3.40 for MDA in model no. 2; VIF < 2.07 in the rest of the models). Therefore, multicollinearity is unlikely to bias our results. All figures were produced using raw data. Fitted lines and associated standard errors were obtained from the respective minimal models (see Supporting Information Appendix S1). All statistical analyses were carried out in R 3.2 (R Core Team, 2015).

To test the OSTA, we constructed model no. 1 (Supporting information Appendix S1: Table S1 section (a)). We used MLSP as response variable, while the five redox markers were entered as explanatory variables. Additionally, body mass and the length of developmental period (sum of incubation and fledging periods) were also added as covariates, as these can strongly influence both the rate of actuarial senescence and the magnitude of oxidative stress (Cohen et al., 2008; de Magalhães & Church, 2006; Speakman, 2005) and could confound the association between longevity and oxidative state (de Magalhães & Church, 2006). We tested whether the results of the latter model are sensitive to inclusion of species with MLSP based on tiny and small sample sizes or MLSP based on captive populations (see also Galván et al., 2015 and Supporting information Appendix S1).

To test the predictions of the OSLH, we constructed the models no. 2–8 (Supporting information Appendix S1: Table S1 section (b)). The first five models (models no. 2–6) address the prediction that increased investment into reproduction entails oxidative stress costs. Therefore, the five redox state markers were set as response variables in separate models and brood value was set as covariate together with the confounding variables such as body mass and redox variables that covary with the response marker. Model no. 7 assessed the prediction that adult annual mortality rate is contingent upon oxidative physiology. This model contained adult mortality rate as dependent variable and body mass as well as redox markers as covariates. Model no. 8 tested the prediction that the covariation of life-history characters along the slow–fast pace-of-life continuum might be governed by oxidative homeostasis. For this, PC1 ("POLS axis") was modelled as a function of oxidative stress parameters, while body mass was omitted as it has been part of the PCA analysis.

3 | RESULTS

3.1 | Lifespan

The model no. 1 that tested OSTA showed that longer lifespan is related to higher antioxidant capacity (TAS) and lower level of oxidative damage to lipids (MDA) (Table 1, Figure 1a,b). Similar results were obtained when we excluded species whose MLSP data are derived from "tiny" and "small" sample sizes, or from captive animals (Supporting information Appendix S1: Table S8).

TABLE 1 Minimal adequate model no. 1 for addressing the hypothesis that longevity is related to oxidative physiology (see Supporting information Appendix S1: Table S1 section (a))

Model no.	Response	Predictor	$\beta \pm SE$	t	p
1	MLSP	Body mass	0.15 ± 0.05	3.30	**0.002**
		TAS	0.80 ± 0.26	3.05	**0.003**
		MDA	−0.62 ± 0.22	2.90	**0.005**

Note. Significant relationships are marked in bold.

