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# Ontogeny of cortisol reaction norms in wild bonobos (*Pan paniscus*)

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Phenotypic plasticity enables animals to adjust physiology, behaviour, morphology and life-history traits in response to changing conditions, either reversibly or through irreversible developmental shifts. In long-lived species, early-life phenotypic changes can have profound consequences if they persist into adulthood. Understanding the balance between stable and flexible trait expression across ontogeny is therefore key. Glucocorticoids (GCs) are central to physiological regulation and known to exhibit plasticity, but little is known about the consistency of GC phenotypes across development. We examined whether bonobos (*Pan paniscus*), a long-lived species, show consistent GC phenotypes as they mature, focusing on individual differences in average urinary cortisol phenotypes (reaction-norm intercepts) and plasticity (reaction-norm slopes) in response to time of day. We applied a reaction-norm approach to assess individual variation in GC intercepts and slopes across ontogeny, using random regression mixed-effects models. Trait repeatability of urinary cortisol was low across and within years, indicating high within-individual variation relative to between-individual variation. Reaction-norm intercepts were moderately repeatable, suggesting stable individual average GC phenotypes across development. By contrast, slopes were weakly repeatable, reflecting flexibility in how individuals modulate GC output across the day. This dual regulatory structure may support adaptive physiological responses to changing demands in a long-lived species.

## 1. Background

Individuals, even when genetically identical, can display a range of different phenotypes in response to environmental variation [1]. This phenotypic plasticity allows organisms to cope with heterogeneous environments [2], which often facilitates fitness [1] and survival [3]. Some responses to environmental conditions are immediate and/or temporary, while others represent long-term shifts that may be irreversible [4,5]. Whether phenotypic changes are transient or persist throughout life, or even across generations, depends both on the individual-level costs associated with a plastic response [6] and the temporal predictability and persistence of environmental change, which shape the evolutionary stability of plasticity within a species [7,8].

Generally, individuals exposed to changing environments throughout their lifetime are expected to show reversible plasticity (i.e. phenotypic flexibility, [9]) primarily while individuals living in stable environments are predicted to display irreversible plasticity [4,7]. In addition, the timing of phenotypic

reactions to environmental conditions in the course of an individual's lifetime may determine whether a plastic change is temporary or permanent: a plastic phenotypic change during the ontogeny of an organism is usually associated with a permanent shift in the phenotype [2,4]. An irreversible phenotypic change relatively early in life can have tremendous long-term implications for the organism later in life (e.g. [10,11]). If such early-life phenotypic adjustments match the individual to future environments, the phenotypic change may be adaptive. However, phenotypic shifts can prove maladaptive, if the adjustment is mismatched to the future environment [12].

Various traits, such as morphological features or the timing of life-history stages, can be expressed plastically [13]. For instance, tadpoles (of the red-eyed treefrog, *Agalychnis callidryas*, and the African clawed frog, *Xenopus laevis*) reared under low temperature conditions developed longer, less ossified legs post-metamorphosis than tadpoles exposed to warmer temperature conditions prior to metamorphosis [14]. Similarly, dung flies (*Scathophaga stercoraria*) experiencing high food abundance and low levels of competition mature later than peers developing under less favourable conditions (e.g. [15]). The expression of this morphological (bone ossification) and life-history (timing of maturation) trait respectively are examples for irreversible plastic shifts in trait expression: the organism responds plastically to environmental cues (temperature and level of competition, respectively), which manifests in irreversible differences in trait expression (longer, less ossified legs and delayed maturation respectively) between individuals.

When it comes to plasticity in more volatile traits, glucocorticoids (GCs), a group of conserved vertebrate steroid hormones, are by far the most studied (e.g. [16–18]). GCs can impact multiple cell types and subsequently influence phenotypes [4,19]. The hypothalamic-pituitary-adrenal (HPA) axis, which mediates GC secretion, operates within an interconnected physiological network, and exerts influence over other physiological circuits, such as the hypothalamic-pituitary-somatotrophic axis associated with growth, or the hypothalamic-pituitary-thyroid axis associated with metabolic adaptations [20–22]. Because of this interconnectedness, GCs exhibit a central role within the physiological network and coordinate responses to different environmental cues [4,23]. Therefore, plastic phenotypic changes in HPA axis functioning during the ontogeny of an organism have received considerable scientific attention [5,24,25]. Even prenatal exposure to GCs (either exogenous or endogenous) shapes cardiovascular, metabolic, reproductive and neurological development and has lasting effects on the maturation and lifelong function of the HPA axis across species, including humans [26–29].

Despite growing knowledge regarding the plastic secretion of GCs in response to environmental factors [17,30,31], relatively little is known about how consistent GC phenotypes are within individuals across ontogeny, particularly in long-lived species [32–35]. The degree of consistency can have major implications for fitness, health and life-history trade-offs, however [31,36,37].

To address this question, reaction-norm approaches provide a powerful framework [34,38,39]. The basis for reaction-norm assessments is a repeated measures design: by sampling individuals repeatedly across a gradient of interest, we can investigate (i) an individual's average response to this gradient (the intercept of the curve), (ii) the degree of plasticity in the individual's response to the gradient (the slope of the curve), and (iii) whether average responses and plasticity are correlated or independent. For example, if intercepts and slopes are correlated, individuals with high intercepts (i.e. higher average phenotypes) may display lower plasticity in their responses to the gradient, and vice versa. In the absence of a correlation between intercept and slope, the two traits (average phenotype and plasticity) vary independently, allowing both individuals with high and low average trait expression to respond plastically to environmental cues. Gradients of interest may be ecologically or physiologically relevant gradients, such as time of day, temperature or social context [17,18,38].

