Effects of the social environment on vertebrate fitness and health in nature: moving beyond the stress axis

Camille Lemonnier[1,2]*, Pierre Bize[3,4], Rudy Boonstra[5], F. Stephen Dobson[2,6], François Criscuolo[2], Vincent A Viblanc[2]

[1]Ecole Normale Supérieur de Lyon, 69342 Lyon, France

[2]Université de Strasbourg, CNRS, IPHC UMR 7178, 67000 Strasbourg, France.

[3]School of Biological Sciences, University of Aberdeen, Aberdeen, UK

[4]Swiss Institute of Ornithology, Sempach, Switzerland

[5]Department of Biological Sciences, University of Toronto Scarborough, Toronto, Canada

[6] Department of Biological Sciences, Auburn University, Auburn, AL, USA

***Correspondance :** Camille Lemonnier; camille.lemonnier@ens-lyon.org

Keywords: dominance, epigenetics, hierarchies, HPA, social determinants of health, social buffers

ABSTRACT

Social interactions are a ubiquitous feature of the lives of vertebrate species. These may be cooperative or competitive, and shape the dynamics of social systems, with profound effects on individual behavior, physiology, health, fitness, and health. On one hand, a wealth of studies on humans, laboratory animal models, and captive species have focused on understanding the relationships between social interactions and individual health within the context of disease and pathology. On the other, ecological studies are attempting an understanding of how social interactions shape individual phenotypes in the wild, and the consequences this entails in terms of adaptation. Whereas numerous studies in wild vertebrates have focused on the relationships between social environments and the stress axis, much remains to be done in understanding how socially-related activation of stress axis coordinates other key physiological functions related to health. Here, we review the state of our current knowledge on the effects that social interactions may have on other markers of vertebrate health. Building upon complementary findings from the biomedical and ecological fields, we identify 6 key physiological functions (cellular metabolism, oxidative stress, cellular senescence, immunity, brain function, and the regulation of biological rhythms) which are intimately related to the stress axis, and likely directly affected by social interactions. Our goal is a holistic understanding of how social environments affect vertebrate fitness and health in the wild. Whereas both social interactions and social environments are recognized as important sources of phenotypic variation, their consequences on vertebrate fitness, and the adaptive nature of social-stress-induced phenotypes, remain unclear. Social flexibility, or the ability of an animal to change its social behavior with resulting changes in social systems in response to fluctuating environments, has emerged as a critical underlying factor that may buffer the beneficial and detrimental effects of social environments on vertebrate fitness and health.

Keywords: dominance, epigenetics, hierarchies, HPA, social buffers, social determinants of health

INTRODUCTION

Social interactions with conspecifics are ubiquitous in vertebrates. These range from interactions between mates, between parents and offspring, among siblings, among members living in groups, or between competitors for space (Wilson, 1975). The evolution of social-living is typically considered in the light of resource competition, kin selection, cooperation, and predation pressure (Hamilton, 1964, 1971; Alexander, 1974; Wilson, 1975; Oli and Armitage, 2003; Silk, 2007; Roulin and Dreiss, 2012); with social biology shaped by a balance between the advantages earned, and costs and constraints paid from aggregating with conspecifics. As a consequence, the optimal frequency, duration, intensity, and quality of social interactions, as well as information exchanged between conspecifics, is expected to vary greatly across species and life-stages depending on the ecology and life history of a species and its surrounding (social, biotic and abiotic) environment (Kappeler et al., 2013; Schradin, 2013; Hofmann et al., 2014). Importantly, deviation from optimal levels of social interactions, whether due to social isolation or overload, can lead to negative effects on vertebrate fitness, often referred to in the literature as 'social stress' (DeVries et al., 2003; Goymann and Wingfield, 2004; Creel et al., 2013; Snyder-Mackler et al., 2020).

Much of the behavioral and physiological changes that occur when vertebrates are faced with predictable or unpredictable stressors – such as when dealing with conspecifics – are orchestrated by the Sympathetic-Adrenal-Medullary axis (SAM) and the Hypothalamic-Pituitary-Adrenal (HPA) axis (hypothalamic pituitary inter-renal HPI in fish) (Smith and Vale, 2006; Romero and Butler, 2007). Thus, it is not surprising that studies on the relationships between social interactions and individual physiology have focused on what has come to be known as the stress axis. These studies have considered both aspects of the stress response: i.e. (1) SAM activity, which occurs within seconds of a disturbance, participates in the "fight-or-flight" response of the organism via the action of catecholamines (adrenaline, noradrenaline),

and can be measured through increases in blood catecholamine levels (Stoddard et al., 1987; Sgoifo and Papi, 1995; Sgoifo et al., 1996), or their effects, such as increased heart rate in response to social stimuli (Eisermann, 1992; Sgoifo et al., 1999; Jong et al., 2000; Wascher et al., 2008; Wascher et al., 2009; Viblanc et al., 2012); and (2) HPA activity which is slower and more sustained, and results in the production of glucocorticoids (GC; cortisol or corticosterone) from the adrenal cortex that promote the mobilization of carbohydrates from body reserves to face the stressful events (Sapolsky et al., 2000).

Whereas the effects of social interactions on the stress axis are now well known, in humans, laboratory/captive animals, and in wild animals, (reviewed at length elsewhere; DeVries et al., 2003; Goymann and Wingfield, 2004; Creel 2004; Creel et al., 2013) it is only more recently that researchers have started to move beyond the stress axis alone in understanding the adaptive consequences of social interactions (Snyder-Mackler et al., 2020). On one hand, a large body of research on human well-being and on laboratory/ captive animals has sought to understand how 'negative' social interactions (competition, hierarchies, work stress, emotional neglect or abuse, etc.) may lead to pathology and disease (DeVries et al., 2003; Cohen, 2004; Epel et al., 2004; Avitsur et al., 2006; Blackburn and Epel, 2012; Norman et al., 2012), whereas 'positive' social interactions may provide a buffer against social challenges, alleviating individual stress, in many cases with positive health outcomes (Turner-Cobb et al., 2000; DeVries et al., 2003; Heinrichs et al., 2003; Cohen, 2004; Rosal et al., 2004; Uchino, 2004; Lutgendorf et al., 2005; Uchino, 2006; Uchino, 2006; Uchino et al., 2012; Bateson, 2016). On the other, numerous studies in wild vertebrates have focused on the relationships between social environments and the stress axis (see Creel et al., 2013) but much remains to be understood about how social environments (often through modulation of the HPA or SAM axes) affect other important physiological functions directly related to individual fitness and health (Sapolsky, 2004, 2005; Snyder-Mackler et al., 2020). Below, we review the current state

of knowledge on the effects that social interactions can have on markers of vertebrate health. We highlight areas where research is needed and propose directions for future studies to broaden our understanding of the consequences that social interactions can have on the health and fitness of wild vertebrates, shaping group dynamics and the evolution of sociality. We see three main reasons why studies would benefit from a more holistic view of vertebrate health, rather than focusing on the activation of the stress axis alone.

First, studies aiming to understand the effects of social environments on individual physiology and stress have mostly focused on how social interactions may affect the function of the SAM or HPA/I axes, and these studies have often used chronically elevated levels of circulating GC as indicative of elevated stress load and a likely marker of disease and pathology (Boonstra 2013). This view is one that stems mostly from the biomedical literature where individuals are pushed beyond their capacity to cope with stressors from the environment. Under such situations, the negative feedback loops that terminate stress responses are impaired, resulting in irreversible "wear and tear" of bodily functions (McEwen and Wingfield, 2003; Romero et al., 2009). Whereas this might occur in natural conditions, it is more likely the exception rather than the rule, stress and chronically elevated GC levels being primarily adaptive responses to deal with ecological challenges, and thus beneficial (Boonstra, 2004, 2005, 2013; Bonier et al., 2009; Beehner and Bergman, 2017). Some confusion may occur when GCs are often and misleadingly referred to negatively and interpreted as "stress hormones" in the literature (including in some of our own papers) (MacDougall-Shackleton et al., 2019). Because GCs are pleiotropic hormones with receptor- and tissue-specific functions, that modulate the expression of some 10% of the genome (Le et al., 2005), the role of GCs cannot be confined to the stress response alone. Unfortunately, several studies using the descriptor "stress hormones" fail to consider the adaptive action of GCs in coordinating other essential physiological functions (for a discussion see Boonstra, 2007, 2013; MacDougall-Shackleton et

al., 2019). Yet studies from wild vertebrates indicate that chronically elevated maternal GCs can adaptively shape the next generation to cope with prevailing social circumstances (Dantzer et al., 2013; Boogert et al., 2014). It is increasingly acknowledged that, in natural conditions, GCs levels alone are not sufficient to evaluate the adaptive nature of physiological stress, especially chronic stress, as such states might reflect the natural and evolutionary conserved response of organisms in coping with the environmental challenges of their daily lives (Boonstra, 2013; MacDougall-Shackleton et al., 2019; Harris, 2020).

Second, focusing solely on the measurement of individual total GC levels in response to social interactions alone can be misleading. GCs circulate in the bloodstream either strongly bound to corticosteroid binding globulin (CBG) or free. The free hormone hypothesis states that only the latter is biologically active and can bind to GC receptors (GR) to trigger cellular signaling pathways and modifications in gene transcription and cell functioning (Mendel, 1989). Because about 90% of blood GCs are bound to CBG in the vast majority of vertebrates (Desantis et al., 2013a; Delehanty et al., 2015; 2020), our understanding of how social interactions affect individual stress requires analyzing total GCs *and* CBG levels, and how these change as a function of the social environment. This could provide information not only on the biologically active fraction of GC (the free GC) and level of activation of the HPA/I axis in response to social challenges, but also on the organism's capacity to buffer elevated levels of circulating GC by binding to CBG. Such information is functionally important in maintaining physiologically acceptable levels of active GCs in the organism (Breuner et al., 2013). Studies from captive animals indicate that socially stressed animals experience a decrease in CBG capacity, leading to higher access of free GCs (Spencer, 1996; Alexander and Irvine, 1998; Tamashiro et al., 2005; Otten et al., 2010). However, those studies usually focus on situations where social conflict is forced, and little is known of social effects on CBG in wild vertebrates. An alternative hypothesis for the relationship between CBG and social interactions is that

150 binding capacity may be increased as a response to chronic social conflicts, *i.e.* to buffer
151 potentially negative effects of chronically elevated GC such as immunodepression (Desantis et
152 al., 2013). Our current studies in colonial king penguins (*Aptenodytes patagonicus*), which are
153 highly aggressive and territorial during reproduction (Côté, 2000), indicate that adults breeding
154 in high social densities exhibit increased CBG levels compared to adults breeding in low social
155 densities (Lemonnier, Schull, Stier, Boonstra, Delahanty, Lefol, Durand, Robin, Criscuolo,
156 Bize, Viblanc; *unpublished data)*. Elevated CBG levels may adaptively allow birds to cope with
157 chronically stressful social environments (Viblanc et al., 2012; Viblanc et al., 2014a), but in
158 general, chronic stress imposed by external stressors (e.g. predation; Boonstra et al. 1998)
159 causes CBG levels to fall, and free levels to increase. Not all researchers agree with the need to
160 measure free levels (see Schoech et al., 2013). The affinity between CBG and GC is subject to
161 local conditions (infection, temperature, other steroid hormones, etc., Cameron et al., 2010; Lin
162 et al., 2010). Therefore, rigor is needed in quantifying CBG and thus free GCs levels (Delehanty
163 et al., 2015; 2020), though it is clear that over the short term (4-24 h depending on the species),
164 CBG levels are stable in plasma. However, extensive evidence indicates that CBG is a key
165 mediator of the biological effects of circulating GCs (Siiteri et al., 1982; Hammond, 1990;
166 Perogamvros et al., 2011; Qian et al., 2011) and thus needs to be quantified to assess how the
167 social environment affects free GC levels.

168 Third, many studies investigating the effects of social environments and social
169 interactions on the vertebrate stress axis in the wild have done so in the context of dominant-
170 subordinate social relationships in social hierarchies (Fox et al., 1997; Kotrschal et al., 1998;
171 Abbott et al., 2003; Goymann and Wingfield, 2004; Muller and Wrangham, 2004; Ostner et al.,
172 2008; Gesquiere et al., 2011; Dantzer et al., 2017). Yet, the relationships between GCs and
173 social rank are not always consistent (Sapolsky and Ray, 1989; Creel, 2001; Creel et al., 2013),
174 and in many cases the endocrine and demographic consequences of social interactions and

social rank do not appear to be mediated by the SAM or HPA/I axes. In particular, studies in cooperative breeding species of effects of the social hierarchy on the stress axis have found that baseline GC concentrations are often higher in dominant than subordinates individuals (or not detectably related to social status). Although direct associations are often found between social hierarchy and various aspects of reproductive function in cooperative breeding species, this body of research has shown that these associations are not necessarily mediated by SAM or HPA/I function, and that other pathways can be involved, such as changes in sex steroid levels or the functioning of the hypothalamic-pituitary-gonadal HPG axis (Schoech et al., 1991; Abbott et al., 1997; Creel et al., 1997; Faulkes and Abbott, 1997; Carlson et al., 2004; reviewed in Montgomery et al., 2018; Creel, 2022 this issue; but see Young et al., 2006; Hart et al., 2022 this issue). These studies indicate that other important physiological mechanisms than the stress (HPA/I or SAM) axis are key to a proper understanding of the effects of social environments on individual phenotypes, and of the adaptive consequences of social interactions and social hierarchies.

For these reasons, our understanding of the fitness consequences that social interactions may have on vertebrates in the wild, and the extent to which they are an important source of phenotypic variation within and amongst social units (siblings, families, matrilines, colonies, etc.), should benefit from a holistic view encompassing key physiological functions, some centrally coordinated by the SAM or HPA/I axes, pertaining to vertebrate health (Sapolsky, 2005, 2004; Snyder-Mackler et al., 2020).

MOVING BEYOND THE STRESS AXES ALONE: SOCIAL EFFECTS ON VERTEBRATE FITNESS AND HEALTH

Health has often been defined as the absence of disease, inflammation or pathology (Blaxter, 2010). This definition, however, can be unpractical, especially when trying to determine how social stressors affect the functioning of organisms, as an overwhelming number of mechanisms and pathways can result in the genesis of diseases (Blaxter, 2010; Ayres, 2020). A more practical definition describes the hallmarks of health as the function of cells and organs, integration of functions and communication at the cellular and organism levels, and normal biological rhythms (López-Otín and Kroemer, 2021) where "normal" allows for allostasis and response to stress with no or minimal long-term deleterious effects, as the organismal response to stressful stimuli is part of the usual and adaptive functioning of organisms (McEwen and Wingfield, 2003; Romero et al., 2009). Below, we review the state of current knowledge on the effects that social environments have on vertebrate health by focusing on key functions. The key functions encompass individual metabolism/energetics, oxidative stress/damage/ageing processes, immunity/inflammation, cognitive/psychological state and decline, and the regulation of biological rhythms (Figure 1).

Social effects on metabolism and energetics: a key role for cellular mitochondria?

