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Review

Do glucocorticoids mediate the link between environmental conditions and telomere dynamics in wild vertebrates? A review

Frédéric Angelier [a,*], David Costantini [b,c], Pierre Blévin [a], Olivier Chastel [a]

[a] Centre d'Etudes Biologiques de Chizé, CNRS-ULR, UMR 7372, Villiers en Bois, France
[b] Muséum National d'Histoire Naturelle, UMR 7221, Paris, France
[c] Behavioural Ecology & Ecophysiology Group, Department of Biology, University of Antwerp, Universiteitsplein 1, 2610 Wilrijk, Belgium

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ABSTRACT

Following the discoveries of telomeres and of their implications in terms of health and ageing, there has been a growing interest into the study of telomere dynamics in wild vertebrates. Telomeres are repeated sequences of non-coding DNA located at the terminal ends of chromosomes and they play a major role in maintaining chromosome stability. Importantly, telomeres shorten over time and shorter telomeres seem to be related with lower survival in vertebrates. Because of this potential link with longevity, it is crucial to understand not only the ecological determinants of telomere dynamics but also the regulatory endocrine mechanisms that may mediate the effect of the environment on telomeres. In this paper, we review the relationships that link environmental conditions, glucocorticoids (GC, the main hormonal mediator of allostasis) and telomere length in vertebrates. First, we review current knowledge about the determinants of inter-individual variations in telomere length. We emphasize the potential strong impact of environmental stressors and predictable life-history events on telomere dynamics. Despite recent progress, we still lack crucial basic data to fully understand the costs of several life-history stages and biotic and abiotic factors on telomere length. Second, we review the link that exists between GCs, oxidative stress and telomere dynamics in vertebrates. Although circulating GC levels may be closely and functionally linked with telomere dynamics, data are still scarce and somewhat contradictory. Further laboratory and field studies are therefore needed not only to better assess the proximate link between GC levels and telomere dynamics, but also to ultimately understand to what extent GCs and telomere length could be informative to measure the fitness costs of specific life-history stages and environmental conditions. Finally, we highlight the importance of exploring the functional links that may exist between coping styles, the GC stress response, and telomere dynamics in a life-history framework. To conclude, we raise new hypotheses regarding the potential of the GC stress response to drive the trade-off between immediate survival and telomere protection.

1. Introduction: The relevance of studying telomeres in an ecological and evolutionary context

In 2009, Elizabeth H. Blackburn, Carol W. Greider and Jack W. Szostack received the Nobel Prize in Physiology and Medicine for the discovery of how chromosomes are protected by telomeres. These discoveries have had an important impact on biomedicine because telomeres and telomere dysfunctions have been closely associated with cancer, ageing and hereditary disease syndromes (Blackburn et al., 2006). Telomeres are well-conserved repeated sequences of non-coding DNA (TTAGGG) located at the terminal ends of chromosomes (Blackburn and Gall, 1978). They play a major role in maintaining chromosome stability during replication processes (Blackburn, 2005). Although telomeres can be restored by the enzyme telomerase, a ribonucleoprotein that adds new sequences onto the ends of chromosomes at each DNA replication (Greider and Blackburn, 1989), telomeres shorten over time and are therefore considered as a molecular marker of cellular ageing (Harley et al., 1990). Importantly, this shortening has been associated with the occurrence of diseases and with increased mortality in humans (Cawthon et al., 2003; Lansdorp, 2009).

Following these discoveries, there has been a growing interest into the study of telomeres in wild animals (Fig. 1). In the early 2000's, Haussmann and his collaborators have initiated the study of telomere biology in an ecological and evolutionary context (Haussmann and Vleck, 2002; Haussmann et al., 2003a; Vleck

* Corresponding author.
E-mail address: angelier@cebc.cnrs.fr (F. Angelier).

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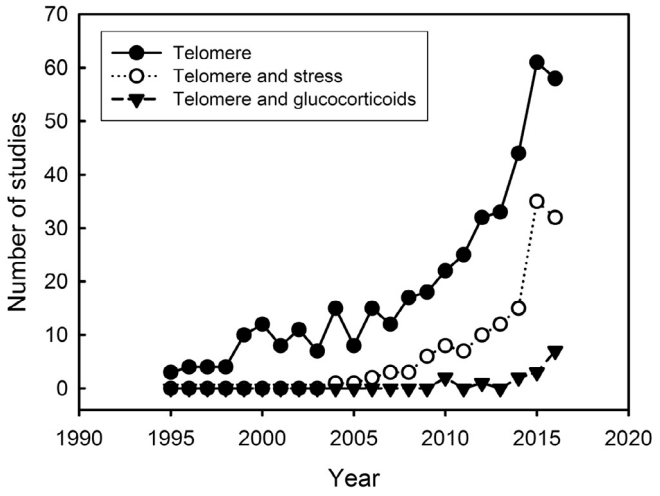

Fig. 1. Trends in the number of studies published each year on different topics from 1995 to 2016. These numbers were found by conducting a search in ISI web of knowledge (search terms, filled circles: telomere, empty circles: telomere and stress, triangles: telomere and glucocorticoids). For all searches, the results were limited to the following categories: ecology, zoology, behavioral sciences, environmental sciences and evolutionary biology.

et al., 2003). By studying telomeres in wild vertebrates, they were the first to emphasize the relevance of this molecular proxy of ageing to explore life-history trade-offs and selection processes (Haussmann et al., 2003b, 2005; Monaghan and Haussmann, 2006; Vleck et al., 2007; Haussmann and Mauck, 2008). Telomere length was initially thought to be a reliable proxy of age (i.e., "mitotic clock", Haussmann and Vleck, 2002), leading to the idea that it could be a useful tool to obtain detailed information on wild populations. Although the relationship between chronological age and telomere length appears more complex than initially thought (Monaghan, 2010; Dunshea et al., 2011; Boonekamp et al., 2013), there is now strong evidence that telomere length and telomere dynamics are tightly linked to fitness (Haussmann and Marchetto, 2010; Bauch et al., 2013; Monaghan 2010, 2014; Ouyang et al., 2016), and especially to longevity and survival in captive and wild vertebrates (e.g. Haussmann et al., 2005; Pauliny et al., 2006; Bize et al., 2009; Salomons et al., 2009; Heidinger et al., 2012; Bauch et al., 2013; Barrett et al., 2013; Asghar et al., 2015a; Fairlie et al., 2016).

These findings have raised a huge interest in measuring telomere length and telomere dynamics in wild animals (Nussey et al., 2014). Specifically, several studies have aimed to understand the ecological and life-history determinants of telomere dynamics. Such studies have led to the idea that telomere dynamics are tightly linked to the occurrence of stressors (reviewed in Haussmann and Marchetto, 2010; Monaghan, 2014; Haussmann and Heidinger, 2015; Bateson, 2016; Fig. 1), and more generally to allostasis (*sensu* McEwen and Wingfield, 2003, i.e. maintaining stability through change). As a consequence, there has recently been a growing interest in understanding the links that may exist between the endocrine regulation of allostasis and telomere dynamics in wild vertebrates (reviewed in Haussmann and Marchetto, 2010; Monaghan, 2014; Fig. 1). In that context, glucocorticoids (GCs) obviously deserve a specific attention because of their involvement in allostasis and the stress response (McEwen and Wingfield, 2003; Wingfield, 2003, 2013; Romero et al., 2009; Angelier and Wingfield, 2013; Wingfield et al., 2015; Romero and Wingfield, 2016). Because of the link between telomeres and survival, integrating telomere dynamics into the framework linking environmental conditions, GCs and life-history decisions may contribute to a better understanding of the life-history/physiology nexus (Zera and Harshman, 2001; Ricklefs and Wikelski, 2002).