Furthermore, by modelling within- and between-individual variance in responses to a gradient repeatedly (repeated exposure to the same gradient, e.g. a temperature gradient over several mating seasons), we can infer (i) whether there are consistent differences between individuals in those responses (trait repeatability) and (ii) whether individuals are consistent in the way they respond to a gradient (reaction-norm repeatability). Trait and reaction-norm repeatability are key prerequisites for assessing whether traits are developmentally canalized (robust) or flexible, and ultimately whether they are potentially subject to selection given the trade-off between robustness and evolvability [4,40]. Trait repeatability of endocrine traits—either in average expression or in plasticity—has been argued to represent an upper bound of heritability [40,41]. High trait repeatability suggests consistent inter-individual differences (e.g. clusters of high and low responders due to heritable factors, maternal styles, or other prenatal and early-life priming factors), whereas low trait repeatability suggests greater developmental flexibility or predominant environmental control. Reaction-norm repeatability indicates how consistent responses to a gradient are within an individual over time. Importantly, repeatable reaction norms (i.e. stable within-individual differences in plasticity or average phenotype) may themselves be targets of selection if they reflect persistent strategies in physiological responsiveness [9,38].

The application of reaction-norm frameworks requires a biologically relevant gradient that (i) affects all individuals within the population, (ii) varies repeatedly within individuals, and (iii) reliably modulates GC levels. One such gradient is the diurnal rhythm: many vertebrates show a diurnal pattern of GC secretion, typically with a morning peak followed by a decline across the day [42,43]. This diurnal GC rhythm emerges during early development—for example, within the first year of life in humans [44], suggesting that plasticity in diurnal GC regulation may itself develop over time.

However, it remains unclear whether individual differences in these diurnal GC profiles are stable across an individual's lifetime or whether they undergo systematic developmental change. Early-life conditions, social environment, or maturation of the HPA axis could lead to persistent or transient modifications in the structure of diurnal GC rhythms, potentially shaping how individuals differ in endocrine regulation as they age [21,45,46]. In other words, repeatability of diurnal rhythms observed within a given year may not necessarily carry over across years, if developmental or environmental influences alter individual endocrine profiles. This raises the open question of how stable or flexible diurnal GC reaction norms remain across ontogeny, and what such changes may reveal about developmental organization and rearing environments.

Bonobos provide an ideal system to address these questions. Bonobos (*Pan paniscus*), as long-lived primates with slow life histories (e.g. [47–49]), experience a range of environmental conditions throughout their lives, including seasonal fluctuations [50], climatic [51], ecological or social changes [52–54]. Such lifespan-encompassing variation may favour reversible forms of plasticity [4,7]. At the same time, bonobos inhabit relatively stable ecological niches at the macrohabitat level [53], where the overall predictability of key environmental features may promote more canalized, irreversible forms of developmental plasticity [2,4]. This combination renders bonobos an ideal model for studying GC reaction norms across ontogeny, particularly in regard to the balance between physiological stability and flexibility.

Recent research monitoring cortisol levels, the main GC in primates, across development in wild bonobos showed that cortisol levels remain relatively stable during ontogeny [35]. This suggests that the developmental trajectory of cortisol secretion may be constrained by stable socioecological conditions. However, such population-level stability does not preclude individual variation in GC phenotypes or plasticity, particularly across developmental stages where individual experiences or physiological maturation may influence responsiveness to diurnal or social gradients. Bonobos also show a pronounced diurnal pattern of cortisol excretion [55], rendering them well-suited for investigating individual differences in diurnal GC regulation. Previous research on adult female bonobos found that average GC phenotype and plasticity across the diurnal gradient were independent, with limited between-individual variation, suggesting low trait repeatability [22]. However, as this study focused exclusively on adults, the question remains open as to whether these traits are stable within individuals across development. Longitudinal data from male chimpanzees suggest that average GC phenotypes are repeatable across years, while plasticity in diurnal responses is less consistent [18]. Additionally, early-life adversity, such as maternal loss, led to temporary changes in GC functioning in immature chimpanzees but did not affect HPA axis functioning into adulthood, supporting the idea that HPA axis plasticity remains responsive across development [33].

In this study, we investigated whether individual bonobos show repeatable GC reaction norms across age. Specifically, we investigated whether average urinary GC levels (reaction-norm intercepts) and/or their plasticity across the diurnal gradient (reaction-norm slopes) are consistent within individuals when sampled at different ages (i.e. across repeated measurements from infancy to adulthood).

Based on life-history theory [4,7,9] and previous comparative findings [18,22,33,35], we predicted that the plastic component of the reaction norm—the diurnal slope—would show low repeatability, reflecting developmental flexibility in GC responsiveness. Statistically, this would be reflected in small among-individual variance in slopes relative to within-individual variance, indicating that the shape of an individual's diurnal cortisol decline is flexible rather than fixed across ages. Biologically, such flexibility would suggest that diurnal cortisol regulation remains responsive to current environmental and social conditions.

For average GC phenotypes (intercepts of the GC curve across the day), we predicted moderate repeatability, due to relatively stable physiological infrastructure (e.g. receptor densities or feedback sensitivity) that may constrain GC levels. This would correspond to detectable among-individual variance in intercepts, implying that individuals maintain characteristic average cortisol levels across development, even as their daily slopes vary.

Given the volatility of GC traits [20,34,41], we further expected low trait repeatability, with within-individual variation exceeding between-individual differences. Such low repeatability would indicate that most of the observed variation arises from short-term, context-dependent factors rather than stable individual properties. However, consistent differences in individual reaction norms, particularly intercepts, may still reflect persistent physiological strategies and represent potential targets of selection.

## 2. Material and methods

### (a) Study species and subjects

The LuiKotale field site (2°47' S, 20°21' E) lies within a continuous stretch of equatorial rainforest at the southwestern border of Salonga National Park in the Democratic Republic of Congo. The region experiences moderate variation in seasonal rainfall patterns with relatively dry seasons between June and August and around February [51]. As part of the long-term data collection of the LuiKotale Bonobo Project, urine samples are gathered routinely and consequently analysed for physiological markers [56]. For the current study, we used samples collected between June 2008 and February 2023 from individuals of all ages, who belonged to the Bompusa East and West communities (i.e. two stable social groups that inhabit neighbouring, partially overlapping home ranges; within each group, individuals maintain long-term social relationships and a fission–fusion grouping pattern).

