



HAL
open science

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

Camille Lemonnier, Pierre Bize, Rudy Boonstra, F. Stephen Dobson, François Criscuolo, Vincent A Viblanc

► 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

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L'archive ouverte pluridisciplinaire **HAL**, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d'enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.

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

3
4 Camille Lemonnier^{1,2*}, Pierre Bize^{3,4}, Rudy Boonstra⁵, F. Stephen Dobson^{2,6}, François
5 Criscuolo², Vincent A Viblanc²

6
7 ¹Ecole Normale Supérieure de Lyon, 69342 Lyon, France

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

9 ³School of Biological Sciences, University of Aberdeen, Aberdeen, UK

10 ⁴Swiss Institute of Ornithology, Sempach, Switzerland

11 ⁵Department of Biological Sciences, University of Toronto Scarborough, Toronto, Canada

12 ⁶Department of Biological Sciences, Auburn University, Auburn, AL, USA

13
14
15 ***Correspondance** : Camille Lemonnier; camille.lemonnier@ens-lyon.org

16
17
18
19 **Keywords:** dominance, epigenetics, hierarchies, HPA, social determinants of health, social
20 buffers

21 **ABSTRACT**

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

45

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

48

49

50 INTRODUCTION

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

66 Much of the behavioral and physiological changes that occur when vertebrates are faced
67 with predictable or unpredictable stressors – such as when dealing with conspecifics – are
68 orchestrated by the Sympathetic-Adrenal-Medullary axis (SAM) and the Hypothalamic-
69 Pituitary-Adrenal (HPA) axis (hypothalamic pituitary inter-renal HPI in fish) (Smith and Vale,
70 2006; Romero and Butler, 2007). Thus, it is not surprising that studies on the relationships
71 between social interactions and individual physiology have focused on what has come to be
72 known as the stress axis. These studies have considered both aspects of the stress response: i.e.
73 (1) SAM activity, which occurs within seconds of a disturbance, participates in the “fight-or-
74 flight” response of the organism via the action of catecholamines (adrenaline, noradrenaline),

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

82 Whereas the effects of social interactions on the stress axis are now well known, in
83 humans, laboratory/captive animals, and in wild animals, (reviewed at length elsewhere;
84 DeVries et al., 2003; Goymann and Wingfield, 2004; Creel 2004; Creel et al., 2013) it is only
85 more recently that researchers have started to move beyond the stress axis alone in
86 understanding the adaptive consequences of social interactions (Snyder-Mackler et al., 2020).
87 On one hand, a large body of research on human well-being and on laboratory/ captive animals
88 has sought to understand how 'negative' social interactions (competition, hierarchies, work
89 stress, emotional neglect or abuse, etc.) may lead to pathology and disease (DeVries et al., 2003;
90 Cohen, 2004; Epel et al., 2004; Avitsur et al., 2006; Blackburn and Epel, 2012; Norman et al.,
91 2012), whereas 'positive' social interactions may provide a buffer against social challenges,
92 alleviating individual stress, in many cases with positive health outcomes (Turner-Cobb et al.,
93 2000; DeVries et al., 2003; Heinrichs et al., 2003; Cohen, 2004; Rosal et al., 2004; Uchino,
94 2004; Lutgendorf et al., 2005; Uchino, 2006; Uchino, 2006; Uchino et al., 2012; Bateson, 2016).
95 On the other, numerous studies in wild vertebrates have focused on the relationships between
96 social environments and the stress axis (see Creel et al., 2013) but much remains to be
97 understood about how social environments (often through modulation of the HPA or SAM
98 axes) affect other important physiological functions directly related to individual fitness and
99 health (Sapolsky, 2004, 2005; Snyder-Mackler et al., 2020). Below, we review the current state

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

106 First, studies aiming to understand the effects of social environments on individual
107 physiology and stress have mostly focused on how social interactions may affect the function
108 of the SAM or HPA/I axes, and these studies have often used chronically elevated levels of
109 circulating GC as indicative of elevated stress load and a likely marker of disease and pathology
110 (Boonstra 2013). This view is one that stems mostly from the biomedical literature where
111 individuals are pushed beyond their capacity to cope with stressors from the environment.
112 Under such situations, the negative feedback loops that terminate stress responses are impaired,
113 resulting in irreversible “wear and tear” of bodily functions (McEwen and Wingfield, 2003;
114 Romero et al., 2009). Whereas this might occur in natural conditions, it is more likely the
115 exception rather than the rule, stress and chronically elevated GC levels being primarily
116 adaptive responses to deal with ecological challenges, and thus beneficial (Boonstra, 2004,
117 2005, 2013; Bonier et al., 2009; Beehner and Bergman, 2017). Some confusion may occur when
118 GCs are often and misleadingly referred to negatively and interpreted as “stress hormones” in
119 the literature (including in some of our own papers) (MacDougall-Shackleton et al., 2019).
120 Because GCs are pleiotropic hormones with receptor- and tissue-specific functions, that
121 modulate the expression of some 10% of the genome (Le et al., 2005), the role of GCs cannot
122 be confined to the stress response alone. Unfortunately, several studies using the descriptor
123 “stress hormones” fail to consider the adaptive action of GCs in coordinating other essential
124 physiological functions (for a discussion see Boonstra, 2007, 2013; MacDougall-Shackleton et

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

132 Second, focusing solely on the measurement of individual total GC levels in response
133 to social interactions alone can be misleading. GCs circulate in the bloodstream either strongly
134 bound to corticosteroid binding globulin (CBG) or free. The free hormone hypothesis states
135 that only the latter is biologically active and can bind to GC receptors (GR) to trigger cellular
136 signaling pathways and modifications in gene transcription and cell functioning (Mendel,
137 1989). Because about 90% of blood GCs are bound to CBG in the vast majority of vertebrates
138 (Desantis et al., 2013a; Delehanty et al., 2015; 2020), our understanding of how social
139 interactions affect individual stress requires analyzing total GCs *and* CBG levels, and how these
140 change as a function of the social environment. This could provide information not only on the
141 biologically active fraction of GC (the free GC) and level of activation of the HPA/I axis in
142 response to social challenges, but also on the organism's capacity to buffer elevated levels of
143 circulating GC by binding to CBG. Such information is functionally important in maintaining
144 physiologically acceptable levels of active GCs in the organism (Breuner et al., 2013). Studies
145 from captive animals indicate that socially stressed animals experience a decrease in CBG
146 capacity, leading to higher access of free GCs (Spencer, 1996; Alexander and Irvine, 1998;
147 Tamashiro et al., 2005; Otten et al., 2010). However, those studies usually focus on situations
148 where social conflict is forced, and little is known of social effects on CBG in wild vertebrates.
149 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

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

189 For these reasons, our understanding of the fitness consequences that social interactions
190 may have on vertebrates in the wild, and the extent to which they are an important source of
191 phenotypic variation within and amongst social units (siblings, families, matriline, colonies,
192 etc.), should benefit from a holistic view encompassing key physiological functions, some
193 centrally coordinated by the SAM or HPA/I axes, pertaining to vertebrate health (Sapolsky,
194 2005, 2004; Snyder-Mackler et al., 2020).

195

196

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

199 Health has often been defined as the absence of disease, inflammation or pathology (Blaxter,
200 2010). This definition, however, can be unpractical, especially when trying to determine how
201 social stressors affect the functioning of organisms, as an overwhelming number of mechanisms
202 and pathways can result in the genesis of diseases (Blaxter, 2010; Ayres, 2020). A more
203 practical definition describes the hallmarks of health as the function of cells and organs,
204 integration of functions and communication at the cellular and organism levels, and normal
205 biological rhythms (López-Otín and Kroemer, 2021) where “normal” allows for allostasis and
206 response to stress with no or minimal long-term deleterious effects, as the organismal response
207 to stressful stimuli is part of the usual and adaptive functioning of organisms (McEwen and
208 Wingfield, 2003; Romero et al., 2009). Below, we review the state of current knowledge on the
209 effects that social environments have on vertebrate health by focusing on key functions. The
210 key functions encompass individual metabolism/energetics, oxidative stress/damage/ageing
211 processes, immunity/inflammation, cognitive/psychological state and decline, and the
212 regulation of biological rhythms (Figure 1).

213

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

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

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

238 There is an intuitive appeal in studying mitochondria to understand vertebrate responses
239 to stressful (social) situations. Mitochondria, often referred to as ‘energy powerhouses’ of
240 eukaryote cells, are organelles responsible for the majority of energy production in vertebrates
241 (Lane, 2006). Their function, morphology and behavior (communication at inter-mitochondrial
242 junctions; Picard et al., 2015) are responsible for determining much of animal performances
243 (Heine and Hood, 2020). In the context of stress, mitochondria play a key role in making
244 cellular energy available under challenging conditions by conforming to local levels of
245 glucocorticoids (Manoli et al., 2007; Picard et al., 2014, 2018). But more importantly,
246 mitochondria are key regulators that are involved in the stress response itself (Picard et al.,
247 2014; Lapp et al., 2019). Following ACTH activation by the adrenal gland, the synthesis of
248 steroids (including glucocorticoids) is limited by the transport of cholesterol across the

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

253 Although the study of mitochondrial function is recently gaining attention in the field
254 of ecology and evolution as a central effector mediating life history trade-offs (Criscuolo et al.,
255 2005; Hood et al., 2018; Havird et al., 2019; Koch et al., 2021), there are yet few studies that
256 have considered mitochondria under the light of organismal stress in wild vertebrates (Stier et
257 al., 2019; Casagrande et al., 2020), except for the well-known role of mitochondria in the
258 response to thermal stress (Criscuolo et al., 2005; Koch et al., 2021). To our knowledge, little
259 is known on the relationships between social environments/social interactions and
260 mitochondrial function in the wild. A recent study in captive rats identified the nucleus
261 accumbens (NAc), a brain region involved in the expression of motivation and anxiety
262 behaviors, as critical in the establishment of social hierarchies (Hollis et al., 2015). Surprisingly,
263 anxious rats had reduced mitochondrial respiratory capacity in the NAc and lower ATP output.
264 More importantly, experimentally inhibiting the activity of respiratory complexes in NAc
265 mitochondria reduced the success of treated animals for winning social contests during the
266 establishment of social hierarchies (Hollis et al., 2015). In addition, studies indicated that
267 mitochondria are sensitive to time-dose dependent effects of stressors (Du et al., 2009; Picard
268 et al., 2018), and may integrate and transduce psychosocial factors into cellular and molecular
269 modifications, which in turn may contribute to the embedding of psychological states affecting
270 social and other behaviors (reviewed in Picard and McEwen, 2018a, 2018b). These studies
271 stemming from the neuroscience literature are important for our understanding of social stress
272 in wild vertebrates, since they indicate a causal mechanistic pathway directly relating

