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## **Glucocorticoids negatively relate to body mass on the short-term in a free-ranging ungulate**

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## 1 **Abstract**

2 Environmental fluctuations force animals to adjust glucocorticoids (GCs) secretion and  
3 release to current conditions. GCs are a widely used proxy of an individual stress level. While  
4 short-term elevation in GCs is arguably beneficial for fitness components, previous studies  
5 have documented that the relationship between long-term baseline GCs elevation and fitness  
6 components can vary according to ecological and individual factors and according to the life-  
7 history of the species studied. Using longitudinal data on roe deer (*Capreolus capreolus*) from  
8 two populations facing markedly different environmental contexts, we tested whether baseline  
9 GC levels negatively correlate with body mass – a trait positively associated with  
10 demographic individual performance – on the short- to long-term. In support, higher baseline  
11 GC concentrations were associated to lighter body mass, both measured during the same  
12 capture event, in adults of both populations. Overall, we showed that despite the marked  
13 environmental and demographic differences between populations and despite the between-sex  
14 differences in life history (*i.e.* reproductive tactics), the relationship between body mass and  
15 GCs is consistent across environmental contexts, but might differ according to the life history  
16 stage of an individual. This work opens promising perspectives to further explore the  
17 relationship between GC and fitness-related traits according to life history stages in free-  
18 ranging mammals across seasonal and environmental contexts. The timing and context-  
19 dependence of GC levels highlight the complexity of studying stress responses in the wild.

20 **Keywords:** stress response, body condition, Faecal Cortisol Metabolites, individual  
21 performance, ungulates

## 22 **Introduction**

23 Throughout their life, individuals have to adjust their morphology, physiology and/or  
24 behaviour to cope with environmental variation. Part of this variation is predictable as  
25 individuals can rely on environmental cues (*e.g.* photoperiod or temperature at the daily or  
26 seasonal scales) to anticipate future conditions (Wingfield 2003, 2008). Whereas predictable  
27 variation is not considered as stressful *per se* (*i.e.* eliciting a stress response), an unpredictable  
28 perturbation can disrupt life cycles and trigger a stress response through the activation of the  
29 hypothalamic-pituitary-adrenal (HPA) axis and the release of glucocorticoids (GCs),  
30 departing them from the baseline level (Reeder and Kramer 2005). The primary role of these  
31 hormones (namely cortisol and corticosterone) is to maintain an organism's energy balance  
32 according to its requirements in its current environment (Wingfield 2013, Hau et al. 2016),  
33 meaning that GCs promote allostasis (*i.e.* achieving stability through change) by ensuring  
34 energy homeostasis despite predictable and unpredictable environmental and life history  
35 variation (McEwen and Wingfield 2003, 2010, Romero et al. 2009). On the short-term, an  
36 elevation of GC concentration in an individual facing a challenging situation is beneficial  
37 because it promotes processes enhancing survival through reallocation of stored energy from  
38 non-immediately essential functions (*e.g.* growth, reproduction) towards cardiovascular  
39 functions or locomotion and foraging activities (Sapolsky et al. 2000). After a temporary  
40 perturbation, the GC concentration often quickly returns to the baseline level (Reeder and  
41 Kramer 2005). However, baseline GC concentration may remain elevated on the long-term  
42 (*i.e.* days to weeks) as a result from exposure to repeated or long-lasting perturbations such as  
43 prolonged inclement weather or food shortage (McEwen and Wingfield 2010, Hau et al.  
44 2016). Such elevated GC concentration in the long-term can have adverse consequences on  
45 individual performance through disruption of the immune function (Dhabhar 2014), inhibition  
46 of growth, or decreased body condition (Reeder and Kramer 2005), with an intensity

47 depending on the species (Boonstra 2013). Indeed, long-term GC elevation can cause  
48 ‘allostatic overload’ (*i.e.* when the energy intake is lower than the energy required to support  
49 daily and seasonal activities and to cope with unpredictable perturbations, McEwen and  
50 Wingfield 2010). Although variations in GC concentration does not reflect only stress  
51 (MacDougall-Shackleton et al. 2019), GC levels are a widely used proxy for evaluating an  
52 individual physiological stress response (Palme 2019).

53 While many studies analysed the effect of an increase of GC beyond the baseline level (*i.e.*  
54 acute stress-induced GC concentration) on individual performance, fewer have considered  
55 how GCs at baseline concentration (*i.e.* homeostatic levels of hormones) affect the  
56 performance of animals in the wild. Results from these rare studies remain equivocal  
57 (Rogovin et al. 2003, 2008, Altmann et al. 2004, Ethan Pride 2005, Pauli and Buskirk 2007,  
58 Cabezas et al. 2007, Pedersen and Greives 2008, Pinho et al. 2019), which could be explained  
59 by the complex influence of environmental conditions (Henderson et al. 2017). When  
60 resources are limited, increased energy demands can be detrimental by resulting in high levels  
61 of GC and allostatic overload. Nevertheless, this detrimental effect may not be observed when  
62 resources are abundant, as high energy input can contribute reducing allostatic load (McEwen  
63 and Wingfield 2003, 2010, Romero et al. 2009). Food availability (*i.e.* energy availability) or  
64 behavioural flexibility stimulating foraging activity can thus compensate for elevated GC  
65 levels and associated higher energy requirements (Henderson et al. 2017). Animals inhabiting  
66 suboptimal habitats (*e.g.* exposed to human disturbance, predation, intra- or interspecific  
67 competition or pathogens) experience continuous stressful conditions and generally show a  
68 higher GC concentration compared to conspecifics inhabiting optimal areas (Dantzer et al.  
69 2013, 2014, Formenti et al. 2018, Boudreau et al. 2019, Dulude-de Broin et al. 2020, Carbillet  
70 et al. 2020).

71 Additionally, there is evidence that the direction of the relationship between GCs and  
72 performance can also depend on individual attributes, such as body condition, sex or  
73 reproductive status and reproductive tactics (Tilbrook et al. 2000, Ricklefs and Wikelski 2002,  
74 Wey et al. 2015, Blumstein et al. 2016, Vuarin et al. 2019). Under high energy demands  
75 triggered by elevated GC concentration, high quality individuals are more likely to prevent  
76 themselves from reaching allostatic overload and may perform better than poor quality ones.  
77 In African striped mice (*Rhabdomys pumilio*), elevated baseline GC concentration was related  
78 to the selective disappearance of light individuals (Vuarin et al. 2019). Since the relationship  
79 between GCs and performance is complex and involves individual attributes and  
80 environmental context, both sources of variation have to be investigated to disentangle their  
81 respective roles (Crespi et al. 2013, Dantzer et al. 2014).