In this review, our aim is to emphasize the links that exist among telomere dynamics, environmental conditions and endocrine stress mechanisms in wild vertebrates. We review current knowledge about (1) the determinants of inter-individual variations in telomere length by emphasizing the role of environmental conditions and life-history stages on telomere dynamics; (2) the link that exists between GCs (i.e. the main hormonal mediator of allostasis), oxidative stress and telomere dynamics in wild vertebrates. Finally, we highlight the importance of exploring the functional link that may exist between the GC stress response and telomere dynamics in a life-history framework.

2. Determinants of inter-individual variation in telomere length

2.1. A large inter-individual variability in telomere dynamics through life

In vertebrates, individual telomere length seems to be determined mainly through three steps: pre-natal telomere dynamics, developmental telomere dynamics, and adult telomere dynamics (Fig. 2). Firstly, telomere length is heritable and also affected by environmental, maternal and epigenetic effects (h^2 varying from 0.18 to 1.23 depending on the species, reviewed in Reichert et al., 2015), meaning that all individuals do not start their life with similar telomere length (Shalev et al., 2013). Thus, biomedical studies have first explored the potential genetic and environmental mechanisms affecting telomere length determination (Njajou et al., 2007; Broer et al., 2013) and, for example, have reported a strong positive influence of paternal age on offspring telomere length in humans (Unryn et al., 2005; Broer et al., 2013). More recently, ecological studies have confirmed that offspring telomere length is

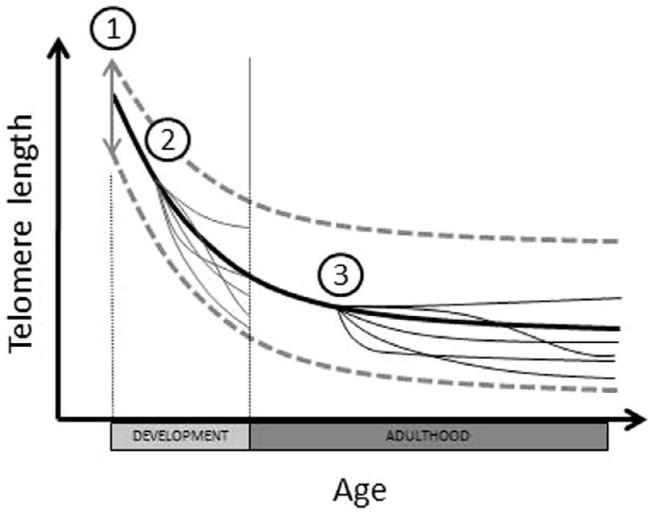

Fig. 2. Schematic representation of individual telomere dynamics through life in wild vertebrates (thick black line): (1) initial telomere length is partly heritable and determined by epigenetic/trans-generational effects; (2) telomeres shorten very quickly during the developmental phase; (3) telomere shortening is much slower during adulthood. For each of these steps, there is an important inter-individual variation in telomere dynamics: (1) Inter-individual variability in initial telomere length is represented by a grey vertical arrow; (2) inter-individual variability in developmental telomere dynamics is represented by a few examples of possible telomere trajectories (thin grey lines); (3) inter-individual variability in adult telomere dynamics is represented by a few examples of possible telomere trajectories (thin black lines). This variability in telomere dynamics translate into large inter-individual differences in telomere length (the theoretical possible range of telomere length is represented by thick grey dashed lines).

related to parents' telomere length through heritability, parental effect (Olsson et al., 2011; Reichert et al., 2015; Asghar et al., 2015b; Atema et al., 2015; Becker et al., 2015), and even transgenerational effects in wild vertebrates (Haussmann and Heidinger, 2015; Bebbington et al., 2016). Secondly, apart from the first steps of the development when the telomeres of the oocytes and early embryo elongate (Liu et al., 2007), growth and development are classically associated with strong telomere attrition in humans (Frenck et al., 1998; Zeichner et al., 1999). Accordingly, most longitudinal studies have found that telomere shortens through growth and development in wild vertebrates (e.g. Pauliny et al., 2006, 2012; Salomons et al., 2009; Boonekamp et al., 2014; but see Ujvari and Madsen, 2009; Ujvari et al., 2016), confirming the idea that development and growth are associated with accelerated cellular ageing in birds (Ricklefs, 2010). Thirdly, after the developmental phase, this shortening seems to slow down as an individual ages and it is even difficult to detect in some species during adulthood (e.g. Foote et al., 2010; Pauliny et al., 2012; Rattiste et al., 2015).

Interestingly, longitudinal studies have clearly shown that telomere dynamics vary between individuals during development and adulthood (e.g. Bize et al., 2009; Foote et al., 2011; Salomons et al., 2009; Barrett et al., 2013; Parolini et al., 2015; Bateson et al., 2015; Stier et al., 2016; Fig. 2). The telomeres of some individuals shorten quickly while the telomeres of other individuals remain steady or even elongate with time (e.g. Bize et al., 2009; Parolini et al., 2015). This large inter-individual variation in telomere dynamics is the main reason why telomere length is not really a reliable predictor of chronological age in wild vertebrates (Dunshea et al., 2011; Pauliny et al., 2012; Boonekamp et al., 2013). However, the strong link between telomere length and remaining lifespan is promising because this suggests not only that telomere length could be a molecular marker of biological age or/and "wear and tear", but also that telomere dynamics and telomere length could help us assess the influence of life-history events and/or environmental conditions on wild vertebrates.