### (b) Urine sample collection, hormone extraction and measurement

We analysed 2245 urine samples from 86 individuals (64 females: 1–99 samples each; 22 males: 5–87 samples each), collected non-invasively between approximately 05.00 and 18.00. Samples were obtained immediately after voiding, stored in liquid nitrogen in the field and later frozen at –20°C until analysis. A subset of 293 samples was measured using liquid chromatography–mass spectrometry (LC–MS) [57–59], and these values strongly correlated with enzyme immunoassay (EIA) measurements of the same samples (Spearman  $r = 0.843$ ,  $p < 0.001$ ), validating the EIA for the full dataset. Cortisol concentrations were corrected for specific gravity [60] and analysed following established protocols [61,62]. Inter- and intra-assay coefficients of variation were 8.1%/11.9% (high/low controls) and 5.6%, respectively. Methodological details, assay validation (including serial dilutions and antibody characteristics) and sample handling procedures are provided in the electronic supplementary material, S1.

### (c) Data preparations

Nine females were pregnant in the course of the sample collection period. Samples collected during a pregnancy were excluded from the dataset as it is challenging to distinguish between glucocorticoids of maternal or fetal origin in a sample [63]. Birth dates of immigrant females are not known. Consequently, samples of 24 females for whom we did not know the age had to be excluded. In total, samples from 40 females (on average  $25 \pm 17.4$  samples per individual, range 1–88 samples) and 22 males (on average  $36.4 \pm 20.4$  samples per individual, range 5–86 samples) were included in our final dataset.

Before running statistical tests, we prepared our data as follows. By subtracting the date of birth from the date of sample collection, we calculated the age of the individual at the time of sample collection, both in days and years (age in days divided by 365.25). In cases, where the birth month was not known, we assumed it to be June. Where the birth day was unknown, we assumed it to be the 15th. This decision was based on the rationale that selecting the middle of the year (June) and the middle of the month (15th) minimizes the potential deviation from the actual birthdate, with a maximum possible error of less than six months in either direction. Assigning a neutral midpoint is unlikely to introduce systematic bias into the analyses. Females were between 1 day and 21 years of age, males between 1 day and 19 years of age. Then, we applied a logarithmic transformation to immunoreactive urinary cortisol (urinary cortisol hereafter) levels (our response variable in the models) to achieve a more symmetrical distribution. We converted the sample collection times into minutes since midnight. Since we wanted to estimate variance in urinary cortisol levels within the individual over time, we created a variable representing each *individual-at-age* (i.e. each individual at a given age). This identifier allowed us to model repeated measures across different ages, resulting in 389 unique *individual-at-age* levels. Lastly, we coded the two communities East and West as 0 and 1 (to allow for subsequent standardizing, see below and [38,64]). Likewise, we transformed sex into a binary variable (females = 0, males = 1). Since it is crucial to standardize variables when examining the magnitude and direction of variance estimates in reaction-norm components [64], we centred all variables that function as predictors (time of sample collection, age at sample collection in days) or control (the community, the sex of the individual) variables in our models. To that end, we subtracted the mean value from each data point, effectively aligning it around zero. Subsequently, we standardized the variables, scaling them to two standard deviation units. This transformation ensures that the data is expressed in a consistent standardized scale [38].

### (d) Statistical analyses

All data preparations, analyses and plotting of data were performed using R [65] in RStudio (version 2023.06.1.524 [66]). To assess individual urinary cortisol reaction norms of female and male bonobos throughout ontogeny, we fitted random-regression-mixed-effects models [20,38,67], using the *lme4* package (version 1.1.34, [68]). To ensure the goodness of our models, we visually inspected fitted versus residual plots, histograms of residuals and quantile–quantile (QQ) plots [69–71]. To assess the goodness-of-fit between two models, we used log-likelihood ratio tests [72].

All our models included log-transformed urinary cortisol levels as a response variable and the time of sample collection, the community (East versus West; included to control for group-level socioecological differences), as well as the interaction term of age at sample and sex (all mean centred and standardized to two standard deviations) as fixed effects. To account for the diurnal rhythm of cortisol excretion, time of day was expressed as minutes since midnight and modelled as a linear predictor. Our samples covered only the daytime period (approx. 05.00–18.00), representing the descending portion of the circadian cortisol cycle. To test whether a nonlinear function would better capture the diurnal pattern, we compared linear, quadratic and natural-spline ( $df = 3$ ) specifications in the random-intercept model (see below). The quadratic term did not improve fit (likelihood ratio test (LRT)  $\Delta \chi^2 = 0.84$ ,  $df = 1$ ,  $p = 0.36$ ), and the spline yielded only a modest Akaike information criterion (AIC) improvement ( $\Delta AIC = 8.2$ ) (see electronic supplementary material, S2, table S1 and figure S1). For parsimony and comparability with published reaction-norm studies [18,22,55], we therefore retained the linear term in the main analyses. To test for potential issues with collinearity between these predictor terms, we calculated variance inflation factors using the *car* package [73].

As there may be variation in cortisol levels between years and months, we included random intercepts for each calendar year (treated as a categorical factor; 2008–2023) and month to all models to account for this potential variation. To estimate *trait repeatability* (following [74]) and the level of short- and long-term between-individual variation in urinary cortisol levels [38], we further added a random intercept for the individual and a random intercept for the *individual-at-age* variable to the model (intercept model). This approach allows decomposing variance in repeated-measures data into short- and long-term components by including hierarchical random effects such as individual and *individual-at-age*, thereby separating variance among individuals from variance within individuals across different ages. In this framework, the reaction norm represents within-individual variation in urinary cortisol levels across the diurnal gradient (time of day). Random intercepts for *individual-at-age*, year and month accounted for repeated measures across ontogeny and temporal structure, ensuring that the reaction norm captured diurnal rather than long-term or annual variation. Reaction-norm slopes therefore describe individual-specific diurnal cortisol profiles, while repeatability estimates quantify the stability of these profiles across different ages of the same individuals. The inclusion of the *individual-at-age* level allowed the estimation of one reaction-norm intercept and one reaction-norm slope for each individual and age, thus quantifying how stable these parameters remained across years of sampling.