82 Focusing on body mass provides a relevant approach to investigate the detrimental effects of  
83 prolonged exposure to a high GC concentration. Indeed, body mass is often positively linked  
84 to fitness components (*e.g.* Gaillard et al., 2000; Ronget et al., 2018) and is a trait easily  
85 measured in the field that constitutes a reliable proxy of body condition in many species,  
86 especially income breeders (Hewison et al. 1996, Andersen et al. 1998). High levels of GCs  
87 are known to have a crucial role on body mass by increasing protein and amino-acid  
88 catabolism and mobilising lipids (promoting gluconeogenesis), consequently decreasing body  
89 mass (Boonstra et al. 1998, Dallman et al. 1999, Hodges et al. 2006, Rabasa and Dickson  
90 2016).

91 However, the relationship between baseline GC concentration and body mass is complex.  
92 Indeed, within seasonal baseline levels, GCs actually result in increased feeding behaviours  
93 potentially promoting fat storing and body mass gain, as opposed to the previously-mentioned  
94 GC actions on protein and amino-acid catabolism and lipid mobilisation (Landys et al. 2006).  
95 Case studies have shown that the relationship between baseline GCs and body mass or

96 condition can be positive, negative or null (*e.g.* George et al. 2014, Wey et al. 2015, Hennin et  
97 al. 2016, Boudreau et al. 2019). For instance, low body mass and poor body condition were  
98 associated with higher baseline cortisol concentrations in Eurasian badgers (*Meles meles*,  
99 George et al., 2014). On the other hand, experimental increase in GC concentration through  
100 predation risk manipulation resulted in no changes in body condition in snowshoe hares  
101 (*Lepus americanus*, Boudreau et al., 2019). Thus, more exploratory research are needed in  
102 natural settings to better understand which factors can modulate this relationship.

103 In this exploratory study, we aimed to contribute to a better understanding of how high  
104 baseline GC concentration relates to body mass in roe deer (*Capreolus capreolus*), an income  
105 breeder for which body mass is a good proxy for body condition (Hewison et al. 1996,  
106 Andersen et al. 2000, Pettorelli et al. 2006). We took advantage of the detailed and intensive  
107 monitoring data collected through capture-mark-recapture for more than a decade at the  
108 individual level in two populations subject to markedly different ecological contexts (*i.e.* one  
109 inhabiting a good quality habitat and one population being strongly food-limited; Pettorelli et  
110 al., 2006). To avoid measuring the capture-induced GC response (Romero & Reed, 2005),  
111 longer-term, indirect measures of GC concentration can be obtained from biological matrices  
112 other than blood (Sheriff et al. 2011). Indeed, blood GC concentration is a point in time  
113 measure and circulating GCs concentration typically increases drastically within 3 minutes  
114 following an individual's capture (Sapolsky et al. 2000, Romero and Reed 2005), which  
115 makes it difficult to obtain reliable measures of the HPA baseline activity in free-ranging  
116 animals (but see Sheriff et al. 2011, Lavergne et al. 2021). In this context, levels of faecal  
117 glucocorticoid metabolites (FGMs) are particularly relevant as they represent an integrative  
118 measure of GC concentration several hours before the capture event (*i.e.* baseline stress;  
119 Palme, 2019). Thus, we measured FGMs, as arguably representative of baseline

120 adrenocortical activity over a few hours, with a time delay of 12h on average in roe deer  
121 (ranging from 6 to 23 hours, Dehnhard et al. 2001).

122 As chronic stress can have both immediate and long-term consequences on fitness  
123 components (Monaghan and Hausmann 2015), we tested the immediate relationship between  
124 concomitant body mass and FGMs (*i.e.* short-term relationship), the relationship between  
125 FGMs in a given year and the change of body mass between two consecutive years (*i.e.*  
126 medium-term relationship) and the relationship between FGM measured on juveniles (*i.e.*  
127 individuals in their first year of life) and body mass during the prime-age and senescent life  
128 stages (*i.e.* long-term relationship). We first analysed the relationship between FGMs and  
129 body mass measured at the same capture event. We expected individuals with higher FGM  
130 concentration to be lighter than individuals with low FGM concentration. As roe deer are  
131 income breeders that do not store fat (Hewison et al. 1996, Andersen et al. 1998), we expected  
132 the catabolic actions of GCs to directly impact body mass and condition and to overcome the  
133 effects of increased feeding behaviours promoted by GCs. Then, since chronically elevated  
134 FGMs are expected to be deleterious on the medium to long-term, we tested whether FGMs  
135 measured at a given capture influenced body mass changes across lifespan. Thus, we first  
136 assessed the relationship between FGMs measured in a given year and a given body mass  
137 change between two consecutive years, separately for growing individuals and prime-aged  
138 adults having reached their full mass (*i.e.* between 4 and 10 years old; Douhard et al., 2017).  
139 We expected that higher FGM concentration should result in a smaller mass gain for growing  
140 individuals, and in a decrease in body mass for prime-aged adults, between two consecutive  
141 years, as chronic stress can be linked to inhibited growth or decreased body condition (Reeder  
142 and Kramer 2005). Finally, we tested for the effect of the stress experienced during the first  
143 year of life on the individual body mass relative to the population and sex at a given age, from  
144 the second year of life onwards. We predicted that individuals with higher FGMs during the



145 first year of life should be lighter later in life, as late-life performance in roe deer is affected  
146 by early-life environmental conditions (Gaillard et al., 2003). Lastly, in all analyses we  
147 expected the negative relationship between FGMs and body mass to be steeper in the food-  
148 limited population due to higher allostatic load (McEwen and Wingfield 2003, Henderson et  
149 al. 2017), and we expected sexes to respond differently to higher FGMs levels due to the  
150 different reproductive tactics of males and females (Ricklefs and Wikelski 2002).

## 151 **Materials and Method**

### 152 **Study population and sample collection**

153 We studied roe deer in two populations inhabiting closed forests at Trois-Fontaines (TF –  
154 1360 ha) in north-eastern France (48°43'N, 4°55'E) and at Chizé (CH – 2614 ha) in western  
155 France (46°05'N, 0°25'W). Roe deer are medium-sized ungulates, weighing around 25 kg and  
156 common in lowland woodlands throughout most of Europe. Roe deer display weak sexual  
157 selection, with adult males only 10% heavier than females and party size less than three  
158 females per buck. They are income breeders (Andersen et al. 2000), and to meet the markedly  
159 increased energy needs during the late gestation and early lactation periods, adult females rely  
160 on food resources rather than body reserves. Both sexes allocate high energy expenditure to  
161 reproduction. Females produce twins every year from 2 to 12 years old (Andersen et al.  
162 1998), and males allocate heavily in territory defence for 5-6 months during the rut period  
163 (Johansson 1996). The TF population is characterised by a homogeneous habitat of good  
164 quality on a broad spatial scale, with high forest productivity due to rich soils. On the other  
165 hand, the CH population inhabits a less suitable heterogeneous habitat comprising three types  
166 of varying quality (Pettorelli et al. 2001), with low forest productivity due to poor soils and  
167 frequent summer droughts (Pettorelli et al. 2006). In both study sites, large carnivores are  
168 absent, but hunting occurs occasionally to control population growth. Since 1975 in TF and