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

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

281

282 **Social effects on oxidative stress, health and ageing**

283 ROS production has both positive and negative effects on vertebrate health: at low levels ROS
284 play a key role as secondary messengers that can affect gene expression and actually promote
285 resistance to stress (Ristow and Zarse, 2010; Costantini, 2014; Hood et al., 2018). At high
286 levels, ROS cause oxidative stress, which has been suggested as an important factor behind
287 organism decline and ageing (Beckman and Ames, 1998; Finkel and Holbrook, 2000; but see
288 Speakman and Selman, 2011; Kirkwood and Kowald, 2012). In recent years, a growing number
289 of studies have focused on the relationships between social interactions and increased oxidative
290 stress in vertebrates. Opposite relationships pointing to beneficial effects of social environments
291 on buffering oxidative stress have also been found (Lardy et al., 2016; Li and Xia, 2020).
292 Interestingly, common to many of those studies is the observation that social instability and
293 social aggression are associated with increased oxidative stress in mammals (Beaulieu et al.,
294 2014; Nation et al., 2008), fish (Border et al., 2019, 2021; Fialkowski et al., 2021; but see
295 Funnell et al., 2022), and birds (Silva et al., 2018; Quque et al., in press).

296 However, it is not only competitive social interactions that are related to increased
297 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 (Schivavone 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

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

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

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

349 Although telomere shortening has been associated with increased exposure to various
350 sources of stress in non-human vertebrates (Chatelain et al., 2020), it is only relatively recently
351 that the effects of social environments and social interactions on telomere length and telomere
352 shortening rates have been considered. In birds, studies have shown that both early-life social
353 adversity (exposure to a large number of dominant competitors; *Sturnus vulgaris*; Nettle et al.,
354 2013), and lack of social contact (social isolation; *Psittacus erithacus erithacus*; Aydinonat et
355 al., 2014) can be associated with accelerated telomere loss. Social crowding also appears to
356 interfere with telomere restoration mechanisms causing shorter telomeres in socially crowded
357 groups (in lab mice, Kotrschal et al., 2007; in birds, Quque et al., *in press*). Moreover, in
358 addition to direct effects of social interactions on telomere dynamics, studies indicate how
359 interactions between social and reproductive strategies may shape ageing trajectories, both in
360 early life and adulthood. For instance, social competition among mature cooperative breeders
361 has been shown to carry long-term costs in terms of increased reproductive senescence (Sharp
362 and Clutton-Brock, 2011). Further, dominant individuals who monopolize reproduction in
363 cooperatively breeding sparrow-weavers (*Plocepasser mahali*) show higher investments into
364 telomere maintenance than subordinates, an association likely to mitigate somatic costs of
365 reproduction (Wood et al., 2021). In juveniles, early social competition for food within litters
366 or juvenile cohorts may enhance telomere loss (Boonekamp et al., 2014; Cram et al., 2017;
367 Nettle et al., 2015; but see van Lieshout et al., 2021), but the presence of cooperative adults
368 helping with reproductive effort may favor telomere maintenance in offspring, suggesting
369 positive social effects on parental care with downstream consequences on offspring health in
370 the wild (Quque et al., 2021). Parents may then act as important buffers to stressful
371 environments for their offspring (Bauer et al., 2015; Gunnar and Hostinar, 2015).

372 Thus, it appears clear that social interactions, status and environments have strong
373 effects on cellular processes related to telomere degradation and maintenance, and overall
374 organismal senescence. However, much remains to be understood about the interplay among
375 social stressors, GCs, oxidative stress, telomere dynamics and their consequences on individual
376 performance in the wild. Notably, telomere length and shortening rate appear to be related to
377 chronic, but not short-term (Zane et al., 2021), modifications in GCs in vertebrates (reviewed
378 in Angelier et al., 2018), though the direction of this relation (shorter telomeres with increased
379 or reduced GCs) is not always consistent. Understanding how telomere biology is regulated by
380 the interplay between GC and ROS production (Casagrande and Hau, 2019) is the next logical
381 step in our understanding of social effects on vertebrate senescence and decline in overall
382 performances in the wild. Particularly, in addition to focusing on telomere loss, it would be of
383 interest to understand how telomerase activity (an enzyme specialized in re-building telomeres)
384 is regulated by social stressors in nature (see Epel et al., 2010; Beery et al., 2012; Deng et al.,
385 2016 for studies on captive animals and humans). In addition, the relationships between social
386 stress and ageing are likely to extend to non-genetic molecular mechanisms as well, as recently
387 demonstrated in wild yellow baboons (*Papio cynocephalus*) where high social rank males
388 experience accelerated epigenetic ageing (Anderson et al., 2021).

389

390 **Social effects on immunity**

391 The immune system is the body's main line of defense against pathogens and parasites (Iwasaki
392 and Medzhitov, 2010). Because social proximity and increased number of direct interactions
393 among individuals increase the risk of pathogen and parasite transmission (Cote and Poulinb,
394 1995; Patterson and Ruckstuhl, 2013; Schmid-Hempel, 2017), sociality and immunity are
395 tightly linked (Kappeler et al., 2015). The immune system can be divided into innate and
396 adaptive responses which, although they interact, differ greatly in their specificity and

397 regulation by the HPA axis and release of GCs (Iwasaki and Medzhitov, 2010). Although GCs
398 are best known for their suppressive effects on the immune system, there is rapidly
399 accumulating evidence that GCs also enhance inflammation and immunity (Cain and
400 Cidlowski, 2017). Accordingly, low doses of GCs are thought to promote localized
401 inflammatory response (stimulating cytokines and complement responses) and suppress
402 adaptive immunity, whereas high doses of GCs will suppress innate and adaptive immunity,
403 therefore preventing excessive and/or prolonged immune responses (Cain and Cidlowski,
404 2017). Hence, the effects of social interactions on GCs and, in turn, immunity, are expected to
405 be complex and to lead to nuanced results. For instance, adaptive immunity was observed to
406 decrease with group size (and social status) in house finches (*Carpodacus mexicanus*) (Hawley
407 et al., 2006), but to increase with group size in other bird species (Minias et al., 2019; Tella et
408 al., 2001; Kamiński et al., 2021). Predicting how social effects, immunity and parasite load are
409 inter-linked is further complicated by the fact that immunity is not regulated by GCs alone
410 (Iwasaki and Medzhitov, 2010), with many other factors besides social interactions and
411 immunity influencing parasite load (Bize et al., 2008). This is well illustrated in studies on
412 social status (Habig and Archie, 2015; Habig et al., 2018). For instance, high rank individuals
413 are expected to be more exposed to parasites, as they often show increased number of social
414 interactions, have greater energy expenditure, and need to feed more frequently (Clutton-Brock
415 and Huchard, 2013). Alternatively, high rank individuals can be more exposed to parasites as
416 they often show greater investment into reproduction that can come at the expense of immune
417 defences. Finally, as low rank individuals experience more frequent defeats in social
418 antagonistic interactions, they are expected to also show lower immunity and higher levels of
419 parasitism caused by social stress and increased circulating GCs. Comparative studies in
420 vertebrates show that social rank has only weak effects on immune functions but often strong
421 effects on parasite load (Habig and Archie, 2015; Habig et al., 2018), with dominant individuals

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

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

435 Finally, it is important to note that the relationship between social behavior and
436 immunity is bidirectional. It is now well demonstrated that the immune system communicates
437 with the brain via the release of proinflammatory cytokines and chemokines, which can impair
438 social behavior by leading to social withdrawal (Dantzer and Kelley, 2007; Kopec et al., 2019).
439 These changes, together with reduced activity, referred to as ‘sickness behavior’, are widely
440 viewed as an adaptive host response that prevents parasite transmission rather than a
441 manipulation of the host behaviour by parasites (Dantzer and Kelley, 2007). A chronic
442 inflammation and immune-neuro modulation of the brain can however lead to social isolation
443 and depression (Raison et al., 2006). Although the immune-neuro modulation of sickness
444 behaviour is evolutionary conserved at least in mammals and birds (Dantzer and Kelley, 2007),
445 we still know very little about effects of inflammation on social interactions in the wild
446 (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

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

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

491

492 **Social effects on biological rhythms**

493 A ubiquitous feature of vertebrate biological functions (including gene expression, physiology
494 and behavior) is their rhythmicity (Aschoff, 1981; Rusak, 1981; Kumar, 2002). Most
495 physiological systems function in pulsatile fashion, with ultradian, circadian, diurnal or
496 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

522 biological rhythms and health have been particularly studied with reference to mood regulation
523 and mental disorders in humans (Grandin et al., 2006; Shen et al., 2008; Margraf et al., 2016;
524 Takaesu, 2018; Sabet et al., 2021). Though studies in other vertebrates have focused on social
525 entrainment of behavioral circadian rhythmicity (Favreau et al., 2009), fewer have focused on
526 the interrelations among social stressors, biological rhythms and health. Yet, social aggression
527 has been found to alter patterns of melatonin secretion (in mammals, Heinzeller et al., 1988; in
528 fish, Larson et al., 2004), a key hormone secreted by the pineal gland responsible for
529 synchronizing daily rhythms to light/dark cycles in vertebrates, perhaps explaining differences
530 in behavioral profiles between subordinate and dominant individuals (Larson et al., 2004).
531 Indeed, melatonin is itself known to play a role in the expression of social aggression (Jasnow
532 et al., 2002; Demas et al., 2004; Munley et al., 2020), and GC and/or adrenoreceptors have been
533 found in the vertebrate pineal gland (e.g. in fish; Benyassi et al., 2001; in rats; Fernandes et al.,
534 2017). These studies suggest that causal pathways might exist from the perception of social
535 stressors to the expression of social behaviors through the regulation of the pineal gland. In
536 addition, the pineal gland and melatonin play a key role in the innate immune response for
537 instance by favoring phagocytosis and modulating inflammation (Majewski et al., 2012;
538 Markus and Ferreira, 2011). Thus, cross-talks between the HPA/SAM axis, the immune system,
539 the pineal gland, and the expression of social behaviors appear to be features that require further
540 exploration in the context of social stressors in wild vertebrates (Couto-Moraes et al., 2009).