2.2. Oxidative stress as a mediator of telomere dynamics

At the proximate level, oxidative stress is thought to be the primary cause of telomere shortening in wild vertebrates (reviewed in Haussmann and Marchetto, 2010). Oxidative stress is a complex biochemical condition of the organism that is dependent on the rate of oxidative damage generation and oxidation of non-protein and protein thiols that regulate the cell oxidative balance (Jones, 2006; Halliwell and Gutteridge, 2015; Costantini and Verhulst, 2009; Sohal and Orr, 2012). Oxidative damage is caused by reactive oxygen species (ROS), such as free radicals, which are mainly produced by metabolic processes and immune cells (Halliwell and Gutteridge, 2015). ROS can oxidize biomolecules, such as lipids, proteins, and DNA, and importantly, telomeres are especially sensitive to ROS because guanine is a dominant site for oxidatively-generated damage (Kawanishi and Oikawa, 2004; Monaghan, 2014). Supporting the importance of oxidative stress in determining telomere dynamics, a few studies have found an association between telomere dynamics and oxidative stress/antioxidant defenses in wild and domestic vertebrates (e.g. Geiger et al., 2012; Haussmann et al., 2012; Stier et al., 2014; Badás et al., 2015; Kim and Velando, 2015; but see Ouyang et al., 2016 and Boonekamp et al., 2017). For example, Geiger et al. (2012) found that small king penguin chicks (*Aptenodytes patagonicus*) that died early during the growth period had the highest level of oxidative damage and the shortest telomere lengths prior to death. Badás et al. (2015) found that supplementation with antioxidants (which protect against oxidative stress) reduced telomere loss a year following treatment in wild blue tits (*Cyanistes caeruleus*). Mechanistic studies have provided stronger evidence for a link between oxidative stress and telomere shortening (reviewed in von Zglinicki, 2002; but see Boonekamp et al., 2017), providing support for a possible causal link of results obtained in wild animals. For example, in cultures of human umbilical vein endothelial cells exposed to oxidative stress, terminal restriction fragment analysis demonstrated faster telomere shortening than in cells not exposed to oxidative stress (Kurz et al., 2004). In addition, telomere dynamics is also certainly dependent on DNA repair mechanisms that include the enzyme telomerase. Here again, most evidence comes from mechanistic studies, which have shown that this enzyme is the main actor of telomere elongation and restoration (Blackburn et al., 2006). Interestingly, oxidative stress induced a rapid and sustained decrease in the activity of this enzyme (Kurz et al., 2004). Moreover, Kurz et al. (2004) also demonstrated a key role for glutathione-dependent redox homeostasis (e.g., glutathione peroxidase) in the preservation of telomere function. Further support for a role of peroxidase activity in the protection of telomeres from oxidation was found in human embryonic kidney 293 (HEK293) cell lines (Aeby et al., 2016). All these mechanisms have rarely been studied in wild vertebrates (but see Haussmann et al., 2007). The connection between oxidative stress and telomere dynamics may also be mediated by glucocorticoids, suggesting one probable route through which environmental stress impacts on cellular ageing. For example, glucocorticoids may affect the oxidative balance through either genomic (Atanasova et al., 2009) or non-genomic (reviewed in Costantini et al., 2011) mechanisms. For example, exposure to dexamethasone enhances the expression of antioxidant genes under some circumstances (Atanasova et al., 2009). Similarly, glucocorticoids can suppress cellular antioxidant defenses though genomic effects in the rat (Kratschmar et al., 2012). Moreover, they may also affect telomere length through a modulation of telomerase activity (Fig. 3).

3. Environmental conditions and telomere shortening

3.1. Telomere dynamics vary between habitats, sites and years

Biomedical studies have first suggested that lifestyle could affect telomere dynamics in humans. For example, obesity and cigarette smoking have been associated with shorter telomeres in humans (Valdes et al., 2005). More recently, ecological studies have explored the links that exist between environmental conditions and telomere dynamics in wild vertebrates (Table 1). A few studies have specifically compared telomere length and telomere dynamics of wild birds living in contrasting habitats. Overall, they found that an *a priori* less suitable habitat is associated with faster telomere attrition, and consequently, shorter telomeres (Angelier et al., 2013; Young et al., 2013; Salmón et al., 2016). Similarly, other studies reported that telomere dynamics or telomere length vary between years or sites that were characterized by contrasting environmental conditions (Watson et al., 2015; Becker et al., 2015; Quirici et al., 2016; Gangoso et al., 2016; Kirby et al., 2017). Moreover, Young et al. (2015) also nicely demonstrated in thick-billed murres that telomere length is linked with spatial habitat use, foraging efficiency and prey selection. Altogether, these studies emphasize that the environment has certainly a strong influence on telomere dynamics. However, all these studies compared some habitats or periods that drastically differed in terms of environmental conditions, and therefore, several biotic and abiotic variables could be responsible for these differences between habitats and periods. We need experimental studies to tease apart the effects of biotic and abiotic variables on telomeres.

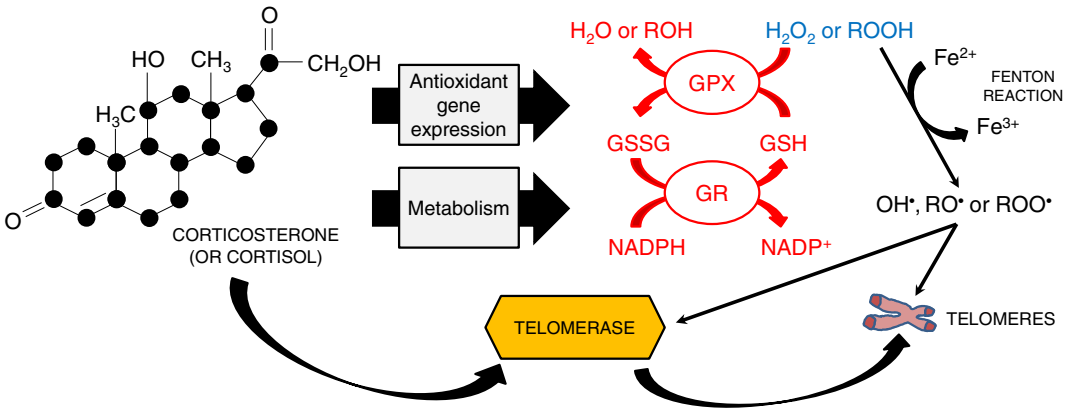

Fig. 3. This illustration shows one hypothetical route through which glucocorticoids may affect telomere dynamics by oxidative stress. Glucocorticoids may influence expression of antioxidant genes involved in glutathione (GSH) redox-cycle, such as those regulating the antioxidant enzyme GPX (glutathione peroxidase). The effect of glucocorticoids on glutathione metabolism may also be non-genomic, such as through increased metabolism and production of reactive oxygen species. The glutathione redox-cycle is used to detoxify cells from accumulation of hydrogen peroxide (H_2O_2) and organic hydroperoxides (ROOH), whose cleavage (Fenton reaction) induced by metal ions (Fe = iron) can generate very reactive free radicals (OH· = hydroxyl radical; RO· = alkoxyl radical; ROO· = alkylperoxyl radical) that damage telomeres. The impact of glucocorticoids and oxidative stress, respectively, on telomere dynamics may also come through a direct effect on the activity of the enzyme telomerase. GSSG = oxidised glutathione; ROH = alcohol; NADPH = nicotinamide adenine dinucleotide phosphate; GR = glutathione reductase. The information used to make this illustration was taken from Colitz et al., 2004; Kurz et al., 2004; Atanasova et al., 2009; Saretzski, 2009; Costantini et al., 2011.

Table 1
Review of the existing studies that report some variations in telomere length or telomere dynamics between study sites, years, cohorts and habitats.

Variable	Relationship with telomere length/dynamics	Stage	Type of study		Common name	Reference
Cohorts	≠	Development	correlative	Bird	Storm petrel	Watson et al. (2015)
Cohorts	≠	Development	correlative	Bird	White-throated dipper	Becker et al. (2015)
Cohorts	≠	Development	correlative	Mammal	Soay sheep	Fairlie et al. (2016)
Cohorts	≠	Development	correlative	Bird	White-tailed eagle	Sletten et al. (2016)
Years	≠	Adulthood	correlative	Bird	Black-tailed gull	Mizutani et al. (2013)
Sites (longitude)	≠	Adulthood	correlative	Bird	Andean condor	Gangoso et al. (2016)
Sites (latitude)	≠	Adulthood	correlative	Bird	Thorn-tailed rayadito	Quirici et al. (2016)
Sites (elevation)	≠	Development	correlative	Bird	Great tit	Stier et al. (2016)
Sites (elevation)	≠	Development	correlative	Bird	Coal tit	Stier et al. (2016)
Sites (latitude)	≠	Adulthood	correlative	Mammal	Black bear	Kirby et al. (2017)
Sites (elevation)	≠	Adulthood	correlative	Reptile	Common lizard	Dupoué et al. (unpublished)
Habitat of low suitability	–	Adulthood	correlative	Bird	American redstart	Angelier et al. (2013)
Habitat of low suitability	–	Adulthood	correlative	Bird	Thick-billed murre	Young et al. (2013)
Habitat of low suitability	–	Development	experimental/correlative	Bird	Great tit	Salmón et al. (2016)
Habitat of low suitability	=	Development	correlative	Bird	Great tit	Biard et al. (2017)

≠ denotes a significant difference in telomere length or telomere dynamics between cohorts, years or sites.
= denotes similar telomere length or telomere dynamics between cohorts, years or sites.
– denotes a negative effect of the variable on telomere length or telomere dynamics.