169 1977 in CH, a Capture-Mark-Recapture (CMR) program has taken place every winter, and  
170 CMR analyses show that both populations were quite below carrying capacity during the  
171 present study period (2010-2021 in TF, 2013-2021 in CH, unpublished data). Captures take  
172 place during 10-12 days each year, which are spread across December (at TF) or January (at  
173 CH) and March (Gaillard et al. 1993) and consists of drive-netting captures with 30-100  
174 beaters pushing individuals towards nets surrounding specific areas of the forests. Given that  
175 captures took place each year over 3 months, we might expect endocrine activity to vary  
176 throughout the capture season according to environmental conditions (*i.e.* populations) and  
177 sex (Dantzer et al. 2010, Sheriff et al. 2012). Therefore, we tested whether FGM levels varied  
178 as a function of the Julian date, population and sex and found that FGMs increased throughout  
179 the season in a population-specific manner (Supporting Information SI). Thus, all FGM  
180 measurements were standardised for the median Julian date of capture (9<sup>th</sup> of February)  
181 specifically for each population. Successive capture events within a capture season may also  
182 have consequences on FGM measurements during the following captures. However, most  
183 capture days were more than 48 hours apart while in roe deer, FGMs peak from 6 to 23 hours  
184 after an ATCH challenge and return to baseline levels between 28 and 31 hours after  
185 treatment (Dehnhard et al. 2001). In some cases, captures took place during two consecutive  
186 days, but were then conducted on opposite areas of the forest to minimise disturbances.  
187 Likewise, morning and afternoon captures of a given day took place in different areas within  
188 both forests. We thus considered that FGM levels were not influenced by previous captures.  
189 Concerning the potential impact of the capture event itself, since roe deer were captured by  
190 drive-netting and most animal manipulations occurred between 1 and 4 hours after capture,  
191 we tested for a possible impact of this delay on FGM levels. We did not detect any increase or  
192 decrease in FGMs (log-transformed) along with the time between capture and sampling  
193 (linear regression accounting for repeated measurements of a given individual:  $0.006 \text{ hour}^{-1} \pm$

194 0.01 SE,  $t = 0.44$ ,  $p = 0.7$ ), even when the delay was  $> 6h$  (mean FGMs 6h onwards after  
195 capture compared to mean FGMs measured within 6h following capture, accounting for  
196 repeated measurement of a given individual,  $-0.06 \pm 0.06$  SE,  $t = -1.05$ ,  $p = 0.3$ ) (Dehnhard et  
197 al. 2001). Individuals of known age (*i.e.* captured within their first year of life) were weighed,  
198 and faecal matter has been collected since 2010. During the capture period, diet is similar and  
199 mostly composed of brambles (*Rubus* sp.) and ivy (*Hedera helix*) in the two deciduous forests  
200 (Tixier and Duncan 1996), so FGM measurements between populations should not be biased  
201 according to diet composition. During this period, females can be gestating, and differences in  
202 female reproductive status could result in FGM variability (Brunton et al. 2008, Dantzer et al.  
203 2010). Nevertheless, we did not account for females' reproductive status in our models, since  
204 i) we have information about reproductive status (*i.e.* pregnant or not and number of foetuses)  
205 only at CH where ultrasounds are performed on captured females, ii) most of them (*i.e.* 92 %)   
206 were gestating at that time and iii) due to the delayed embryonic implantation (Aitken 1974),  
207 gestation is at a very early stage during the capture season. Body mass measured on juveniles  
208 (*i.e.* at about 8 months of age) depends on the date of capture. Juveniles gained on average 12  
209 g/day and 24 g/day in CH and TF, respectively, during the capture period (Douhard et al.  
210 2017). Therefore, we standardised juvenile body mass with a linear regression, using the  
211 above-mentioned Julian date-body mass relationship. Thus, we computed the individual body  
212 mass expected on the 9<sup>th</sup> of February, the median date of captures at CH and TF. In these two  
213 populations, cohort quality is reliably measured by the cohort-specific average juvenile mass  
214 corrected for the Julian date of capture (Gaillard et al. 1996). This proxy of environmental  
215 quality from the birth of an individual (typically in May) to its capture as juvenile in winter  
216 was obtained for all years, and will be referred to as 'cohort quality' hereafter.

## 217 **Faecal glucocorticoid metabolites (FGMs)**

218 Baseline adrenocortical activity was estimated through FGMs (Palme 2019). We collected  
219 faecal matter rectally at capture from 2010 to 2021. Faeces were frozen at -20 °C within 24  
220 hours prior to 2013 in CH and 2017 in TF, and immediately frozen at -80 °C after collection  
221 since then. The time between faecal sampling and freezing, as well as freezing temperature,  
222 may impact FGM values, with longer delays and higher temperature likely to result in lower  
223 FGM levels due to bacterial activity (Lexen et al. 2008, Hadinger et al. 2015, Carbillet et al.  
224 2023b). In CH, the first FGM values available in our dataset are post-2013, so all samples  
225 were immediately frozen at -80 °C. However, in TF, FGM values are effectively higher when  
226 samples were immediately frozen ( $0.43 \pm 0.06$  SE,  $t = 7.62$ ,  $p < 0.0001$ ). This is accounted for  
227 in our models by adding the year of capture as a random effect. Extraction of FGM followed a  
228 methanol-based procedure and the analysis was performed using a group-specific 11-  
229 oxoetiocholanolone enzyme immunoassay (EIA), a method previously described in details  
230 (Möstl et al. 2002) and validated for roe deer (Zbyryt et al. 2018). In brief, 0.5 g ( $\pm 0.005$ ) of  
231 faeces were vortexed in 5 mL of 80% methanol before being centrifuged for 15 minutes at  
232 2500 g (Palme et al. 2013). The amount of FGM was determined in an aliquot of the  
233 supernatant diluted 10 times in assay buffer. Measurements were done in duplicate with intra-  
234 and inter-assay coefficients lower than 10% and 15%, respectively. FGMs are expressed as  
235 nanograms per grams of wet faeces (ng/g). The data were then log-transformed for the  
236 statistical analyses (henceforth called FGMs).

## 237 **Statistical analyses**

238 All analyses were performed using R version 4.2.2 (R Core Team 2022). Two raw FGM  
239 measures were unusually high: 5192 and 6194 ng/g, for a female in CH aged 10, and a female  
240 in TF aged 1, respectively (see SI). Results exclude these extreme values (analyses including

241 them are reported in SI), but we specify whether adding them yielded different conclusions or  
242 not.