In a second model (slope model), we added random slopes for the centred and standardized time of day variable (diurnal pattern of urinary cortisol) for the individual as well as for the dummy variable *individual-at-age*. Subsequently, variance estimates derived from this model were used to calculate *reaction-norm repeatability* (following [38]) of individuals throughout ontogeny.

### 3. Results

The maximal variance inflation factor of a model including all our main effects (time of day, community, age at sample collection and sex of the individual) was 1.04, indicating no collinearity between these variables. Therefore, we proceeded by running our intercept model (table 1), which was significantly better ( $\chi^2 = 14.33$ ,  $df = 1$ ,  $p < 0.001$ ) than the same model without the random intercept for individual, suggesting that individuals differ regarding their average urinary cortisol excretion.

To estimate individual plasticity in cortisol levels across the circadian gradient, within and between years, we fitted a random slope model that contained the same main and random effects as the intercept model, but additionally contained random slopes for the time of sample collection for each individual and each *individual-at-age* and the correlation between the random intercepts and random slopes. Since this model had a singular fit warning, we simplified the model structure by excluding the correlations between intercepts and slopes to improve model stability. A Bayesian reanalysis confirmed that the intercept–slope correlation was weakly supported by the data and that the simplified model provided an equivalent fit (see electronic supplementary material, S3 and table S2). Although including random slopes for time of sample collection for the individual ( $\chi^2 = 0.03$ ,  $df = 1$ ,  $p = 0.857$ ) and *individual-at-age* ( $\chi^2 = 0.02$ ,  $df = 1$ ,  $p = 0.651$ ) did not improve model fit, we retained this random slope structure (table 1) to quantify the magnitude and repeatability of slope variation.

From the random intercept model, we estimated that long-term (between years) urinary cortisol trait repeatability was 0.04 (lower confidence interval (lCI) = 0.02, upper confidence interval (uCI) = 0.05). Short-term (within years) urinary cortisol trait repeatability was 0.06 (lCI = 0.05, uCI = 0.07). The estimates for both short-term and long-term trait repeatability were low, which suggests that the relative between-individual variation in cortisol levels is low as compared with the within individual level variation. Furthermore, the low within-year trait repeatability indicates that differences in average cortisol levels among individuals were not influenced by varying environmental conditions that would fluctuate between different years but affect individuals differently. In summary, the low estimate for the long-term trait repeatability indicates that neither genetic nor prenatal priming factors are likely to shape urinary cortisol expression phenotypes in bonobos. The low short-term trait repeatability estimate suggests that either environmental conditions across years were very consistent or that individuals reacted to changes in environmental conditions in a similar fashion.

Calculating reaction-norm repeatability from the random slope model, we found a repeatability score of 0.58 (lCI = 0.48, uCI = 0.66) for individual intercepts and a score of 0.13 (lCI = 0.09, uCI = 0.17) for the time gradient slope within individuals. Thus, within individuals, bonobos seem to be rather consistent in the expression of their average cortisol excretion phenotypes (figure 1), but show marked variation throughout ontogeny regarding the plasticity of urinary cortisol responses across the circadian gradient (figure 2). Overall, these findings show that, while individual bonobos exhibit consistent urinary cortisol intercepts (consistent in regard to average phenotypes), the plasticity (slope) of their circadian urinary cortisol responses varies considerably throughout development.

### 4. Discussion

Our results show that individual bonobos exhibit low trait repeatability in urinary cortisol levels, both across and within years, indicating that most variation in urinary cortisol levels occurs within individuals rather than between them. However, the reaction-norm intercepts, representing each individual's average urinary cortisol phenotype across the diurnal gradient, showed stable among-individual differences across ages. As outlined by Araya-Ajoy and colleagues [38–40,75], repeatability is formally defined at the population level, as the proportion of total variance attributable to consistent among-individual differences across repeated measures. Biologically, however, reaction-norm repeatability reflects the stability of individual-specific intercepts and slopes across contexts or time. In this sense, the moderate repeatability of intercepts in our data indicates that individuals maintained stable average cortisol phenotypes across ages, whereas the low repeatability of slopes suggests little evidence for consistent among-individual differences in diurnal plasticity. This suggests that individuals are fairly consistent in this trait expression as they mature. By contrast, reaction-norm slopes, capturing individual plasticity in response to time of day, showed weak repeatability, indicating that diurnal urinary cortisol secretion is highly flexible within individuals across development.

Taken together, these findings point to (i) a minor role of genetic and priming factors in shaping diurnal urinary cortisol phenotypes and (ii) a combination of phenotypic stability and plasticity in diurnal urinary cortisol reaction-norm traits. Bonobos maintain stable individual patterns in overall urinary cortisol expression, but the way they modulate urinary cortisol output in response to temporal cues remains flexible throughout ontogeny. This dissociation between stable intercepts and flexible slopes within individuals across years suggests that HPA axis regulation in bonobos combines a consistent phenotypic trait (reaction-norm intercept) with a flexible component (reaction-norm slope). The ability to adjust diurnal cortisol responses (varying degrees of plasticity), regardless of individuals' overall phenotypes (stable reaction-norm intercepts), may provide a mechanism for coping with fluctuating environmental conditions such as seasonal changes, social complexity or ecological pressures. In other words, this pattern may reflect an adaptive balance between physiological consistency and context-dependent flexibility in a long-lived species facing heterogeneous environments across its lifespan. The moderate repeatability of reaction-norm intercepts may indicate canalized, robust aspects of HPA axis function that develop early and persist throughout ontogeny, whereas the low repeatability of reaction-norm slopes suggests a capacity for reversible, context-dependent adjustment to temporal and environmental variation. Such a dual structure—stable baseline regulation paired with flexible modulation—may be particularly advantageous for long-lived species like bonobos, which experience predictable and dynamic elements in their ecological and social environment.