Regulating the organism's energy balance, and the rate at which organisms acquire and expend energy and materials, is central to maintaining life and governs several ecological processes (Brown et al., 2004). Because physical activity is associated with an increase in oxygen and energy substrate consumption that are needed to fuel muscular activity, it follows that social interactions are often associated with an increase in activity and therefore in metabolic rate. For instance, territorial defense against social conspecifics, the maintenance of a position in the social hierarchy, or vocal advertising during competitive displays have all been associated with increased metabolic rates in birds (Nephew and Romero, 2003; Lindström et al., 2005; Viera et al., 2011), reptiles (Marler et al., 1995), amphibians (Wells and Taigen, 1986), and fish

(Metcalfe et al., 1995; Sloman et al., 2000; Reid et al., 2011). However, it is not clear that increased metabolic rate in response to social stressors is due to increased physical activity alone. In brown trout (*Salmo trutta*) for instance, subordinate fish paired with dominant fish showed higher standard metabolic rate despite displaying overall lower levels of activity (Sloman et al., 2000). In king penguins (*Aptenodytes patagonicus*), bird daily heart rate at rest (a proxy to resting energy expenditure; Groscolas et al., 2010) is positively associated with increasing social density in breeding colonies (Viblanc et al., 2014b). Interestingly, higher social densities in penguins are also associated with higher baseline GC levels in individual birds (Viblanc et al., 2014a), suggesting a mechanistic link between social stressors, GCs, and metabolic rate. Besides their complex role in regulating glucose metabolism and the availability of energy substrates (Peckett et al., 2011; Kuo et al., 2015;), studies have indicated that GC play a role in stimulating the biogenesis of mitochondria and mitochondrial DNA transcription (Weber et al., 2002; Hunter et al., 2016; Morgan et al., 2016), which might account for elevated resting metabolic rates under stressful conditions.

There is an intuitive appeal in studying mitochondria to understand vertebrate responses to stressful (social) situations. Mitochondria, often referred to as 'energy powerhouses' of eukaryote cells, are organelles responsible for the majority of energy production in vertebrates (Lane, 2006). Their function, morphology and behavior (communication at inter-mitochondrial junctions; Picard et al., 2015) are responsible for determining much of animal performances (Heine and Hood, 2020). In the context of stress, mitochondria play a key role in making cellular energy available under challenging conditions by conforming to local levels of glucocorticoids (Manoli et al., 2007; Picard et al., 2014, 2018). But more importantly, mitochondria are key regulators that are involved in the stress response itself (Picard et al., 2014; Lapp et al., 2019). Following ACTH activation by the adrenal gland, the synthesis of steroids (including glucocorticoids) is limited by the transport of cholesterol across the

mitochondrial membrane for conversion into pregnanolone (Bose et al., 2002; Clark, 2016), and later conversion into GCs (corticosterone or cortisol) in the mitochondrial matrix before their release into the general circulation (Picard et al., 2018). Thus, mitochondria not only respond to variations in local GCs levels, but also play an essential role in their production.

Although the study of mitochondrial function is recently gaining attention in the field of ecology and evolution as a central effector mediating life history trade-offs (Criscuolo et al., 2005; Hood et al., 2018; Havird et al., 2019; Koch et al., 2021), there are yet few studies that have considered mitochondria under the light of organismal stress in wild vertebrates (Stier et al., 2019; Casagrande et al., 2020), except for the well-known role of mitochondria in the response to thermal stress (Criscuolo et al., 2005; Koch et al., 2021). To our knowledge, little is known on the relationships between social environments/social interactions and mitochondrial function in the wild. A recent study in captive rats identified the nucleus accumbens (NAc), a brain region involved in the expression of motivation and anxiety behaviors, as critical in the establishment of social hierarchies (Hollis et al., 2015). Surprisingly, anxious rats had reduced mitochondrial respiratory capacity in the NAc and lower ATP output. More importantly, experimentally inhibiting the activity of respiratory complexes in NAc mitochondria reduced the success of treated animals for winning social contests during the establishment of social hierarchies (Hollis et al., 2015). In addition, studies indicated that mitochondria are sensitive to time-dose dependent effects of stressors (Du et al., 2009; Picard et al., 2018), and may integrate and transduce psychosocial factors into cellular and molecular modifications, which in turn may contribute to the embedding of psychological states affecting social and other behaviors (reviewed in Picard and McEwen, 2018a, 2018b). These studies stemming from the neuroscience literature are important for our understanding of social stress in wild vertebrates, since they indicate a causal mechanistic pathway directly relating

mitochondrial function to the expression of social behaviors and social interactions, which has yet to be explored in nature.

There is at least one other aspect in which mitochondria are relevant to the study of social stress, and that is because they can be a source of cellular reactive oxygen species (ROS) production (Balaban et al., 2005; but see Zhang and Wong, 2021). ROS are by-products of normal cellular respiration, but can damage and disrupt the function of important biomolecules such as lipids, proteins and DNA, if their production outweighs the detoxifying physiological antioxidant systems organisms use to keep them in check (Monaghan et al., 2009).

Social effects on oxidative stress, health and ageing

ROS production has both positive and negative effects on vertebrate health: at low levels ROS play a key role as secondary messengers that can affect gene expression and actually promote resistance to stress (Ristow and Zarse, 2010; Costantini, 2014; Hood et al., 2018). At high levels, ROS cause oxidative stress, which has been suggested as an important factor behind organism decline and ageing (Beckman and Ames, 1998; Finkel and Holbrook, 2000; but see Speakman and Selman, 2011; Kirkwood and Kowald, 2012). In recent years, a growing number of studies have focused on the relationships between social interactions and increased oxidative stress in vertebrates. Opposite relationships pointing to beneficial effects of social environments on buffering oxidative stress have also been found (Lardy et al., 2016; Li and Xia, 2020). Interestingly, common to many of those studies is the observation that social instability and social aggression are associated with increased oxidative stress in mammals (Beaulieu et al., 2014; Nation et al., 2008), fish (Border et al., 2019, 2021; Fialkowski et al., 2021; but see Funnell et al., 2022), and birds (Silva et al., 2018; Quque et al., in press).

However, it is not only competitive social interactions that are related to increased oxidative stress. In certain cooperative breeders, such as the Damaraland mole rat (*Fukomys*

298 *damarensis*), increased contributions to cooperative behavior (in this case burrowing activity)
299 is associated with increased oxidative damage (in blood and ejaculates) (Mendonça et al., 2020).
300 Interestingly, in males, oxidative damage was biased towards the germline, indicating increased
301 protection of somatic cells at the expense of future reproduction, whereas in general, females
302 appeared to be better equipped in dealing with the oxidative costs of increased cooperation
303 (Mendonça et al., 2020). This study, however, manipulated burrowing (physical) effort, so that
304 increased oxidative stress was more likely the result of increased metabolism than of increased
305 social interactions *per se*. To date, we have little information on whether social interactions
306 intensify or ameliorate individual oxidative stress by other means than increasing metabolism.
307 As discussed above, modifications in the organisms' production of ROS might be mediated by
308 the interaction between GCs and mitochondrial function. In addition, GCs may have non-
309 metabolic effects on oxidative stress by down-regulating antioxidant defenses (Briehl and
310 Baker, 1996; McIntosh et al., 1998). Not surprisingly, increased oxidative stress has been
311 associated with increased GCs in vertebrates, at both baseline levels (Costantini et al., 2011)
312 and acute levels in response to a stressor (Majer et al., 2019; Stier et al., 2019; Casagrande et
313 al., 2020). Within the context of social interactions, however, the interplay among GCs,
314 mitochondrial function, ROS production, and antioxidant defense regulation remains under-
315 studied (Epel, 2009).

316 Studying the intricate relationship between social interactions, oxidative stress and the
317 stress axis in wild vertebrates may benefit from insights into laboratory studies on social
318 isolation. In socially isolated rats, enhanced oxidative stress in the brain seems due to an
319 increased expression of a ROS-generating family of enzymes known as of NADPH oxidases,
320 especially NOX-2 (Schiavone et al., 2009; Colaianna et al., 2013). NOX2-mediated oxidative
321 production appears to be an early trigger of HPA activation, since social isolation increases the
322 expression of NOX2 *before* the elevation of corticotropin-releasing hormone in the

hypothalamus or of ACTH in the blood. The latter initiate the stress response (mechanisms reviewed in Li and Xia, 2020). In addition, NOX2-mediated oxidative stress also appears to have important excitation functions in the sympathetic nervous system (SNS), specifically in the rostral ventrolateral medulla, a region of the brain responsible for control of sympathetic and cardiovascular function (Chan et al., 2005; Bai et al., 2009). Though we still lack information on the extent to which social isolation or social interactions may modulate SNS activity through NOX2-mediated oxidative stress, it is quite clear that ROS play a central role in shaping both HPA and SNS activity and the response to stress (Campese et al., 2004; for a review see Li and Xia, 2020). Thus, studies on wild vertebrates will benefit from investigations into the complex interplay that appears to exist between oxidative stress and GCs in shaping both HPA and SAM responses to social stressors.

From a health perspective, the study of individual oxidative stress in response to social interactions is of further interest because of the challenges ROS molecules pose to biological tissues. Increased accumulated damage resulting from free radicals over the lifetime of individuals is one of main theories proposed for explaining ageing processes in aerobic organisms (Harman, 1956; Finkel and Holbrook, 2000; also see Speakman and Selman, 2011; Kirkwood and Kowald, 2012). Specifically, reactive oxygen species are known to interact with DNA bases (notably guanine) which form the repeated non-coding DNA sequences that cap, stabilize and protect chromosome ends, known as telomeres (von Zglinicki, 2002; Kawanishi and Oikawa, 2004; Reichert and Stier, 2017). Telomere shortening has been associated with cell senescence and overall organism ageing (Blackburn, 2000; Blackburn and Epel, 2012) and thus may constitutes a good proxy for individual health and longevity. There is clear evidence from studies in humans that adverse social conditions (domestic violence, caregiving, poverty) and psychosocial stress associated with peer-pressure and poverty accelerate telomere loss and

cellular ageing (Epel et al., 2004; Entringer et al., 2011; Blackburn and Epel, 2012; Oliveira et al., 2016; Rentscher et al., 2020).

Although telomere shortening has been associated with increased exposure to various sources of stress in non-human vertebrates (Chatelain et al., 2020), it is only relatively recently that the effects of social environments and social interactions on telomere length and telomere shortening rates have been considered. In birds, studies have shown that both early-life social adversity (exposure to a large number of dominant competitors; *Sturnus vulgaris*; Nettle et al., 2013), and lack of social contact (social isolation; *Psittacus erithacus erithacus*; Aydinonat et al., 2014) can be associated with accelerated telomere loss. Social crowding also appears to interfere with telomere restoration mechanisms causing shorter telomeres in socially crowded groups (in lab mice, Kotrschal et al., 2007; in birds, Quque et al., *in press*). Moreover, in addition to direct effects of social interactions on telomere dynamics, studies indicate how interactions between social and reproductive strategies may shape ageing trajectories, both in early life and adulthood. For instance, social competition among mature cooperative breeders has been shown to carry long-term costs in terms of increased reproductive senescence (Sharp and Clutton-Brock, 2011). Further, dominant individuals who monopolize reproduction in cooperatively breeding sparrow-weavers (*Plocepasser mahali*) show higher investments into telomere maintenance than subordinates, an association likely to mitigate somatic costs of reproduction (Wood et al., 2021). In juveniles, early social competition for food within litters or juvenile cohorts may enhance telomere loss (Boonekamp et al., 2014; Cram et al., 2017; Nettle et al., 2015; but see van Lieshout et al., 2021), but the presence of cooperative adults helping with reproductive effort may favor telomere maintenance in offspring, suggesting positive social effects on parental care with downstream consequences on offspring health in the wild (Quque et al., 2021). Parents may then act as important buffers to stressful environments for their offspring (Bauer et al., 2015; Gunnar and Hostinar, 2015).

Thus, it appears clear that social interactions, status and environments have strong effects on cellular processes related to telomere degradation and maintenance, and overall organismal senescence. However, much remains to be understood about the interplay among social stressors, GCs, oxidative stress, telomere dynamics and their consequences on individual performance in the wild. Notably, telomere length and shortening rate appear to be related to chronic, but not short-term (Zane et al., 2021), modifications in GCs in vertebrates (reviewed in Angelier et al., 2018), though the direction of this relation (shorter telomeres with increased or reduced GCs) is not always consistent. Understanding how telomere biology is regulated by the interplay between GC and ROS production (Casagrande and Hau, 2019) is the next logical step in our understanding of social effects on vertebrate senescence and decline in overall performances in the wild. Particularly, in addition to focusing on telomere loss, it would be of interest to understand how telomerase activity (an enzyme specialized in re-building telomeres) is regulated by social stressors in nature (see Epel et al., 2010; Beery et al., 2012; Deng et al., 2016 for studies on captive animals and humans). In addition, the relationships between social stress and ageing are likely to extend to non-genetic molecular mechanisms as well, as recently demonstrated in wild yellow baboons (*Papio cynocephalus*) where high social rank males experience accelerated epigenetic ageing (Anderson et al., 2021).

Social effects on immunity

The immune system is the body's main line of defense against pathogens and parasites (Iwasaki and Medzhitov, 2010). Because social proximity and increased number of direct interactions among individuals increase the risk of pathogen and parasite transmission (Cote and Poulinb, 1995; Patterson and Ruckstuhl, 2013; Schmid-Hempel, 2017), sociality and immunity are tightly linked (Kappeler et al., 2015). The immune system can be divided into innate and adaptive responses which, although they interact, differ greatly in their specificity and

regulation by the HPA axis and release of GCs (Iwasaki and Medzhitov, 2010). Although GCs are best known for their suppressive effects on the immune system, there is rapidly accumulating evidence that GCs also enhance inflammation and immunity (Cain and Cidlowski, 2017). Accordingly, low doses of GCs are thought to promote localized inflammatory response (stimulating cytokines and complement responses) and suppress adaptive immunity, whereas high doses of GCs will suppress innate and adaptive immunity, therefore preventing excessive and/or prolonged immune responses (Cain and Cidlowski, 2017). Hence, the effects of social interactions on GCs and, in turn, immunity, are expected to be complex and to lead to nuanced results. For instance, adaptive immunity was observed to decrease with group size (and social status) in house finches (*Carpodacus mexicanus*) (Hawley et al., 2006), but to increase with group size in other bird species (Minias et al., 2019; Tella et al., 2001; Kamiński et al., 2021). Predicting how social effects, immunity and parasite load are inter-linked is further complicated by the fact that immunity is not regulated by GCs alone (Iwasaki and Medzhitov, 2010), with many other factors besides social interactions and immunity influencing parasite load (Bize et al., 2008). This is well illustrated in studies on social status (Habig and Archie, 2015; Habig et al., 2018). For instance, high rank individuals are expected to be more exposed to parasites, as they often show increased number of social interactions, have greater energy expenditure, and need to feed more frequently (Clutton-Brock and Huchard, 2013). Alternatively, high rank individuals can be more exposed to parasites as they often show greater investment into reproduction that can come at the expense of immune defences. Finally, as low rank individuals experience more frequent defeats in social antagonistic interactions, they are expected to also show lower immunity and higher levels of parasitism caused by social stress and increased circulating GCs. Comparative studies in vertebrates show that social rank has only weak effects on immune functions but often strong effects on parasite load (Habig and Archie, 2015; Habig et al., 2018), with dominant individuals

(especially males) showing higher levels of parasitism. These findings suggest that rank-associated variations in parasitism are primarily influenced by exposure to parasites and trade-offs between reproduction and immunity. In addition, the relationship between the social environment and immunity is complex as social structures and social relations are conditioned by trade-offs between the benefits of sharing social information and the costs of transmitting pathogens (Romano et al., 2020, 2021).

Socially interacting individuals can also limit the spread of parasites when both the individuals who initiated the social interaction and the recipients enjoy greater protection, such as by allogrooming, referred to in the literature as collective defences or social immunity (Cotter and Kilner, 2010). Interestingly, low levels of GCs have been suggested to favour proactive behaviour (Raulo and Dantzer, 2018), that in turn could favour the occurrence of allo-grooming in social interacting species. The role of GCs in shaping social immunity versus personal immunity remains to be investigated in detail.