541

542 **WHERE NEXT?**

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

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

564 First, the evaluation of the various health markers described above in a wider range of
565 species encountering different habitats, social environments and having different life-histories
566 is necessary to assess how these different markers respond to various types of social and non-
567 social environments. Such assessments will provide a better understanding of their associations
568 with individual survival, reproduction, life history, and fitness. Implementing the evaluation of
569 such markers in field-based ecological studies is challenging, considering the methodological
570 and ethical limitations inherent to the study of wild animals. However, available tools in both
571 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 even more salient by recent
596 technological advances allowing the acquisition of detailed information on social interactions

597 in situations where direct visual observation is not always possible (Rutz et al., 2012; Smith
598 and Pinter-Wollman, 2021). The use of such tools and the fine resolution of data collected are
599 promising avenues for measuring metrics related to energy metabolism (e.g. via heart rate and
600 ECG, and accelerometry; Green et al., 2009), immunity (e.g. via body temperature; Jax et al.,
601 2021), rhythmicity (e.g. via accelerometry and activity patterns); and brain activity (via EEG;
602 Vyssotski et al., 2006), with regards to varying social interactions, social contexts, and
603 hierarchies.

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

620 Studies of mechanisms responsible for intergenerational consequences of social
621 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.

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

658

659 ACKNOWLEDGMENTS

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

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

675

676 REFERENCES

- 677 Abbott, D.H., Saltzman, W., Schultz-Darken, N.J., Smith, T.E., 1997. Specific
678 neuroendocrine mechanisms not involving generalized stress mediate social regulation
679 of female reproduction in cooperatively breeding marmoset monkeys. *Ann. NY Acad.*
680 *Sci.* 807, 219–238.
- 681 Abbott, D.H., Keverne, E.B., Bercovitch, F.B., Shively, C.A., Mendoza, S.P., Saltzman, W.,
682 Snowdon, C.T., Ziegler, T.E., Banjevic, M., Garland, T., Sapolsky, R.M., 2003. Are
683 subordinates always stressed? A comparative analysis of rank differences in cortisol
684 levels among primates. *Horm. Behav.* 43, 67–82.
- 685 Alexander, R.D., 1974. The evolution of social behavior. *Ann. Rev. Ecol. Systematics* 5, 325–
686 383.
- 687 Alexander, S., Irvine, C., 1998. The effect of social stress on adrenal axis activity in horses:
688 the importance of monitoring corticosteroid-binding globulin capacity. *J. Endocrinol.*
689 157, 425–432.
- 690 Anderson, J.A., Johnston, R.A., Lea, A.J., Campos, F.A., Voyles, T.N., Akinyi, M.Y.,
691 Alberts, S.C., Archie, E.A., Tung, J., 2021. High social status males experience
692 accelerated epigenetic aging in wild baboons. *eLife* 10, e66128.
- 693 Angelier, F., Costantini, D., Blévin, P., Chastel, O., 2018. Do glucocorticoids mediate the link
694 between environmental conditions and telomere dynamics in wild vertebrates? A
695 review. *Gen. Comp. Endocrinol.* 256, 99–111.
- 696 Aplin, L.M., Farine, D.R., Morand-Ferron, J., Cockburn, A., Thornton, A., Sheldon, B.C.,
697 2015. Experimentally induced innovations lead to persistent culture via conformity in
698 wild birds. *Nature* 518, 538–541.
- 699 Aschoff, J. (Ed.), 1981. *Biological rhythms*. Springer US, Boston, MA.
- 700 Avitsur, R., Padgett, D.A., Sheridan, J.F., 2006. Social interactions, stress, and immunity.
701 *Neurol. Clinics* 24, 483–491.
- 702 Aydinonat, D., Penn, D.J., Smith, S., Moodley, Y., Hoelzl, F., Knauer, F., Schwarzenberger,
703 F., 2014. Social isolation shortens telomeres in African grey parrots (*Psittacus*
704 *erithacus erithacus*). *PLoS ONE* 9, e93839.
- 705 Ayres, J.S., 2020. The biology of physiological health. *Cell* 181, 250–269.
- 706 Bai, Y., Jabbari, B., Ye, S., Campese, V.M., Vaziri, N.D., 2009. Regional Expression of
707 NAD(P)H Oxidase and Superoxide Dismutase in the Brain of Rats with Neurogenic
708 Hypertension. *Am. J. Nephrol.* 29, 483–492.
- 709 Balaban, R.S., Nemoto, S., Finkel, T., 2005. Mitochondria, oxidants, and aging. *Cell* 120,
710 483–495.
- 711 Bateson, M., 2016. Cumulative stress in research animals: Telomere attrition as a biomarker
712 in a welfare context? *BioEssays* 38, 201–212.
- 713 Bauer, C.M., Hayes, L.D., Ebensperger, L.A., Ramírez-Estrada, J., León, C., Davis, G.T.,
714 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

814 model of psychosocial stress: relation to oxidative stress. *Antiox. Redox Signal.* 18,
815 1385–1399.

816 Costantini, D., 2014. *Oxidative stress and hormesis in evolutionary ecology and physiology:*
817 *A marriage between mechanistic and evolutionary approaches*, 1st ed. Springer,
818 Berlin, Heidelberg.

819 Costantini, D., Marasco, V., Møller, A.P., 2011. A meta-analysis of glucocorticoids as
820 modulators of oxidative stress in vertebrates. *J. Comp. Physiol. B.*

821 Cote, I.M., Poulinb, R., 1995. Parasitism and group size in social animals: a meta-analysis.
822 *Behavi. Ecol.* 6, 159–165.

823 Côté, S.D., 2000. Aggressiveness in king penguins in relation to reproductive status and
824 territory location. *Anim. Behav.* 59, 813–821.

825 Cotter, S.C., Kilner, R.M., 2010. Personal immunity versus social immunity. *Behav. Ecol.* 21,
826 663–668.

827 Couto-Moraes, R., Palermo-Neto, J., Markus, R.P., 2009. The immune-pineal axis: Stress as a
828 modulator of pineal gland function. *Ann. NY Acad. Sci.* 1153, 193–202.

829 Cram, D.L., Monaghan, P., Gillespie, R., Clutton-Brock, T., 2017. Effects of early-life
830 competition and maternal nutrition on telomere lengths in wild meerkats. *Proc. R. Soc.*
831 *B.* 284, 20171383.

832 Creel, S., 2001. Social dominance and stress hormones. *Trends Ecol. Evol.* 16, 491–497.

833 Creel, S., 2022. A retrospective view of early research on dominance, stress and reproduction
834 in cooperatively breeding carnivores. *Horm. Behav.* 140, 105119.

835 Creel, S., Creel, N.M., Mills, M.G.L., Monfort, S.L., 1997. Rank and reproduction in
836 cooperatively breeding African wild dogs: behavioral and endocrine correlates. *Behav.*
837 *Ecol.* 8, 298–306.

838 Creel, S., Dantzer, B., Goymann, W., Rubenstein, D.R., 2013. The ecology of stress: effects
839 of the social environment. *Functional Ecology* 27, 66–80.

840 Criscuolo, F., Gonzalez-Barroso, M. del M., Bouillaud, F., Ricquier, D., Miroux, B., Sorci,
841 G., 2005. Mitochondrial uncoupling proteins: new perspectives for evolutionary
842 ecologists. *Am. Nat.* 166, 686–699.

843 Cunliffe, V.T., 2016. The epigenetic impacts of social stress: how does social adversity
844 become biologically embedded? *Epigenomics* 8, 1653–1669.

845 D’Agostino, S., Testa, M., Aliperti, V., Venditti, M., Minucci, S., Aniello, F., Donizetti, A.,
846 2019. Expression pattern dysregulation of stress- and neuronal activity-related genes
847 in response to prenatal stress paradigm in zebrafish larvae. *Cell Stress Chaperones* 24,
848 1005–1012.

849 Dantzer, B., Newman, A.E.M., Boonstra, R., Palme, R., Boutin, S., Humphries, M.M.,
850 McAdam, A.G., 2013. Density triggers maternal hormones that increase adaptive
851 offspring growth in a wild mammal. *Science* 340, 1215–1217.

852 Dantzer, B., Bennett, N.C., Clutton-Brock, T.H., 2017. Social conflict and costs of
853 cooperation in meerkats are reflected in measures of stress hormones. *Behav. Ecol.* 28,
854 1131–1141.

855 Dantzer, R., Kelley, K.W., 2007. Twenty years of research on cytokine-induced sickness
856 behavior. *Brain Behav. Immun.* 21, 153–160.

857 Delehanty, B., Hossain, S., Jen, C.C., Crawshaw, G.J., Boonstra, R., 2015. Measurement of
858 free glucocorticoids: Quantifying corticosteroid-binding globulin binding affinity and
859 its variation within and among mammalian species. *Conserv. Physiol.* 3(1), cov020

860 Delehanty, B., Bossart, G.D., Champagne, C., Crocker, D.E., Elliott, K.H., Fair, P.A., Houser,
861 D., Newman, A.E.M., Boonstra, R., 2020. Measurement of free glucocorticoids:
862 quantifying corticosteroid binding capacity and its variation within and among
863 mammal and bird species. *Conserv. Physiol.* 8(1), coaa057.