**Table 1.** Results of the random intercept and the random slope model. Both models contained the same fixed effects.

	random intercept model	random slope model
<b>fixed effects</b>	<b><math>\beta</math> (95% CI)</b>	<b><math>\beta</math> (95% CI)</b>
intercept	3.846 (3.570, 4.122)	3.845 (3.569, 4.121)
time of sample collection	−0.540 (−0.637, −0.443)	−0.531 (−0.632, −0.431)
community	0.121 (−0.032, 0.274)	0.119 (−0.034, 0.273)
age at sample collection	0.073 (−0.068, 0.214)	0.071 (−0.071, 0.213)
sex	−0.015 (−0.173, 0.143)	−0.015 (−0.170, 0.139)
age–sex interaction	0.361 (0.098, 0.625)	0.358 (0.095, 0.621)
<b>random effects</b>	<b><math>\beta</math> (s.d.)</b>	<b><math>\beta</math> (s.d.)</b>
variance <i>individual-at-age</i> intercept	0.036 (0.189)	0.035 (0.18573)
variance <i>individual-at-age</i> slope for time		0.030 (0.17177)
variance individual intercept	0.038 (0.196)	0.038 (0.19491)
variance individual slope for time		0.005 (0.06677)
variance year intercept	0.243 (0.493)	0.244 (0.49399)
variance month intercept	0.018 (0.134)	0.018 (0.13427)
residual variance	0.999 (0.9995)	0.992 (0.99576)

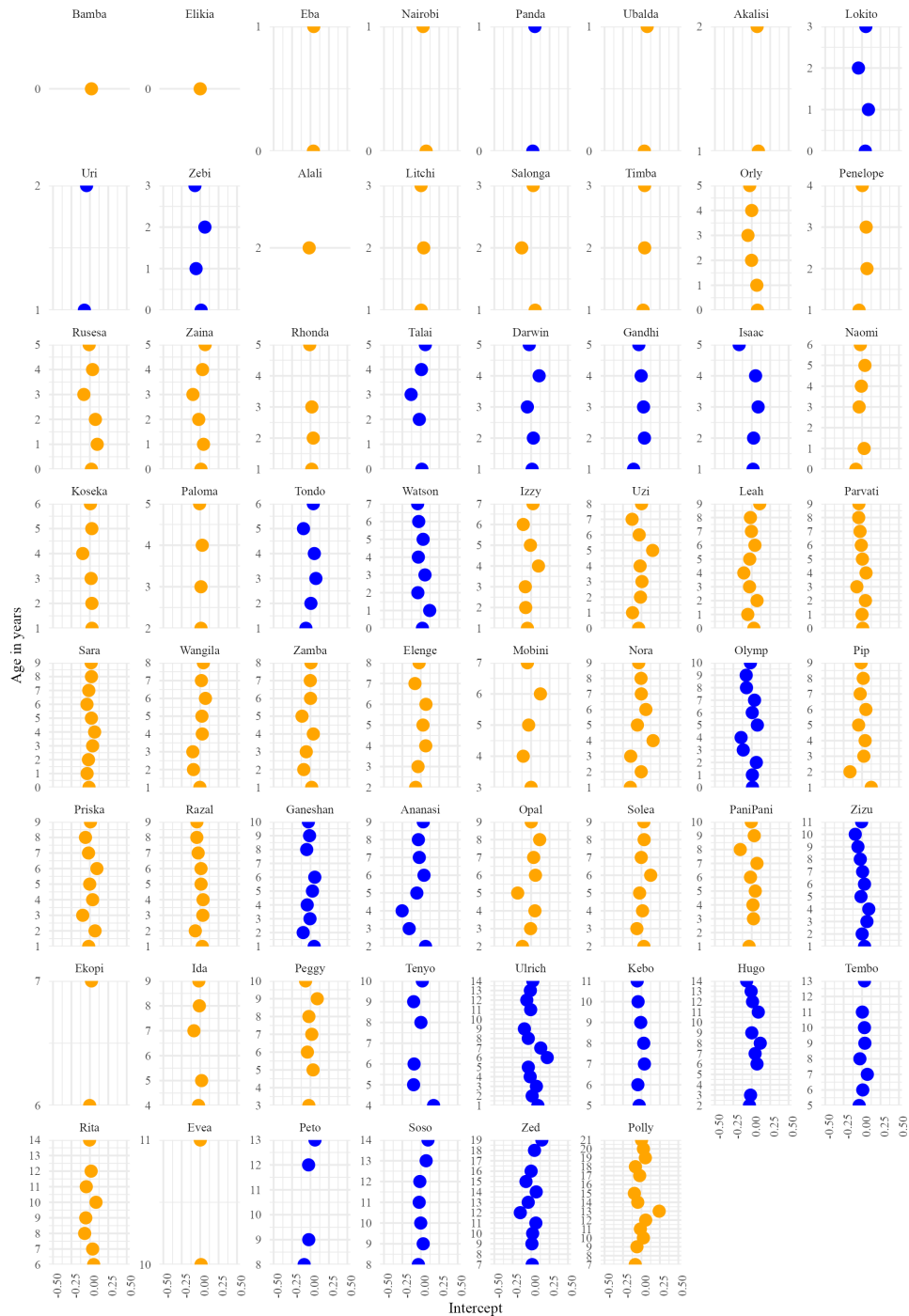
### (a) Within-individual variation in urinary cortisol levels exceeds between-individual variation (low trait repeatability)

Our results show that the majority of variance in urinary cortisol concentrations is attributable to within-individual variation, with only a small proportion explained by stable between-individual differences. This implies that urinary cortisol levels in wild bonobos exhibit low trait repeatability, meaning that individuals do not differ consistently from each other in their urinary cortisol output. This finding is consistent with a growing body of literature demonstrating that GC measures often lack strong repeatability across time and contexts [18,37,76]. Schoenemann & Bonier [76] found that average repeatability estimates for baseline GC levels across vertebrate taxa were modest ( $R \approx 0.23$ ), and similarly low for integrated measures such as urinary, faecal or feather GCs ( $R \approx 0.32$ ). Such values reflect a relatively small proportion of variance attributable to stable between-individual differences. This pattern was especially pronounced in birds and mammals, including primates, whereas amphibians showed somewhat higher repeatability.