Finally, it is important to note that the relationship between social behavior and immunity is bidirectional. It is now well demonstrated that the immune system communicates with the brain via the release of proinflammatory cytokines and chemokines, which can impair social behavior by leading to social withdrawal (Dantzer and Kelley, 2007; Kopec et al., 2019). These changes, together with reduced activity, referred to as 'sickness behavior', are widely viewed as an adaptive host response that prevents parasite transmission rather than a manipulation of the host behaviour by parasites (Dantzer and Kelley, 2007). A chronic inflammation and immune-neuro modulation of the brain can however lead to social isolation and depression (Raison et al., 2006). Although the immune-neuro modulation of sickness behaviour is evolutionary conserved at least in mammals and birds (Dantzer and Kelley, 2007), we still know very little about effects of inflammation on social interactions in the wild (Stockmaier et al., 2018; Hamilton et al., 2020) and their consequences on health and fitness.

447

448 **Social effects on the brain and psychological states**

449 Effects of (social) stress on brain development, morphology and plasticity (Blanchard et al.,
450 2001; Oitzl et al., 2010; Madalena and Lerch, 2017; Cameron and Schoenfeld, 2018), and in
451 turn on psychological states (anxiety and depression) (Lukkes et al., 2009; Teo et al., 2013),
452 cognition (memory, learning) (Modlinska et al., 2018; Hesse et al., 2019; Lambert and Guillette,
453 2021), and social competences (Taborsky, 2016; Reyes-Contreras et al., 2019) have been
454 extensively studied, especially, in fish, rodents and humans. For example, using the mouse
455 model for studying depression, chronic social stress was found to increase the permeability of
456 the blood brain barrier, which in turn increases the infiltration of peripheral inflammatory
457 signals into the brain, leading to neuroinflammations and depression-like behaviors (Menard et
458 al., 2017). Beside immune signaling to the brain (see 'social effects on immunity'), GCs are
459 also known to strongly influence learning abilities, and memory formation and maintenance by
460 binding to glucocorticoid receptors (GR) and mineralocorticoid receptors (MR) widely
461 distributed throughout the brain. The (dis)balance between GR:MR receptors in particular is
462 thought to play a central role in altering information processing in the neural circuits underlying
463 fear, reward, social behaviour and resilience, and subsequently in altering the behaviours,
464 psychological states and cognitive abilities of individuals (Oitzl et al., 2010). As GCs have been
465 shown to promote aversive memories following social contests (Tertil et al., 2018), they can
466 play a role in shaping individual social memory and social competence, that is, the expression
467 of context-relevant behaviors (*e.g.*, subordinates showing submissive behavior in presence of
468 higher-ranking individuals), via neurological and behavioral processes. Consistent with this, in
469 a cooperatively breeding fish, experimental exposure to GCs during the juvenile stage resulted
470 in persistent down-regulation of MR but not GR gene expression in the brain (telencephalon)
471 and reduced social competence (Reyes-Contreras et al., 2019). Although research on the effects

of social stress on brain development, morphology and plasticity is likely to remain scarce in wild vertebrates due to the invasive nature of such studies (and the associated ethical challenges), much remains to be done to study the effect of the social environment on psychological states associated with fear or phobias (such as neophobia) (Kelly et al., 2020) and cognition (Heinen et al., 2021) in natural populations, as well as possible connections with markers of heath as oxidative stress and mitochondrial function (Hoffmann and Spengler, 2018) (see also 'Social effects on metabolism and energetics: a key role for cellular mitochondria?' and 'Social effects on oxidative stress, health and ageing').

Finally, social interactions can also have positive effects by facilitating learning (Aplin et al., 2015), and also by helping to buffer stress associated with negative memories (fear) (Leblanc and Ramirez, 2020; Mikami et al., 2020). Hence, social effects on the brain and especially behavior and cognition, can be seen as a mechanism promoting 'social homeostasis', that is to say, the maintenance of a degree of social connection necessary for the normal function of organisms and maintenance of health (Matthews and Tye, 2019). In line with this, a systematic review has shown that more, better, or more diverse opportunities for social experiences early in life lead in most cases to better social skills in the same individuals measured later in life (Taborsky, 2016). As a result, social learning and memory of social interactions are expected to be important mechanisms shaping future behavior and long-term health and fitness outcomes in natural populations.

Social effects on biological rhythms

A ubiquitous feature of vertebrate biological functions (including gene expression, physiology and behavior) is their rhythmicity (Aschoff, 1981; Rusak, 1981; Kumar, 2002). Most physiological systems function in pulsatile fashion, with ultradian, circadian, diurnal or infradian patterns of activation (*e.g.*, hormonal secretion), often synchronized by environmental

497 cues known as *zeitgebers* (photoperiod, temperature, exercise, social interactions) (Aschoff,
498 1981; Kumar, 2002). For instance, GC secretion follows circadian and ultradian biological
499 rhythms important in regulating metabolism, inflammation, mood, cognition and stress
500 responsiveness (Focke and Iremonger, 2020). The HPA axis itself – especially the adrenal gland
501 – plays a key role in transmitting biological rhythms to the entire body (Kalsbeek et al., 2012;
502 Rao and Androulakis, 2019).

503 The importance of biological rhythmicity to health and fitness is made clear by the
504 observation in humans and laboratory animals that chronic disruptions of biological rhythms
505 can lead to pathological dysregulation of physiological systems. These range from systemic
506 immune/ metabolic disorders and cognitive impairment (Takahashi et al., 2008; Delezie and
507 Challet, 2011; Karatsoreos et al., 2011; Cermakian et al., 2014; Rao and Androulakis, 2019),
508 central nervous system disorders including anxiety, schizophrenia, depression, and bi-polarity
509 amongst others (Lamont et al., 2007; McClung, 2007; Benca et al., 2009), to cell senescence
510 (Grosbellet et al., 2015) and cancer (Sephton and Spiegel, 2003; Shilts et al., 2018). However,
511 it is only recently, and mostly in the context of anthropogenic disturbance and urbanization
512 (Dominoni et al., 2013, 2016; Kolbe et al., 2021; Secondi et al., 2021; Ziegler et al., 2021), that
513 studies on wild vertebrates have focused on the extent to which chronic disruption of biological
514 rhythms may occur in nature. Yet, social interactions have been suggested to play a key role in
515 the regulation of biological rhythms and health (Ehlers et al., 1988; Mistlberger and Skene,
516 2004). Studying the effects social environments may have on the dysregulation of biological
517 rhythmicity may be important for better understanding how physiological costs of social
518 relationships arise, for instance between parents and offspring, mates, or dominants and
519 subordinates. The social zeitgeber theory (Ehlers et al., 1988) proposes that disruptions in
520 individual social routines can cause dysregulation in biological rhythms, themselves leading to
521 the deleterious health consequences mentioned above. The influence of social routines on

biological rhythms and health have been particularly studied with reference to mood regulation and mental disorders in humans (Grandin et al., 2006; Shen et al., 2008; Margraf et al., 2016; Takaesu, 2018; Sabet et al., 2021). Though studies in other vertebrates have focused on social entrainment of behavioral circadian rhythmicity (Favreau et al., 2009), fewer have focused on the interrelations among social stressors, biological rhythms and health. Yet, social aggression has been found to alter patterns of melatonin secretion (in mammals, Heinzeller et al., 1988; in fish, Larson et al., 2004), a key hormone secreted by the pineal gland responsible for synchronizing daily rhythms to light/dark cycles in vertebrates, perhaps explaining differences in behavioral profiles between subordinate and dominant individuals (Larson et al., 2004). Indeed, melatonin is itself known to play a role in the expression of social aggression (Jasnow et al., 2002; Demas et al., 2004; Munley et al., 2020), and GC and/or adrenoreceptors have been found in the vertebrate pineal gland (e.g. in fish; Benyassi et al., 2001; in rats; Fernandes et al., 2017). These studies suggest that causal pathways might exist from the perception of social stressors to the expression of social behaviors through the regulation of the pineal gland. In addition, the pineal gland and melatonin play a key role in the innate immune response for instance by favoring phagocytosis and modulating inflammation (Majewski et al., 2012; Markus and Ferreira, 2011). Thus, cross-talks between the HPA/SAM axis, the immune system, the pineal gland, and the expression of social behaviors appear to be features that require further exploration in the context of social stressors in wild vertebrates (Couto-Moraes et al., 2009).

WHERE NEXT?

The sheer breadth of studies presented above highlights how important social effects can be in affecting the stress, health, and fitness of vertebrates. On one hand, many of these studies have emerged from the biomedical community (Epel et al., 2004; Entringer et al., 2011; Blackburn and Epel, 2012; Mitchell et al., 2014) with the growing recognition that societal factors (broadly

including work stress, emotional neglect and abuse, financial hardship, income, gender, education, unemployment, early childhood development, housing, social exclusion, social support networks, social gradients, war, etc.) are key determinants of individual health in human societies (World Health Organization 2022). On the other, studies from the ecological community have started to delve into the intricacies of socially-induced consequences on individual physiology in wild vertebrates, but so far, have – by and large – mostly focused on social effects on the stress axis. Yet, much remains to be learned about the way in which social environments might affect individual health and phenotypes *sensu largo*, with joint examination of individual anatomy, physiology and behavior. We believe that cross-talks between the biomedical and ecological communities will take us further in our understanding of how evolution has shaped similar or specific phenotypic responses to social interactions, ubiquitous across vertebrates. Integrating the study of social stressors and their mechanistic effects on animal phenotypes at the level of organs, cells, organelles, proteins and gene expression, with joint examination of relevant ecological (*e.g.*, density, predation pressure, parasite pressure, resource abundance) and life history (*e.g.*, trade-off between reproduction and self-maintenance) factors affecting wild animals, will allow shedding lights on the actual benefits and costs of sociality.

First, the evaluation of the various health markers described above in a wider range of species encountering different habitats, social environments and having different life-histories is necessary to assess how these different markers respond to various types of social and non-social environments. Such assessments will provide a better understanding of their associations with individual survival, reproduction, life history, and fitness. Implementing the evaluation of such markers in field-based ecological studies is challenging, considering the methodological and ethical limitations inherent to the study of wild animals. However, available tools in both the ecological and the biomedical fields can, and are already, being used. For instance, there is

572 an increasing interest in the measure of mitochondrial respiration or aerobic metabolism and
573 the evaluation of mitochondrial function in wild vertebrates (reviewed in Koch et al., 2021).
574 Evaluating mitochondrial function is complex for field ecologists, as sampling protocols can be
575 invasive and biological samples must be assayed in a dedicated laboratory within hours of
576 collection. However, in some taxa (i.e. birds, amphibians and reptiles), recent methodological
577 developments now allow measuring mitochondrial function longitudinally and in a minimally
578 invasive way (Stier et al., 2013; 2015; 2017). These developments allow for new insights on
579 the cascading effects of social stress on GCs, mitochondrial traits, health and fitness
580 (Casagrande et al., 2020b; Stier et al., 2019). Similarly, methodological as well as ethical
581 limitations greatly reduce the possibility of assessing brain function in wild vertebrates, which
582 are mainly being done through indirect observations such as learning behavior and capacity,
583 and memory (see methods in Hesse et al., 2019; Modlinska et al., 2018; Reyes-Contreras et al.,
584 2019; White et al., 2012). Evaluating brain function of free ranging vertebrates would gain from
585 the development of minimally invasive and validated methods from the neurological and
586 biomedical fields and the development of sampling or recording techniques of relevant
587 parameters (brain activity, transcriptomics, proteomics).

588 In this regard, bio-loggers, which are already being extensively used in the field of
589 ecophysiology in free ranging animals, may provide an interesting and practical method for
590 studying individual health in ecological settings (Jax et al., 2021). Bio-loggers are monitoring
591 devices that allow acquiring fine-resolution data ranging from activity (movement, energy
592 expenditures, acceleration), to foraging, temperature, or sleep, on periods ranging from days to
593 months. Bio-loggers are honed for specific study species, parameters, and durations of
594 monitoring (Ropert-Coudert et al., 2012; Wilmers et al., 2015). The relevance of bio-loggers to
595 the study of social interaction – health relationships is rendered event more salient by recent
596 technological advances allowing the acquisition of detailed information on social interactions

in situations where direct visual observation is not always possible (Rutz et al., 2012; Smith and Pinter-Wollman, 2021). The use of such tools and the fine resolution of data collected are promising avenues for measuring metrics related to energy metabolism (e.g. via heart rate and ECG, and accelerometry; Green et al., 2009), immunity (e.g. via body temperature; Jax et al., 2021), rhythmicity (e.g. via accelerometry and activity patterns); and brain activity (via EEG; Vyssotski et al., 2006), with regards to varying social interactions, social contexts, and hierarchies.

Second, the adaptive foundations of stress responses have been little investigated, compared to the wealth of knowledge of how mechanisms that produce stress responses operate. Specifically, studies of differences in reproduction and survival of individuals that respond appropriately to ecological and social stressors might be expected to produce nuanced results. The primary difficulty will be to demonstrate the evolutionary benefits of stress responses in fitness terms. A key to our understanding of these evolutionary benefits will be in integrating the study of phenotypic plasticity to social environments within the lifetime of an individual (Levins, 1968). Phenotypic plasticity is the ability of one genotype to produce multiple phenotypes over a life time as the environment, or that of the offspring, changes (Charmantier et al., 2008; Chevin and Hoffmann, 2017). Its study is particularly relevant to the issue of social change. Animals moving through life, from infant to subadult to adult, or from subordinate to dominant, will experience changes in their social environment. Being able to adjust to these changes adaptively will result in fitness benefits. For example, as food supply fluctuates in red squirrel along with social competition for limited resources, maternal hormones result in offspring with different growth trajectories and different survival prospects in the long run (Dantzer et al., 2013).

Studies of mechanisms responsible for intergenerational consequences of social environments across vertebrates will prove particularly exciting. Such studies will reveal the

622 mechanisms through which social environments encountered by parents, early in their lives (in
623 the womb, egg, den, nest), influence the development of future (offspring) phenotypes. This
624 intergenerational embedding of influences of parental environments (including, but not limited
625 to, social experiences) are today known to occur through transmission of maternal hormones in
626 the yolk, placenta or milk (Mazuc et al., 2003; Kaiser and Sachser, 2005; Guibert et al., 2010;
627 Edwards et al., 2021; Stead et al., 2022), or epigenetic inheritance (Cunliffe, 2016; Venney et
628 al., 2020), sometimes mediated via variations in parental care (Champagne, 2008, 2010).
629 Prenatal exposure to ecological or social stressors (whether competition or social isolation)
630 experienced by parents while offspring are in the womb or in the egg, may directly affect
631 offspring neuroendocrine function (Sheriff et al., 2010; Love et al., 2013). For instance,
632 mediations may occur via alterations of the expression/transcription of specific genes/proteins
633 related to the programming/functioning of the stress axis (Marasco et al., 2016; D'Agostino et
634 al., 2019; Mueller et al., 2021; Haq et al., 2021). Yet, the extent to which such effects are
635 adaptive or not in ecological environments is unclear, and still under intense scrutiny (Sheriff
636 and Love, 2013; Sheriff et al., 2017; Sopinka et al., 2017; Yin et al., 2019; Sánchez-Tójar et al.,
637 2020; Zhang et al., 2020). Here again, there is an urgent need for studies where the fitness
638 consequences (encompassing both parental and offspring reproduction and survival) of early
639 social environments can be evaluated (Fagundes et al., 2013). Long-term field studies of wild
640 vertebrates (where physiological, behavioral data and life-history data can be acquired) will
641 prove particularly useful in doing so, though assessing the mechanistic (physiological)
642 pathways relating social environments to phenotypes and fitness in longitudinal monitoring
643 schemes is faced with the considerable challenge of doing so in a minimally invasive way (but
644 see Anderson et al., 2021). Research into methodological refinements for longitudinal measures
645 of physiological markers at organ, cellular and subcellular levels is thus needed.