### 243 *FGM repeatability*

244 Individual FGM repeatability was calculated for all individuals, and separately for each  
245 population, using the ‘rptR’ package (Stoffel et al. 2017). Repeatability analysis included  
246 individuals sampled only once to improve estimates of the within-individual variance (Martin  
247 et al. 2011). Within-individual FGM repeatability was detectable but weak in both CH ( $r =$   
248  $0.14$ , 95% CI =  $[0.03, 0.26]$ ) and TF ( $r = 0.15$   $[0.01, 0.29]$ ).

### 249 *Model structure*

250 We tested the hypothesis that body mass is related to FGM levels, and that this relationship  
251 may depend on individual (*i.e.* sex) and environmental (*i.e.* population and condition at birth)  
252 characteristics, using linear mixed effect models (LMMs) with a normal error distribution. All  
253 continuous covariates were mean-centred so that the intercept is the relative individual body  
254 mass for the mean cohort quality and/or mean FGMs. Visual inspection of model residuals  
255 confirmed that they fulfilled the assumptions of Gaussian distribution and homoscedasticity.

### 256 *Short-term relationships between FGMs and body mass measured at the same capture event*

257 We first assessed the relationship between FGMs and body mass measured the same year (*i.e.*  
258 at the same capture) to test the hypothesis that higher baseline FGMs result in short-term  
259 adverse consequences on body mass. Individuals in their first year of life (*i.e.* juveniles) were  
260 analysed separately from individuals in their second year of life onwards, because juveniles  
261 have to allocate to growth and are much more susceptible to any environmental harshness  
262 than adults, making the first year of life is the critical period of roe deer population dynamics  
263 (Hamel et al. 2009, Gaillard et al. 2013). In addition, metabolic rate is generally higher in  
264 juveniles than in adults due to the costs of growth (Glazier 2005) and metabolic rates can alter

265 hormones metabolization and excretion, making it difficult to compare juvenile and adult  
266 FGM concentrations (Goymann 2012). Since juveniles are not yet reproductively active, they  
267 also likely display different endocrine profiles than adults (Dantzer et al. 2010). Finally, adult  
268 roe deer habituate faster than juveniles to stress, which can in turn affect the relationship  
269 between FGM and fitness-related trait (Bonnot et al. 2018). Body mass varies with age, and at  
270 a given age, varies between sexes and according to environmental conditions at birth (*i.e.*  
271 cohort effects, Hamel et al. 2016). We thus tested the relationship between FGMs measured in  
272 a given year and individual body mass relative to the average body mass of all individuals of  
273 the same age, population and sex, considering only ages for which we had data from at least 3  
274 individuals. Therefore, the average body mass was calculated up to 10 and 15 years old in  
275 males and females at CH, respectively, and up to 12 and 13 years old in males and females at  
276 TF, respectively. For juveniles, we analysed the relationship between FGMs and body mass  
277 measured the same year on 368 juveniles (78 females and 96 males in CH, 91 females and  
278 103 males in TF). The response variable was the mass corrected for the date of capture (see  
279 above) and fixed explanatory variables included FGMs (corrected for the date of capture and  
280 the population), cohort quality, population, sex and the two-way interactions between FGMs  
281 and the other covariables. Cohort quality was expressed as a relative cohort quality (*i.e.* the  
282 difference between mean cohort quality in each population and an individual cohort quality),  
283 so there is no redundancy with the population variable. The year of birth of the individuals  
284 (*i.e.* cohort) was included as a random effect. For individuals aged 2 years or older, the dataset  
285 included 655 observations on 377 individuals: 104 females (218 observations) and 83 males  
286 (136 obs.) in CH, and 106 females (164 obs.) and 84 males (137 obs.) in TF. The response  
287 variable used to test for the FGMs-mass relationship was the individual body mass minus the  
288 average mass of roe deer of the same population, same sex and same age, regardless of their  
289 year of capture (hereafter 'relative body mass'). Fixed effects included continuous variation in

290 FGMs, relative cohort quality, population, sex and the two-way interactions between FGMs  
291 and other covariables. Random effects of the cohort and of the year of capture were also  
292 included. Random effect of the individual identity (ID) was also included to account for  
293 repeated measurements on the same individuals.

294 *Medium-term relationships between FGMs and body mass change between two consecutive*  
295 *years*

296 Baseline FGMs can reflect medium-term consequences of GCs on body mass. The  
297 relationship between FGMs and the change of body mass between two consecutive years was  
298 analysed separately for early-growing individuals (*i.e.* between age 1 and 2), late-growing  
299 individuals (*i.e.* between the second year of life and adulthood at 4 years old, Hewison et al.  
300 2011) and for prime-aged adults which had reached their full body mass (*i.e.* from 4 to 10  
301 years old, Hewison et al., 2011). The response variable was the change in relative body mass.  
302 Briefly, the change in relative mass expresses the change in body mass of a given individual  
303 between two ages in relation to the change in body mass of all individuals of the same sex and  
304 population between the same ages. The relative mass was calculated as the difference between  
305 the mass of an individual at age  $x$ , and the mean mass of all individuals of age  $t$  of the same  
306 sex and the same population. The change in relative body mass was calculated as the  
307 difference between the relative mass at age  $t + 1$  and the relative mass at age  $t$ . Fixed effects  
308 included relative cohort quality, population, sex, FGMs (either measured at age  $t$  or  
309 considered as the average of the FGM values measured at age  $t$  and age  $t + 1$ ), body mass at  
310 age  $t$  (*i.e.* initial body mass) and all two-way interactions between FGMs and the other  
311 covariables. Random effect of the cohort was included, and for late-growing and prime-aged  
312 individuals, random effects of the year of capture and individual ID were also included. For  
313 growing individuals, the dataset included 99 individuals with FGMs measured as juveniles,  
314 and 67 individuals when including FGMs measured during the first and second years of life to

315 calculate the mean value. For both late-growing individuals and adults, we kept a single  
316 observation per individual since the inclusion of individual ID as a random effect created  
317 singularities. For individuals with several observations, we kept the one for which the  
318 individual's age was the closest to the mean age of all individuals with a unique observation  
319 according to sex and population, among observations with complete data. The final dataset  
320 comprised 85 individuals in their late-growth period and 90 prime-aged adults with FGMs  
321 measured at age  $t$ , and 63 late-growing individuals and 71 prime-aged adults for which we  
322 had measurements at age  $t$  and  $t+1$ .