In our bonobo dataset, the low trait repeatability estimate of  $R = 0.03$  reflects a high degree of within-individual variation in urinary cortisol levels relative to lower between-individual variation. From a physiological perspective, this is consistent with the role of GCs in mediating allostasis—dynamic adjustments to changing social and ecological demands [77,78]. Thus, cortisol levels may vary more in response to immediate environmental conditions than reflect stable between-individual differences. This high within-individual flexibility may limit the utility of GC levels as indicators of individual, ‘selective traits’, unless multiple measurements are obtained across standardized contexts. Low repeatability also constrains the potential heritability of GC phenotypes, since repeatability sets an upper bound for heritability estimates [38,75]. Consequently, although GCs play key roles in physiology and behaviour, the evolutionary potential of average GC levels may be limited unless more stable individual differences (e.g. in HPA axis reactivity or feedback sensitivity) are captured by other metrics, such as repeatable reaction norms.

### (b) Ontogenetic stability in cortisol intercepts

The moderate repeatability of reaction-norm intercepts ( $R = 0.58$ ) across ontogeny suggests that individual bonobos express relatively stable average urinary cortisol phenotypes over time. While we found no evidence for trait repeatability in diurnal urinary cortisol reaction norms (that is, persistent between-individual differences), the consistency in intercepts indicates that individuals tend to maintain similar average phenotypes across developmental stages. Similar patterns have been reported in other species. For instance, in humans, measures such as the cortisol awakening response and diurnal area under the curve show moderate temporal stability from early childhood into adolescence (e.g. [79–81]). In other primates, including Assamese macaques and chimpanzees, early maternal and environmental influences appear to shape long-term HPA axis activity, potentially anchoring endocrine phenotypes through developmental programming [32,35]. Such patterns align with recent proposals that GC phenotypes may exhibit ontogenetically constrained plasticity—stable within individuals but sensitive to early-life cues—thereby contributing to individual specialization in coping strategies [4]. Within this framework, the repeatability of intercepts does not imply that individuals are inflexible or canalized. Rather, it reflects the fact that individuals tend to express similar endocrine baselines (in the statistical sense of a reaction-norm intercept) across life stages, even if their responsiveness to immediate challenges (i.e. plasticity or slope) remains variable. According to Dantzer [4], such stability in the structure of endocrine phenotypes could reflect phenotypic integration across life-history stages, shaped by intrinsic developmental dynamics and early environmental inputs. The HPA axis, viewed as a central hub within a larger regulatory