- 864 Delezie, J., Challet, E., 2011. Interactions between metabolism and circadian clocks:
865 reciprocal disturbances: Circadian disruption and metabolic dysfunction. *Ann. NY*
866 *Acad. Sci.* 1243, 30–46.
- 867 Demas, G.E., Polacek, K.M., Durazzo, A., Jasnow, A.M., 2004. Adrenal hormones mediate
868 melatonin-induced increases in aggression in male Siberian hamsters (*Phodopus*
869 *sungorus*). *Horm. Behav.* 46, 582–591.
- 870 Deng, W., Cheung, S.T., Tsao, S.W., Wang, X.M., Tiwari, A.F.Y., 2016. Telomerase activity
871 and its association with psychological stress, mental disorders, lifestyle factors and
872 interventions: A systematic review. *Psychoneuroendocrinology* 64, 150–163.
- 873 Desantis, L.M., Delehanty, B., Weir, J.T., Boonstra, R., 2013. Mediating free glucocorticoid
874 levels in the blood of vertebrates: are corticosteroid-binding proteins always
875 necessary? *Funct. Ecol.* 27, 107–119.
- 876 DeVries, A.C., Glasper, E.R., Detillion, C.E., 2003. Social modulation of stress responses.
877 *Physiol. Behav.* 79, 399–407.
- 878 Dobson, F.S., 1982. Competition for mates and predominant juvenile male dispersal in
879 mammals. *Anim. Behav.* 30, 1183–1192.
- 880 Dominoni, D., Quetting, M., Partecke, J., 2013. Artificial light at night advances avian
881 reproductive physiology. *Proc. R. Soc. B.* 280, 20123017.
- 882 Dominoni, D.M., Borniger, J.C., Nelson, R.J., 2016. Light at night, clocks and health: from
883 humans to wild organisms. *Biol. Lett.* 12, 20160015.
- 884 Du, J., McEwen, B., Manji, H.K., 2009. Glucocorticoid receptors modulate mitochondrial
885 function. *Comm. Integr. Biol.* 2, 350–352.
- 886 Edwards, P.D., Lavergne, S.G., McCaw, L.K., Wijenayake, S., Boonstra, R., McGowan, P.O.,
887 Holmes, M.M., 2021. Maternal effects in mammals: Broadening our understanding of
888 offspring programming. *Front. Neuroendocrinol.* 62, 100924.
- 889 Ehlers, C.L., Frank, E., Kupfer, D.J., 1988. A unified approach to understanding the etiology
890 of depression. *Social Zeitgebers and Biological Rhythms* 45, 948-952.
- 891 Eisermann, K., 1992. Long-term heart rate responses to social stress in wild European rabbits:
892 Predominant effect of rank position. *Physiol. Behav.* 52, 33–36.
- 893 Entringer, S., Epel, E.S., Kumsta, R., Lin, J., Hellhammer, D.H., Blackburn, E.H., Wust, S.,
894 Wadhwa, P.D., 2011. Stress exposure in intrauterine life is associated with shorter
895 telomere length in young adulthood. *Proc. Natl. Acad. Sci. USA* 108, E513-E518.
- 896 Epel, E.S., Blackburn, E.H., Lin, J., Dhabhar, F.S., Adler, N.E., Morrow, J.D., Cawthon,
897 R.M., 2004. Accelerated telomere shortening in response to life stress. *Proc. Natl.*
898 *Acad. Sci. USA* 101, 17312–17315.
- 899 Epel, E.S., 2009. Psychological and metabolic stress: A recipe for accelerated cellular aging?
900 *Hormones* 8, 7–22.
- 901 Epel, E.S., Lin, J., Dhabhar, F.S., Wolkowitz, O.M., Puterman, E., Karan, L., Blackburn,
902 E.H., 2010. Dynamics of telomerase activity in response to acute psychological stress.
903 *Brain Behav. Immun.* 24, 531–539.
- 904 Fagundes, C.P., Glaser, R., Kiecolt-Glaser, J.K., 2013. Stressful early life experiences and
905 immune dysregulation across the lifespan. *Brain Behav. Immun.* 27, 8–12.
- 906 Faulkes, C.G., Abbott, D.H., 1997. The physiology of a reproductive dictatorship: Regulation
907 of male and female reproduction by a single breeding female in colonies of naked
908 mole-rats, in: French, J.A., Solomon, N.G. (Eds.), *Cooperative breeding in mammals*.
909 Cambridge University Press, Cambridge, pp. 302–334.
- 910 Favreau, A., Richard-Yris, M.-A., Bertin, A., Houdelier, C., Lumineau, S., 2009. Social
911 influences on circadian behavioural rhythms in vertebrates. *Anim. Behav.* 77, 983–
912 989.

- 913 Fernandes, P.A., Tamura, E.K., D'Argenio-Garcia, L., Muxel, S.M., da Silveira Cruz-
914 Machado, S., Marçola, M., Carvalho-Sousa, C.E., Cecon, E., Ferreira, Z.S., Markus,
915 R.P., 2017. Dual effect of catecholamines and corticosterone crosstalk on pineal gland
916 melatonin synthesis. *Neuroendocrinology* 104, 126–134.
- 917 Fialkowski, R., Aufdemberge, P., Wright, V., Dijkstra, P., 2021. Radical change: temporal
918 patterns of oxidative stress during social ascent in a dominance hierarchy. *Behav.*
919 *Ecol. Sociobiol.* 75, 43.
- 920 Finkel, T., Holbrook, N.J., 2000. Oxidants, oxidative stress and the biology of ageing. *Nature*
921 408, 239–247.
- 922 Focke, C.M.B., Iremonger, K.J., 2020. Rhythmicity matters: Circadian and ultradian patterns
923 of HPA axis activity. *Mol. Cell. Endocrinol.* 501, 110652.
- 924 Fox, H.E., White, S.A., Kao, M.H., Fernald, R.D., 1997. Stress and dominance in a social
925 fish. *J. Neurosci.* 17, 6463–6469.
- 926 Funnell, T.R., Fialkowski, R.J., Dijkstra, P.D., 2022. Social dominance does not increase
927 oxidative stress in a female dominance hierarchy of an African cichlid fish. *Ethology*
928 128, 15–25.
- 929 Gersick, A.S., Rubenstein, D.I., 2017. Physiology modulates social flexibility and collective
930 behaviour in equids and other large ungulates. *Phil. Trans. R. Soc. B* 372, 20160241.
- 931 Gesquiere, L.R., Learn, N.H., Simao, M.C.M., Onyango, P.O., Alberts, S.C., Altmann, J.,
932 2011. Life at the top: rank and stress in wild male baboons. *Science* 333, 357–360.
- 933 Goymann, W., Wingfield, J.C., 2004. Allostatic load, social status and stress hormones: the
934 costs of social status matter. *Anim. Behavi.* 67, 591–602.
- 935 Grandin, L.D., Alloy, L.B., Abramson, L.Y., 2006. The social zeitgeber theory, circadian
936 rhythms, and mood disorders: Review and evaluation. *Clin. Psychol. Rev.* 26, 679–
937 694.
- 938 Green, J.A., Halsey, L.G., Wilson, R.P., Frappell, P.B., 2009. Estimating energy expenditure
939 of animals using the accelerometry technique: activity, inactivity and comparison with
940 the heart-rate technique. *J. Exp. Biol.* 212, 471–482.
- 941 Greenwood, P.J., 1980. Mating systems, philopatry and dispersal in birds and mammals.
942 *Anim. Behav.* 28, 1140–1162.
- 943 Grosbellet, E., Zahn, S., Arrivé, M., Dumont, S., Gourmelen, S., Pévet, P., Challet, E.,
944 Criscuolo, F., 2015. Circadian desynchronization triggers premature cellular aging in a
945 diurnal rodent. *FASEB j.* 29, 4794–4803.
- 946 Groscolas, R., Viera, V., Guerin, N., Handrich, Y., Côté, S.D., 2010. Heart rate as a predictor
947 of energy expenditure in undisturbed fasting and incubating penguins. *J. Exp. Biol.*
948 213, 153–160.
- 949 Guibert, F., Richard-Yris, M.-A., Lumineau, S., Kotrschal, K., Guémené, D., Bertin, A.,
950 Möstl, E., Houdelier, C., 2010. Social Instability in Laying Quail: Consequences on
951 Yolk Steroids and Offspring's Phenotype. *PLoS ONE* 5, e14069.
- 952 Gunnar, M.R., Hostinar, C.E., 2015. The social buffering of the hypothalamic–pituitary–
953 adrenocortical axis in humans: Developmental and experiential determinants. *Soc.*
954 *Neurosci.* 10, 479–488.
- 955 Habig, B., Archie, E.A., 2015. Social status, immune response and parasitism in males: a
956 meta-analysis. *Phil. Trans. R. Soc. B* 370, 20140109.
- 957 Habig, B., Doellman, M.M., Woods, K., Olansen, J., Archie, E.A., 2018. Social status and
958 parasitism in male and female vertebrates: a meta-analysis. *Sci. Rep.* 8, 3629.
- 959 Hamilton, D.G., Jones, M.E., Cameron, E.Z., Kerlin, D.H., McCallum, H., Storfer, A.,
960 Hohenlohe, P.A., Hamede, R.K., 2020. Infectious disease and sickness behaviour:
961 tumour progression affects interaction patterns and social network structure in wild
962 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.

