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### ► To cite this version:

Camille Lemonnier, Pierre Bize, Rudy Boonstra, F. Stephen Dobson, François Criscuolo, et al.. Effects of the social environment on vertebrate fitness and health in nature: moving beyond the stress axis. *Hormones and Behaviour*, 2022, 145, pp.105232. 10.1016/j.yhbeh.2022.105232 . hal-03838854

**HAL Id: hal-03838854**

**<https://hal.science/hal-03838854>**

Submitted on 3 Nov 2022

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

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**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.

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## 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



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low social densities (Lemonnier, Schull, Stier, Boonstra, Delahanty, Lefol, Durand, Robin, Criscuolo, Bize, Viblanc; *unpublished data*).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

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

(Metcalf 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 (Viblanco et al., 2014b). Interestingly, higher social densities in penguins are also associated with higher baseline GC levels in individual birds (Viblanco 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*

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

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

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

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

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

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

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

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## 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.

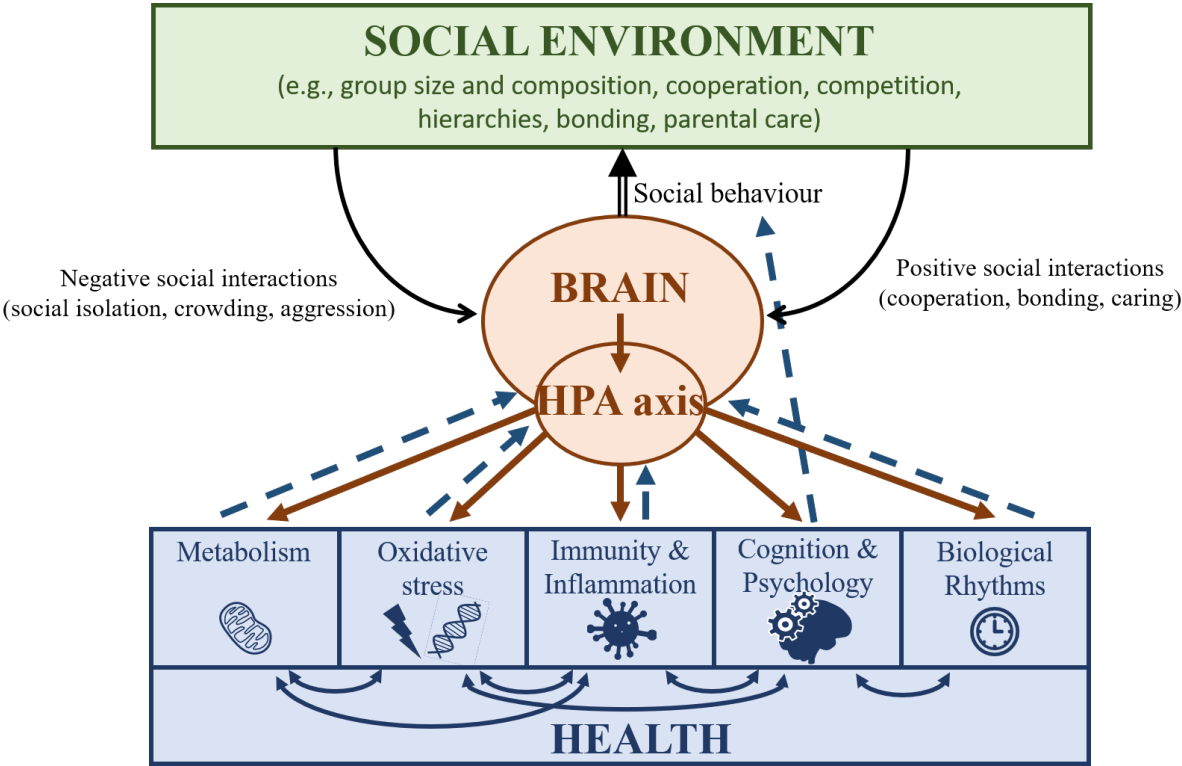
Marker of Health	Effect	Mechanism	Example	
			Laboratory	Wild
Metabolism	Increased metabolic rate	Increase in physical activity		birds <sup>1,2</sup> , fish <sup>3,4</sup> , reptiles <sup>5</sup> , amphibians <sup>6</sup>
		Without increase in physical activity (HPA/SAM activity)		fish <sup>4</sup> ,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

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



1509 **FIGURES**

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1512 **Figure 1.**

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