3.2. Ecological determinants of telomere dynamics

In that context, other correlative and experimental studies have shed some light on the impact of specific environmental variables on telomere length. Infection and diseases may be associated with reduced telomere length in wild animals (Table 2). For example, Ilmonen et al. (2008) found that repeated exposure to bacteria was associated with rapid telomere attrition in wild-derived mice (*Mus musculus*). More recently, Asghar et al. (2015a) found that chronic malaria infection was associated with significantly faster telomere attrition in a wild bird species, the great reed warbler (*Acrocephalus arundinaceus*). However, Sebastiano et al. (2017) did not find any association between telomere length and herpes viral disease in nestling Magnificent frigatebirds (*Fregata magnificens*).

Although psychological stress and associated disorders have been correlated with reduced telomere length in humans (Epel et al., 2004; Shalev et al., 2013), the influence of social variables on telomere dynamics has been overlooked in wild vertebrates and we currently lack data on the influence of social ranks and social bonds on telomere dynamics (Lewin et al., 2015). To our knowledge, a single study has tested the influence of psychological stress on telomere dynamics in birds and it found that social isolation was associated with fast telomere attrition in African grey parrots (*Psithaccus erithacus erithacus*), a highly sociable species (Aydinonat et al., 2014). In addition to pathogens and social factors, nutritional deficit could also represent a major constraint for wild animals. Several studies have shown that nutritional constraints during the developmental phase are associated with short telomeres in birds (see Table 2). However, the influence of nutritional constraints (food abundance and food quality) on adult telomere dynamics has rarely been tested in adults, especially in wild vertebrates (Table 2). Recently, Hoelzl et al. (2016) showed that food supplementation reduces telomere attrition and is even associated with telomere elongation in a wild mammal species, the dormouse (*Glis glis*).

Human-induced disturbance may also cause a reduction of telomere length in wild vertebrates. For instance, urban noise has been experimentally shown to reduce telomere length in house sparrow chicks (*Passer domesticus*, Meillère et al., 2015). Similarly,

Table 2
Review of the existing studies that report an influence of different ecological variables on telomere length or telomere dynamics.

Variable	Relationship with telomere length/dynamics	Stage	Type of study		Common name	Reference
Infection/pathogens/parasites						
Multiple infections	–	Adulthood	experimental	Mammal	House mouse	Ilmonen et al. (2008)
Tuberculosis infection	–	Adulthood	correlative	Mammal	European badger	Beirne et al. (2014)
Malaria infection	0	Adulthood	experimental	Bird	Blue tit	Badás et al. (2015)
Malaria infection	–	Adulthood	experimental	Bird	Great reed warbler	Asghar et al. (2015a)
Immune challenge	–	adulthood	experimental	Bird	European blackbird	Hau et al. (2015)
Herpes virus infection	0	Development	correlative	Bird	Magnificent frigatebird	Sebastiano et al. (2017)
Social factors						
Social isolation	–	Adulthood	correlative	Bird	African grey parrot	Aydinonat et al. (2014)
Dominance rank	+	Adulthood	correlative	Mammal	Spotted hyena	Lewin et al. (2015)
Nutritional factors						
Nutritional antioxidant	–*	Adulthood	experimental	Bird	Blue tit	Badás et al. (2015)
Nutritional micronutrient supplementation	0	Development	experimental	Bird	Zebra finch	Noguera et al. (2015)
Nutritional micronutrient supplementation	+/0**	Adulthood	experimental	Bird	Zebra finch	Noguera et al. (2015)
Nutritional antioxidant	+/0***	Development	experimental	Bird	Yellow-legged gull	Kim and Velando (2015)
Food availability	0	Adulthood	correlative	Mammal	Spotted hyena	Lewin et al. (2015)
Food availability	+	Adulthood	correlative	Bird	Seychelles warbler	Bebbington et al. (2016)
Food supplementation	+	Adulthood	experimental	Mammal	Edible dormouse	Hoelzl et al. (2016)
Developmental factors						
Maternal age	+	Development	correlative	Bird	Great reed warbler	Asghar et al. (2015b)
Maternal age	+	Development	correlative	Bird	White-throated dipper	Becker et al. (2015)
Parental age	0	Development	correlative	Bird	European shag	Heidinger et al. (2016)
Parental age	+	Development	correlative	Bird	Black-browed albatross	Dupont et al. (unpublished)
Brood competition	0	Development	experimental	Bird	Collared flycatcher	Voillemot et al. (2012)
Brood competition	–	Development	experimental	Bird	European starling	Nettle et al. (2013)
Brood competition	–	Development	experimental	Bird	Jackdaw	Boonekamp et al. (2014)
Brood competition	–	Development	experimental	Bird	European starling	Bateson et al. (2015)
Brood competition	–	Development	experimental	Bird	European starling	Nettle et al. (2015)
Brood competition	–	Development	experimental	Bird	Zebra finch	Reichert et al. (2015)
Brood competition	–	Development	correlative	Bird	Magpie/Great spotted cuckoo	Soler et al. (2015)
Brood competition	–	Development	experimental	Bird	European starling	Nettle et al. (2016)
Brood competition	–	Development	correlative	Bird	European shag	Heidinger et al. (2016)
Brood competition	–	Development	correlative	Bird	Black-tailed gull	Mizutani et al. (2016)
Brood competition	–	Development	experimental	Bird	Barn swallow	Costanzo et al. (2017)
Brood competition disadvantage	–	Development	correlative	Bird	Great tit	Stier et al. (2015)
Growth	+	Development	correlative	Bird	Barn swallow	Caprioli et al. (2013)
Growth	0	Development	correlative	Bird	Black-backed gull	Foote et al. (2011)
Growth	+	Development	correlative	Bird	Barn swallow	Parolini et al. (2015)
Growth	+	Development	correlative	Bird	American redstart	Angelier et al. (2015)
Accelerated growth	–	Development	correlative	Bird	King penguin	Geiger et al. (2012)
Accelerated growth	0	Development	experimental	Fish	Brown trout	Näslund et al. (2015)
Slow growth	–	Development	correlative	Bird	Black-tailed gull	Mizutani et al. (2016)
Developmental constraints	–	Development	correlative	Bird	King penguin	Stier et al. (2014)
Reproductive factors						
Reproductive effort	–	Adulthood	experimental	Mammal	House mouse	Kotrschal et al. (2007)
Reproductive effort	0	Adulthood	experimental	Bird	Adélie penguin	Beaulieu et al. (2011)
Reproductive effort	–	Adulthood	correlative	Reptile	Leatherback turtle	Plot et al. (2012)
Reproductive effort	–	Adulthood	correlative	Bird	Common tern	Bauch et al. (2013)
Reproductive effort	–	Adulthood	experimental	Bird	Zebra finch	Reichert et al. (2014)
Reproductive effort	–	Adulthood	experimental	Bird	Blue tit	Sudyka et al. (2014)
Reproductive effort	0	Adulthood	experimental	Bird	Zebra finch	Sudyka et al. (2016)
Climatic factors						
Climate (El Nino)	+	Adulthood	correlative	Bird	Black-tailed gull	Mizutani et al. (2013)
Reduced temperature	–	Development	experimental	Fish	Eastern mosquitofish	Rollings et al. (2014)
Increased temperature	–	Adulthood	experimental	Fish	Siberian sturgeon	Simide et al. (2016)
Increased temperature	–	Adulthood	experimental	Fish	Brown trout	Debes et al. (2016)
Stressors						
Crowding	–	adulthood	experimental	Mammal	House mouse	Kotrschal et al. (2007)
Handling	–	Development	experimental	Bird	European shag	Herborn et al. (2014)
Disturbance	–	adulthood	experimental	Bird	European blackbird	Hau et al. (2015)
Noise	–	Development	experimental	Bird	House sparrow	Meillère et al. (2015)
Contamination						
Heavy metals	–	Development	correlative	Bird	Great tit	Stauffer et al. (2017)
Heavy metals	0	Development	correlative	Bird	Pied flycatcher	Stauffer et al. (2017)
Heavy metals	0	Adulthood	correlative	Bird	Great tit	Stauffer et al. (2017)
Heavy metals	0	Adulthood	correlative	Bird	Pied flycatcher	Stauffer et al. (2017)
Chlordanes	–/0****	Adulthood	correlative	Bird	Black-legged kittiwake	Blévin et al. (2016)
Persistent organic pollutants (ΣPOPs)	0	Adulthood	correlative	Bird	Black-legged kittiwake	Blévin et al. (2016)