### 323 *Long-term relationships between FGMs during early life and body mass later in life*

324 As elevated baseline FGMs are expected to have negative effects on body mass on the long-  
325 term, and as environmental conditions during the first year of life have carry-over effects on  
326 performance later in life in roe deer (Gaillard et al. 2003), we tested whether FGMs during  
327 development are associated with adult body mass. Only individuals for which we measured  
328 FGMs as juvenile and body mass beyond the second year of life were analysed. We used 345  
329 observations on 159 individuals for which we measured FGMs as juvenile and body mass as  
330 adult: 34 females (78 obs.) and 44 males (87 obs.) in CH, and 42 females (99 obs.) and 39  
331 males (81 obs.) in TF. The response variable was the relative individual body mass as defined  
332 above. Fixed effects included FGMs measured when juvenile, relative cohort quality,  
333 population, sex and the two-way interactions between FGMs and the other covariables.  
334 Random effects of the cohort and the year of capture were included, as well as individual ID  
335 to account for repeated measurements on the same individuals.

### 336 *Model selection*

337 Final models were selected based on the second order Akaike Information Criterion (AICc,  
338 *i.e.* Akaike Information Criterion corrected for small sample sizes). We compared all sub-



339 models included in the full model described above and for each model AICc scores were  
340 computed with the “*MuMIn*” package (v. 1.47.5, Bartoń 2023). We retained the best-fitting  
341 model as the one with the lowest AICc score (Burnham and Anderson 2002), or the simplest  
342 model (*i.e.* with the lowest number of parameters) within the set of models within 7  $\Delta$ AICc.  
343 Indeed, models within 7  $\Delta$ AICc are plausible models, become increasingly equivocal up to 14  
344  $\Delta$ AICc, and implausible afterwards (Burnham et al. 2011). For each variable we estimated its  
345 effect size ( $\beta$ ) with 95% confidence intervals (95% CI) and calculated marginal and  
346 conditional  $R^2$ . We provide full model selection tables in SI.

## 347 **Results**

348 FGM concentrations ranged from 8 to 3428 ng/g with a median value of 689 ng/g. The  
349 median FGMs was 674 ng/g (range: 34-3275 ng/g) in CH, and 715 ng/g (range: 8-3428 ng/g)  
350 in TF.

351

### 352 **Short-term relationships between FGMs and body mass measured at the same capture** 353 **event**

#### 354 *Juveniles*

355 The retained model highlighted the expected positive relationship between cohort quality and  
356 body mass ( $\beta_{\text{Cohort quality}} = 1.08 [0.84, 1.32]$ , Table 1, SI), but no relationship between FGMs  
357 and body mass could be evidenced (Figure 1(a), Table 1, SI). Results were similar when  
358 including female of TF with a high FGM value (SI). Note that since the random effect of the  
359 cohort created singularities, it was removed and models therefore consisted of linear models.

#### 360 *Adults*

361 We found support for a short-term negative relationship between individual relative body  
362 mass and FGMs in adults ( $\beta_{\text{FGM}} = -0.32 [-0.46, -0.18]$ , Figure 1(b), Table 1, SI). Results were

363 similar when including the high FGM value measured on a female of CH (SI). Since the  
364 random effect of the cohort created singularities, the models only included individual ID and  
365 year of capture as random effects.

366 **Medium-term relationships between FGMs and change in body mass between two**  
367 **consecutive years**

368 *Early-growing individuals*

369 We did not find any evidence for a relationship between FGMs, either measured in juveniles  
370 or considered as the mean FGM value between the first and second years of life, and the  
371 change in relative body mass for growing individuals (Figure 2(a), Table 1, SI). Results were  
372 similar when including the female of CH with the highest FGM value (SI). All models  
373 included cohort as random effect.

374 *Late-growing individuals*

375 We found a negative relationship between body mass gain and initial mass ( $\beta_{\text{mass}} = -0.15$  [-  
376 0.25, -0.05], Table 1, SI) for the dataset accounting for FGMs measured in juveniles. With the  
377 dataset including the mean FGM value between FGMs measured at age  $t$  and age  $t+1$ , we also  
378 found a negative relationship between late growth and body mass measured at age  $t$  ( $\beta_{\text{mass}} = -$   
379 0.17 [-0.27, -0.07], Table 1, SI). However, in both cases, we found no support for a  
380 relationship between FGMs and change in relative body mass (Figure 2(b), Table 1, SI).  
381 Models resulted in singularities when the random effect of the cohort was included so that  
382 only the year of capture was included as a random effect in the models.

383 *Prime-aged adults*

384 In no cases (*i.e.* neither when accounting for FGM measured at age  $t$  or for the mean FGM  
385 value between age  $t$  and age  $t+1$ ) was a relationship found between FGMs and change in

386 relative body mass (Figure 2(c), Table 1, SI). Similarly, we found no evidence that mass  
387 measured at age  $t$ , cohort quality, population or sex were related to changes in relative body  
388 mass (Table 1, SI). Models resulted in singularities when the random effect of the cohort was  
389 included, thus, only the random effect of the year of capture was included in the models.

390 **Long-term relationships between FGMs during early-life and body mass later in life**

391 We found no evidence for a relationship between FGM measured in juveniles, cohort quality,  
392 population or sex, and adult relative body mass (Figure 3, Table 1, SI). Results were similar  
393 when including the three observations of the adult female of CH with the highest FGM value  
394 measured as juvenile (SI). We only included individual ID and year of capture as random  
395 effects, because including the cohort created singularities.

396 **Table 1.** Linear and linear mixed effect models selected for the short-, medium- and  
 397 long-term relationships between relative body mass and faecal glucocorticoid  
 398 metabolites (FGMs). Models accounted for sex, population and for relative cohort  
 399 quality (Qcohort, the difference between the mean cohort quality in each population  
 400 and an individual cohort quality). Models were selected through model selection  
 401 based on AICc. 95%CI: 95% confidence intervals, V: variance, SD: standard-  
 402 deviation.

SHORT-TERM								
Juveniles			Adults					
Random effects	V	SD	Random effects	V	SD			
			Individual ID	3.45	1.86			
			Year of capture	0.29	0.54			
Fixed effects	Estimate	95%CI	Fixed effects	Estimate	95%CI			
Intercept	-0.06	[-0.28, 0.16]	Intercept	-0.02	[-0.39, 0.35]			
Qcohort	1.08	[0.84, 1.32]	FGM	-0.32	[-0.46, -0.18]			
Marginal R <sup>2</sup>	0.18		Marginal R <sup>2</sup>	0.02				
Conditional R <sup>2</sup>			Conditional R <sup>2</sup>	0.81				

MEDIUM-TERM								
FGM measured at age <i>t</i>								
Early-growth			Late-growth			Prime-aged		
Random effects	V	SD	Random effects	V	SD	Random effects	V	SD
Cohort	0.65	0.81	Year of capture	0.41	0.64	Year of capture	0.24	0.49
Fixed effects	Estimate	95%CI	Fixed effects	Estimate	95%CI	Fixed effects	Estimate	95%CI
Intercept	-0.35	[-0.92, 0.22]	Intercept	-0.55	[-1.02, -0.08]	Intercept	-0.19	[-0.60, 0.22]
			Initial mass	-0.15	[-0.25, -0.05]			
Marginal R <sup>2</sup>	-		Marginal R <sup>2</sup>	0.09		Marginal R <sup>2</sup>	-	
Conditional R <sup>2</sup>	0.21		Conditional R <sup>2</sup>	0.32		Conditional R <sup>2</sup>	0.15	