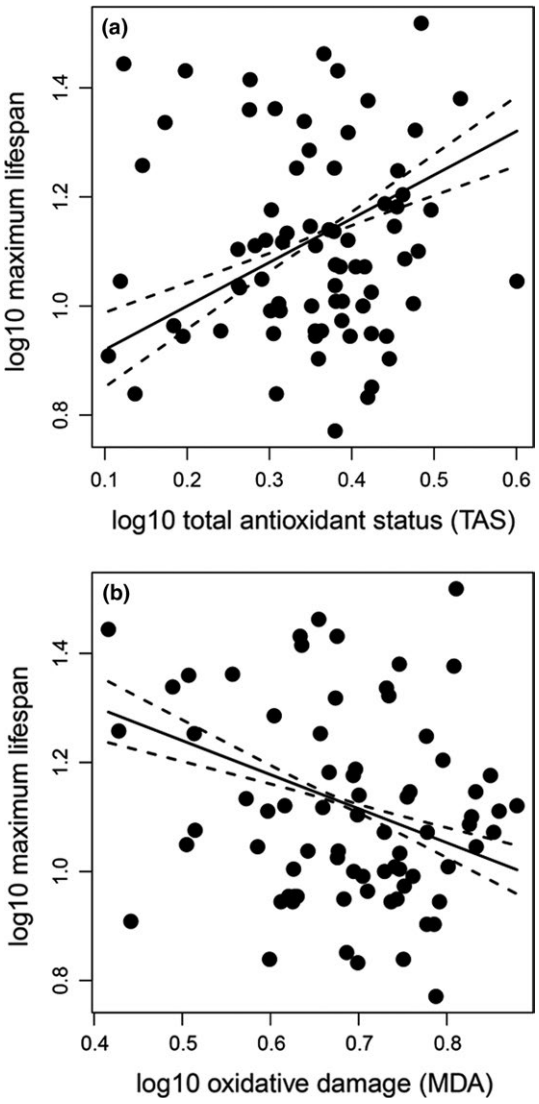

FIGURE 1 Longevity and oxidative state. Maximum lifespan potential is positively associated with total antioxidant status, TAS (a) and negatively with oxidative damage to lipids, MDA (b). Model fits ± *SE* (continuous and dashed lines, respectively) are based on model no. 1 (see the Materials and Methods)

3.2 | Life histories

We tested the oxidative stress cost of reproduction in model no. 2 through 6. These models showed that species that prioritize allocation towards current reproductive effort (i.e., have higher brood value) have significantly lower TAS, residual TAS (i.e., TASua) and tGSH levels, but increased UA concentration (Table 2, Figure 2). The degree of oxidative damage to lipids (MDA) was unrelated to brood value (Table 2).

We tested whether higher level of oxidative stress is associated with an increased mortality (model no. 7). Adult annual mortality rate was not related to any oxidative stress marker but only to body mass; larger species had lower annual mortality rates (Table 3). In addition, we tested the covariation of life-history pace and oxidative state (model no. 8). This model indicated that species featuring a faster pace-of-life syndrome (i.e., have lower PC1 value) suffer higher oxidative damage to lipids (MDA) (Table 3, Figure 3).

4 | DISCUSSION

4.1 | Oxidative stress theory of ageing

According to our study, species with longer lifespan suffer less oxidative damage to lipids. This finding is probably the consequence of long-lived species having membranes that are exposed to less ROS (Barja, 2013; Buttemer et al., 2010; Delhaye et al., 2016) and that are constitutively less vulnerable to oxidation insults due to their lower PUFA content (Buttemer et al., 2008; Galván et al., 2015; Hulbert et al., 2007; Naudí et al., 2013). This finding supports a cornerstone prediction of the OSTA and has important implications.

Lipids are one of the major targets of oxidation processes (Monaghan et al., 2009; Pamplona & Barja, 2011). Peroxidative damage to phospholipids can induce mitochondrial dysfunction via altered membrane fluidity and proton gradient, ultimately contributing to ageing (Paradies, Petrosillo, Paradies, & Ruggiero, 2010). Carbonyl compounds are reactive products of lipid peroxidation (e.g., dialdehydes as MDA), have longer half-life than ROS, can migrate through membranes with ease to cause damage distant to their place of formation and can damage cellular macromolecules by forming adducts with proteins, DNA and membrane lipids (e.g., advanced lipoxidation and glycation end products; Kudryavtseva et al., 2016; Monaghan et al., 2009; Pamplona & Barja, 2011; Sohal & Orr, 2012). Longevity was indeed found to be inversely related to advanced glycation end products (Sell et al., 1996), MDA–lysine adducts (Ruiz et al., 2005) and damages to mitochondrial DNA (Pamplona & Barja, 2011; Sanz et al., 2006) as well as proteins (Shi et al., 2010; Sohal & Orr, 2012). Finally, the adducts of lipid peroxidation products and regulatory proteins can derange virtually all important physiological and metabolic pathways that are functionally linked to ageing (reviewed by Kudryavtseva et al., 2016).