(*continued on next page*)

Table 2 (*continued*)

Variable	Relationship with telomere length/dynamics	Stage	Type of study		Common name	Reference
Polychlorinated biphenyls (ΣPCBs)	0	Adulthood	correlative	Bird	Black-legged kittiwake	Blévin et al. (2016)
Hexachlorobenzen (HCBs)	0	Adulthood	correlative	Bird	Black-legged kittiwake	Blévin et al. (2016)
Dichlorodiphenyldichloroethylen (pp'-DDE)	0	Adulthood	correlative	Bird	Black-legged kittiwake	Blévin et al. (2016)
Perfluoroalkylated substances (PFASs)	+	Adulthood	correlative	Bird	Black-legged kittiwake	Blévin et al. (2017)
Mercury	+/0**	Adulthood	correlative	Bird	Black-legged kittiwake	Blévin et al. (unpublished)
Organochlorine pesticides (OCPs)	0	Development	correlative	Bird	White-tailed eagle	Sletten et al. (2016)
Perfluoroalkylated substances (PFASs)	0	Development	correlative	Bird	White-tailed eagle	Sletten et al. (2016)
Other factors						
Tail regrowth	–	Adulthood	correlative	Reptile	Sand lizard	Olsson et al. (2010)

*This negative effect is indirectly linked to increased reproductive effort in experimental birds.
**The effect was apparent in females only.
***The positive effect of antioxidant supplementation was only apparent for bold chicks.
****The effect was apparent for oxychlordanes in females only.

exposure to handling stress was associated with telomere shortening in European shag nestlings (*Phalacrocorax aristotelis,* Herborn et al., 2014). However, there is currently no direct or experimental data on the influence of multiple stressors on telomere dynamics that are known to occur frequently in wild vertebrate populations (e.g. predation risk). Work done on captive animals suggests that exposure to multiple stressors may influence telomere dynamics. Hau et al. (2015) exposed hand-raised adult Eurasian blackbirds (*Turdus merula*) to a combination of repeated immune and disturbance stressors for over one year to determine the effects of chronic stress on telomeres. By the end of the experiment, stress-exposed birds showed greater decreases in telomere lengths as compared to controls, suggesting that repeated exposure to experimental stressors might affect the rate of biological ageing (Hau et al., 2015).

Although a few studies have examined the impact of rising ambient temperature on telomere dynamics in fish (Rollings et al., 2014; Debes et al., 2016; Simide et al., 2016), there is currently little data on the effect of climatic variables on telomere dynamics or telomere length in wild birds or mammals. In black-tailed gulls (*Larus crassirostris*), mild weather and low sea-surface temperature were associated with a slow telomere shortening (Mizutani et al., 2013). This suggests that climate could affect telomere dynamics but additional studies are clearly needed. Finally, a few studies have reported that environmental contaminants can potentially have detrimental effects on telomere dynamics in free-living birds (Sletten et al., 2016; Stauffer et al., 2017). For instance, Blévin et al. (2016) recently found that oxychlordane contamination, an organochlorine pesticide known to be very toxic for wildlife, is associated with shorter telomeres in female black-legged kittiwakes (*Rissa tridacyla*). Our current world is facing multiple sources of contamination and their effects on physiology and fitness are often dose-dependent. Therefore, further experimental and correlative studies linking telomere dynamics and contamination are necessary to better understand the impact of anthropogenic pollution on wild vertebrates.

As detailed in the previous paragraphs, many studies have found that telomere dynamics and telomere length are tightly connected to a broad panel of environmental conditions that are experienced by individuals. Importantly, this connection is apparent both during development and adulthood, demonstrating that environmental conditions are probably one of the main drivers of telomere dynamics in wild vertebrates. Further studies are however necessary to better understand not only the link between specific overlooked environmental conditions and telomere dynamics but also the potential additive, interactive, and antagonistic effects of the occurrence of multiple environmental constraints on telomere dynamics. Overall, all these results demonstrate that studying telomere dynamics or telomere length can be appropriate to assess the survival costs of specific environmental conditions in wild animals.

4. Telomere shortening through the life cycle

In addition to environmental conditions, predictable life-history events are also likely to have a strong impact on telomere dynamics, especially when they are associated with physiological and metabolic modifications (Monaghan, 2014). During its life, an organism will face obligatory stages such as development and growth in early life and breeding, wintering, migration, or molt during adulthood (Wingfield, 2008). Although necessary for reproduction and survival, all these stages can be associated with specific physiological constraints that may affect telomere dynamics.

There is now strong evidence that telomeres shorten very quickly during early life in most wild vertebrates (e.g. Salomons et al., 2009; Foote et al., 2011; Geiger et al., 2012; Heidinger et al., 2012; Reichert et al., 2015; Hammers et al., 2015; Asghar et al., 2015a,b). The pace of early life shortening may differ between males and females (Barrett and Richardson, 2011; Parolini et al., 2015) and seem to be dependent on developmental conditions (Table 2). For instance, the rate of early-life telomere shortening is overall faster when sibling competition is harsher (e.g. Boonekamp et al., 2014; Nettle et al., 2015; Stier et al., 2015; Costanzo et al., 2017, Table 2). Because replication is known to accelerate telomere attrition, such shortening certainly results from the replication processes that are inherent to growth and development of the organism. Moreover, growth is also associated with strong metabolic modification and with the production of oxidative stress (Smith et al., 2016) that is known to accelerate telomere attrition (von Zglinicki, 2002; Kawanishi and Oikawa, 2004). In comparison, telomere attrition is much slower during adulthood (e.g. Rattiste et al., 2015), but can still reflect exposure to chronic stress (Hau et al., 2015). Because offspring telomere length could be related to survival probability and lifespan (Pauliny et al., 2006; Heidinger et al., 2012; Barrett et al., 2013; Fairlie et al., 2016), this developmental telomere attrition supports the existence of a trade-off between growth and longevity (Ricklefs, 2006; Lee et al., 2013). Therefore, comparing developmental telomere dynamics among species could help to understand the importance of this trade-off in mediating inter-specific life-history strategies (e.g. fast vs. slow development). Moreover, at the individual level, developmental telomere dynamics could

help better assessing the influence of early developmental events on the future performances and the fitness of individuals (Monaghan, 2014; Boonekamp et al., 2014).