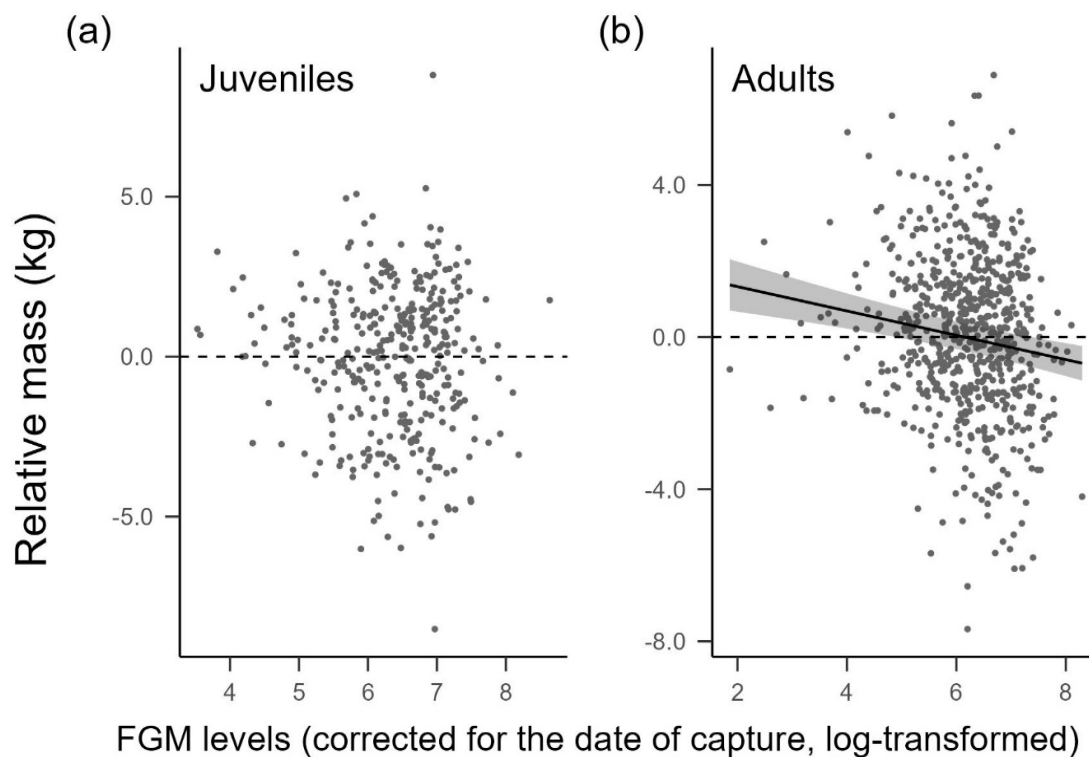
Mean FGM (FGM <sub><i>t</i></sub> and FGM <sub><i>t+1</i></sub> )								
Early-growth			Late-growth			Prime-aged		
Random effects	V	SD	Random effects	V	SD	Random effects	V	SD
Cohort	0.90	0.95	Year of capture	0.32	0.56	Year of capture	0.18	0.42
Fixed effects	Estimate	95%CI	Fixed effects	Estimate	95%CI	Fixed effects	Estimate	95%CI
Intercept	-0.37	[-1.11, 0.37]	Intercept	-0.47	[-0.94, -0.00]	Intercept	-0.28	[-0.71, 0.15]
			Initial mass	-0.17	[-0.27, -0.07]			
Marginal R <sup>2</sup>	-		Marginal R <sup>2</sup>	0.13		Marginal R <sup>2</sup>	-	
Conditional R <sup>2</sup>	0.26		Conditional R <sup>2</sup>	0.32		Conditional R <sup>2</sup>	0.10	

LONG-TERM		
Random effects	V	SD
Individual ID	3.65	1.91
Year of capture	0.63	0.79

Fixed effects	Estimate	95%CI
Intercept	-0.06	[-0.63, 0.51]
Marginal R <sup>2</sup>	-	
Conditional R <sup>2</sup>	0.83	

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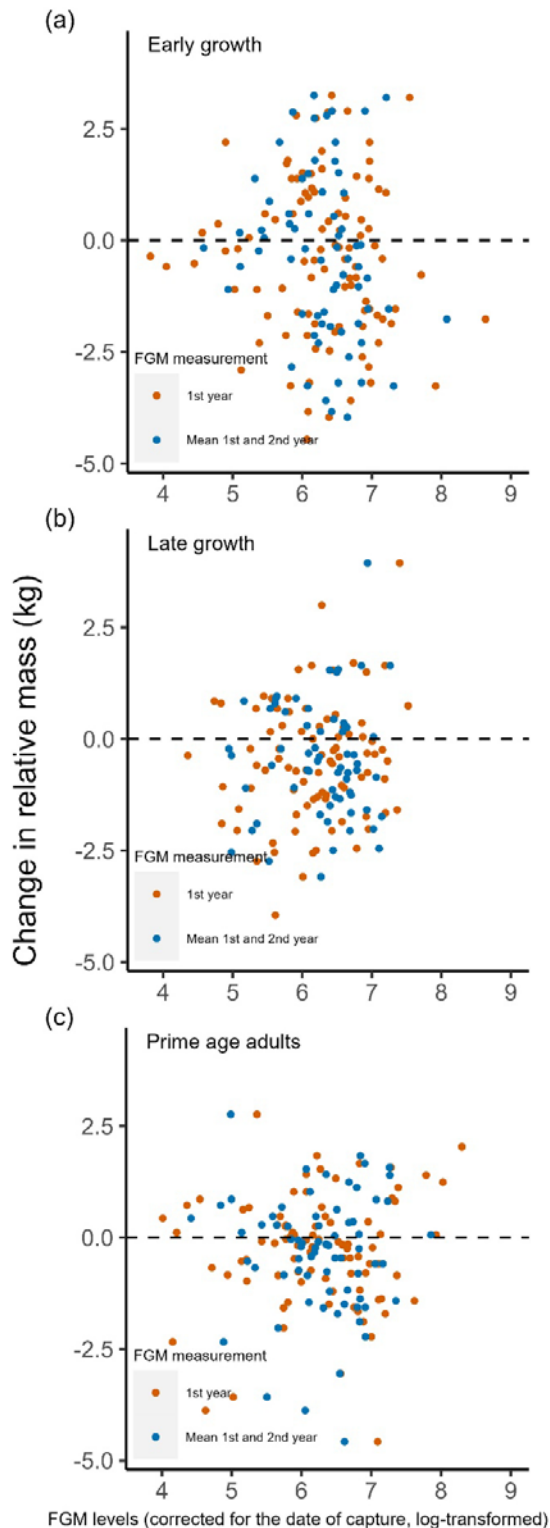
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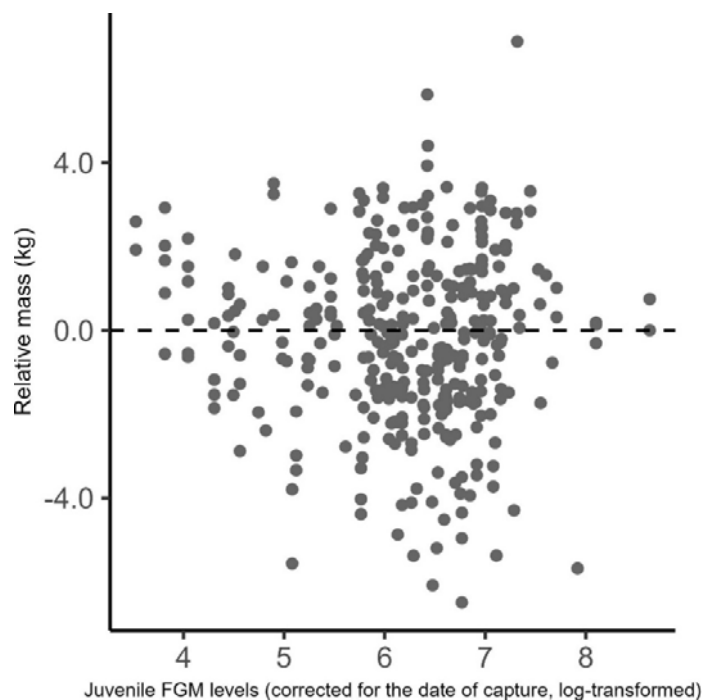
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**Figure 1.** Relationship between faecal glucocorticoid metabolites (log-transformed FGMs, corrected for the date of capture and population) and individual relative body mass (individual body mass (corrected for the date of capture for juveniles) minus the average body mass of all individuals of the same age, population and sex) for (a) juveniles (N = 368 individuals) and for (b) adults aged 2 years old onwards (N = 655 observations on 377 individuals). Points are raw data and line is the prediction from the retained model with 95% confidence intervals.