Most of the earlier cross-species analyses showed a negative association between MLSP and antioxidant levels, opposing the prediction of the OSTA (reviewed by Barja, 2013; Sanz et al., 2006). This was interpreted through an evolutionary lens in which antioxidants are tracking the level of oxidative stress, and since long-lived species experience less oxidative stress, they are not selected for constitutively elevated antioxidant defence (Barja, 2013; Cohen et al., 2008; Pamplona & Barja, 2011). However, most previous comparative studies were based on a small sample of species and did not correct for confounding effects of body mass and phylogeny (except Cohen et al., 2008) or blood sampling was not strictly limited to the breeding stage. On the other hand, the level of antioxidants was also found to be positively related to longevity (reviewed by Buttemer et al., 2010; Salmon et al., 2010), especially when ROS production is less pronounced (Kirkwood & Kowald, 2012), such as in species with long lifespan (Barja, 2013). Our finding lines up beside these studies by showing that under physiologically challenging conditions, such as breeding, species with longer lifespan have elevated non-enzymatic

TABLE 2 Minimal adequate models no 2–6 for testing the prediction that reproduction has oxidative costs (see Supporting information Appendix S1: Table S1 section (b))

Model No.	Response	Predictor	$\beta \pm SE$	t	p
2	TAS	Body mass	−0.02 ± 0.02	0.72	0.473
		UA	0.23 ± 0.13	1.82	0.073
		MDA	0.37 ± 0.14	2.73	**0.008**
		Brood value	−0.10 ± 0.05	2.21	**0.031**
3	UA	Body mass	0.01 ± 0.02	0.68	0.500
		TAS	0.26 ± 0.12	2.15	**0.036**
		MDA	0.70 ± 0.10	6.77	**<0.001**
		Brood value	0.09 ± 0.04	2.28	**0.027**
4	TASua	Body mass	−0.02 ± 0.10	0.19	0.854
		MDA	1.00 ± 0.37	2.69	**0.009**
		Brood value	−0.66 ± 0.21	3.23	**0.002**
5	tGSH	Body mass	0.21 ± 0.06	3.32	**0.002**
		Brood value	−0.45 ± 0.13	3.47	**0.001**
6	MDA	Body mass	−0.05 ± 0.03	1.85	0.069
		TAS	0.30 ± 0.11	2.83	**0.006**
		UA	0.57 ± 0.08	6.73	**<0.001**

Note. Significant relationships are marked in bold.