Several studies have focused on the influence of reproductive effort on telomere dynamics and they have reported contrasting results (Table 2). Although some of them found that the reproductive phase, and especially the parental phase, is correlated with quick telomere shortening (Bauch et al., 2013; Sudyka et al., 2014; Reichert et al., 2014), others did not find such an effect (Beaulieu et al., 2011; Sudyka et al., 2016). Interestingly, some of these studies have experimentally shown that parental effort is associated with quick telomere attrition (Sudyka et al., 2014; Reichert et al., 2014). This supports the idea that reproduction is a demanding life-history stage for wild vertebrates and it illustrates perfectly the trade-off that exists between reproduction and survival (or at least organism's "wear and tear"). Surprisingly, the influence of other life-history stages on telomere dynamics has been much less studied in wild vertebrates. For example, the influence of molt on telomere dynamics has to our knowledge never been studied. The influence of migration on telomere dynamics has been studied in a few wild bird species so far. By comparing residents and migratory dark-eyed juncos (*Junco hyemalis*), Bauer et al. (2016) found that migration was associated with shorter telomeres, emphasizing a potential important cost of migration at least in this passerine species. Regarding wintering, studies are also scarce. Quick telomere attrition has been associated with a short time spent on the wintering ground in black-legged kittiwakes (Schultner et al., 2014) and with an increased wintering foraging activity in thick-billed murres (Young et al., 2017). This suggests that this non-breeding stage may actually be associated with telomere preservation, at least when foraging conditions are appropriate. Such telomere preservation probably occurs because wintering individuals may face low energetic demands and may allocate a large part of their resources to protecting processes that limit ageing and telomere shortening. Interestingly and supporting this interpretation, hibernation has also been studied in wild mammals and torpor seems to slow down telomere attrition in dormice (Hoelzl et al., 2016) and even to be associated with telomere elongation in sub-adult djungarian hamsters (*Phodopus sungorus,* Turbill et al., 2012, 2013). Overall, we currently lack crucial basic data to fully understand the costs of several life-history stages on telomere length, and thus organism's longevity. Moreover, to our knowledge, no study has examined the dynamics of telomeres through a whole annual life cycle in wild animals. Such an approach could be especially relevant to assess the relative costs of each life stage (reproduction, migration, wintering, molt, etc.). Finally, studying the impact of specific life-history stages on the telomere dynamics of individuals living in contrasting environments could shed some light on the costs and benefits of specific life-history decisions under contrasting environmental conditions.

5. Glucocorticoids and telomere dynamics

5.1. Are glucocorticoids the link between environmental constraints and telomere dynamics?

In the previous paragraphs, we reviewed the important links that exist between life-history demands, environmental conditions and telomere dynamics in wild vertebrates. Overall, demanding life-history stages and constraining environmental conditions seem to be associated with a rapid rate of telomere attrition and there is also a clear connection between stress and telomere attrition in humans, rodents and wild vertebrates (Epel et al., 2004; Kotrschal et al., 2007; Haussmann and Marchetto, 2010; Shalev et al., 2013; Monaghan, 2014; Hau et al., 2015; Bateson, 2016). This suggests that telomere dynamics could be tightly related to allostatic load (*sensu* McEwen and Wingfield, 2003; Romero et al., 2009) in wild vertebrates. When facing predictable and unpredictable events, organisms develop a suite of behavioral and physiological adjustments to maintain their homeostasis (Wingfield, 2003; Angelier and Wingfield, 2013; Romero and Wingfield, 2016). These adjustments are under control of a few central neurological and endocrine mechanisms (Romero et al., 2009). Among them, the Hypothalamus-Pituitary-Adrenal (HPA) axis appears especially relevant not only because it regulates several physiological functions but also because it is considered as a proxy of allostatic load in wild vertebrates (Romero et al., 2009). Supporting the relevance of this HPA axis when focusing on telomere dynamics, the activation of this axis and the resulting secretion of GCs are functionally linked with the proximate mechanisms regulating telomere dynamics (Fig. 3). Short-term exposure to increased GCs may increase oxidation but also antioxidant protection to limit spread of oxidative damage (reviewed in Costantini et al., 2011). On the other hand, prolonged exposure to high GCs results in increased oxidative stress and reduction of antioxidant defenses in vertebrates (reviewed in Costantini et al., 2011; see also work on wild marmots *Marmota marmota* by Costantini et al. (2012); and work on wild brown trouts *Salmo trutta* by Birnie-Gauvin et al., 2017). Importantly, biomedical studies have also shown that glucocorticoids may affect telomerase activity. Specifically, exposure to elevated cortisol levels is associated with a down-regulation of telomerase activity (Choi et al., 2008) and glutathione peroxidase (Patel et al., 2002), but mild increases in cortisol levels rather seems to up-regulate telomerase activity (Epel et al., 2010).

In addition, GCs are involved in several physiological systems that are also linked to oxidative stress or/and telomere shortening. For instance, GCs are involved in metabolic processes (reviewed in Landys et al., 2006) and an activation of these processes results in an increased production of ROS (Costantini, 2014). Specifically, increased circulating GCs levels are associated with protein catabolism and are thought to promote glucose and lipid mobilization (reviewed in Landys et al., 2006). Moreover, GCs are closely connected to immune activation (Martin, 2009) that is known to be associated with the production of ROS and with telomere attrition (Ilmonen et al., 2008; Asghar et al., 2015a). Increased circulating GC levels are known to enhance parts of the innate and adaptive immune responses in the short-term while the pathological maintenance of elevated GC levels during a prolonged period is rather associated with immune suppression (reviewed in Martin, 2009). Similarly, circulating GC levels are associated to disturbance and the occurrence of acute and chronic stressors (Dickens and Romero, 2013; Madliger and Love, 2016) that also seem to accelerate telomere attrition (Epel et al., 2004; Kotrschal et al., 2007; Herborn et al., 2014; Hau et al., 2015). Furthermore, a few studies have also shown that environmental contaminants can both disrupt GC regulation (Nordstad et al., 2012; Tartu et al., 2015; Meillère et al., 2016), and affect telomere dynamics in wild vertebrates (Blévin et al., 2016; Stauffer et al., 2017). Finally, circulating GC levels are known to fluctuate during the life-history cycle (reviewed in Romero, 2002), and interestingly, telomere shortening also seems greater during stages when GC levels are elevated. For example, GC levels are elevated during reproduction (Romero, 2002), which is associated with fast telomere attrition (Bauch et al., 2013; Sudyka et al., 2014; Reichert et al., 2014). Moreover, reproductive effort is positively associated with both elevated GCs (e.g. large brood size, Bonier et al., 2011; Love et al., 2014) and rapid telomere attrition (Sudyka et al., 2014; Reichert et al., 2014), emphasizing this potential link between telomere dynamics and GCs.