Finally, studies in evolutionary ecology would benefit from integrating the concepts of social stress and social health in thinking about the evolution of group-living. Many vertebrate species show some degree of intra-specific social flexibility ranging from solitary to group living, depending on season or environmental contexts (Schradin, 2013), so that most individuals are subject to variable social contexts within their lifetimes. Several studies have considered how individual physiological requirements may shape social systems (e.g. in equids; Gersick and Rubenstein, 2017), or shown how social group fission or natal dispersal may arise from competition for resources and mates, as well as limited breeding opportunities (Greenwood, 1980; Dobson, 1982; Waser, 1985). However, the extent to which social flexibility arises as a response to the intensification of social competition in other contexts, or alleviates individuals from the costs of social isolation, at given periods of time, remains to be considered.

ACKNOWLEDGMENTS

We wish to thank M Homes and PD Edwards for inviting us to contribute to the special issue on Hormones & Hierarchies. We are grateful to two anonymous reviewers for insightful and constructive comments on a previous version of this review paper. This paper is a product from the ENS-Lyon MSc review assignment of CL, and the "Social stress and early environmental effects workshop" held at the Institut Pluridisciplinaire Hubert Curien, UMR 7178 CNRS in July 2017. We are grateful to Richard Bon, Palmyre Boucherie, Josefa Bleu, Ivan Puga-Gonzalez, Charlotte Récapet, Carsten Schradin, Quentin Schull, Bernard Thierry, and Antoine Stier for insightful discussions during the workshop. The workshop was supported by a CNRS-PICS grant to VA Viblanc (PICS-07143 SOSTRESS Understanding the oxidative and pro-ageing consequences of social stress in wild animal populations). FSD and VAV were supported by a fellowship grant from the Institute of Advanced Studies of the University of

Strasbourg on the effects of social environments on individual stress. FSD thanks the Région Grand Est and Eurométropole de Strasbourg for the award of a Gutenberg Excellence Chair on those questions. C Lemonnier was supported by the ENS de Lyon (Ministère de l'Enseignement Supérieur et de la Recherche) during the time of writing.

REFERENCES

Abbott, D.H., Saltzman, W., Schultz-Darken, N.J., Smith, T.E., 1997. Specific neuroendocrine mechanisms not involving generalized stress mediate social regulation of female reproduction in cooperatively breeding marmoset monkeys. Ann. NY Acad. Sci. 807, 219–238.

Abbott, D.H., Keverne, E.B., Bercovitch, F.B., Shively, C.A., Mendoza, S.P., Saltzman, W., Snowdon, C.T., Ziegler, T.E., Banjevic, M., Garland, T., Sapolsky, R.M., 2003. Are subordinates always stressed? A comparative analysis of rank differences in cortisol levels among primates. Horm. Behav. 43, 67–82.

Alexander, R.D., 1974. The evolution of social behavior. Ann. Rev. Ecol. Systematics 5, 325–383.

Alexander, S., Irvine, C., 1998. The effect of social stress on adrenal axis activity in horses: the importance of monitoring corticosteroid-binding globulin capacity. J. Endocrinol. 157, 425–432.

Anderson, J.A., Johnston, R.A., Lea, A.J., Campos, F.A., Voyles, T.N., Akinyi, M.Y., Alberts, S.C., Archie, E.A., Tung, J., 2021. High social status males experience accelerated epigenetic aging in wild baboons. eLife 10, e66128.

Angelier, F., Costantini, D., Blévin, P., Chastel, O., 2018. Do glucocorticoids mediate the link between environmental conditions and telomere dynamics in wild vertebrates? A review. Gen. Comp. Endocrinol. 256, 99–111.

Aplin, L.M., Farine, D.R., Morand-Ferron, J., Cockburn, A., Thornton, A., Sheldon, B.C., 2015. Experimentally induced innovations lead to persistent culture via conformity in wild birds. Nature 518, 538–541.

Aschoff, J. (Ed.), 1981. Biological rhythms. Springer US, Boston, MA.

Avitsur, R., Padgett, D.A., Sheridan, J.F., 2006. Social interactions, stress, and immunity. Neurol. Clinics 24, 483–491.

Aydinonat, D., Penn, D.J., Smith, S., Moodley, Y., Hoelzl, F., Knauer, F., Schwarzenberger, F., 2014. Social isolation shortens telomeres in African grey parrots (*Psittacus erithacus erithacus*). PLoS ONE 9, e93839.

Ayres, J.S., 2020. The biology of physiological health. Cell 181, 250–269.

Bai, Y., Jabbari, B., Ye, S., Campese, V.M., Vaziri, N.D., 2009. Regional Expression of NAD(P)H Oxidase and Superoxide Dismutase in the Brain of Rats with Neurogenic Hypertension. Am. J. Nephrol. 29, 483–492.

Balaban, R.S., Nemoto, S., Finkel, T., 2005. Mitochondria, oxidants, and aging. Cell 120, 483–495.

Bateson, M., 2016. Cumulative stress in research animals: Telomere attrition as a biomarker in a welfare context? BioEssays 38, 201–212.

Bauer, C.M., Hayes, L.D., Ebensperger, L.A., Ramírez-Estrada, J., León, C., Davis, G.T., Romero, L.M., 2015. Maternal stress and plural breeding with communal care affect

715 development of the endocrine stress response in a wild rodent. Horm. Behav. 75, 18–
716 24.
717 Beaulieu, M., Mboumba, S., Willaume, E., Kappeler, P.M., Charpentier, M.J.E., 2014. The
718 oxidative cost of unstable social dominance. J. Exp. Biol. 217, 2629–2632.
719 Beckman, K.B., Ames, B.N., 1998. The free radical theory of aging matures. Physiol. Rev.
720 78, 547–581.
721 Beehner, J.C., Bergman, T.J., 2017. The next step for stress research in primates: To identify
722 relationships between glucocorticoid secretion and fitness. Horm. Behav. 91, 68–83.
723 Beery, A.K., Lin, J., Biddle, J.S., Francis, D.D., Blackburn, E.H., Epel, E.S., 2012. Chronic
724 stress elevates telomerase activity in rats. Biol. Lett. 8, 1063–1066.
725 Benca, R., Duncan, M.J., Frank, E., McClung, C., Nelson, R.J., Vicentic, A., 2009. Biological
726 rhythms, higher brain function, and behavior: Gaps, opportunities, and challenges.
727 Brain Res. Rev. 62, 57–70.
728 Benyassi, A., Schwartz, C., Ducouret, B., Falcón, J., 2001. Glucocorticoid receptors and
729 serotonin N-acetyltransferase activity in the fish pineal organ: Neuroreport 12, 889–
730 892.
731 Bize, P., Jeanneret, C., Klopfenstein, A., Roulin, A., 2008. What makes a host profitable?
732 Parasites balance host nutritive resources against immunity. Am. Nat. 171, 107–118.
733 Blackburn, E.H., 2000. Telomere states and cell fates. Nature 408, 53–56.
734 Blackburn, E.H., Epel, E.S., 2012. Telomeres and adversity: Too toxic to ignore. Nature 490,
735 169–171.
736 Blanchard, R.J., McKittrick, C.R., Blanchard, D.C., 2001. Animal models of social stress:
737 effects on behavior and brain neurochemical systems. Physiol. Behav. 73, 261–271.
738 Blaxter, M., 2010. Health, 2nd ed. Polity Press, Cambridge, UK.
739 Bonier, F., Martin, P.R., Moore, I.T., Wingfield, J.C., 2009. Do baseline glucocorticoids
740 predict fitness? Trends Ecol. Evol. 24, 634–642.
741 Boogert, N.J., Farine, D.R., Spencer, K.A., 2014. Developmental stress predicts social
742 network position. Biol. Lett. 10, 20140561.
743 Boonekamp, J.J., Mulder, G.A., Salomons, H.M., Dijkstra, C., Verhulst, S., 2014. Nestling
744 telomere shortening, but not telomere length, reflects developmental stress and
745 predicts survival in wild birds. Proc. Roy. Soc. B 281, 20133287.
746 Boonstra, R., Hik, D., Singleton, G.R., Tinnikov, A., 1998. The impact of predator-induced
747 stress on the snowshoe hare cycle. Ecol. Monogr. 68, 371-394.
748 Boonstra, R., 2004. Coping with changing northern environments: The role of the stress axis
749 in birds and mammals. Integr. Comp. Biol. 44, 95–108.
750 Boonstra, R., 2005. Equipped for life: the adaptive role of the stress axis in male mammals. J.
751 Mammal. 86, 236–247.
752 Boonstra, R., Barker, J.M., Castillo, J., Fletcher, Q.E., 2007. The role of the stress axis in life-
753 history adaptations. Rodent societies: an ecological and evolutionary perspective, 139-
754 149.
755 Boonstra, R., 2013. Reality as the leading cause of stress: rethinking the impact of chronic
756 stress in nature. Funct. Ecol. 27, 11–23.
757 Border, S.E., DeOliveira, G.M., Janeski, H.M., Piefke, T.J., Brown, T.J., Dijkstra, P.D., 2019.
758 Social rank, color morph, and social network metrics predict oxidative stress in a
759 cichlid fish. Behav. Ecol. 30, 490–499.
760 Border, S.E., Piefke, T.J., Funnell, T.R., Fialkowski, R.F., Sawecki, J., Dijkstra, P.D., 2021.
761 Social instability influences rank-specific patterns of oxidative stress in a cichlid fish.
762 J. Exp. Biol. 224, jeb237172.
763 Bose, H.S., Lingappa, V.R., Miller, W.L., 2002. Rapid regulation of steroidogenesis by
764 mitochondrial protein import. Nature 417, 87–91.

765 Breuner, C.W., Delehanty, B., Boonstra, R., 2013. Evaluating stress in natural populations of
766 vertebrates: total CORT is not good enough. Funct. Ecol. 27, 24–36.
767 Breuner, C.W., Orchinik, M., Hahn, T.P., Meddle, S.L., Moore, I.T., Owen-Ashley, N.T.,
768 Sperry, T.S., Wingfield, J.C., 2003. Differential mechanisms for regulation of the
769 stress response across latitudinal gradients. Am. J. Physiol. Reg. Integr. Comp.
770 Physiol. 285, R594–R600.
771 Briehl, M.M., Baker, A.F., 1996. Modulation of the antioxidant defence as a factor in
772 apoptosis. Cell Death Differ. 3, 63–70.
773 Brown, J.H., Gillooly, J.F., Allen, A.P., Savage, V.M., West, G.B., 2004. Toward a metabolic
774 theory of ecology. Ecology 85, 1771–1789.
775 Cain, D.W., Cidlowski, J.A., 2017. Immune regulation by glucocorticoids. Nat. Rev.
776 Immunol. 17, 233–247.
777 Cameron, A., Henley, D., Carrell, R., Zhou, A., Clarke, A., Lightman, S., 2010. Temperature-
778 responsive release of cortisol from its binding globulin: a protein thermocouple. J.
779 Clin. Endocrinol. Metab. 95, 4689–4695.
780 Cameron, H.A., Schoenfeld, T.J., 2018. Behavioral and structural adaptations to stress. Front.
781 Neuroendocrinol. 49, 106–113.
782 Campese, V.M., Ye, S., Zhong, H., Yanamadala, V., Ye, Z., Chiu, J., 2004. Reactive oxygen
783 species stimulate central and peripheral sympathetic nervous system activity. Am. J.
784 Physiol. Heart Circulat. Physiol. 287, H695–H703.
785 Carlson, A.A., Young, A.J., Russell, A.F., Bennett, N.C., McNeilly, A.S., Clutton-Brock, T.,
786 2004. Hormonal correlates of dominance in meerkats (*Suricata suricatta*). Horm.
787 Behav. 46, 141–150.
788 Casagrande, S., Hau, M., 2019. Telomere attrition: metabolic regulation and signalling
789 function? Biol. Lett. 15, 20180885.
790 Casagrande, S., Stier, A., Monaghan, P., Loveland, J.L., Boner, W., Lupi, S., Trevisi, R., Hau,
791 M., 2020. Increased glucocorticoid concentrations in early life cause mitochondrial
792 inefficiency and short telomeres. J. Exp. Biol. 223, jeb.222513.
793 Cermakian, N., Westfall, S., Kiessling, S., 2014. Circadian clocks and inflammation:
794 reciprocal regulation and shared mediators. Arch. Immunol. Ther. Exp. 62, 303–318.
795 Champagne, F.A., 2010. Epigenetic influence of social experiences across the lifespan.
796 Develop. Psychobiol. 52, 299–311.
797 Chan, S.H.H., Hsu, K.-S., Huang, C.-C., Wang, L.-L., Ou, C.-C., Chan, J.Y.H., 2005.
798 NADPH oxidase–derived superoxide anion mediates angiotensin II–induced pressor
799 effect via activation of p38 mitogen–activated protein kinase in the rostral
800 ventrolateral medulla. Circulat. Res. 97, 772–780.
801 Charmantier, A., McCleery, R.H., Cole, L.R., Perrins, C., Kruuk, L.E.B., Sheldon, B.C.,
802 2008. Adaptive Phenotypic Plasticity in Response to Climate Change in a Wild Bird
803 Population. Science 320, 800–803.
804 Chatelain, M., Drobniak, S.M., Szulkin, M., 2020. The association between stressors and
805 telomeres in non-human vertebrates: a meta-analysis. Ecology Letters 23, 381–398.
806 Chevin, L.-M., Hoffmann, A.A., 2017. Evolution of phenotypic plasticity in extreme
807 environments. Phil. Trans. R. Soc. B 372, 20160138.
808 Clark, B.J., 2016. ACTH Action on StAR Biology. Front. Neurosci. 10, 547.
809 Clutton-Brock, T.H., Huchard, E., 2013. Social competition and selection in males and
810 females. Phil. Trans. R. Soc. B 368, 20130074.
811 Cohen, S., 2004. Social Relationships and Health. Am. Psychol. 59, 676–684.
812 Colaianna, M., Schiavone, S., Zotti, M., Tucci, P., Morgese, M.G., Bäckdahl, L., Holmdahl,
813 R., Krause, K.-H., Cuomo, V., Trabace, L., 2013. Neuroendocrine profile in a rat