- 1012 Hood, W R, Zhang, Y., Mowry, A.V., Hyatt, H.W., Kavazis, A.N., 2018. Life history trade-
1013 offs within the context of mitochondrial hormesis. *Integr. Comp. Biol.* 58, 567–577.
- 1014 Hunter, R.G., Seligsohn, M., Rubin, T.G., Griffiths, B.B., Ozdemir, Y., Pfaff, D.W., Datson,
1015 N.A., McEwen, B.S., 2016. Stress and corticosteroids regulate rat hippocampal
1016 mitochondrial DNA gene expression via the glucocorticoid receptor. *Proc. Natl. Acad.*
1017 *Sci. USA* 113, 9099–9104.
- 1018 Iwasaki, A., Medzhitov, R., 2010. Regulation of adaptive immunity by the innate immune
1019 system. *Science* 327, 291–295.
- 1020 Jasnow, A.M., Huhman, K.L., Bartness, T.J., Demas, G.E., 2002. Short days and exogenous
1021 melatonin increase aggression of male Syrian hamsters (*Mesocricetus auratus*). *Horm.*
1022 *Behav.* 42, 13–20.
- 1023 Jax, E., Müller, I., Börno, S., Borlinghaus, H., Eriksson, G., Fricke, E., Timmermann, B.,
1024 Pendl, H., Fiedler, W., Klein, K., Schreiber, F., Wikelski, M., Magor, K.E., Kraus,
1025 R.H.S., 2021. Health monitoring in birds using bio-loggers and whole blood
1026 transcriptomics. *Sci. Rep.* 11, 10815.
- 1027 Jong, I.C. de, Sgoifo, A., Lambooj, E., Korte, S.M., Blokhuis, H.J., Koolhaas, J.M., 2000.
1028 Effects of social stress on heart rate and heart rate variability in growing pigs. *Can. J.*
1029 *Anim. Sci.* 80, 273–280.
- 1030 Kaiser, S., Sachser, N., 2005. The effects of prenatal social stress on behaviour: mechanisms
1031 and function. *Neurosci. Biobehav. Rev.* 29, 283–294.
- 1032 Kalsbeek, A., van der Spek, R., Lei, J., Endert, E., Buijs, R.M., Fliers, E., 2012. Circadian
1033 rhythms in the hypothalamo–pituitary–adrenal (HPA) axis. *Mol. Cell. Endocrinol.*
1034 349, 20–29.
- 1035 Kamiński, M., Janiszewski, T., Indykiewicz, P., Nowakowski, J.J., Kowalski, J., Dulisz, B.,
1036 Minias, P., 2021. Density-dependence of nestling immune function and physiological
1037 condition in semi-precocial colonial bird: a cross-fostering experiment. *Front. Zool.*
1038 18, 7.
- 1039 Kappeler, P.M., Barrett, L., Blumstein, D.T., Clutton-Brock, T.H., 2013. Constraints and
1040 flexibility in mammalian social behaviour: introduction and synthesis. *Phil. Trans. R.*
1041 *Soc. B* 368, 20120337.
- 1042 Kappeler, P.M., Cremer, S., Nunn, C.L., 2015. Sociality and health: impacts of sociality on
1043 disease susceptibility and transmission in animal and human societies. *Phil. Trans. R.*
1044 *Soc. B* 370, 20140116.
- 1045 Karatsoreos, I.N., Bhagat, S., Bloss, E.B., Morrison, J.H., McEwen, B.S., 2011. Disruption of
1046 circadian clocks has ramifications for metabolism, brain, and behavior. *Proc. Natl.*
1047 *Acad. Sci. USA* 108, 1657–1662.
- 1048 Kawanishi, S., Oikawa, S., 2004. Mechanism of Telomere Shortening by Oxidative Stress.
1049 *Ann. NY Acad. Sci.* 1019, 278–284.
- 1050 Kelly, T.R., Kimball, M.G., Stansberry, K.R., Lattin, C.R., 2020. No, you go first: phenotype
1051 and social context affect house sparrow neophobia. *Biol. Lett.* 16, 20200286.
- 1052 Kirkwood, T.B.L., Kowald, A., 2012. The free-radical theory of ageing - older, wiser and still
1053 alive: Modelling positional effects of the primary targets of ROS reveals new support.
1054 *Bioessays* 34, 692–700.
- 1055 Koch, R.E., Buchanan, K.L., Casagrande, S., Crino, O., Dowling, D.K., Hill, G.E., Hood,
1056 W.R., McKenzie, M., Mariette, M.M., Noble, D.W.A., Pavlova, A., Seebacher, F.,
1057 Sunnucks, P., Udino, E., White, C.R., Salin, K., Stier, A., 2021. Integrating
1058 mitochondrial aerobic metabolism into ecology and evolution. *Trends Ecol. Evol.* 36,
1059 321–332.

- 1060 Kolbe, J.J., Moniz, H.A., Lapiedra, O., Thawley, C.J., 2021. Bright lights, big city: an
 1061 experimental assessment of short-term behavioral and performance effects of artificial
 1062 light at night on *Anolis* lizards. *Urban Ecosyst.* 24, 1035–1045.
- 1063 Kopec, A.M., Smith, C.J., Bilbo, S.D., 2019. Neuro-immune mechanisms regulating social
 1064 behavior: Dopamine as mediator? *Trends Neurosci.* 42, 337–348.
- 1065 Kotrschal, A., Ilmonen, P., Penn, D.J., 2007. Stress impacts telomere dynamics. *Biol. Lett.* 3,
 1066 128–130.
- 1067 Kotrschal, K., Hirschenhauser, K., Möstl, E., 1998. The relationship between social stress and
 1068 dominance is seasonal in greylag geese. *Anim. Behav.* 55, 171–176.
- 1069 Kumar, V., 2002. *Biological rhythms*. Springer Narosa Publishing House, New Delhi.
- 1070 Kuo, T., McQueen, A., Chen, T.-C., Wang, J.-C., 2015. Regulation of glucose homeostasis by
 1071 glucocorticoids, in: Wang, J.-C., Harris, C. (Eds.), *Glucocorticoid signaling, Advances*
 1072 *in experimental medicine and biology*. Springer New York, New York, pp. 99–126.
- 1073 Lambert, C.T., Guillette, L.M., 2021. The impact of environmental and social factors on
 1074 learning abilities: a meta-analysis. *Biol. Rev.* 96, 2871–2889.
- 1075 Lamont, E.W., Legault-Coutu, D., Cermakian, N., Boivin, D.B., 2007. The role of circadian
 1076 clock genes in mental disorders. *Dialogues Clin. Neurosci.* 9, 333–342.
- 1077 Lane, N., 2006. *Power, sex, suicide: mitochondria and the meaning of life*. Oxford University
 1078 Press, Oxford.
- 1079 Lapp, H.E., Bartlett, A.A., Hunter, R.G., 2019. Stress and glucocorticoid receptor regulation
 1080 of mitochondrial gene expression. *J. Mol. Endocrinol.* 62, R121–R128.
- 1081 Lardy, S., Rey, B., Salin, K., Voituron, Y., Cohas, A., 2016. Beneficial effects of group size
 1082 on oxidative balance in a wild cooperative breeder. *Behav. Ecol.* 27, 1820–1825.
- 1083 Larson, E.T., Winberg, S., Mayer, I., Lepage, O., Summers, C.H., Øverli, Ø., 2004. Social
 1084 stress affects circulating melatonin levels in rainbow trout. *Gen. Comp. Endocrinol.*
 1085 136, 322–327.
- 1086 Le, P.P., Friedman, J.R., Schug, J., Brestelli, J.E., Parker, J.B., Bochkis, I.M., Kaestner, K.H.,
 1087 2005. Glucocorticoid Receptor-Dependent Gene Regulatory Networks. *PLOS*
 1088 *Genetics* 1, 16.
- 1089 Leblanc, H., Ramirez, S., 2020. Linking Social Cognition to Learning and Memory. *J.*
 1090 *Neurosci.* 40, 8782–8798.
- 1091 Levins, R., 1968. *Evolution in changing environments: some theoretical explorations*,
 1092 *Monographs in population biology*. Princeton Univ. Pr, Princeton, NJ.
- 1093 Li, H., Xia, N., 2020. The role of oxidative stress in cardiovascular disease caused by social
 1094 isolation and loneliness. *Redox Biol.* 37, 101585.
- 1095 Lin, H.-Y., Muller, Y.A., Hammond, G.L., 2010. Molecular and structural basis of steroid
 1096 hormone binding and release from corticosteroid-binding globulin. *Mol. Cell.*
 1097 *Endocrinol.* 316, 3–12.
- 1098 Lindström, K.M., Hasselquist, D., Wikelski, M., 2005. House sparrows (*Passer domesticus*)
 1099 adjust their social status position to their physiological costs. *Horm. Behav.* 48, 311–
 1100 320.
- 1101 López-Otín, C., Kroemer, G., 2021. Hallmarks of Health. *Cell* 184, 33–63.
- 1102 Love, O.P., McGowan, P.O., Sheriff, M.J., 2013. Maternal adversity and ecological stressors
 1103 in natural populations: the role of stress axis programming in individuals, with
 1104 implications for populations and communities. *Funct. Ecol.* 27, 81–92.
- 1105 Lukkes, J., Watt, M., Lowry, C., Forster, G., 2009. Consequences of post-weaning social
 1106 isolation on anxiety behavior and related neural circuits in rodents. *Front. Behav.*
 1107 *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.