5.2. Telomere length and circulating glucocorticoids levels: A context-dependent link?

All these results strongly suggest that HPA regulation probably plays a major role in determining telomere dynamics in wild vertebrates. However, only a few studies have examined the link between circulating GCs levels and telomere dynamics in wild vertebrates (Table 3). In thorn-tailed rayaditos (*Aphrastura spinicauda*), telomere length was negatively correlated with circulating corticosterone levels. Similarly, birds with higher corticosterone levels had shorter telomeres in black-browed albatrosses (*Thallassarche melanophrys*, Angelier et al., unpublished data) and in Andean condors (Gangoso et al., 2016). Interestingly, Young et al. (2016) found that the link between telomere length and corticosterone levels was inconsistent in thick-billed murres (*Uria lomvia*). Indeed, corticosterone levels were positively correlated with telomere length in one colony but negatively correlated in another one. Other studies found no correlation between circulating GC levels and telomere length (Young et al., 2016; Ouyang et al., 2016). Similarly, Bauch et al. (2016) found that corticosterone levels were negatively correlated with telomere length in male but not in female Common terns. All these studies suggest that the relationship between GCs and telomere length is complex and context dependent (Young et al., 2016; Bauch et al., 2016). This is not so surprising because GCs can be positively correlated to individual quality in some circumstances (the CORT-adaptation hypothesis, Bonier et al., 2009) and negatively in others (the CORT-fitness hypothesis, Bonier et al., 2009). Thus, elevated GC levels can be a proxy of the inability of an individual to cope with its environment, leading to the idea that elevated GC levels should be found in individuals of low quality (Angelier et al., 2010), and thus in individuals with short telomeres (Quirici et al., 2016; Angelier et al., unpublished data; Young et al., 2016; Gangoso et al., 2016). On the other hand, telomere length and GC levels could be positively correlated when GC levels are positively correlated with individual quality. For instance, elevated GC levels are necessary to sustain the energetic demands of reproduction, and therefore, elevated GC levels should be only found in individuals that are able to allocate resources to reproduction, i.e. individuals of high quality with long telomeres (Bauch et al., 2016; Young et al., 2016).

Importantly, a few experimental studies have also examined the link between GCs and telomere dynamics (Monaghan, 2014). In developing offspring, telomere attrition was accelerated by handling stress but an additional experimental increase of circulating GCs levels did not amplify this pattern (Herborn et al., 2014). In domestic chickens (*Gallus gallus*), an embryonic exposure to GCs resulted in shorter telomeres 25 days after hatching (Haussmann et al., 2012). In adults, two experimental studies have to our knowledge examined the impact of corticosterone on telomere dynamics. In black-legged kittiwakes, a temporary increase in corticosterone levels was clearly associated with a faster rate of telomere attrition over a year (Schultner et al., 2014). In captive zebra finches (*Taeniopygia guttata*), injection of corticosterone induced a faster rate of attrition in reproductive females (Tissier et al., 2014). All these studies clearly demonstrate that there is a strong functional link between the HPA axis and telomere dynamics. However, most studies have focused on circulating GC levels without exploring the whole and complex functioning of the HPA axis. The relevance of circulating GC levels as a proxy of physiological stress or fitness is currently debated (Bonier et al., 2009; Dickens and Romero, 2013; Love et al., 2014) because the actions of GCs on physiology and behavior are regulated at multiple levels (e.g. receptors, corticosterone-binding globulins, negative feedback, etc. Romero, 2004). Therefore, further laboratory and field studies are mandatory not only to better assess the proximate link between the HPA axis and telomere dynamics, but also to ultimately understand to what extent GCs and telomere length could be informative to measure the fitness costs of specific life-history stages and environmental conditions.

5.3. Coping styles, the glucocorticoid stress response and telomere dynamics

So far, most studies have focused on baseline circulating GC levels that provide information on the energetic status of individuals (i.e. allostatic load, McEwen and Wingfield, 2003; Romero et al., 2009). However, the link between the physiological sensibility to stressors and telomere dynamics has been overlooked. In wild vertebrates, individuals adopt different strategies to cope with unpredictable events and these coping styles have been linked to the HPA axis (Wingfield, 2003, 2013; Wingfield et al., 2015). In response to an unpredictable event, the HPA axis is activated and this results in the rapid and transitory acute secretion of GCs in the bloodstream (Romero, 2004). These increased circulat-

Table 3
Review of the existing studies that focus on the link between GCs and telomere length.

GC measurement	Relationship with telomere length	Stage	Taxon	Common name	Reference
correlative studies					
Feather corticosterone	+	Adulthood	Bird	Thick-billed murre	Young et al. (2017)
Plasma corticosterone	–	Adulthood	Bird	Thorn-tailed rayadito	Quirici et al. (2016)
Plasma corticosterone	–*	Adulthood	Bird	Common tern	Bauch et al. (2016)
Plasma corticosterone	+	Adulthood	Bird	Thick-billed murre	Young et al. (2016)
Plasma corticosterone	0	Adulthood	Bird	Thick-billed murre	Young et al. (2016)
Plasma corticosterone	–	Adulthood	Bird	Thick-billed murre	Young et al. (2016)
Plasma corticosterone	–	Adulthood	Bird	Black-browed albatross	Angelier et al. (unpublished)
Feather corticosterone	–	Adulthood	Bird	Andean condor	Gangoso et al. (2016)
Plasma corticosterone	0	Adulthood	Bird	Tree swallow	Ouyang et al. (2016)
+ and – respectively denote a positive and negative relationship between GC levels and telomere length					
0 denotes no significant relationship between GC levels and telomere length					
* the relationship was apparent in males only					
Experimental studies					
Embryonic injection of corticosterone	–	Development	Bird	Domestic chicken	Haussmann et al. (2012)
Ingestion of corticosterone	–	Development	Bird	European shag	Herborn et al. (2014)
Corticosterone implants	–	Adulthood	Bird	Black-legged kittiwake	Schultner et al. (2014)
Injection of corticosterone	–	Adulthood	Bird	Zebra finch	Tissier et al. (2014)

– denotes a negative effect of the experiment on telomere length.

ing GC levels act on specific behavioral and physiological systems to activate an emergency life-history stage (ELHS) that aims to promote immediate survival at the expense of other life-history components, such as reproduction (Wingfield et al., 1998). Importantly, there is a large inter-individual variation in this so called GC stress response (Cockrem, 2013) and this variability is associated to different stress sensitivity and coping styles. Although this GC stress response is known to mediate life-history decisions (Wingfield and Sapolsky, 2003; Lendvai et al., 2007; Krause et al., 2016) and to have important fitness consequences (Breuner et al., 2008), its link with telomere dynamics has to our knowledge never been examined. Here, we develop a theoretical framework and several hypotheses that may stimulate future studies.

Because elevated GC levels are linked with oxidative stress (Costantini et al., 2011) and reduced telomerase activity (Choi et al., 2008), mounting a GC stress response could be associated with increased DNA damage and telomere shortening. One hidden cost of an increased sensitivity to stress could therefore be an acceleration of telomere shortening, and more generally, ageing. Because telomere length is positively associated with longevity (Pauliny et al., 2006; Bize et al., 2009; Salomons et al., 2009; Heidinger et al., 2012; Barrett et al., 2013; Fairlie et al., 2016), the GC stress response may therefore mediate the trade-off between immediate survival and long-term survival (Hypothesis 1). Thus, a high sensitivity to stress (and a strong GC stress response) could promote immediate survival at the expense of telomere protection, and thus, longevity (Fig. 4A).