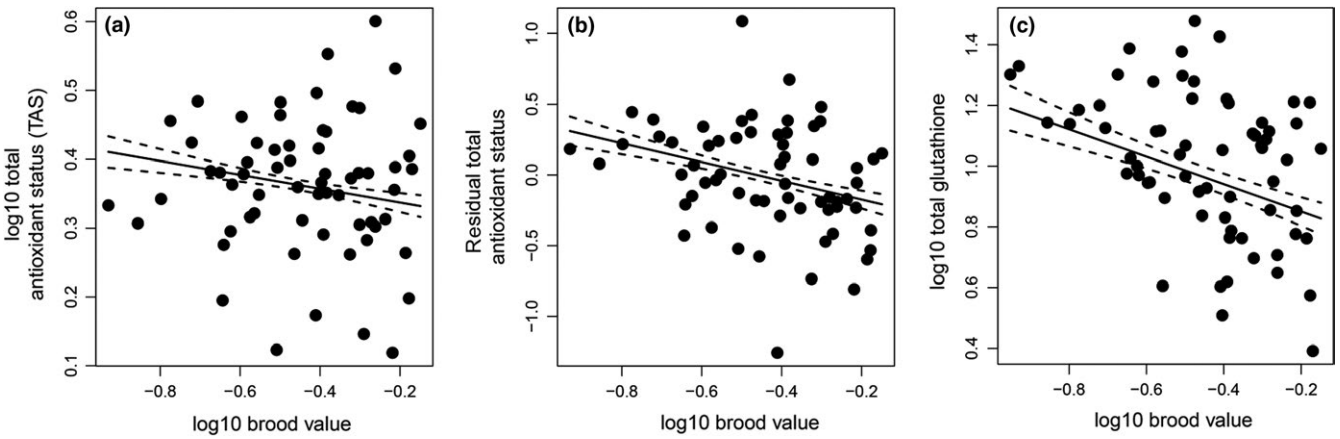

FIGURE 2 The oxidative cost of reproduction. Higher brood value (i.e., higher investment into current reproduction) is associated with significantly lower total antioxidant status, TAS (a), residual total antioxidant status, TASua (b) and total glutathione level, tGSH (c). Model fits ± *SE* (continuous and dashed lines, respectively) are based on models no. 2, 4 and 5, respectively

TABLE 3 Minimal adequate models no. 7 and 8 for testing the predictions that survival rate is inversely related to oxidative stress and pace-of-life is positively related to oxidative stress (see Supporting information Appendix S1: Table S1 section (b))

Model No.	Response	Predictor	$\beta \pm SE$	t	p
7	Adult mortality	Body mass	−0.13 ± 0.02	5.92	**<0.001**
8	PC1 ("POLS axis")	MDA	−1.01 ± 0.30	3.37	**0.001**

Note. Significant relationships are marked in bold.

antioxidant levels. This higher antioxidant capacity can help them to avoid somatic damage during metabolically stressful periods and to safeguard future fitness potential of the parents. Besides, this higher defence capacity might also shield the offspring, given that the oxidative state of parents can be passed down to their young, e.g., via germ cell damages (Barja, 2013; Blount et al., 2016; Costantini et al., 2010). Both the higher residual reproductive value of parents and the prime early-life conditions of their young are crucial in species that evolved slow-paced life histories.

Longevity was positively related only to TAS, but not to UA or GSH. UA was proposed to play a key role in scavenging oxidants (Vleck et al., 2007), such as lipid peroxidation products (Cohen et

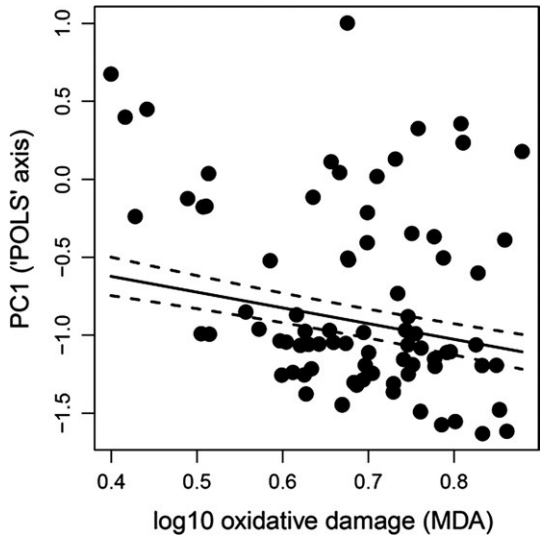

FIGURE 3 Pace-of-life in relation to oxidative state. PC1 (an inverse "pace-of-life axis") is negatively related to oxidative damage to lipids, MDA. Model fit ± *SE* (continuous and dashed lines, respectively) is based on model no. 8

al., 2008). Nonetheless, UA levels might better reflect the degree of protein catabolism and have limited antioxidant function (Hõrak & Cohen, 2010). Our study, in concert with the study of Cohen and co-authors (Cohen et al., 2008), suggests that UA cannot explain lifespan variation among species. GSH is thought to be a versatile intracellular antioxidant due to its thiol groups, as co-factor of glutathione peroxidase and via the GSH:GSSG redox ratio (Bokov et al., 2004; Sohal & Orr, 2012). However, our study does not support this view. The association of longevity with UA and GSH might not be simple because both of these antioxidants might also cause, rather than diminish, mitochondrial oxidative damages and can generate further radicals (Cadenas & Davies, 2000; Dröge, 2002).