model of psychosocial stress: relation to oxidative stress. Antiox. Redox Signal. 18, 1385–1399.
Costantini, D., 2014. Oxidative stress and hormesis in evolutionary ecology and physiology: A marriage between mechanistic and evolutionary approaches, 1st ed. Springer, Berlin, Heidelberg.
Costantini, D., Marasco, V., Møller, A.P., 2011. A meta-analysis of glucocorticoids as modulators of oxidative stress in vertebrates. J. Comp. Physiol. B.
Cote, I.M., Poulinb, R., 1995. Parasitism and group size in social animals: a meta-analysis. Behavi. Ecol. 6, 159–165.
Côté, S.D., 2000. Aggressiveness in king penguins in relation to reproductive status and territory location. Anim. Behav. 59, 813–821.
Cotter, S.C., Kilner, R.M., 2010. Personal immunity versus social immunity. Behav. Ecol. 21, 663–668.
Couto-Moraes, R., Palermo-Neto, J., Markus, R.P., 2009. The immune-pineal axis: Stress as a modulator of pineal gland function. Ann. NY Acad. Sci. 1153, 193–202.
Cram, D.L., Monaghan, P., Gillespie, R., Clutton-Brock, T., 2017. Effects of early-life competition and maternal nutrition on telomere lengths in wild meerkats. Proc. R. Soc. B. 284, 20171383.
Creel, S., 2001. Social dominance and stress hormones. Trends Ecol. Evol. 16, 491–497.
Creel, S., 2022. A retrospective view of early research on dominance, stress and reproduction in cooperatively breeding carnivores. Horm. Behav. 140, 105119.
Creel, S., Creel, N.M., Mills, M.G.L., Monfort, S.L., 1997. Rank and reproduction in cooperatively breeding African wild dogs: behavioral and endocrine correlates. Behav. Ecol. 8, 298–306.
Creel, S., Dantzer, B., Goymann, W., Rubenstein, D.R., 2013. The ecology of stress: effects of the social environment. Functional Ecology 27, 66–80.
Criscuolo, F., Gonzalez-Barroso, M. del M., Bouillaud, F., Ricquier, D., Miroux, B., Sorci, G., 2005. Mitochondrial uncoupling proteins: new perspectives for evolutionary ecologists. Am. Nat. 166, 686–699.
Cunliffe, V.T., 2016. The epigenetic impacts of social stress: how does social adversity become biologically embedded? Epigenomics 8, 1653–1669.
D'Agostino, S., Testa, M., Aliperti, V., Venditti, M., Minucci, S., Aniello, F., Donizetti, A., 2019. Expression pattern dysregulation of stress- and neuronal activity-related genes in response to prenatal stress paradigm in zebrafish larvae. Cell Stress Chaperones 24, 1005–1012.
Dantzer, B., Newman, A.E.M., Boonstra, R., Palme, R., Boutin, S., Humphries, M.M., McAdam, A.G., 2013. Density triggers maternal hormones that increase adaptive offspring growth in a wild mammal. Science 340, 1215–1217.
Dantzer, B., Bennett, N.C., Clutton-Brock, T.H., 2017. Social conflict and costs of cooperation in meerkats are reflected in measures of stress hormones. Behav. Ecol. 28, 1131–1141.
Dantzer, R., Kelley, K.W., 2007. Twenty years of research on cytokine-induced sickness behavior. Brain Behav. Immun. 21, 153–160.
Delehanty, B., Hossain, S., Jen, C.C., Crawshaw, G.J., Boonstra, R., 2015. Measurement of free glucocorticoids: Quantifying corticosteroid-binding globulin binding affinity and its variation within and among mammalian species. Conserv. Physiol. 3(1), cov020
Delehanty, B., Bossart, G.D., Champagne, C., Crocker, D.E., Elliott, K.H., Fair, P.A., Houser, D., Newman, A.E.M., Boonstra, R., 2020. Measurement of free glucocorticoids: quantifying corticosteroid binding capacity and its variation within and among mammal and bird species. Conserv. Physiol. 8(1), coaa057.

Delezie, J., Challet, E., 2011. Interactions between metabolism and circadian clocks: reciprocal disturbances: Circadian disruption and metabolic dysfunction. Ann. NY Acad. Sci. 1243, 30–46.
Demas, G.E., Polacek, K.M., Durazzo, A., Jasnow, A.M., 2004. Adrenal hormones mediate melatonin-induced increases in aggression in male Siberian hamsters (*Phodopus sungorus*). Horm. Behav. 46, 582–591.
Deng, W., Cheung, S.T., Tsao, S.W., Wang, X.M., Tiwari, A.F.Y., 2016. Telomerase activity and its association with psychological stress, mental disorders, lifestyle factors and interventions: A systematic review. Psychoneuroendocrinology 64, 150–163.
Desantis, L.M., Delehanty, B., Weir, J.T., Boonstra, R., 2013. Mediating free glucocorticoid levels in the blood of vertebrates: are corticosteroid-binding proteins always necessary? Funct. Ecol. 27, 107–119.
DeVries, A.C., Glasper, E.R., Detillion, C.E., 2003. Social modulation of stress responses. Physiol. Behav. 79, 399–407.
Dobson, F.S., 1982. Competition for mates and predominant juvenile male dispersal in mammals. Anim. Behav. 30, 1183–1192.
Dominoni, D., Quetting, M., Partecke, J., 2013. Artificial light at night advances avian reproductive physiology. Proc. R. Soc. B. 280, 20123017.
Dominoni, D.M., Borniger, J.C., Nelson, R.J., 2016. Light at night, clocks and health: from humans to wild organisms. Biol. Lett. 12, 20160015.
Du, J., McEwen, B., Manji, H.K., 2009. Glucocorticoid receptors modulate mitochondrial function. Comm. Integr. Biol. 2, 350–352.
Edwards, P.D., Lavergne, S.G., McCaw, L.K., Wijenayake, S., Boonstra, R., McGowan, P.O., Holmes, M.M., 2021. Maternal effects in mammals: Broadening our understanding of offspring programming. Front. Neuroendocrinol. 62, 100924.
Ehlers, C.L., Frank, E., Kupfer, D.J., 1988. A unified approach to understanding the etiology of depression. Social Zeitgebers and Biological Rhythms 45, 948-952.
Eisermann, K., 1992. Long-term heart rate responses to social stress in wild European rabbits: Predominant effect of rank position. Physiol. Behav. 52, 33–36.
Entringer, S., Epel, E.S., Kumsta, R., Lin, J., Hellhammer, D.H., Blackburn, E.H., Wust, S., Wadhwa, P.D., 2011. Stress exposure in intrauterine life is associated with shorter telomere length in young adulthood. Proc. Natl. Acad. Sci. USA 108, E513-E518.
Epel, E.S., Blackburn, E.H., Lin, J., Dhabhar, F.S., Adler, N.E., Morrow, J.D., Cawthon, R.M., 2004. Accelerated telomere shortening in response to life stress. Proc. Natl. Acad. Sci. USA 101, 17312–17315.
Epel, E.S., 2009. Psychological and metabolic stress: A recipe for accelerated cellular aging? Hormones 8, 7–22.
Epel, E.S., Lin, J., Dhabhar, F.S., Wolkowitz, O.M., Puterman, E., Karan, L., Blackburn, E.H., 2010. Dynamics of telomerase activity in response to acute psychological stress. Brain Behav. Immun. 24, 531–539.
Fagundes, C.P., Glaser, R., Kiecolt-Glaser, J.K., 2013. Stressful early life experiences and immune dysregulation across the lifespan. Brain Behav. Immun. 27, 8–12.
Faulkes, C.G., Abbott, D.H., 1997. The physiology of a reproductive dictatorship: Regulation of male and female reproduction by a single breeding female in colonies of naked mole-rats, in: French, J.A., Solomon, N.G. (Eds.), Cooperative breeding in mammals. Cambridge University Press, Cambridge, pp. 302–334.
Favreau, A., Richard-Yris, M.-A., Bertin, A., Houdelier, C., Lumineau, S., 2009. Social influences on circadian behavioural rhythms in vertebrates. Anim. Behav. 77, 983–989.

Fernandes, P.A., Tamura, E.K., D'Argenio-Garcia, L., Muxel, S.M., da Silveira Cruz-Machado, S., Marçola, M., Carvalho-Sousa, C.E., Cecon, E., Ferreira, Z.S., Markus, R.P., 2017. Dual effect of catecholamines and corticosterone crosstalk on pineal gland melatonin synthesis. Neuroendocrinology 104, 126–134.

Fialkowski, R., Aufdemberge, P., Wright, V., Dijkstra, P., 2021. Radical change: temporal patterns of oxidative stress during social ascent in a dominance hierarchy. Behav. Ecol. Sociobiol. 75, 43.

Finkel, T., Holbrook, N.J., 2000. Oxidants, oxidative stress and the biology of ageing. Nature 408, 239–247.

Focke, C.M.B., Iremonger, K.J., 2020. Rhythmicity matters: Circadian and ultradian patterns of HPA axis activity. Mol. Cell. Endocrinol. 501, 110652.

Fox, H.E., White, S.A., Kao, M.H., Fernald, R.D., 1997. Stress and dominance in a social fish. J. Neurosci. 17, 6463–6469.

Funnell, T.R., Fialkowski, R.J., Dijkstra, P.D., 2022. Social dominance does not increase oxidative stress in a female dominance hierarchy of an African cichlid fish. Ethology 128, 15–25.

Gersick, A.S., Rubenstein, D.I., 2017. Physiology modulates social flexibility and collective behaviour in equids and other large ungulates. Phil. Trans. R. Soc. B 372, 20160241.

Gesquiere, L.R., Learn, N.H., Simao, M.C.M., Onyango, P.O., Alberts, S.C., Altmann, J., 2011. Life at the top: rank and stress in wild male baboons. Science 333, 357–360.

Goymann, W., Wingfield, J.C., 2004. Allostatic load, social status and stress hormones: the costs of social status matter. Anim. Behavi. 67, 591–602.

Grandin, L.D., Alloy, L.B., Abramson, L.Y., 2006. The social zeitgeber theory, circadian rhythms, and mood disorders: Review and evaluation. Clin. Psychol. Rev. 26, 679–694.

Green, J.A., Halsey, L.G., Wilson, R.P., Frappell, P.B., 2009. Estimating energy expenditure of animals using the accelerometry technique: activity, inactivity and comparison with the heart-rate technique. J. Exp. Biol. 212, 471–482.

Greenwood, P.J., 1980. Mating systems, philopatry and dispersal in birds and mammals. Anim. Behav. 28, 1140–1162.

Grosbellet, E., Zahn, S., Arrivé, M., Dumont, S., Gourmelen, S., Pévet, P., Challet, E., Criscuolo, F., 2015. Circadian desynchronization triggers premature cellular aging in a diurnal rodent. FASEB j. 29, 4794–4803.

Groscolas, R., Viera, V., Guerin, N., Handrich, Y., Côté, S.D., 2010. Heart rate as a predictor of energy expenditure in undisturbed fasting and incubating penguins. J. Exp. Biol. 213, 153–160.

Guibert, F., Richard-Yris, M.-A., Lumineau, S., Kotrschal, K., Guémené, D., Bertin, A., Möstl, E., Houdelier, C., 2010. Social Instability in Laying Quail: Consequences on Yolk Steroids and Offspring's Phenotype. PLoS ONE 5, e14069.

Gunnar, M.R., Hostinar, C.E., 2015. The social buffering of the hypothalamic–pituitary–adrenocortical axis in humans: Developmental and experiential determinants. Soc. Neurosci. 10, 479–488.

Habig, B., Archie, E.A., 2015. Social status, immune response and parasitism in males: a meta-analysis. Phil. Trans. R. Soc. B 370, 20140109.

Habig, B., Doellman, M.M., Woods, K., Olansen, J., Archie, E.A., 2018. Social status and parasitism in male and female vertebrates: a meta-analysis. Sci. Rep. 8, 3629.

Hamilton, D.G., Jones, M.E., Cameron, E.Z., Kerlin, D.H., McCallum, H., Storfer, A., Hohenlohe, P.A., Hamede, R.K., 2020. Infectious disease and sickness behaviour: tumour progression affects interaction patterns and social network structure in wild Tasmanian devils. Proc. R. Soc. B. 287, 20202454.

963 Hamilton, W.D., 1964. The genetical evolution of social behaviour. I & II. J. Theor. Biol. 7,
964 1–52.
965 Hamilton, W.D., 1971. Geometry for the selfish herd. J. Theor. Biol. 31, 295–311.
966 Haq, S.U., Bhat, U.A., Kumar, A., 2021. Prenatal stress effects on offspring brain and
967 behavior: Mediators, alterations and dysregulated epigenetic mechanisms. J. Biosci.
968 46, 34.
969 Harman, D., 1956. Aging: A Theory Based on Free Radical and Radiation Chemistry. Sci.
970 Aging Knowl. Environ. 11.
971 Harris, B.N., 2020. Stress hypothesis overload: 131 hypotheses exploring the role of stress in
972 tradeoffs, transitions, and health. Gen. Co. Endocrinol. 288, 113355.
973 Hart, D.W., van Vuuren, A.K.J., Erasmus, A., Süess, T., Hagenah, N., Ganswindt, A.,
974 Bennett, N.C., 2022. The endocrine control of reproductive suppression in an
975 aseasonally breeding social subterranean rodent, the Mahali mole-rat (*Cryptomys*
976 *hottentotus mahali*). Horm. Behav. 142, 105155.
977 Havird, J.C., Weaver, R.J., Milani, L., Ghiselli, F., Greenway, R., Ramsey, A.J., Jimenez,
978 A.G., Dowling, D.K., Hood, W.R., Montooth, K.L., Estes, S., Schulte, P.M.,
979 Sokolova, I.M., Hill, G.E., 2019. Beyond the Powerhouse: Integrating Mitonuclear
980 Evolution, Physiology, and Theory in Comparative Biology. Integr. Comp. Biol. 59,
981 856–863.
982 Hawley, D.M., Lindström, K., Wikelski, M., 2006. Experimentally increased social
983 competition compromises humoral immune responses in house finches. Horm. Behav.
984 49, 417–424.
985 Heine, K.B., Hood, W.R., 2020. Mitochondrial behaviour, morphology, and animal
986 performance. Biol. Rev. 95, 730–737.
987 Heinen, V.K., Benedict, L.M., Pitera, A.M., Sonnenberg, B.R., Bridge, E.S., Pravosudov,
988 V.V., 2021. Social dominance has limited effects on spatial cognition in a wild food-
989 caching bird. Proc. R. Soc. B. 288, 20211784.
990 Heinrichs, M., Baumgartner, T., Kirschbaum, C., Ehlert, U., 2003. Social support and
991 oxytocin interact to suppress cortisol and subjective responses to psychosocial stress.
992 Biol. Psychiatry 54, 1389–1398.
993 Heinzeller, Th., Joshi, B.N., Nürnberger, F., Reiter, R.J., 1988. Effects of aggressive
994 encounters on pineal melatonin formation in male gerbils (*Meriones unguiculatus,*
995 *Cricetidae*). J. Comp. Physiol. 164, 91–94.
996 Hesse, S., Sandmann, S., Bakker, T.C.M., Thünken, T., 2019. Impact of social rearing-
997 environment on performance in a complex maze in females of a cichlid fish. Behav.
998 Proc. 167, 103915.
999 Hoffmann, A., Spengler, D., 2018. The mitochondrion as potential interface in early-life
1000 stress brain programming. Front. Behav. Neurosci. 12, 306.
1001 Hofmann, H.A., Beery, A.K., Blumstein, D.T., Couzin, I.D., Earley, R.L., Hayes, L.D., Hurd,
1002 P.L., Lacey, E.A., Phelps, S.M., Solomon, N.G., Taborsky, M., Young, L.J.,
1003 Rubenstein, D.R., 2014. An evolutionary framework for studying mechanisms of
1004 social behavior. Trends Ecol. Evol. 29, 581–589.
1005 Hollis, F., van der Kooij, M.A., Zanoletti, O., Lozano, L., Cantó, C., Sandi, C., 2015.
1006 Mitochondrial function in the brain links anxiety with social subordination. Proc. Natl.
1007 Acad. Sci. USA 112, 15486–15491.
1008 Hood, Wendy R, Austad, S.N., Bize, P., Jimenez, A.G., Montooth, K.L., Schulte, P.M., Scott,
1009 G.R., Sokolova, I., Treberg, J.R., Salin, K., 2018. The mitochondrial contribution to
1010 animal performance, adaptation, and life-history variation. Integr. Comp. Biol. 58,
1011 480–485.