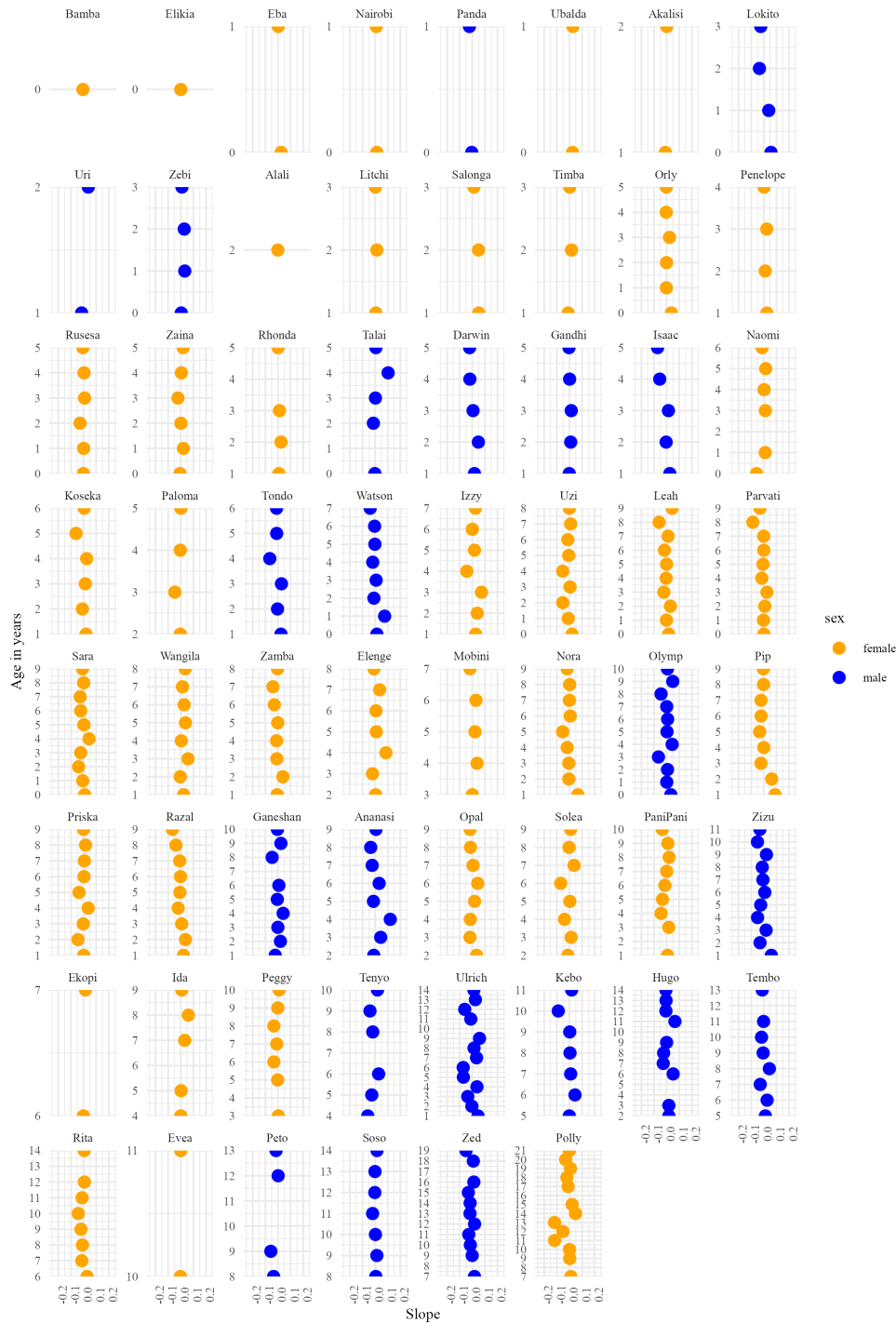


**Figure 1.** Reaction-norm intercepts of maturing bonobos. The  $x$ -axis shows the individual intercept estimate (low to high intercepts) for a given age (as shown on the  $y$ -axis). Each box represents an individual, with males in blue and females in yellow. The intercepts for each individual at a given age were extracted from the random slope model. Although there is variation in intercepts within individuals, bonobos display relatively stable urinary cortisol intercepts across age, suggesting that individuals show repeatable reaction-norm intercepts during ontogeny.

network, may contribute to this integration by aligning endocrine outputs with individual state and developmental timing. Thus, our findings support the notion that urinary cortisol intercepts may represent a developmentally organized component of the endocrine phenotype—stable within individuals but not necessarily distinct between them—and may provide a foundation for exploring the eco-evolutionary significance of HPA axis profiles in natural populations.

### (c) Within-individual plasticity in diurnal slopes

The low repeatability of reaction-norm slopes across ontogeny indicates that diurnal plasticity in cortisol secretion is not a stable individual characteristic in bonobos, but rather a flexible component of HPA axis functioning which changes over time. This finding aligns with the notion that plasticity itself may be developmentally labile, particularly during the extended ontogeny in long-lived species, such as bonobos [4,82]. The ability to adjust diurnal cortisol dynamics across developmental stages may reflect physiological recalibration in response to shifting ecological and social contexts, without altering overall cortisol output. By contrast to the stability observed in reaction-norm intercepts, which probably reflect longer-term developmental organization, diurnal slope plasticity appears more sensitive to transient factors. This pattern is consistent with previous studies



**Figure 2.** Reaction-norm slopes of maturing bonobos. The *x*-axis shows the individual slope estimate (steep to flat slopes) for a given age (as shown on the *y*-axis). Each box represents an individual, where male individuals are coloured in blue and female individuals are depicted in yellow. Slopes for each individual at a given age were extracted from the random slope model. Unlike the reaction-norm intercepts, the slopes of urinary cortisol levels across age are not repeatable within individuals, which suggests a high level of plasticity in cortisol responses to the circadian gradient within individuals and consequently high individual flexibility in this trait during ontogeny.

in humans and non-human primates, where diurnal slopes exhibit lower temporal stability than average phenotypes of GC level expression [18,79,81]. In bonobos and other great apes, diurnal slopes have been shown to flatten under conditions of social disruption, such as group transfers, and steepen under routine conditions [83], suggesting that slope plasticity reflects acute, state-dependent regulation.

Due to convergence issues, we were unable to retain the correlation term between intercepts and slopes in our model, and therefore cannot directly assess whether individual average urinary cortisol phenotypes are statistically linked to plasticity in diurnal slope expression within our dataset. However, a recent study on adult bonobos found no correlation between reaction-norm intercepts and slopes [22], suggesting that these components of endocrine regulation may vary independently. Although we could not formally test this relationship here, our findings mirror this broader pattern: intercepts were moderately repeatable across ontogeny, while slopes showed low repeatability, implying that stable average urinary cortisol levels and flexible diurnal modulation may represent distinct aspects of HPA axis regulation. Reaction-norm frameworks are particularly

suiting to decomposing such multidimensional traits [4,20,38], and future longitudinal studies across life stages may clarify whether intercept–slope independence holds consistently across contexts and developmental trajectories.

State-dependent plasticity in reaction-norm slope expression has been linked to ecologically and fitness-relevant outcomes in other taxa. In red squirrels and house sparrows, for example, individuals differ consistently in their GC reaction-norm slopes under repeated challenges, with steeper responsiveness predicting enhanced survival or reproductive success under certain conditions [16,84]. While bonobos inhabit relatively stable environments compared with these species [53], their complex social structures and developmental transitions may impose equally salient regulatory demands, calling for temporally fine-tuned endocrine responses [54,56].

Together, our findings suggest that while bonobos maintain stable reaction-norm intercepts across ontogeny, their diurnal cortisol plasticity remains flexible and responsive to environmental and maturational factors. This dissociation between stability in reaction-norm intercepts and flexibility in diurnal dynamics supports recent calls to decompose endocrine traits into component dimensions—such as within-individual average phenotypes and within-individual slopes—to better understand physiological regulation under naturalistic conditions [4,34,38]. Our results thus underscore the need to consider not only average hormone levels, but how they change over time and in response to environmental cues, particularly across critical developmental periods.

## 5. Conclusions

In conclusion, our results demonstrate that GC phenotypes in wild bonobos reflect both physiological consistency and developmental plasticity. While average cortisol phenotypes appear moderately repeatable throughout development, probably due to anatomical or physiological constraints such as receptor density or feedback mechanism, cortisol plasticity in response to the circadian gradient varies markedly within individuals across ontogeny. This dissociation between stability and flexibility suggests that the HPA axis can provide both long-term consistency and short-term adaptability, enabling individuals to maintain physiological homeostasis while responding to environmental or social fluctuations. Individual bonobos with both high and low cortisol intercepts showed varying degrees of plasticity in their cortisol levels along the diurnal gradient. These findings highlight the utility of using reaction-norm approaches in capturing the complex dynamics of hormone regulation across ontogeny. Finally, by adopting a reaction-norm framework in a wild primate population, our findings contribute to a growing body of research emphasizing the ecological relevance of hormone plasticity and the developmental processes shaping traits in natural settings. But, to what extent are intercepts and slopes predictive of fitness-relevant outcomes such as resilience, survival or reproductive success? Addressing these questions will require integrative, longitudinal designs that link endocrine profiles to ecological, behavioural and life-history data. Expanding this approach to additional hormonal axes and species will be essential to better understand how physiological flexibility evolves and operates across taxa. In the context of rapid environmental change, such work is critical for identifying mechanisms of resilience in long-lived species like bonobos.