4.2 | Oxidative stress hypothesis of life histories (OSLH)

Although reproduction can be the most demanding life-history stage (Speakman, 2008), there is surprisingly little evidence on whether it causes oxidative stress or not (Metcalfe & Monaghan, 2013; Monaghan et al., 2009). This is partly because the majority of earlier studies did not measure ROS production or oxidative damage, but only antioxidants (Monaghan et al., 2009). For instance, it was shown that zebra finches *Taeniopygia guttata* that lay more clutches per lifetime and raise more offspring per clutch pay a cost in terms of diminished antioxidant defences (Alonso-Alvarez et al., 2006, 2004 ; Wiersma et al., 2004). Our results also indicate that an increased investment into current reproduction (i.e., high brood value) coevolved with dampened antioxidant defence across birds (but see Cohen et al., 2008). Interestingly, in contrast with the adverse effects of reproduction on TAS and GSH, brood value was positively related to UA levels, which supports the view that UA might better reflect protein catabolism than antioxidant capacity (Hõrak & Cohen, 2010). The effect of reproduction on ROS production and oxidative damage is more debated. A recent review by Selman and co-authors emphasized that most studies found no association between reproductive effort and lipid peroxidation and concluded that "oxidative damage is not a key mediator of life-history trade-offs across diverse taxa" (Selman et al., 2012). We also found no support for an increased level of peroxidative lipid damage in species with high brood value. Nevertheless, there is also evidence that opposes this conclusion. Experimental stimulation of high egg production renders fruit flies less resistant against free radical attack (Salmon, Marx, & Harshman, 2001; Wang, Salmon, & Harshman, 2001). In zebra finches, there is a positive genetic correlation between resistance against ROS and number of breeding events throughout life (Kim, Velando, Sorci, & Alonso-Alvarez, 2010). Experimentally increased brood size resulted in temporary elevation of MDA in female barn swallows *Hirundo rustica*, yet this effect appeared to be transient (Pap et al., 2018).

Evidence for a role of oxidative stress in survival is also equivocal and largely correlative. In alpine swifts *Tachymarptis melba*, there is positive selection for higher resistance against oxidative stress as more resistant individuals live longer (Bize et al., 2014). In disagreement with the OSLH, a comparative study on American birds found that antioxidant levels were inversely related to survival rate (Cohen et al., 2008). Contrary to the latter, we found that mortality rate is unrelated to oxidative damage. Annual survival/mortality reflects the mean lifespan, which is thought to be mostly dependent on extrinsic environmental factors rather than intrinsic milieu such as oxidative state (Barja, 2013; Flatt & Schmidt, 2009). Indeed, oxidative damage was found to influence MLSP but not mean lifespan (reviewed by Sastre, Pallardó, & Viña, 2003). We found that annual adult mortality rate is inversely related to body size, which is in accordance with the view that mortality rates are mostly affected by extrinsic factors, as these factors are known to be weaker in larger-sized species (Ricklefs, 2008).

Surprisingly few studies assessed the covariation between life-history pace and oxidative damage on a large sample of free-living species. What we know so far is that accelerated early-life growth rate increases MDA levels and adversely affects GSH and its biosynthesis (Metcalfe & Alonso-Alvarez, 2010). Our comparative study is the first to show that fast life-history pace (i.e., lower PC1 value) covaries with increased lipid peroxidation. However, we found that pace-of-life is unrelated to either GSH, or other antioxidants, despite GSH being suggested to mediate life-history trade-offs (Isaksson et al., 2011). Increased oxidative damage in fast-living species might arise because reproduction directly constrains survival via either physiological consequences or antagonistic pleiotropic effects (Flatt, 2011; Flatt & Schmidt, 2009; Monaghan et al., 2009). There is evidence in support of direct reproduction-induced oxidative damages (Flatt & Schmidt, 2009; Isaksson et al., 2011; Monaghan et al., 2009; Pap et al., 2018; Speakman, 2008). Antagonistic pleiotropy is also plausible as ROS are adaptively generated by enzymes because they stimulate signalling pathways that are responsible for activating sexual reproduction or initiating reproductive activity, and in parallel,