- 1156 Mendonça, R., Vullioud, P., Katlein, N., Vallat, A., Glauser, G., Bennett, N.C., Helfenstein,
1157 F., 2020. Oxidative costs of cooperation in cooperatively breeding Damaraland mole-
1158 rats. *Proc. Roy. Soc. B* 287, 20201023.
- 1159 Metcalfe, N.B., Taylor, A.C., Thorpe, J.E., 1995. Metabolic rate, social status and life-history
1160 strategies in Atlantic salmon. *Anim. Behav.* 49, 431–436.
- 1161 Mikami, K., Kiyokawa, Y., Ishii, A., Takeuchi, Y., 2020. Social buffering enhances
1162 extinction of conditioned fear responses by reducing corticosterone levels in male rats.
1163 *Horm. Behavi.* 118, 104654.
- 1164 Minias, P., Gach, K., Włodarczyk, R., Janiszewski, T., 2019. Colony size affects nestling
1165 immune function: a cross-fostering experiment in a colonial waterbird. *Oecologia* 190,
1166 333–341.
- 1167 Mistlberger, R.E., Skene, D.J., 2004. Social influences on mammalian circadian rhythms:
1168 animal and human studies. *Biol. Rev.* 79, 533–556.
- 1169 Mitchell, C., Hobcraft, J., McLanahan, S.S., Siegel, S.R., Berg, A., Brooks-Gunn, J.,
1170 Garfinkel, I., Notterman, D., 2014. Social disadvantage, genetic sensitivity, and
1171 children’s telomere length. *Proc. Natl. Acad. Sci.* 111, 5944–5949.
- 1172 Modlinska, K., Stryjek, R., Chrzanowska, A., Pisula, W., 2018. Social environment as a factor
1173 affecting exploration and learning in pre-juvenile rats. *Behav. Proc.* 153, 77–83.
- 1174 Monaghan, P., Metcalfe, N.B., Torres, R., 2009. Oxidative stress as a mediator of life history
1175 trade-offs: mechanisms, measurements and interpretation. *Ecol. Lett.* 12, 75–92.
- 1176 Montgomery, T.M., Pendleton, E.L., Smith, J.E., 2018. Physiological mechanisms mediating
1177 patterns of reproductive suppression and alloparental care in cooperatively breeding
1178 carnivores. *Physiol. Behav.* 193, 167–178.
- 1179 Morgan, D.J., Poolman, T.M., Williamson, A.J.K., Wang, Z., Clark, N.R., Ma’ayan, A.,
1180 Whetton, A.D., Brass, A., Matthews, L.C., Ray, D.W., 2016. Glucocorticoid receptor
1181 isoforms direct distinct mitochondrial programs to regulate ATP production. *Sci. Rep.*
1182 6, 26419.
- 1183 Mueller, I., Shakiba, N., Brown, M.A., Crowel, S.E., Conradt, E., 2021. Epigenetic effects of
1184 prenatal stress, in: Wazana, A., Székely, E., Oberlander, T.F. (Eds.), *Prenatal stress
1185 and child development*. Springer International Publishing, Cham, pp. 89–111.
- 1186 Muller, M.N., Wrangham, R.W., 2004. Dominance, cortisol and stress in wild chimpanzees
1187 (*Pan troglodytes schweinfurthii*). *Behav. Ecol. Sociobiol.* 55, 332–340.
- 1188 Munley, K.M., Deyoe, J.E., Ren, C.C., Demas, G.E., 2020. Melatonin mediates seasonal
1189 transitions in aggressive behavior and circulating androgen profiles in male Siberian
1190 hamsters. *Horm. Behav.* 117, 104608.
- 1191 Nation, D.A., Gonzales, J.A., Mendez, A.J., Zaias, J., Szeto, A., Brooks, L.G., Paredes, J.,
1192 D’Angola, A., Schneiderman, N., McCabe, P.M., 2008. The effect of social
1193 environment on markers of vascular oxidative stress and inflammation in the
1194 Watanabe heritable hyperlipidemic rabbit. *Psychosomatic Med.* 70, 269–275.
- 1195 Nephew, B.C., Romero, L.M., 2003. Behavioral, physiological, and endocrine responses of
1196 starlings to acute increases in density. *Horm. Behav.* 44, 222–232.
- 1197 Nettle, D., Monaghan, P., Boner, W., Gillespie, R., Bateson, M., 2013. Bottom of the heap:
1198 having heavier competitors accelerates early-life telomere loss in the european
1199 starling, *Sturnus vulgaris*. *PLoS ONE* 8, e83617.
- 1200 Nettle, D., Monaghan, P., Gillespie, R., Brilot, B., Bedford, T., Bateson, M., 2015. An
1201 experimental demonstration that early-life competitive disadvantage accelerates
1202 telomere loss. *Proc. R. Soc. B.* 282, 20141610.
- 1203 Norman, R.E., Byambaa, M., De, R., Butchart, A., Scott, J., Vos, T., 2012. The long-term
1204 health consequences of child physical abuse, emotional abuse, and neglect: A
1205 systematic review and meta-analysis. *PLoS Medicine* 9, 1001349.

- 1206 Oitzl, M.S., Champagne, D.L., van der Veen, R., de Kloet, E.R., 2010. Brain development
1207 under stress: Hypotheses of glucocorticoid actions revisited. *Neurosci. Biobehav. Rev.*
1208 34, 853–866.
- 1209 Oli, M.K., Armitage, K.B., 2003. Sociality and individual fitness in yellow-bellied marmots:
1210 Insights from a long-term study (1962–2001). *Oecologia* 136, 543–550.
- 1211 Oliveira, B.S., Zunzunegui, M.V., Quinlan, J., Fahmi, H., Tu, M.T., Guerra, R.O., 2016.
1212 Systematic review of the association between chronic social stress and telomere
1213 length: A life course perspective. *Ageing Res. Rev.* 26, 37–52.
- 1214 Ostner, J., Heistermann, M., Schülke, O., 2008. Dominance, aggression and physiological
1215 stress in wild male Assamese macaques (*Macaca assamensis*). *Horm. Behav.* 54, 613–
1216 619.
- 1217 Otten, W., Kanitz, E., Couret, D., Veissier, I., Prunier, A., Merlot, E., 2010. Maternal social
1218 stress during late pregnancy affects hypothalamic-pituitary-adrenal function and brain
1219 neurotransmitter systems in pig offspring. *Domestic Anim. Endocrinol.* 38, 146–156.
- 1220 Patterson, J.E.H., Ruckstuhl, K.E., 2013. Parasite infection and host group size: a meta-
1221 analytical review. *Parasitology* 140, 803–813.
- 1222 Peckett, A.J., Wright, D.C., Riddell, M.C., 2011. The effects of glucocorticoids on adipose
1223 tissue lipid metabolism. *Metabolism* 60, 1500–1510.
- 1224 Perogamvros, I., Ray, D.W., Trainer, P.J., 2012. Regulation of cortisol bioavailability —
1225 effects on hormone measurement and action. *Nat. Rev. Endocrinol.* 8, 717–727.
- 1226 Picard, M., Juster, R.-P., McEwen, B.S., 2014. Mitochondrial allostatic load puts the “gluc”
1227 back in glucocorticoids. *Nat. Rev. Endocrinol.* 10, 303–310.
- 1228 Picard, M., McManus, M.J., Csordás, G., Várnai, P., Dorn II, G.W., Williams, D., Hajnóczky,
1229 G., Wallace, D.C., 2015. Trans-mitochondrial coordination of cristae at regulated
1230 membrane junctions. *Nat. Commun.* 6, 6259.
- 1231 Picard, M., McEwen, B.S., 2018a. Psychological stress and mitochondria: a conceptual
1232 framework. *Psychosom. Med.* 80, 126–140.
- 1233 Picard, M., McEwen, B.S., Epel, E.S., Sandi, C., 2018b. An energetic view of stress: Focus on
1234 mitochondria. *Front. Neuroendocrinol.* 49, 72–85. h
- 1235 Qian, X., Droste, S.K., Gutierrez-Mecinas, M., Collins, A., Kersante, F., Reul, J., Linthorst,
1236 A.C.E., 2011. A rapid release of corticosteroid-binding globulin from the liver
1237 restrains the glucocorticoid hormone response to acute stress. *Endocrinol.* 152, 3738–
1238 3748.
- 1239 Quque, M., Paquet, M., Zahn, S., Théron, F., Faivre, B., Sueur, C., Criscuolo, F., Doutrelant,
1240 C., Covas, R., 2021. Contrasting associations between nestling telomere length and
1241 pre and postnatal helpers’ presence in a cooperatively breeding bird. *Oecologia* 196,
1242 Quque, M., Ferreira, C., Sosa, S., Schull, Q., Zahn, S., Criscuolo, F., Bleu, J., Viblanc, V.A.,
1243 2022. Cascading effects of conspecific aggression on oxidative status and telomere length in
1244 zebra finches. *Physiol. Biochem. Zool.*, in press.
- 1245 Raison, C.L., Capuron, L., Miller, A.H., 2006. Cytokines sing the blues: inflammation and the
1246 pathogenesis of depression. *Trends Immunol.* 27, 24–31.
- 1247 Rao, R., Androulakis, I.P., 2019. The physiological significance of the circadian dynamics of
1248 the HPA axis: Interplay between circadian rhythms, allostasis and stress resilience.
1249 *Horm. Behav.* 110, 77–89.
- 1250 Raulo, A., Dantzer, B., 2018. Associations between glucocorticoids and sociality across a
1251 continuum of vertebrate social behavior. *Ecol. Evol.* 8, 7697–7716.
- 1252 Reichert, S., Stier, A., 2017. Does oxidative stress shorten telomeres *in vivo*? A review. *Biol.*
1253 *Lett.* 13, 20170463.

- 1254 Reid, D., Armstrong, J.D., Metcalfe, N.B., 2011. Estimated standard metabolic rate interacts
1255 with territory quality and density to determine the growth rates of juvenile Atlantic
1256 salmon: SMR, density, territory and growth. *Funct. Ecol.* 25, 1360–1367.
- 1257 Rentscher, K.E., Carroll, J.E., Mitchell, C., 2020. Psychosocial Stressors and Telomere
1258 Length: A Current Review of the Science. *Annu. Rev. Public Health* 41, 223–245.
- 1259 Reyes-Contreras, M., Glauser, G., Rennison, D.J., Taborsky, B., 2019. Early-life
1260 manipulation of cortisol and its receptor alters stress axis programming and social
1261 competence. *Phil. Trans. Roy. Soc. B* 374, 20180119.
- 1262 Ristow, M., Zarse, K., 2010. How increased oxidative stress promotes longevity and
1263 metabolic health: The concept of mitochondrial hormesis (mitohormesis). *Exp.*
1264 *Gerontol.* 45, 410–418.
- 1265 Romano, V., MacIntosh, A.J.J., Sueur, C., 2020. Stemming the flow: Information, infection,
1266 and social evolution. *Trends Ecol. Evol.* 35, 849–853.
- 1267 Romano, V., Sueur, C., MacIntosh, A.J.J., 2021. The tradeoff between information and
1268 pathogen transmission in animal societies. *Oikos* 08290.
- 1269 Romero, L.M., Butler, L.K., 2007. Endocrinology of Stress. *Int. J. Comp. Psychol.* 20(2).
- 1270 Romero, L.M., Dickens, M.J., Cyr, N.E., 2009. The reactive scope model — A new model
1271 integrating homeostasis, allostasis, and stress. *Horm. Behav.* 55, 375–389.
- 1272 Ropert-Coudert, Y., Kato, A., Grémillet, D., Crenner, F., 2012. Bio-logging: recording the
1273 ecophysiology and behaviour of animals moving freely in their environment, in:
1274 *Sensors for Ecology*. pp. 17–41.
- 1275 Rosal, M.C., King, J., Ma, Y., Reed, G.W., 2004. Stress, social support, and cortisol: inverse
1276 associations? *Behav. Med.* 30, 11–22.
- 1277 Roulin, A., Dreiss, A.N., 2012. Sibling competition and cooperation over parental care, in:
1278 *The evolution of parental care*. Oxford University Press.
- 1279 Rusak, B., 1981. Vertebrate behavioral rhythms, in: Aschoff, J. (Ed.), *Biological rhythms*.
1280 Springer US, Boston, MA, pp. 183–213.
- 1281 Rutz, C., Burns, Z.T., James, R., Ismar, S.M.H., Burt, J., Otis, B., Bowen, J., St Clair, J.J.H.,
1282 2012. Automated mapping of social networks in wild birds. *Curr. Biol.* 22, R669–
1283 R671.
- 1284 Sabet, S.M., Dautovich, N.D., Dzierzewski, J.M., 2021. The rhythm is gonna get you: social
1285 rhythms, sleep, depressive, and anxiety symptoms. *J. Affect. Dis.* 286, 197–203.
- 1286 Sánchez-Tójar, A., Lagisz, M., Moran, N.P., Nakagawa, S., Noble, D.W.A., Reinhold, K.,
1287 2020. The jury is still out regarding the generality of adaptive ‘transgenerational’
1288 effects. *Ecol. Lett.* 23, 1715–1718.
- 1289 Sapolsky, R.M., Ray, J.C., 1989. Styles of dominance and their endocrine correlates among
1290 wild olive baboons (*Papio anubis*) *Am. J. Primatology* 18:1-13
- 1291 Sapolsky, R.M., Romero, L.M., Munck, A.U., 2000. How do glucocorticoids influence stress
1292 responses? Integrating permissive, suppressive, stimulatory, and preparative actions.
1293 *Endocr. Rev.* 21, 55–89.
- 1294 Sapolsky, R.M., 2004. Social status and health in humans and other animals. *Ann. Rev.*
1295 *Anthropol.* 33, 393–418.
- 1296 Sapolsky, R.M., 2005. The influence of social hierarchy on primate health. *Science* 308, 648–
1297 652.
- 1298 Schiavone, S., Sorce, S., Dubois-Dauphin, M., Jaquet, V., Colaianna, M., Zotti, M., Cuomo,
1299 V., Trabace, L., Krause, K.-H., 2009. Involvement of NOX2 in the development of
1300 behavioral and pathologic alterations in isolated rats. *Biol. Psychiatry* 66, 384–392.
- 1301 Schmid-Hempel, P., 2017. Parasites and their social hosts. *Trends Parasitol.* 33, 453–462.