Hood, W R, Zhang, Y., Mowry, A.V., Hyatt, H.W., Kavazis, A.N., 2018. Life history trade-offs within the context of mitochondrial hormesis. Integr. Comp. Biol. 58, 567–577.
Hunter, R.G., Seligsohn, M., Rubin, T.G., Griffiths, B.B., Ozdemir, Y., Pfaff, D.W., Datson, N.A., McEwen, B.S., 2016. Stress and corticosteroids regulate rat hippocampal mitochondrial DNA gene expression via the glucocorticoid receptor. Proc. Natl. Acad. Sci. USA 113, 9099–9104.
Iwasaki, A., Medzhitov, R., 2010. Regulation of adaptive immunity by the innate immune system. Science 327, 291–295.
Jasnow, A.M., Huhman, K.L., Bartness, T.J., Demas, G.E., 2002. Short days and exogenous melatonin increase aggression of male Syrian hamsters (*Mesocricetus auratus*). Horm. Behav. 42, 13–20.
Jax, E., Müller, I., Börno, S., Borlinghaus, H., Eriksson, G., Fricke, E., Timmermann, B., Pendl, H., Fiedler, W., Klein, K., Schreiber, F., Wikelski, M., Magor, K.E., Kraus, R.H.S., 2021. Health monitoring in birds using bio-loggers and whole blood transcriptomics. Sci. Rep. 11, 10815.
Jong, I.C. de, Sgoifo, A., Lambooij, E., Korte, S.M., Blokhuis, H.J., Koolhaas, J.M., 2000. Effects of social stress on heart rate and heart rate variability in growing pigs. Can. J. Anim. Sci. 80, 273–280.
Kaiser, S., Sachser, N., 2005. The effects of prenatal social stress on behaviour: mechanisms and function. Neurosci. Biobehav. Rev. 29, 283–294.
Kalsbeek, A., van der Spek, R., Lei, J., Endert, E., Buijs, R.M., Fliers, E., 2012. Circadian rhythms in the hypothalamo–pituitary–adrenal (HPA) axis. Mol. Cell. Endocrinol. 349, 20–29.
Kamiński, M., Janiszewski, T., Indykiewicz, P., Nowakowski, J.J., Kowalski, J., Dulisz, B., Minias, P., 2021. Density-dependence of nestling immune function and physiological condition in semi-precocial colonial bird: a cross-fostering experiment. Front. Zool. 18, 7.
Kappeler, P.M., Barrett, L., Blumstein, D.T., Clutton-Brock, T.H., 2013. Constraints and flexibility in mammalian social behaviour: introduction and synthesis. Phil. Trans. R. Soc. B 368, 20120337.
Kappeler, P.M., Cremer, S., Nunn, C.L., 2015. Sociality and health: impacts of sociality on disease susceptibility and transmission in animal and human societies. Phil. Trans. R. Soc. B 370, 20140116.
Karatsoreos, I.N., Bhagat, S., Bloss, E.B., Morrison, J.H., McEwen, B.S., 2011. Disruption of circadian clocks has ramifications for metabolism, brain, and behavior. Proc. Natl. Acad. Sci. USA 108, 1657–1662.
Kawanishi, S., Oikawa, S., 2004. Mechanism of Telomere Shortening by Oxidative Stress. Ann. NY Acad. Sci. 1019, 278–284.
Kelly, T.R., Kimball, M.G., Stansberry, K.R., Lattin, C.R., 2020. No, you go first: phenotype and social context affect house sparrow neophobia. Biol. Lett. 16, 20200286.
Kirkwood, T.B.L., Kowald, A., 2012. The free-radical theory of ageing - older, wiser and still alive: Modelling positional effects of the primary targets of ROS reveals new support. Bioessays 34, 692–700.
Koch, R.E., Buchanan, K.L., Casagrande, S., Crino, O., Dowling, D.K., Hill, G.E., Hood, W.R., McKenzie, M., Mariette, M.M., Noble, D.W.A., Pavlova, A., Seebacher, F., Sunnucks, P., Udino, E., White, C.R., Salin, K., Stier, A., 2021. Integrating mitochondrial aerobic metabolism into ecology and evolution. Trends Ecol. Evol. 36, 321–332.

Kolbe, J.J., Moniz, H.A., Lapiedra, O., Thawley, C.J., 2021. Bright lights, big city: an experimental assessment of short-term behavioral and performance effects of artificial light at night on Anolis lizards. Urban Ecosyst. 24, 1035–1045.
Kopec, A.M., Smith, C.J., Bilbo, S.D., 2019. Neuro-immune mechanisms regulating social behavior: Dopamine as mediator? Trends Neurosci. 42, 337–348.
Kotrschal, A., Ilmonen, P., Penn, D.J., 2007. Stress impacts telomere dynamics. Biol. Lett. 3, 128–130.
Kotrschal, K., Hirschenhauser, K., Möstl, E., 1998. The relationship between social stress and dominance is seasonal in greylag geese. Anim. Behav. 55, 171–176.
Kumar, V., 2002. Biological rhythms. Springer Narosa Publishing House, New Delhi.
Kuo, T., McQueen, A., Chen, T.-C., Wang, J.-C., 2015. Regulation of glucose homeostasis by glucocorticoids, in: Wang, J.-C., Harris, C. (Eds.), Glucocorticoid signaling, Advances in experimental medicine and biology. Springer New York, New York, pp. 99–126.
Lambert, C.T., Guillette, L.M., 2021. The impact of environmental and social factors on learning abilities: a meta-analysis. Biol. Rev. 96, 2871–2889.
Lamont, E.W., Legault-Coutu, D., Cermakian, N., Boivin, D.B., 2007. The role of circadian clock genes in mental disorders. Dialogues Clin. Neurosci. 9, 333–342.
Lane, N., 2006. Power, sex, suicide: mitochondria and the meaning of life. Oxford University Press, Oxford.
Lapp, H.E., Bartlett, A.A., Hunter, R.G., 2019. Stress and glucocorticoid receptor regulation of mitochondrial gene expression. J. Mol. Endocrinol. 62, R121–R128.
Lardy, S., Rey, B., Salin, K., Voituron, Y., Cohas, A., 2016. Beneficial effects of group size on oxidative balance in a wild cooperative breeder. Behav. Ecol. 27, 1820-1825.
Larson, E.T., Winberg, S., Mayer, I., Lepage, O., Summers, C.H., Øverli, Ø., 2004. Social stress affects circulating melatonin levels in rainbow trout. Gen. Comp. Endocrinol. 136, 322–327.
Le, P.P., Friedman, J.R., Schug, J., Brestelli, J.E., Parker, J.B., Bochkis, I.M., Kaestner, K.H., 2005. Glucocorticoid Receptor-Dependent Gene Regulatory Networks. PLOS Genetics 1, 16.
Leblanc, H., Ramirez, S., 2020. Linking Social Cognition to Learning and Memory. J. Neurosci. 40, 8782–8798.
Levins, R., 1968. Evolution in changing environments: some theoretical explorations, Monographs in population biology. Princeton Univ. Pr, Princeton, NJ.
Li, H., Xia, N., 2020. The role of oxidative stress in cardiovascular disease caused by social isolation and loneliness. Redox Biol. 37, 101585.
Lin, H.-Y., Muller, Y.A., Hammond, G.L., 2010. Molecular and structural basis of steroid hormone binding and release from corticosteroid-binding globulin. Mol. Cell. Endocrinol. 316, 3–12.
Lindström, K.M., Hasselquist, D., Wikelski, M., 2005. House sparrows (*Passer domesticus*) adjust their social status position to their physiological costs. Horm. Behav. 48, 311–320.
López-Otín, C., Kroemer, G., 2021. Hallmarks of Health. Cell 184, 33–63.
Love, O.P., McGowan, P.O., Sheriff, M.J., 2013. Maternal adversity and ecological stressors in natural populations: the role of stress axis programming in individuals, with implications for populations and communities. Funct. Ecol. 27, 81–92.
Lukkes, J., Watt, M., Lowry, C., Forster, G., 2009. Consequences of post-weaning social isolation on anxiety behavior and related neural circuits in rodents. Front. Behav. Neurosci. 3, 18.

1108 Lutgendorf, S.K., Sood, A.K., Anderson, B., McGinn, S., Maiseri, H., Dao, M., Sorosky, J.I.,
1109 De Geest, K., Ritchie, J., Lubaroff, D.M., 2005. Social support, psychological distress,
1110 and natural killer cell activity in ovarian cancer. J. Clin. Oncol. 23, 7105–7113.
1111 MacDougall-Shackleton, S.A., Bonier, F., Romero, L.M., Moore, I.T., 2019. Glucocorticoids
1112 and "stress" are not synonymous. Integr. Organism. Biol. 1, obz017.
1113 Madalena, K.M., Lerch, J.K., 2017. The effect of glucocorticoid and glucocorticoid receptor
1114 interactions on brain, spinal cord, and glial cell plasticity. Neural Plasticity 2017,
1115 e8640970.
1116 Majer, A.D., Fasanello, V.J., Tindle, K., Frenz, B.J., Ziur, A.D., Fischer, C.P., Fletcher, K.L.,
1117 Seecof, O.M., Gronsky, S., Vassallo, B.G., Reed, W.L., Paitz, R.T., Stier, A.,
1118 Haussmann, M.F., 2019. Is there an oxidative cost of acute stress? Characterization,
1119 implication of glucocorticoids and modulation by prior stress experience. Proc. R.
1120 Soc. B. 286, 20191698.
1121 Majewski, P., Markowska, M., Pawlak, J., Piesiewicz, A., Turkowska, E., Skwarlo-Sonta, K.,
1122 2012. Pineal gland and melatonin: Impact on the seasonality of immune defence in
1123 mammals and birds. Adv. Neuroimmun. Biol. 3, 95–108.
1124 Manoli, I., Alesci, S., Blackman, M.R., Su, Y.A., Rennert, O.M., Chrousos, G.P., 2007.
1125 Mitochondria as key components of the stress response. Trends Endocrinol. Metabol.
1126 18, 190–198.
1127 Marasco, V., Herzyk, P., Robinson, J., Spencer, K.A., 2016. Pre- and post-natal stress
1128 programming: developmental exposure to glucocorticoids causes long-term brain-
1129 region specific changes to transcriptome in the precocial Japanese quail. J.
1130 Neuroendocrinol. 28, 5.
1131 Margraf, J., Lavallee, K., Zhang, X., Schneider, S., 2016. Social rhythm and mental health: a
1132 cross-cultural comparison. PLoS ONE 11, e0150312.
1133 Markus, R.P., Ferreira, Z.S., 2011. The immune-pineal axis: the role of pineal and extra-
1134 pineal melatonin in modulating inflammation. Adv. Neuroimmun. Biol. 1, 95–104.
1135 Marler, C A., Walsberg, G., White, M.L., Moore, M., Marler, C. A., 1995. Increased energy
1136 expenditure due to increased territorial defense in male lizards after phenotypic
1137 manipulation. Behav. Ecol. Sociobiol. 37, 225–231.
1138 Matthews, G.A., Tye, K.M., 2019. Neural mechanisms of social homeostasis. Ann. NY Acad.
1139 Sci. 1457, 5–25.
1140 Mazuc, J., Bonneaud, C., Chastel, O., Sorci, G., 2003. Social environment affects female and
1141 egg testosterone levels in the house sparrow (*Passer domesticus*): Effect of social
1142 environment on female and egg testosterone levels. Ecol. Lett. 6, 1084–1090.
1143 McClung, C.A., 2007. Circadian genes, rhythms and the biology of mood disorders.
1144 Pharmacol. Therapeut. 114, 222–232.
1145 McEwen, B.S., Wingfield, J.C., 2003. The concept of allostasis in biology and biomedicine.
1146 Horm. Behav. 43, 2–15.
1147 McIntosh, L.J., Hong, K.E., Sapolsky, R.M., 1998. Glucocorticoids may alter antioxidant
1148 enzyme capacity in the brain: baseline studies. Brain Res. 791, 209–214.
1149 Menard, C., Pfau, M.L., Hodes, G.E., Kana, V., Wang, V.X., Bouchard, S., Takahashi, A.,
1150 Flanigan, M.E., Aleyasin, H., LeClair, K.B., Janssen, W.G., Labonté, B., Parise, E.M.,
1151 Lorsch, Z.S., Golden, S.A., Heshmati, M., Tamminga, C., Turecki, G., Campbell, M.,
1152 Fayad, Z.A., Tang, C.Y., Merad, M., Russo, S.J., 2017. Social stress induces
1153 neurovascular pathology promoting depression. Nat. Neurosci. 20, 1752–1760.
1154 Mendel, C.M., 1989. the free hormone hypothesis: a physiologically based mathematical
1155 model. Endocr. Rev. 10, 232–274.

Mendonça, R., Vullioud, P., Katlein, N., Vallat, A., Glauser, G., Bennett, N.C., Helfenstein, F., 2020. Oxidative costs of cooperation in cooperatively breeding Damaraland mole-rats. Proc. Roy. Soc. B 287, 20201023.
Metcalfe, N.B., Taylor, A.C., Thorpe, J.E., 1995. Metabolic rate, social status and life-history strategies in Atlantic salmon. Anim. Behav. 49, 431–436.
Mikami, K., Kiyokawa, Y., Ishii, A., Takeuchi, Y., 2020. Social buffering enhances extinction of conditioned fear responses by reducing corticosterone levels in male rats. Horm. Behavi. 118, 104654.
Minias, P., Gach, K., Włodarczyk, R., Janiszewski, T., 2019. Colony size affects nestling immune function: a cross-fostering experiment in a colonial waterbird. Oecologia 190, 333–341.
Mistlberger, R.E., Skene, D.J., 2004. Social influences on mammalian circadian rhythms: animal and human studies. Biol. Rev. 79, 533–556.
Mitchell, C., Hobcraft, J., McLanahan, S.S., Siegel, S.R., Berg, A., Brooks-Gunn, J., Garfinkel, I., Notterman, D., 2014. Social disadvantage, genetic sensitivity, and children's telomere length. Proc. Natl. Acad. Sci. 111, 5944–5949.
Modlinska, K., Stryjek, R., Chrzanowska, A., Pisula, W., 2018. Social environment as a factor affecting exploration and learning in pre-juvenile rats. Behav. Proc. 153, 77–83.
Monaghan, P., Metcalfe, N.B., Torres, R., 2009. Oxidative stress as a mediator of life history trade-offs: mechanisms, measurements and interpretation. Ecol. Lett. 12, 75–92.
Montgomery, T.M., Pendleton, E.L., Smith, J.E., 2018. Physiological mechanisms mediating patterns of reproductive suppression and alloparental care in cooperatively breeding carnivores. Physiol. Behav. 193, 167–178.
Morgan, D.J., Poolman, T.M., Williamson, A.J.K., Wang, Z., Clark, N.R., Ma'ayan, A., Whetton, A.D., Brass, A., Matthews, L.C., Ray, D.W., 2016. Glucocorticoid receptor isoforms direct distinct mitochondrial programs to regulate ATP production. Sci. Rep. 6, 26419.
Mueller, I., Shakiba, N., Brown, M.A., Crowel, S.E., Conradt, E., 2021. Epigenetic effects of prenatal stress, in: Wazana, A., Székely, E., Oberlander, T.F. (Eds.), Prenatal stress and child development. Springer International Publishing, Cham, pp. 89–111.
Muller, M.N., Wrangham, R.W., 2004. Dominance, cortisol and stress in wild chimpanzees (*Pan troglodytes schweinfurthii*). Behav. Ecolo. Sociobiol. 55, 332–340.
Munley, K.M., Deyoe, J.E., Ren, C.C., Demas, G.E., 2020. Melatonin mediates seasonal transitions in aggressive behavior and circulating androgen profiles in male Siberian hamsters. Horm. Behav. 117, 104608.
Nation, D.A., Gonzales, J.A., Mendez, A.J., Zaias, J., Szeto, A., Brooks, L.G., Paredes, J., D'Angola, A., Schneiderman, N., McCabe, P.M., 2008. The effect of social environment on markers of vascular oxidative stress and inflammation in the Watanabe heritable hyperlipidemic rabbit. Psychosomatic Med. 70, 269–275.
Nephew, B.C., Romero, L.M., 2003. Behavioral, physiological, and endocrine responses of starlings to acute increases in density. Horm. Behav. 44, 222–232.
Nettle, D., Monaghan, P., Boner, W., Gillespie, R., Bateson, M., 2013. Bottom of the heap: having heavier competitors accelerates early-life telomere loss in the european starling, *Sturnus vulgaris*. PLoS ONE 8, e83617.
Nettle, D., Monaghan, P., Gillespie, R., Brilot, B., Bedford, T., Bateson, M., 2015. An experimental demonstration that early-life competitive disadvantage accelerates telomere loss. Proc. R. Soc. B. 282, 20141610.
Norman, R.E., Byambaa, M., De, R., Butchart, A., Scott, J., Vos, T., 2012. The long-term health consequences of child physical abuse, emotional abuse, and neglect: A systematic review and meta-analysis. PLoS Medicine 9, 1001349.