the excess ROS that escape neutralization represent a havoc (Flatt, 2011; Metcalfe & Alonso-Alvarez, 2010).

5 | CONCLUSION

Our results suggest that avian species that live longer and slower have a better capacity to carry out effective somatic maintenance in terms of oxidative homeostasis. This capacity appears to be adaptive because mortality in long-lived species is more likely due to intrinsic causes, for example, somatic dysfunction, while short-lived species more often die due to extrinsic causes, for example, predation (Ricklefs, 2008). Our findings suggest that oxidative stress is not a mere epiphenomenon of ageing (de Magalhães & Church, 2006) as developmental time did not confound the association between oxidative stress and lifespan. Future studies should verify whether oxidative stress is also relevant in free-living mammals that clearly differ in oxidative physiology and lifespan from size-matched birds (Costantini, 2008; Hulbert et al., 2007).

ACKNOWLEDGEMENTS

We owe a debt of gratitude to Attila Marton, Lőrinc Bărbos, Judit Veres-Szászka, Krisztina Sándor and Attila Fülöp for their help on the field, to Manuela Banciu and Alina Sesarman for biochemical advice and to Jácint Tökölyi for his help with statistics and for his constructive criticism on an earlier version of this manuscript. We thank the directorate of the Botanical Garden Cluj-Napoca for permitting bird capturing. The constructive comments raised by the Associate Editor and three anonymous reviewers considerably improved the manuscript. This research was supported by the European Union and the State of Hungary, co-financed by the European Social Fund in the framework of TÁMOP 4.2.4.A/2-11-1-2012-0001 "National Excellence Program" and Hungarian Research Foundation (OTKA Grant no. K 113108). CIV was financed by the János Bolyai Research Scholarship of the Hungarian Academy of Sciences and by a postdoctoral grant of the Hungarian National Research, Development and Innovation Office (no. PD 121166), OV by the Hungarian Eötvös Scholarship (MÁEÖ2017_16/156845) awarded by the Tempus Public Foundation, PLP by the János Bolyai Research Scholarship of the Hungarian Academy of Sciences and by an Exploratory Research Grant of the Romanian Ministry of Research and Innovation (no. PN-III-P4-ID-PCE-2016-0404) and ZB by the Higher Education Institutional Excellence Programme of the Ministry of Human Capacities in Hungary, within the framework of the FIK-Lendület Behavioural Ecology Research Group thematic programme of the University of Debrecen. We declare no conflict of interest.

AUTHORS' CONTRIBUTIONS

C.I.V., Z.B. and P.L.P. conceived the project; C.I.V., O.V., O.G. and P.L.P. collected the blood samples; O.V. and J.P. collected literature data; L.P. carried out the biochemical assays; C.I.V. and O.V. analysed the data with input from M.F.H., Z.B. and P.L.P.; C.I.V. wrote the manuscript with significant input from O.V., M.F.H., Z.B. and P.L.P. All authors gave final approval for publication and agree to be accountable for the aspects of work that they conducted.

DATA ACCESSIBILITY

Data deposited in the Dryad Digital Repository: https://doi.org/10.5061/dryad.q3r06g1 (Vágási et al., 2018).

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

How to cite this article: Vágási CI, Vincze O, Pătraș L, et al. Longevity and life history coevolve with oxidative stress in birds. *Funct Ecol*. 2018;00:1–10. https://doi.org/10.1111/1365-2435.13228