- 1302 Schoech, S.J., Mumme, R.L., Moore, M.C., 1991. Reproductive endocrinology and
 1303 mechanisms of breeding inhibition in cooperatively breeding Florida scrub jays
 1304 (*Aphelocoma C. Coerulescens*). *The Condor* 93, 354–364.
- 1305 Schoech, S.J., Romero, L.M., Moore, I.T., Bonier, F., 2013. Constraints, concerns and
 1306 considerations about the necessity of estimating free glucocorticoid concentrations for
 1307 field endocrine studies. *Funct. Ecol.* 27, 1100–1106.
- 1308 Schradin, C., 2013. Intraspecific variation in social organization by genetic variation,
 1309 developmental plasticity, social flexibility or entirely extrinsic factors. *Phil. Trans. R.*
 1310 *Soc. B* 368, 20120346.
- 1311 Secondi, J., Mondy, N., Gippet, J.M.W., Touzot, M., Gardette, V., Guillard, L., Lengagne, T.,
 1312 2021. Artificial light at night alters activity, body mass, and corticosterone level in a
 1313 tropical anuran. *Behav. Ecol.* 32, 932–940.
- 1314 Sephton, S., Spiegel, D., 2003. Circadian disruption in cancer: a neuroendocrine-immune
 1315 pathway from stress to disease? *Brain, Behav. Immun.* 17, 321–328.
- 1316 Sgoifo, A., Papi, F., 1995. Effects of social and non-social acute stressors on plasma levels of
 1317 catecholamines and corticosterone in wild rats. *Rend. Fis. Acc. Lincei* 6, 289.
- 1318 Sgoifo, A., De Boer, S.F., Haller, J., Koolhaas, J.M., 1996. Individual differences in plasma
 1319 catecholamine and corticosterone stress responses of wild-type rats: relationship with
 1320 aggression. *Physiol. Behav.* 60, 1403–1407.
- 1321 Sgoifo, A., Koolhaas, J., De Boer, S., Musso, E., Stilli, D., Buwalda, B., Meerlo, P., 1999.
 1322 Social stress, autonomic neural activation, and cardiac activity in rats. *Neurosci.*
 1323 *Biobehav. Rev.* 23, 915–923.
- 1324 Sharp, S.P., Clutton-Brock, T.H., 2011. Competition, breeding success and ageing rates in
 1325 female meerkats: Competition and senescence in meerkats. *J. Evol. Biol.* 24, 1756–
 1326 1762.
- 1327 Shen, G.H., Alloy, L.B., Abramson, L.Y., Sylvia, L.G., 2008. Social rhythm regularity and
 1328 the onset of affective episodes in bipolar spectrum individuals. *Bipolar Disorders* 10,
 1329 520–529.
- 1330 Sheriff, M.J., Krebs, C.J., Boonstra, R., 2010. The ghosts of predators past: population cycles
 1331 and the role of maternal programming under fluctuating predation risk. *Ecology* 91,
 1332 2983–2994.
- 1333 Sheriff, M.J., Love, O.P., 2013. Determining the adaptive potential of maternal stress. *Ecol.*
 1334 *Lett.* 16, 271–280.
- 1335 Sheriff, M.J., Bell, A., Boonstra, R., Dantzer, B., Lavergne, S.G., McGhee, K.E., MacLeod,
 1336 K.J., Winandy, L., Zimmer, C., Love, O.P., 2017. Integrating ecological and
 1337 evolutionary context in the study of maternal stress. *Integr. Comp. Biol.* 57, 437–449.
- 1338 Shilts, J., Chen, G., Hughey, J.J., 2018. Evidence for widespread dysregulation of circadian
 1339 clock progression in human cancer. *PeerJ* 6, e4327.
- 1340 Siiteri, P.K., Murai, J.T., Hammond, G.L., Nisker, J.A., Raymoure, W.J., Kuhn, R.W., 1982.
 1341 The serum transport of steroid-hormones. *Recent Prog. Horm. Res.* 38, 457-503
- 1342 Silk, J.B., 2007. Social components of fitness in primate groups. *Science* 317, 1347–1351.
- 1343 Silva, L.R., Lardy, S., Ferreira, A.C., Rey, B., Doutrelant, C., Covas, R., 2018. Females pay
 1344 the oxidative cost of dominance in a highly social bird. *Anim. Behav.* 144, 135–146.
- 1345 Sloman, K.A., Motherwell, G., O'Connor, K.I., Taylor, A.C., 2000. The effect of social stress
 1346 on the standard metabolic rate (SMR) of brown trout, *Salmo trutta*. *Fish Physiol.*
 1347 *Biochem.* 23, 49–53.
- 1348 Smith, J.E., Pinter-Wollman, N., 2021. Observing the unwatchable: Integrating automated
 1349 sensing, naturalistic observations and animal social network analysis in the age of big
 1350 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., Gaydos, 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., Hausmann, 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., Donazar, 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.

- 1400 Teo, A.R., Lerrigo, R., Rogers, M.A.M., 2013. The role of social isolation in social anxiety
1401 disorder: A systematic review and meta-analysis. *J. Anx. Dis.* 27, 353–364.
- 1402 Tertilt, M., Skupio, U., Barut, J., Dubovyk, V., Wawrzczak-Bargiela, A., Soltys, Z., Golda, S.,
1403 Kudla, L., Wiktorowska, L., Szklarczyk, K., Korostynski, M., Przewlocki, R., Slezak,
1404 M., 2018. Glucocorticoid receptor signaling in astrocytes is required for aversive
1405 memory formation. *Transl. Psychiatry* 8, 1–11.
- 1406 Turner-Cobb, J.M., Sephton, S.E., Koopman, C., Blake-Mortimer, J., Spiegel, D., 2000.
1407 Social support and salivary cortisol in women with metastatic breast cancer.
1408 *Psychosom. Med.* 62, 337–345.
- 1409 Uchino, B.N., 2004. Social support and physical health: Understanding the health
1410 consequences of relationships. Yale University Press.
- 1411
- 1412 Uchino, B.N., 2006. Social support and health: a review of physiological processes potentially
1413 underlying links to disease outcomes. *J. Behav. Med.* 29, 377–387.
- 1414 Uchino, B.N., Cawthon, R.M., Smith, T.W., Light, K.C., McKenzie, J., Carlisle, M., Gunn,
1415 H., Birmingham, W., Bowen, K., 2012. Social relationships and health: is feeling
1416 positive, negative, or both (ambivalent) about your social ties related to telomeres?
1417 *Health Psychol.* 31, 789–796.
- 1418 van Lieshout, S.H.J., Badás, E.P., Bright Ross, J.G., Bretman, A., Newman, C., Buesching,
1419 C.D., Burke, T., Macdonald, D.W., Dugdale, H.L., 2021. Early-life seasonal, weather
1420 and social effects on telomere length in a wild mammal. *Mol. Ecol. mec.* 16014.
- 1421 Venney, C.J., Love, O.P., Drown, E.J., Heath, D.D., 2020. DNA Methylation Profiles Suggest
1422 Intergenerational Transfer of Maternal Effects. *Mol. Biol. Evol.* 37, 540–548.
- 1423 Viblanc, V.A., Valette, V., Kauffmann, M., Malosse, N., Groscolas, R., 2012. Coping with
1424 social stress: heart rate responses to agonistic interactions in king penguins. *Behav.*
1425 *Ecol.* 23, 1178–1185.
- 1426 Viblanc, V.A., Gineste, B., Stier, A., Robin, J.-P., Groscolas, R., 2014a. Stress hormones in
1427 relation to breeding status and territory location in colonial king penguin: a role for
1428 social density? *Oecologia* 175, 763–772.
- 1429 Viblanc, V.A., Saraux, C., Malosse, N., Groscolas, R., 2014b. Energetic adjustments in freely
1430 breeding-fasting king penguins: does colony density matter? *Funct. Ecol.* 28, 621–631.
- 1431 Viera, V.M., Viblanc, V.A., Filippi-Codaccioni, O., Côté, S.D., Groscolas, R., 2011. Active
1432 territory defence at a low energy cost in a colonial seabird. *Anim. Behav.* 82, 69–76.
- 1433 von Zglinicki, T., 2002. Oxidative stress shortens telomeres. *Trends Biochem. Sci.* 27, 339–
1434 344.
- 1435 Vyssotski, A.L., Serkov, A.N., Itskov, P.M., Dell’Omo, G., Latanov, A.V., Wolfer, D.P.,
1436 Lipp, H.-P., 2006. Miniature neurologgers for flying pigeons: multichannel EEG and
1437 action and field potentials in combination with GPS recording. *J. Neurophysiol.* 95,
1438 1263–1273.
- 1439 Wascher, C.A.F., Arnold, W., Kotrschal, K., 2008. Heart rate modulation by social contexts
1440 in greylag geese (*Anser anser*). *J. Comp. Psychol.* 122, 100–107.
- 1441 Wascher, C.A.F., Scheiber, I.B.R., Weiß, B.M., Kotrschal, K., 2009. Heart rate responses to
1442 agonistic encounters in greylag geese, *Anser anser*. *Anim. Behav.* 77, 955–961.
- 1443 Waser, P.M., 1985. Does competition drive dispersal? *Ecology* 66, 1170–1175.
- 1444 Weber, K., Brück, P., Mikes, Z., Küpper, J.-H., Klingenspor, M., Wiesner, R.J., 2002.
1445 Glucocorticoid hormone stimulates mitochondrial biogenesis specifically in skeletal
1446 muscle. *Endocrinology* 143, 177–184.
- 1447 Wells, K.D., Taigen, T.L., 1986. The effect of social interactions on calling energetics in the
1448 gray treefrog (*Hyla versicolor*). *Behav. Ecol. Sociobiol.* 19, 9–18.