**Figure 2.** Relationship between faecal glucocorticoid metabolites (log-transformed FGMs, corrected for the date of capture and population) and change in relative body mass between two consecutive years for (a) early-growing individuals (between the first and second years of life), with FGMs either measured the first year of life (N = 99, orange points) or considered as the mean FGMs between the first and second years (N = 67, blue points); for (b) late-growing individuals (between 2 and 4 years old) with FGMs either measured at age  $t$  (N = 85, orange points) or considered as the mean FGMs between years  $t$  and  $t+1$  (N = 63, blue points) and for (c) prime-aged adults which had reached their full body mass (from 4 years old to 10 years old), with FGMs either measured at a given age  $t$  (N = 90, orange points) or considered as the mean FGM between age  $t$  and age  $t+1$  (N = 71, blue points). Points are raw data



435

436

437 **Figure 3.** Relationship between faecal glucocorticoid metabolites (log-transformed FGMs,  
438 corrected for the date of capture and population) measured in juveniles and individual relative  
439 body mass (*i.e.* individual body mass minus the average body mass of all individuals of the  
440 same age, sex and population) (N = 345 observations on 159 individuals). Points are raw  
441 data.

441

## 442 **Discussion**

443 The relationship between GCs and fitness-related traits such as body mass is yet to be fully

444 deciphered. Recent reviews indicate that studies in the wild have led to contrasting results by

445 showing either negative, positive or no relationships between measures of stress and studied

446 traits (Bonier et al. 2009, Crespi et al. 2013, Dantzer et al. 2014). A salient conclusion from

447 these studies is that the relationship depends on an individual's ecological context and internal

448 state. In the present study, the longitudinal monitoring of individual roe deer in two

449 populations exposed to markedly different environmental contexts – one facing limiting

450 resources and the other benefiting from a higher availability and quality of resources –

451 allowed us to analyse the relationship between FGMs and body mass, while accounting for

452 ecological factors and individual attributes such as sex and age. On the short-term, we found

453 clear evidence for a negative relationship between baseline adult FGMs and adult body mass,  
454 an important driver of fitness in this species (Gaillard et al. 2000).

455 We could not detect any relationship between FGMs and the change in relative body mass  
456 between two consecutive years, neither in growing or prime-aged individuals. In late-growing  
457 individuals only, a negative relationship between initial body mass and relative body mass  
458 gain appeared, so that lighter individuals of age  $t$  tended to gain more weight from one year to  
459 the other, suggesting the existence of compensatory growth mechanisms in these populations  
460 (Metcalf and Monaghan 2001). Likewise, we found no support for a relationship between  
461 early-life FGM levels and adult body mass. Indeed, we found that FGMs and body mass were  
462 negatively associated only on the short-term and in adult individuals only. This suggest that  
463 FGMs relate to body mass, but without any carry-over effects, and that FGMs are related to  
464 body mass differently according to life-history stages. High viability selection at the juvenile  
465 stage in the two populations (Toigo et al. 2006, Douhard et al. 2014) could explain that we  
466 could not observe any carry-over effects of FGMs on body mass, and an absence of evidence  
467 for a short-term relationship between FGMs and body mass in juveniles.

468 Roe deer body mass correlated negatively with FGM levels on the short-term, in adults, with  
469 no detectable population or sex differences. This result suggests that the relationship between  
470 FGM and body mass is consistent across individuals and between environmental contexts in  
471 this species. Roe deer are income breeders, relying mostly on acquired resources rather than  
472 on stored energy (Andersen et al. 2000). As GCs stimulate activity through the mobilisation  
473 of energy stored (Sapolsky et al. 2000, Reeder and Kramer 2005), the physiological costs of  
474 elevated GC levels are expected to directly impair body condition as individuals do not have  
475 body reserves to buffer elevated energy requirements (Henderson et al. 2017). However,  
476 given marked between-population differences in environmental context we would have  
477 expected individuals at TF to be able to mitigate the physiological costs of high GC levels by