Oitzl, M.S., Champagne, D.L., van der Veen, R., de Kloet, E.R., 2010. Brain development under stress: Hypotheses of glucocorticoid actions revisited. Neurosci. Biobehav. Rev. 34, 853–866.
Oli, M.K., Armitage, K.B., 2003. Sociality and individual fitness in yellow-bellied marmots: Insights from a long-term study (1962-2001). Oecologia 136, 543–550.
Oliveira, B.S., Zunzunegui, M.V., Quinlan, J., Fahmi, H., Tu, M.T., Guerra, R.O., 2016. Systematic review of the association between chronic social stress and telomere length: A life course perspective. Ageing Res. Rev .26, 37–52.
Ostner, J., Heistermann, M., Schülke, O., 2008. Dominance, aggression and physiological stress in wild male Assamese macaques (*Macaca assamensis*). Horm. Behav. 54, 613–619.
Otten, W., Kanitz, E., Couret, D., Veissier, I., Prunier, A., Merlot, E., 2010. Maternal social stress during late pregnancy affects hypothalamic-pituitary-adrenal function and brain neurotransmitter systems in pig offspring. Domestic Anim. Endocrinol. 38, 146–156.
Patterson, J.E.H., Ruckstuhl, K.E., 2013. Parasite infection and host group size: a meta-analytical review. Parasitology 140, 803–813.
Peckett, A.J., Wright, D.C., Riddell, M.C., 2011. The effects of glucocorticoids on adipose tissue lipid metabolism. Metabolism 60, 1500–1510.
Perogamvros, I., Ray, D.W., Trainer, P.J., 2012. Regulation of cortisol bioavailability — effects on hormone measurement and action. Nat. Rev. Endocrinol. 8, 717-727.
Picard, M., Juster, R.-P., McEwen, B.S., 2014. Mitochondrial allostatic load puts the “gluc” back in glucocorticoids. Nat. Rev E.ndocrinol. 10, 303–310.
Picard, M., McManus, M.J., Csordás, G., Várnai, P., Dorn II, G.W., Williams, D., Hajnóczky, G., Wallace, D.C., 2015. Trans-mitochondrial coordination of cristae at regulated membrane junctions. Nat. Commun. 6, 6259.
Picard, M., McEwen, B.S., 2018a. Psychological stress and mitochondria: a conceptual framework. Psychosom. Med. 80, 126–140.
Picard, M., McEwen, B.S., Epel, E.S., Sandi, C., 2018b. An energetic view of stress: Focus on mitochondria. Front. Neuroendocrinol. 49, 72–85. h
Qian, X., Droste, S.K., Gutierrez-Mecinas, M., Collins, A., Kersante, F., Reul, J., Linthorst, A.C.E., 2011. A rapid release of corticosteroid-binding globulin from the liver restrains the glucocorticoid hormone response to acute stress. Endocrinol. 152, 3738-3748.
Quque, M., Paquet, M., Zahn, S., Théron, F., Faivre, B., Sueur, C., Criscuolo, F., Doutrelant, C., Covas, R., 2021. Contrasting associations between nestling telomere length and pre and postnatal helpers’ presence in a cooperatively breeding bird. Oecologia 196,
Quque, M., Ferreira, C., Sosa, S., Schull, Q., Zahn, S., Criscuolo, F., Bleu, J., Viblanc, V.A., 2022. Cascading effects of conspecific aggression on oxidative status and telomere length in zebra finches. Physiol. Biochem. Zool., in press.
Raison, C.L., Capuron, L., Miller, A.H., 2006. Cytokines sing the blues: inflammation and the pathogenesis of depression. Trends Immunol. 27, 24–31.
Rao, R., Androulakis, I.P., 2019. The physiological significance of the circadian dynamics of the HPA axis: Interplay between circadian rhythms, allostasis and stress resilience. Horm. Behav. 110, 77–89.
Raulo, A., Dantzer, B., 2018. Associations between glucocorticoids and sociality across a continuum of vertebrate social behavior. Ecol. Evol. 8, 7697–7716.
Reichert, S., Stier, A., 2017. Does oxidative stress shorten telomeres *in vivo* ? A review. Biol. Lett. 13, 20170463.

Reid, D., Armstrong, J.D., Metcalfe, N.B., 2011. Estimated standard metabolic rate interacts with territory quality and density to determine the growth rates of juvenile Atlantic salmon: SMR, density, territory and growth. Funct. Ecol. 25, 1360–1367.
Rentscher, K.E., Carroll, J.E., Mitchell, C., 2020. Psychosocial Stressors and Telomere Length: A Current Review of the Science. Annu. Rev. Public Health 41, 223–245.
Reyes-Contreras, M., Glauser, G., Rennison, D.J., Taborsky, B., 2019. Early-life manipulation of cortisol and its receptor alters stress axis programming and social competence. Phil. Trans. Roy. Soc. B 374, 20180119.
Ristow, M., Zarse, K., 2010. How increased oxidative stress promotes longevity and metabolic health: The concept of mitochondrial hormesis (mitohormesis). Exp. Gerontol. 45, 410–418.
Romano, V., MacIntosh, A.J.J., Sueur, C., 2020. Stemming the flow: Information, infection, and social evolution. Trends Ecol. Evol. 35, 849–853.
Romano, V., Sueur, C., MacIntosh, A.J.J., 2021. The tradeoff between information and pathogen transmission in animal societies. Oikos 08290.
Romero, L.M., Butler, L.K., 2007. Endocrinology of Stress. Int. J. Comp. Psychol. 20(2).
Romero, L.M., Dickens, M.J., Cyr, N.E., 2009. The reactive scope model — A new model integrating homeostasis, allostasis, and stress. Horm. Behav. 55, 375–389.
Ropert-Coudert, Y., Kato, A., Grémillet, D., Crenner, F., 2012. Bio-logging: recording the ecophysiology and behaviour of animals moving freely in their environment, in: Sensors for Ecology. pp. 17–41.
Rosal, M.C., King, J., Ma, Y., Reed, G.W., 2004. Stress, social support, and cortisol: inverse associations? Behav. Med. 30, 11–22.
Roulin, A., Dreiss, A.N., 2012. Sibling competition and cooperation over parental care, in: The evolution of parental care. Oxford University Press.
Rusak, B., 1981. Vertebrate behavioral rhythms, in: Aschoff, J. (Ed.), Biological rhythms. Springer US, Boston, MA, pp. 183–213.
Rutz, C., Burns, Z.T., James, R., Ismar, S.M.H., Burt, J., Otis, B., Bowen, J., St Clair, J.J.H., 2012. Automated mapping of social networks in wild birds. Curr. Biol. 22, R669–R671.
Sabet, S.M., Dautovich, N.D., Dzierzewski, J.M., 2021. The rhythm is gonna get you: social rhythms, sleep, depressive, and anxiety symptoms. J. Affect. Dis. 286, 197–203.
Sánchez-Tójar, A., Lagisz, M., Moran, N.P., Nakagawa, S., Noble, D.W.A., Reinhold, K., 2020. The jury is still out regarding the generality of adaptive 'transgenerational' effects. Ecol. Lett. 23, 1715–1718.
Sapolsky, R.M., Ray, J.C., 1989. Styles of dominance and their endocrine correlates among wild olive baboons (*Papio anubis*) Am. J. Primatology 18:1-13
Sapolsky, R.M., Romero, L.M., Munck, A.U., 2000. How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions. Endocr. Rev. 21, 55–89.
Sapolsky, R.M., 2004. Social status and health in humans and other animals. Ann. Rev. Anthropol. 33, 393–418.
Sapolsky, R.M., 2005. The influence of social hierarchy on primate health. Science 308, 648–652.
Schiavone, S., Sorce, S., Dubois-Dauphin, M., Jaquet, V., Colaianna, M., Zotti, M., Cuomo, V., Trabace, L., Krause, K.-H., 2009. Involvement of NOX2 in the development of behavioral and pathologic alterations in isolated rats. Biol. Psychiatry 66, 384–392.
Schmid-Hempel, P., 2017. Parasites and their social hosts. Trends Parasitol. 33, 453–462.

Schoech, S.J., Mumme, R.L., Moore, M.C., 1991. Reproductive endocrinology and mechanisms of breeding inhibition in cooperatively breeding Florida scrub jays (*Aphelocoma C. Coerulescens*). The Condor 93, 354–364.
Schoech, S.J., Romero, L.M., Moore, I.T., Bonier, F., 2013. Constraints, concerns and considerations about the necessity of estimating free glucocorticoid concentrations for field endocrine studies. Funct. Ecol. 27, 1100–1106.
Schradin, C., 2013. Intraspecific variation in social organization by genetic variation, developmental plasticity, social flexibility or entirely extrinsic factors. Phil. Trans. R. Soc. B 368, 20120346.
Secondi, J., Mondy, N., Gippet, J.M.W., Touzot, M., Gardette, V., Guillard, L., Lengagne, T., 2021. Artificial light at night alters activity, body mass, and corticosterone level in a tropical anuran. Behav. Ecol. 32, 932–940.
Sephton, S., Spiegel, D., 2003. Circadian disruption in cancer: a neuroendocrine-immune pathway from stress to disease? Brain, Behav. Immun. 17, 321–328.
Sgoifo, A., Papi, F., 1995. Effects of social and non-social acute stressors on plasma levels of catecholamines and corticosterone in wild rats. Rend. Fis. Acc. Lincei 6, 289.
Sgoifo, A., De Boer, S.F., Haller, J., Koolhaas, J.M., 1996. Individual differences in plasma catecholamine and corticosterone stress responses of wild-type rats: relationship with aggression. Physiol. Behav. 60, 1403–1407.
Sgoifo, A., Koolhaas, J., De Boer, S., Musso, E., Stilli, D., Buwalda, B., Meerlo, P., 1999. Social stress, autonomic neural activation, and cardiac activity in rats. Neurosci. Biobehav. Rev. 23, 915–923.
Sharp, S.P., Clutton-Brock, T.H., 2011. Competition, breeding success and ageing rates in female meerkats: Competition and senescence in meerkats. J. Evol. Biol. 24, 1756–1762.
Shen, G.H., Alloy, L.B., Abramson, L.Y., Sylvia, L.G., 2008. Social rhythm regularity and the onset of affective episodes in bipolar spectrum individuals. Bipolar Disorders 10, 520–529.
Sheriff, M.J., Krebs, C.J., Boonstra, R., 2010. The ghosts of predators past: population cycles and the role of maternal programming under fluctuating predation risk. Ecology 91, 2983–2994.
Sheriff, M.J., Love, O.P., 2013. Determining the adaptive potential of maternal stress. Ecol. Lett. 16, 271–280.
Sheriff, M.J., Bell, A., Boonstra, R., Dantzer, B., Lavergne, S.G., McGhee, K.E., MacLeod, K.J., Winandy, L., Zimmer, C., Love, O.P., 2017. Integrating ecological and evolutionary context in the study of maternal stress. Integr. Comp. Biol. 57, 437–449.
Shilts, J., Chen, G., Hughey, J.J., 2018. Evidence for widespread dysregulation of circadian clock progression in human cancer. PeerJ 6, e4327.
Siiteri, P.K., Murai, J.T., Hammond, G.L., Nisker, J.A., Raymoure, W.J., Kuhn, R.W., 1982. The serum transport of steroid-hormones. Recent Prog. Horm. Res. 38, 457-503
Silk, J.B., 2007. Social components of fitness in primate groups. Science 317, 1347–1351.
Silva, L.R., Lardy, S., Ferreira, A.C., Rey, B., Doutrelant, C., Covas, R., 2018. Females pay the oxidative cost of dominance in a highly social bird. Anim. Behav. 144, 135–146.
Sloman, K.A., Motherwell, G., O'Connor, K.I., Taylor, A.C., 2000. The effect of social stress on the standard metabolic rate (SMR) of brown trout, *Salmo trutta*. Fish Physiol. Biochem. 23, 49–53.
Smith, J.E., Pinter-Wollman, N., 2021. Observing the unwatchable: Integrating automated sensing, naturalistic observations and animal social network analysis in the age of big data. J. Anim. Ecol. 90, 62–75.

1351 Smith, S.M., Vale, W.W., 2006. The role of the hypothalamic-pituitary-adrenal axis in
1352 neuroendocrine responses to stress. Dialogues Clin. Neurosci. 8, 383.
1353 Snyder-Mackler, N., Burger, J.R., Gaydosh, L., Belsky, D.W., Noppert, G.A., Campos, F.A.,
1354 Bartolomucci, A., Yang, Y.C., Aiello, A.E., O'Rand, A., Harris, K.M., Shively, C.A.,
1355 Alberts, S.C., Tung, J., 2020. Social determinants of health and survival in humans
1356 and other animals. Science 368, eaax9553.
1357 Sopinka, N.M., Capelle, P.M., Semeniuk, C.A.D., Love, O.P., 2017. Glucocorticoids in fish
1358 eggs: variation, interactions with the environment, and the potential to shape offspring
1359 fitness. Physiol. Biochem. Zool. 90, 15–33.
1360 Speakman, J.R., Selman, C., 2011. The free-radical damage theory: Accumulating evidence
1361 against a simple link of oxidative stress to ageing and lifespan. Bioessays 33, 255–
1362 259.
1363 Spencer, R., 1996. Chronic social stress produces reductions in available splenic type II
1364 corticosteroid receptor binding and plasma corticosteroid binding globulin levels.
1365 Psychoneuroendocrinology 21, 95–109.
1366 Stead, S.M., Bădescu, I., Boonstra, R., 2022. Of mammals and milk: how maternal stress
1367 affects nursing offspring. Mam. Rev .52, 129–147.
1368 Stier, A., Bize, P., Schull, Q., Zoll, J., Singh, F., Geny, B., Gros, F., Royer, C., Massemin, S.,
1369 Criscuolo, F., 2013. Avian erythrocytes have functional mitochondria, opening novel
1370 perspectives for birds as animal models in the study of ageing. Front. Zool., 10, 33.
1371 Stier, A., Reichert, S., Criscuolo, F., & Bize, P., 2015. Red blood cells open promising
1372 avenues for longitudinal studies of ageing in laboratory, non-model and wild animals.
1373 Exp. Gerontol., 71, 118-134.
1374 Stier, A., Romestaing, C., Schull, Q., Lefol, E., Robin, J.-P., Roussel, D., Bize, P., 2017. How
1375 to measure mitochondrial function in birds using red blood cells: a case study in the
1376 king penguin and perspectives in ecology and evolution. Meth. Ecol. Evol. 8, 1172–
1377 1182.
1378 Stier, A., Schull, Q., Bize, P., Lefol, E., Haussmann, M., Roussel, D., Robin, J.-P., Viblanc,
1379 V.A., 2019. Oxidative stress and mitochondrial responses to stress exposure suggest
1380 that king penguins are naturally equipped to resist stress. Sci. Rep. 9, 8545.
1381 Stockmaier, S., Bolnick, D.I., Page, R.A., Carter, G.G., 2018. An immune challenge reduces
1382 social grooming in vampire bats. Anim. Behav. 140, 141–149.
1383 Stoddard, S.L., Bergdall, V.K., Conn, P.S., Levin, B.E., 1987. Increases in plasma
1384 catecholamines during naturally elicited defensive behavior in the cat. J. Autonom.
1385 Nerv. Syst. 19, 189–197.
1386 Taborsky, B., 2016. Opening the black box of developmental experiments: behavioural
1387 mechanisms underlying long-term effects of early social experience. Ethology 122,
1388 267–283
1389 Takaesu, Y., 2018. Circadian rhythm in bipolar disorder: A review of the literature.
1390 Psychiatry Clin. Neurosci. 72, 673–682.
1391 Takahashi, J.S., Hong, H.-K., Ko, C.H., McDearmon, E.L., 2008. The genetics of mammalian
1392 circadian order and disorder: implications for physiology and disease. Nat. Rev.
1393 Genet. 9, 764–775.
1394 Tamashiro, K.L.K., Nguyen, M.M.N., Sakai, R.R., 2005. Social stress: From rodents to
1395 primates. Front. Neuroendocrinol. 26, 27–40.
1396 Tella, J.L., Forero, M.G., Bertellotti, M., Donázar, J.A., Blanco, G., Ceballos, O., 2001.
1397 Offspring body condition and immunocompetence are negatively affected by high
1398 breeding densities in a colonial seabird: a multiscale approach. Proc. R. Soc. Lond. B
1399 268, 1455–1461.