1449 White, D.J., Gersick, A.S., Snyder-Mackler, N., 2012. Social networks and the development
1450 of social skills in cowbirds. *Phil. Trans. Roy. Soc. B* 367, 1892–1900.

1451 Wilmers, C.C., Nickel, B., Bryce, C.M., Smith, J.A., Wheat, R.E., Yovovich, V., 2015. The
1452 golden age of bio-logging: how animal-borne sensors are advancing the frontiers of
1453 ecology. *Ecology* 96, 1741–1753.

1454 Wilson, E.O., 1975. *Sociobiology: The new synthesis*. Harvard University Press.

1455 Wood, E.M., Capilla-Lasheras, P., Cram, D.L., Walker, L.A., York, J.E., Lange, A.,
1456 Hamilton, P.B., Tyler, C.R., Young, A.J., 2021. Social dominance and rainfall predict
1457 telomere dynamics in a cooperative arid-zone bird. *Mol. Ecol. mec.*15868.

1458 Yin, J., Zhou, M., Lin, Z., Li, Q.Q., Zhang, Y., 2019. Transgenerational effects benefit
1459 offspring across diverse environments: a meta-analysis in plants and animals. *Ecol.*
1460 *Lett.* 22, 1976–1986.

1461 Young, A.J., Carlson, A.A., Monfort, S.L., Russell, A.F., Bennett, N.C., Clutton-Brock, T.,
1462 2006. Stress and the suppression of subordinate reproduction in cooperatively
1463 breeding meerkats. *Proc. Natl. Acad. Sci. USA* 103, 12005–12010.

1464 Zane, L., Ensminger, D.C., Vázquez-Medina, J.P., 2021. Short-term elevations in
1465 glucocorticoids do not alter telomere lengths: A systematic review and meta-analysis of non-
1466 primate vertebrate studies. *PLoS ONE* 16(10), e0257370.

1467 Zhang, Y., Wong, H.S., 2021. Are mitochondria the main contributor of reactive oxygen
1468 species in cells? *J. Exp. Biol.* 224, jeb221606.

1469 Zhang, Y., Yin, J., Zhou, M., Lin, Z., Li, Q.Q., 2020. Adaptive transgenerational effects
1470 remain significant. *Ecol. Lett.* 23, 1719–1720.

1471 Ziegler, A.-K., Watson, H., Hegemann, A., Meitern, R., Canoine, V., Nilsson, J.-Å., Isaksson,
1472 C., 2021. Exposure to artificial light at night alters innate immune response in wild
1473 great tit nestlings. *J. Exp. Biol.* 224, jeb239350.

1474

1475

1476

1477 **FIGURE CAPTIONS**

1478 **Figure 1. A comprehensive view of the effects of the social environment on key biological**
1479 **functions related to vertebrate health.** Inputs from the social environment (or social stimuli)
1480 are integrated in the brain as negative or positive events, and this integration leads to
1481 modifications in key physiological functions including metabolism, oxidative stress, ageing,
1482 the rhythmicity of biological functions, immune responses, and brain function and
1483 psychological states. These functions can be directly affected by social stimuli, or indirectly,
1484 orchestrated by the stress axes including the sympathetic-adrenal-medullary (SAM) axis and
1485 the Hypothalamic-Pituitary-Adrenal (HPA) axis (hypothalamic pituitary inter-renal HPI in
1486 fish). Note that cross-talks between biological functions, either via or independently, of the
1487 stress axes, make an integration of functions necessary to properly understand the effects of
1488 social environments on vertebrate health.

1489

1490

1491 **TABLES**

1492

1493 **Table 1.** Summary of the known effects of the social environment on key markers of vertebrates

1494 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	Increase in 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

1495

1496 **Footnote :** ¹Nephew et al., 2003; ²Viera et al., 2011; ³Metcalf et al., 1995; ⁴Sloman et al., 2000; ⁵Marler et al.,
 1497 1995; ⁶Wells et al., 1986 ; ⁷Viblanco et al., 2014b ; ⁸Hollis et al., 2015 ; ⁹Beaulieu et al., 2014 ; ¹⁰Border et al.,
 1498 2019 ; ¹¹Funnel et al., 2022 ; ¹²Silva et al., 2018 ; ¹³Colaïanna et al., 2013 ; ¹⁴Lardy et al., 2016 ; ¹⁵Kotrschal et al.,
 1499 2007 ; ¹⁶Epel et al., 2004 ; ¹⁷Nettle et al., 2013 ; ¹⁸Anderson et al., 2021 ; ¹⁹Sharp & Clutton-Brock 2011 ; ²⁰Quque
 1500 et al., 2021 ; ²¹Bauer et al., 2015 ; ²²Hawley et al. ; 2006 ; ²³Minias et al., 2019 ; ²⁴Tella et al., 2001 ; ²⁵Kamiński
 1501 et al., 2021; ²⁶Habig et al., 2018 ; ²⁷Raulo & Dantzer, 2018 ; ²⁸Hamilton et al., 2020 ; ²⁹Dantzer & Kelley, 2007 ;
 1502 ³⁰Kopec et al., 2019 ; ³¹Blanchard et al., 2001 ; ³²Lukkes et al., 2009 ; ³³Teo et al. ,2013 ; ³⁴Menard et al., 2017 ;
 1503 ³⁵Tertil et al., 2018 ; ³⁶Reyes-Contreras et al., 2019 ; ³⁷Heinen et al., 2021 ; ³⁸Leblanc & Ramirez, 2020 ;
 1504 ³⁹Taborsky, 2016 ; ⁴⁰Favreau et al., 2016 ; ⁴¹Heinzeller et al., 1988 ; ⁴²Larson et al., 2004

1505

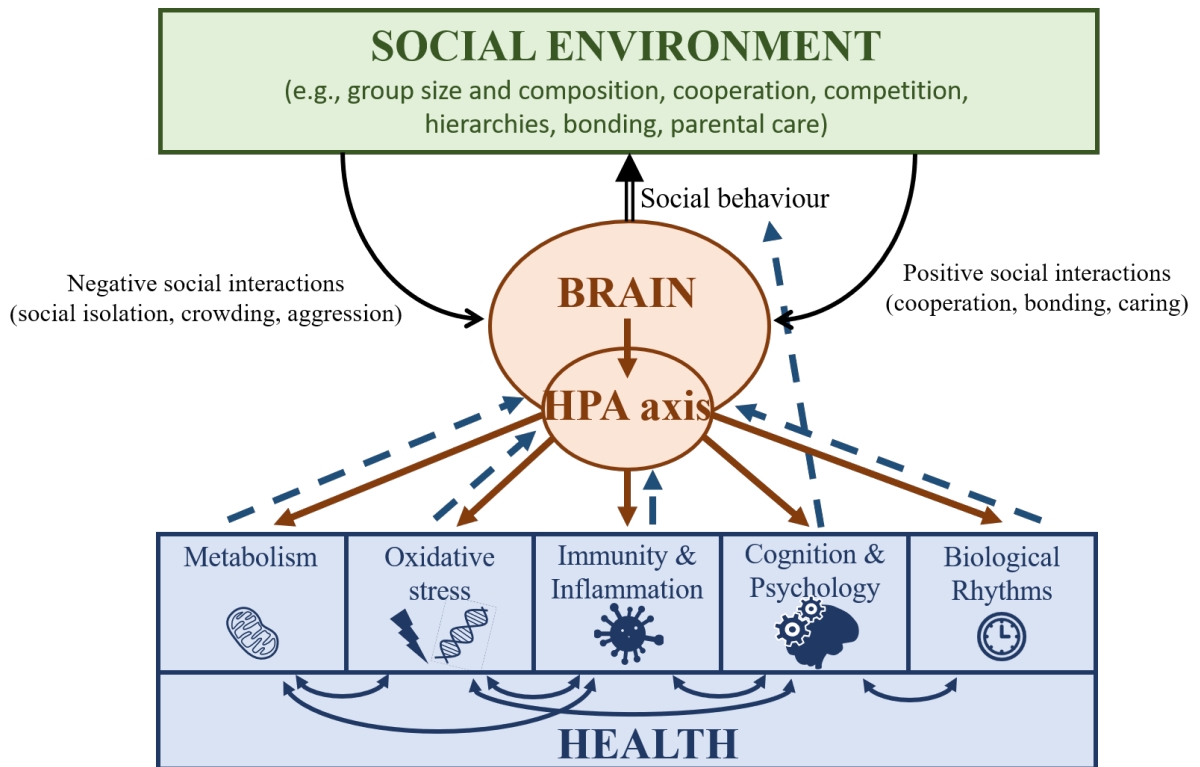
1506

1507

1508

1509 **FIGURES**

1510



1511

1512 **Figure 1.**

1513