Importantly, this trade-off between immediate survival and telomere preservation may be exacerbated or alleviated depending on the environmental context and/or individual quality. Individual quality involves multiple phenotypic traits that are positively correlated with fitness (Wilson and Nussey, 2010). These phenotypic traits certainly involve behavior, physiology and morphological components that can benefit to fitness under specific circumstances. Overall, we may expect individuals of higher quality to have a better access to resources and to be able to allocate more resources to multiple competing traits. If individuals of high quality or individuals living in a highly suitable environment have for instance better antioxidant defenses or better DNA repair mechanisms, they may be able to limit the oxidative damages that are associated with elevated GC levels. Therefore, they may bear less telomere shortening when mounting an intense stress response (Hypothesis 2). Under that scenario, the link between telomere dynamics and the intensity of the GC stress response would depend not only on the environmental context, but also on the state of the individual (Fig. 4B).

Finally, the situation could even be more complex when individuals are engaged in a specific life-history stage. The GC stress response mediates behavioral and physiological adjustments that shift the organism from specific stages to an ELHS, which prioritizes immediate survival at the expense of other demanding activities (Wingfield et al., 1998). When these demanding activities are associated with oxidative stress and telomere shortening, the disruption of these activities may actually be associated with a slowing down of the rate of telomere attrition. Therefore, a high sensitivity to stress (and a strong GC stress response) could be counter-intuitively associated with reduced telomere attrition under some circumstances (Hypothesis 3, Fig. 4C). Given our current knowledge of the links between GCs, telomere shortening and reproductive effort, the best support for this hypothesis comes from studies that have focused on the reproductive stage. During reproduction, an intense GC stress response is classically associated with a substantial reduction of parental effort (Wingfield and Sapolsky, 2003; Lendvai et al., 2007; Krause et al., 2016), which is known to translate into a reduction of the rate of telomere attrition (Bauch et al., 2013; Sudyka et al., 2014; Reichert et al., 2014).

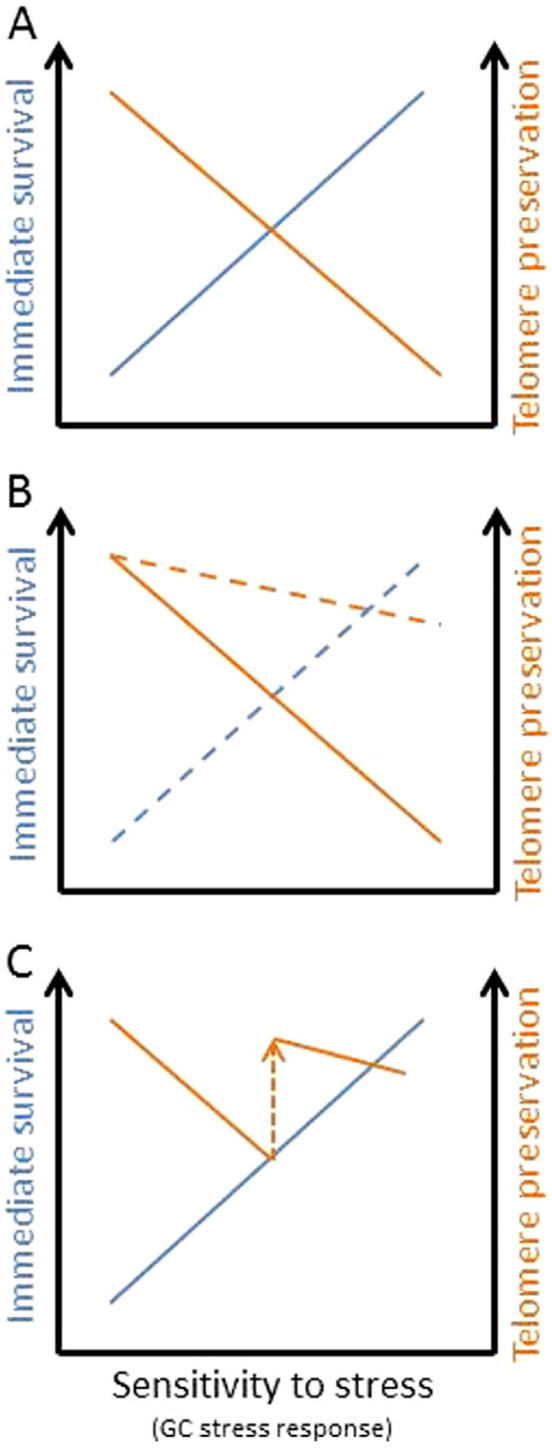

Fig. 4. Theoretical links between the GC stress response, immediate survival and telomere preservation. A: An intense GC stress response is theoretically associated with an improved immediate survival when an acute stressor occurs (blue line) but also with a faster telomere attrition (orange line). B: The relationship between the intensity of the GC stress response and telomere dynamics may be different in high-quality individuals or in individuals living in a highly suitable environment. Because of better antioxidant defenses or DNA repair mechanisms, these individuals may be able to limit the negative effect of elevated GC levels on telomere length (the solid and the dashed line respectively represent individuals of low and high quality). C: Elevated GC levels can switch individual from a specific life-history stage (e.g. breeding stage) to an emergency life-history stage (e.g. non breeding stage). This switch is associated with reduced demanding activities, and therefore, with limited telomere attrition. This switch from a specific life-history stage to an emergency life history stage is represented by the orange arrow. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

In contrast, a weak stress response is usually associated with the maintenance of parental effort (Wingfield and Sapolsky, 2003; Lendvai et al., 2007; Krause et al., 2016), and thus with potential significant survival costs (“the cost of reproduction”) that would be associated to an important telomere attrition (Bauch et al., 2013; Sudyka et al., 2014; Reichert et al., 2014).

The link between the GC stress response and telomere attrition is however probably not so straightforward and it certainly depends on the costs and benefits of activating an ELHS that are certainly species and context dependent. Specifically, these costs and benefits certainly depend on what aspects of behavior or physiology are up or down-regulated when an ELHS is activated. For instance, high stress sensitivity could be associated with a reduced rate of telomere shortening in breeding individuals if it limits the negative impact of breeding activities on telomere dynamics. However, high stress sensitivity could also be associated with a risk of mounting numerous and repeated GC stress responses that are known to accelerate telomere attrition. Under some circumstances, the negative impact of high stress sensitivity on telomere dynamics may therefore outweigh the benefits of a reduced breeding effort on telomere dynamics, especially when the costs of reproduction are limited. Therefore, it appears crucial to better assess the costs and benefits of elevated GC levels and specific life-history stages on telomere dynamics in multiple species with contrasted life-history strategies and further studies are clearly needed to better understand the functional link between the GC stress response and telomere dynamics in wild vertebrates.

Although the GC stress response is known to be heritable and repeatable (Evans et al., 2006; Cockrem et al., 2009; Angelier et al., 2011; Jenkins et al., 2014), it is also flexible because it can be actively modulated by individuals (Wingfield and Sapolsky, 2003; Lendvai et al., 2007; Krause et al., 2016). The ability of individuals to adaptively modulate this GC stress response is thought to be involved in their capacity to cope with a changing world (Angelier and Wingfield, 2013; Wingfield et al., 2015). Exploring the link between the GC stress response, its flexibility, and telomere dynamics should further shed some light on the costs and benefits of the hormonal sensitivity to stress. Therefore, we believe that combining GCs and telomere dynamics measurements in several ecological contexts should open some exciting and promising new research areas in environmental endocrinology.

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