478 reducing their allostatic load (McEwen and Wingfield 2010, Henderson et al. 2017),  
479 compared to individuals at CH which were expected to enter allostatic overload, thus  
480 expecting different allostatic load in the two populations (McEwen and Wingfield 2010). Both  
481 populations differ in terms of overall habitat quality, with CH being characterised by a low  
482 forest productivity due to poor soils, mild winters, but frequent droughts during summer, and  
483 TF by a high forest productivity due to rich soils and relatively wet summers (Pettorelli et al.  
484 2006). Accordingly, life history parameters differ between populations. This is especially the  
485 case for juvenile survival (Gaillard et al. 1993), body condition (Gilot-Fromont et al. 2012),  
486 senescence patterns (haematological parameters: Jégo et al. 2014, body mass: Douhard et al.  
487 2017, immune system: Cheynel et al. 2017), and the relation between FGMs and immune  
488 traits (Carbillet et al. 2023a). Thus, differences in the relationship between FGMs and body  
489 mass was expected to occur between populations, with the strongest negative association in  
490 CH, the poorer habitat in which individuals would be more at risk of allostatic overload  
491 (McEwen and Wingfield 2010, Henderson et al. 2017). Red deer (*Cervus elaphus*) showed  
492 different allostatic loads across populations with different densities and environmental  
493 conditions, with individuals facing high density or poor-quality habitat having higher FGM  
494 levels and lower body mass (Caslini et al. 2016). Our results rather suggest that both roe deer  
495 populations are in a state of allostatic overload. During the winter season, roe deer face more  
496 severe winters at TF than at CH, which can in turn mitigate, during this period, the overall  
497 better resource quality in TF. This could partly explain why no population differences were  
498 detected. It also suggests that allostatic load can change across seasons, as shown in wild grey  
499 mouse lemurs (*Microcebus murinus*, Hämäläinen et al. 2015), and that this variation could  
500 operate in a population-specific way. It is therefore of great interest to assess the GC-fitness  
501 relationship across different environmental contexts, including different years or climatic  
502 conditions, and through various life history stages. Finally, the absence of large predators and

503 the weak hunting pressure in both population might at least partly account for the absence of  
504 support for a steeper relationship at CH. Studies of roe deer populations facing varying  
505 conditions in terms of presence/absence of large predators and hunting pressure, in Poland,  
506 showed that hunted populations without large predators had highest FGM levels with largest  
507 variation (Zbyryt et al. 2018). In support, observed variation in FGM levels was high in both  
508 populations (CV = 59.7 % and 55.3 % in CH and TF, respectively), which could mitigate the  
509 relationship between body mass and FGMs and differences between populations.

510 Similarly, we could not detect any sex difference, despite the marked differences in the stress  
511 response in males and females reported in mammals, due in part to the interaction between the  
512 HPA and reproductive axes (Viau 2002, Wilson et al. 2005, and see Toufexis et al. 2014,  
513 Novais et al. 2016 for reviews). This absence of sex-differences also suggests that during this  
514 period of the year, female reproductive status does not influence the relationship between  
515 FGMs and body mass. In reindeer (*Rangifer tarandus*), cortisol levels did not differ between  
516 males, non-pregnant or pregnant females, but showed seasonal variation (Bubenik et al.  
517 1998). Accordingly, red deer showed no sex-differences in their levels of FGMs but also  
518 showed seasonal variation (Huber et al. 2003). Thus, it would be interesting to collect FGM  
519 data of roe deer during different seasons as the stress response can vary accordingly (Bubenik  
520 et al. 1998, Vera et al. 2011). It would also provide an opportunity to investigate whether the  
521 different reproductive tactics of males and females can affect the relationship between GC and  
522 fitness-related traits. Females of an iteroparous, long-lived species such as roe deer are  
523 expected to trade reproduction for survival when environmental conditions are poor  
524 (Hirshfield and Tinkle 1975). Thus, females are expected to be stress-responsive during their  
525 reproductive period to be able to switch between alternative physiological states according to  
526 whether they reproduce or not, due to their future opportunities of reproducing (Ricklefs and  
527 Wikelski 2002). On the other hand, males allocate heavily to reproduction by defending

528 territories for half of the year (Johansson 1996). Territory defence results in elevated  
529 physiological damages, whatever their reproductive success. Therefore, females could show a  
530 larger variability in the way they respond to GC than males which are more constrained. This  
531 provides promising perspectives to better understand the relationship between GC levels and  
532 fitness-related traits according to sex-specific life history stages. It would also be interesting  
533 to investigate whether offspring have different life-history trajectories according to their  
534 mothers' baseline FGMs during gestation, as shown in North American red squirrels  
535 (*Tamiasciurus hudsonicus*, Dantzer et al. 2013), or according to whether mother were  
536 captured or not during gestation to further investigate the impact of capture on individuals.

537 FGMs have been previously used as proxies to evaluate the homeostatic level or allostatic  
538 load in wild populations, but the results should be interpreted with caution. FGMs do not fully  
539 reflect an individual's stress response because GCs only represent one part of the complex  
540 endocrine pathway involved in this response (MacDougall-Shackleton et al. 2019). Moreover,  
541 although GCs are often referred to as "stress hormones", at baseline levels, their primary role  
542 is to acquire, deposit and mobilise energy (Busch and Hayward 2009). Thus, unlike acute GC  
543 concentrations, baseline GCs do not only provide information on how an individual responds  
544 to stressors, but also on its physiology and activity (Reeder and Kramer 2005). It has been  
545 reported that the acute GC response tends to be attenuated in individuals repeatedly exposed  
546 to stressors (*e.g.* Ader et al. 1968, Kant et al. 1983, Vera et al. 2011). However, baseline GC  
547 levels can also, but not always, be lower in individuals chronically exposed to environmental  
548 stressors, making it difficult to interpret FGM levels in the wild. Indeed, whether low baseline  
549 GCs actually represent a non-stressed individual rather than an individual chronically exposed  
550 to stressors is difficult to tell (Davis and Maney 2018).

551 In conclusion, we found that high baseline FGMs may have immediate adverse consequences  
552 on body mass and that the relationship between body mass and FGM levels seems to depend

553 on an individual life-stage, rather than on the environmental context or sex. Through this  
554 work, we therefore emphasise the need to account for both environmental and individual  
555 factors, including life history traits, to better capture the relationship between the stress  
556 response and fitness-related traits. We also put emphasis on the idea to collect FGM across  
557 different temporal and environmental contexts to evaluate how the GC-fitness relationship can  
558 be modulated according to the seasonal and environmental conditions. To do so, it appears  
559 critical to have access to longitudinal data. Measuring proxies of stress together with fitness-  
560 related traits throughout the life of individuals is likely to be the only way to properly refine  
561 our understanding of the implications of stress for fitness.

## 562 **Conflict of interest**

563 The authors declare having no conflict of interest.

## 564 **Ethics**

565 The research presented in this manuscript was done according to all institutional and/or  
566 national guidelines. For both populations (Trois-Fontaines and Chizé), the protocol of capture  
567 of roe deer, under the authority of the Office Français de la Biodiversité (OFB), was approved  
568 by the Director of Food, Agriculture and Forest (Prefectoral order 2009-14 from Paris). All  
569 procedures were approved by the Ethical Committee of Lyon 1 University (project DR2014-  
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