Teo, A.R., Lerrigo, R., Rogers, M.A.M., 2013. The role of social isolation in social anxiety disorder: A systematic review and meta-analysis. J. Anx. Dis. 27, 353–364.
Tertil, M., Skupio, U., Barut, J., Dubovyk, V., Wawrzczak-Bargiela, A., Soltys, Z., Golda, S., Kudla, L., Wiktorowska, L., Szklarczyk, K., Korostynski, M., Przewlocki, R., Slezak, M., 2018. Glucocorticoid receptor signaling in astrocytes is required for aversive memory formation. Transl. Psychiatry 8, 1–11.
Turner-Cobb, J.M., Sephton, S.E., Koopman, C., Blake-Mortimer, J., Spiegel, D., 2000. Social support and salivary cortisol in women with metastatic breast cancer. Psychosom. Med. 62, 337–345.
Uchino, B.N., 2004. Social support and physical health: Understanding the health consequences of relationships. Yale University Press.

Uchino, B.N., 2006. Social support and health: a review of physiological processes potentially underlying links to disease outcomes. J. Behav. Med. 29, 377–387.
Uchino, B.N., Cawthon, R.M., Smith, T.W., Light, K.C., McKenzie, J., Carlisle, M., Gunn, H., Birmingham, W., Bowen, K., 2012. Social relationships and health: is feeling positive, negative, or both (ambivalent) about your social ties related to telomeres? Health Psychol. 31, 789–796.
van Lieshout, S.H.J., Badás, E.P., Bright Ross, J.G., Bretman, A., Newman, C., Buesching, C.D., Burke, T., Macdonald, D.W., Dugdale, H.L., 2021. Early-life seasonal, weather and social effects on telomere length in a wild mammal. Mol. Ecol. mec.16014.
Venney, C.J., Love, O.P., Drown, E.J., Heath, D.D., 2020. DNA Methylation Profiles Suggest Intergenerational Transfer of Maternal Effects. Mol. Biol. Evol. 37, 540–548.
Viblanc, V.A., Valette, V., Kauffmann, M., Malosse, N., Groscolas, R., 2012. Coping with social stress: heart rate responses to agonistic interactions in king penguins. Behav. Ecol. 23, 1178–1185.
Viblanc, V.A., Gineste, B., Stier, A., Robin, J.-P., Groscolas, R., 2014a. Stress hormones in relation to breeding status and territory location in colonial king penguin: a role for social density? Oecologia 175, 763–772.
Viblanc, V A., Saraux, C., Malosse, N., Groscolas, R., 2014b. Energetic adjustments in freely breeding-fasting king penguins: does colony density matter? Funct. Ecol. 28, 621–631.
Viera, V.M., Viblanc, V.A., Filippi-Codaccioni, O., Côté, S.D., Groscolas, R., 2011. Active territory defence at a low energy cost in a colonial seabird. Anim. Behav. 82, 69–76.
von Zglinicki, T., 2002. Oxidative stress shortens telomeres. Trends Biochem. Sci. 27, 339–344.
Vyssotski, A.L., Serkov, A.N., Itskov, P.M., Dell'Omo, G., Latanov, A.V., Wolfer, D.P., Lipp, H.-P., 2006. Miniature neurologgers for flying pigeons: multichannel EEG and action and field potentials in combination with GPS recording. J. Neurophysiol. 95, 1263–1273.
Wascher, C.A F., Arnold, W., Kotrschal, K., 2008. Heart rate modulation by social contexts in greylag geese (*Anser anser*). J. Comp. Psychol. 122, 100–107.
Wascher, C.A.F., Scheiber, I.B.R., Weiß, B.M., Kotrschal, K., 2009. Heart rate responses to agonistic encounters in greylag geese, *Anser anser*. Anim. Behav. 77, 955–961.
Waser, P.M., 1985. Does competition drive dispersal? Ecology 66, 1170–1175.
Weber, K., Brück, P., Mikes, Z., Küpper, J.-H., Klingenspor, M., Wiesner, R.J., 2002. Glucocorticoid hormone stimulates mitochondrial biogenesis specifically in skeletal muscle. Endocrinology 143, 177–184.
Wells, K.D., Taigen, T.L., 1986. The effect of social interactions on calling energetics in the gray treefrog (*Hyla versicolor*). Behav. Ecol. Sociobiol. 19, 9–18.

White, D.J., Gersick, A.S., Snyder-Mackler, N., 2012. Social networks and the development of social skills in cowbirds. Phil. Trans. Roy. Soc. B 367, 1892–1900.
Wilmers, C.C., Nickel, B., Bryce, C.M., Smith, J.A., Wheat, R.E., Yovovich, V., 2015. The golden age of bio-logging: how animal-borne sensors are advancing the frontiers of ecology. Ecology 96, 1741–1753.
Wilson, E.O., 1975. Sociobiology: The new synthesis. Harvard University Press.
Wood, E.M., Capilla-Lasheras, P., Cram, D.L., Walker, L.A., York, J.E., Lange, A., Hamilton, P.B., Tyler, C.R., Young, A.J., 2021. Social dominance and rainfall predict telomere dynamics in a cooperative arid-zone bird. Mol. Ecol. mec.15868.
Yin, J., Zhou, M., Lin, Z., Li, Q.Q., Zhang, Y., 2019. Transgenerational effects benefit offspring across diverse environments: a meta-analysis in plants and animals. Ecol. Lett. 22, 1976–1986.
Young, A.J., Carlson, A.A., Monfort, S.L., Russell, A.F., Bennett, N.C., Clutton-Brock, T., 2006. Stress and the suppression of subordinate reproduction in cooperatively breeding meerkats. Proc. Natl. Acad. Sci. USA 103, 12005–12010.
Zane, L., Ensminger, D.C., Vázquez-Medina, J.P., 2021. Short-term elevations in glucocorticoids do not alter telomere lengths: A systematic review and meta-analysis of non-primate vertebrate studies. PLoS ONE 16(10), e0257370.
Zhang, Y., Wong, H.S., 2021. Are mitochondria the main contributor of reactive oxygen species in cells? J. Exp. Biol. 224, jeb221606.
Zhang, Y., Yin, J., Zhou, M., Lin, Z., Li, Q.Q., 2020. Adaptive transgenerational effects remain significant. Ecol. Lett. 23, 1719–1720.
Ziegler, A.-K., Watson, H., Hegemann, A., Meitern, R., Canoine, V., Nilsson, J.-Å., Isaksson, C., 2021. Exposure to artificial light at night alters innate immune response in wild great tit nestlings. J. Exp. Biol. 224, jeb239350.

FIGURE CAPTIONS

Figure 1. A comprehensive view of the effects of the social environment on key biological functions related to vertebrate health. Inputs from the social environment (or social stimuli) are integrated in the brain as negative or positive events, and this integration leads to modifications in key physiological functions including metabolism, oxidative stress, ageing, the rhythmicity of biological functions, immune responses, and brain function and psychological states. These functions can be directly affected by social stimuli, or indirectly, orchestrated by the stress axes including the sympathetic-adrenal-medullary (SAM) axis and the Hypothalamic-Pituitary-Adrenal (HPA) axis (hypothalamic pituitary inter-renal HPI in fish). Note that cross-talks between biological functions, either via or independently, of the stress axes, make an integration of functions necessary to properly understand the effects of social environments on vertebrate health.

TABLES

Table 1. Summary of the known effects of the social environment on key markers of vertebrates health.

Marker of Health	Effect	Mechanism	Example	
			Laboratory	Wild
Metabolism	Increased metabolic rate	Increase in physical activity		birds[1,2], fish[3,4], reptiles[5], amphibians[6]
		Without increase in physical activity (HPA/SAM activity)		fish[4], birds[7]
	Mitochondria respiratory capacities (in NAC)		rats[8]	
Oxidative stress & Ageing	Increased oxidative stress	Increased metabolic rate + enhance HPA activity + increased expression NADPH oxidases	rats[13]	mammals[9], fish[10,11], birds[12]
	Buffered oxidative stress			mammals[14]
	Accelerated telomere loss	Modification in GCs (trend direction not always consistent) + metabolic activity + telomerase activity	mice[15]	humans[16], birds[17], mammals[18,19]
	Reduced telomere loss in offspring			birds[20], mammals[21]
Immunity & Inflammation	Decreased adaptive immunity			house finches[22]
	Increased adaptive immunity			other birds species[23-25]
	Parasite load		meta-analysis in vertebrate species[26]	
	Increased social immunity	Allo-grooming, sickness behavior	humans[29,30]	mammals[27,28], birds[27]
Cognition & Psychology	Brain morphology & plasticity	Modification of MR and GR expression in brain	rats[31], mice[31]	mammals[31]
	Psychological state (depression-like behavior, anxiety)	Increased permeability of the Blood Brain Barrier	rats[32], mice[32,33], humans[34]	
	Learning & Memory	Role of GCs in formation of aversive memory	mice[35], fish[36]	birds[37], mammals[38]
	Social Competence		fish[36]	review on vertebrates[39]
Biological Rhythms	Social zeitgebers theory	Modification in patterns of hormone secretion (e.g. , melatonin)		humans and non-humans mammals[40]
	Social entrainment		fish[42]	mammals[41]

Footnote : [1]Nephew et al., 2003; [2]Viera et al., 2011; [3]Metcalfe et al., 1995; [4]Sloman et al., 2000; [5]Marler et al., 1995; [6]Wells et al., 1986 ; [7]Viblanc et al., 2014b ; [8]Hollis et al., 2015 ; [9]Beaulieu et al., 2014 ; [10]Border et al., 2019 ; [11]Funnel et al., 2022 ; [12]Silva et al., 2018 ; [13]Colaianna et al., 2013 ; [14]Lardy et al., 2016 ; [15]Kotrschal et al., 2007 ; [16]Epel et al., 2004 ; [17]Nettle et al., 2013 ; [18]Anderson et al., 2021 ; [19]Sharp & Clutton-Brock 2011 ; [20]Quque et al., 2021 ; [21]Bauer et al., 2015 ; [22]Hawley et al. ; 2006 ; [23]Minias et al., 2019 ; [24]Tella et al., 2001 ; [25]Kamiński et al., 2021; [26]Habig et al., 2018 ; [27]Raulo & Dantzer, 2018 ; [28]Hamilton et al., 2020 ; [29]Dantzer & Kelley, 2007 ; [30]Kopec et al., 2019 ; [31]Blanchard et al., 2001 ; [32]Lukkes et al., 2009 ; [33]Teo et al. ,2013 ; [34]Menard et al., 2017 ; [35]Tertil et al., 2018 ; [36]Reyes-Contreras et al., 2019 ; [37]Heinen et al., 2021 ; [38]Leblanc & Ramirez, 2020 ; [39]Taborsky, 2016 ; [40]Favreau et al., 2016 ; [41]Heinzeller et al., 1988 ; [42]Larson et al., 2004

FIGURES

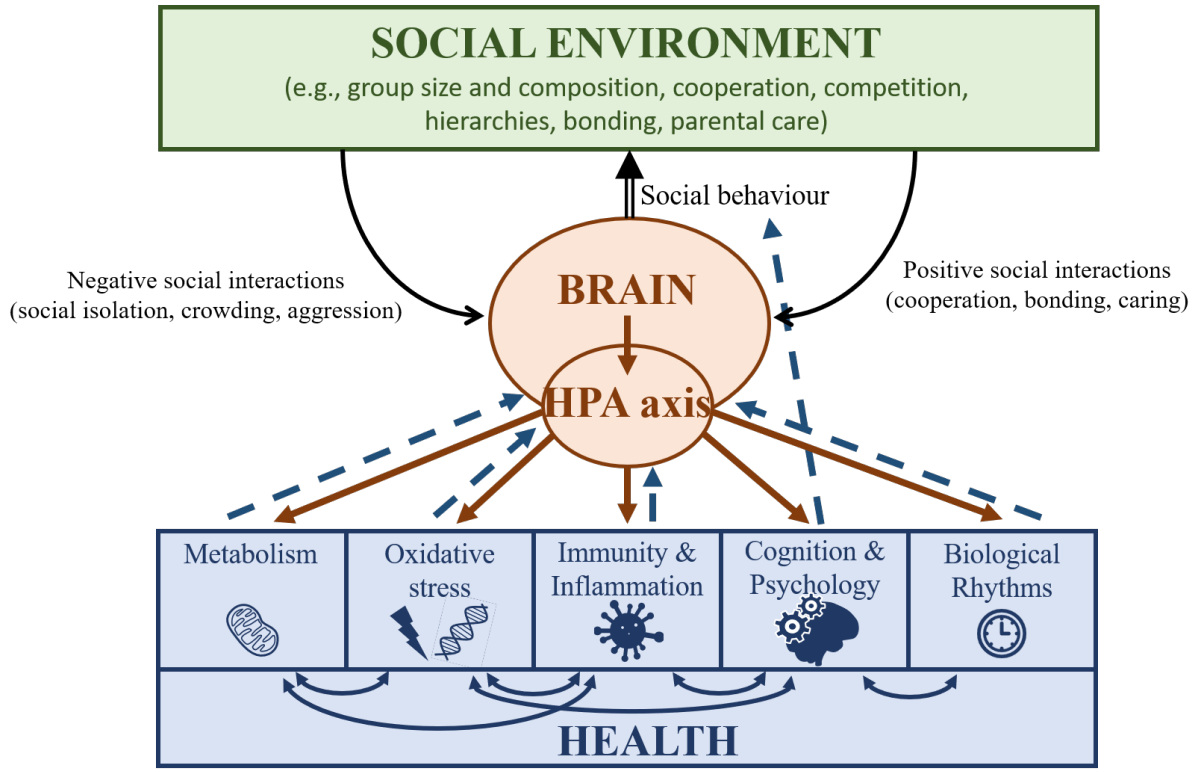

Figure 1.