**Ethics.** We followed a strict protocol to ensure a non-invasive, non-contact approach with wild animals. Research in LuiKotale, Salonga National Park, Democratic Republic of Congo, is authorized by the Institut Congolaise pour la Conservation de la Nature (ICCN) under Permission Number: 0683/ICCN/DG/ADG/014/KV/2012. Additionally, the ICCN has granted permits (Permit Number: 0521/ICCN/DG/CWB/05/01/2014) for the export of urine samples from the Democratic Republic of Congo, while the German Ministry for Social Affairs and Consumer Protection has issued the necessary permits for their importation.

**Data accessibility.** The dataset and analysis code supporting this study are publicly available in the Göttingen Research Online (GRO) repository [85]. Supplementary material is available online [86].

**Declaration of AI use.** AI was used to improve readability and language of some sections of the manuscript and to aid with developing R code to visualize (plot) results.

**Authors' contributions.** R.S.: conceptualization, formal analysis, investigation, visualization, writing—original draft; G.H.: conceptualization, funding acquisition, investigation, project administration, resources, writing—review and editing; T.D.: methodology, validation, writing—review and editing; B.F.: funding acquisition, project administration, resources, writing—review and editing; M.H.: methodology, validation, writing—review and editing; V.B.: conceptualization, funding acquisition, investigation, methodology, validation, writing—review and editing.

All authors gave final approval for publication and agreed to be held accountable for the work performed therein.

**Conflict of interest declaration.** We declare we have no competing interests.

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## References

1. Fordyce JA. 2006 The evolutionary consequences of ecological interactions mediated through phenotypic plasticity. *J. Exp. Biol.* **209**, 2377–2383. (doi:10.1242/jeb.02271)

2. Taborsky B. 2017 Chapter three - developmental plasticity: preparing for life in a complex world. In *Advances in the study of behavior* (eds M Naguib, J Podos, LW Simmons, L Barrett, SD Healy, M Zuk), pp. 49–99. Cambridge, MA: Academic Press. (doi:10.1016/bs.asb.2016.12.002)
3. Chevin LM, Lande R. 2010 When do adaptive plasticity and genetic evolution prevent extinction of a density-regulated population? *Evolution* **64**, 1143–1150. (doi:10.1111/j.1558-5646.2009.00875.x)
4. Dantzer B. 2023 Frank Beach award winner: the centrality of the hypothalamic-pituitary-adrenal axis in dealing with environmental change across temporal scales. *Horm. Behav.* **150**, 105311. (doi:10.1016/j.yhbeh.2023.105311)
5. Nepomnaschy P, Flinn M. 2009 Early life influences on the ontogeny of the neuroendocrine stress response in the human child. In *Endocrinology of social relationships* (eds PT Ellison, PB Gray), pp. 364–382. Cambridge, MA: Harvard University Press. (doi:10.2307/j.ctv22jns8.19)
6. Snell-Rood EC. 2013 An overview of the evolutionary causes and consequences of behavioural plasticity. *Anim. Behav.* **85**, 1004–1011. (doi:10.1016/j.anbehav.2012.12.031)
7. Botero CA, Weissing FJ, Wright J, Rubenstein DR. 2015 Evolutionary tipping points in the capacity to adapt to environmental change. *Proc. Natl Acad. Sci. USA* **112**, 184–189. (doi:10.1073/pnas.1408589111)
8. Leimar O, McNamara JM. 2015 The evolution of transgenerational integration of information in heterogeneous environments. *Am. Nat.* **185**, E55–69. (doi:10.1086/679575)
9. Piersma T, Drent J. 2003 Phenotypic flexibility and the evolution of organismal design. *Trends Ecol. Evol.* **18**, 228–233. (doi:10.1016/s0169-5347(03)00036-3)
10. Lu A, Petrucci L, Carrera S, Feder J, Schneider-Crease I, Snyder-Mackler N. 2019 Developmental responses to early-life adversity: evolutionary and mechanistic perspectives. *Evol. Anthropol. Issues News Rev.* **28**, 249–266. (doi:10.1002/evan.21791)
11. Whitman D, Agrawal A. 2009 What is phenotypic plasticity and why is it important? In *Phenotypic plasticity of insects: mechanisms and consequences* (ed. DW Whitman). Boca Raton, FL: Science Publishers Inc. (doi:10.1201/b10201-2)
12. Sheriff MJ, Love OP. 2013 Determining the adaptive potential of maternal stress. *Ecol. Lett.* **16**, 271–280. (doi:10.1111/ele.12042)
13. Gotthard K, Nylin S. 1995 Adaptive plasticity and plasticity as an adaptation: a selective review of plasticity in animal morphology and life history. *Oikos* **74**, 3–17. (doi:10.2307/3545669)
14. Gomez-Mestre I, Saccoccio VL, Iijima T, Collins EM, Rosenthal GG, Warkentin KM. 2010 The shape of things to come: linking developmental plasticity to post-metamorphic morphology in anurans. *J. Evol. Biol.* **23**, 1364–1373. (doi:10.1111/j.1420-9101.2010.02016.x)
15. Blanckenhorn WU. 1998 Adaptive phenotypic plasticity in growth, development, and body size in the yellow dung fly. *Evolution* **52**, 1394–1407. (doi:10.1111/j.1558-5646.1998.tb02021.x)
16. Guindre-Parker S. 2020 Individual variation in glucocorticoid plasticity: considerations and future directions. *Integr. Comp. Biol.* **60**, 79–88. (doi:10.1093/icb/icaa003)
17. Hau M, Deimel C, Moiron M. 2022 Great tits differ in glucocorticoid plasticity in response to spring temperature. *Proc. R. Soc. B* **289**, 20221235. (doi:10.1098/rspb.2022.1235)
18. Sonnweber R *et al.* 2018 Circadian rhythms of urinary cortisol levels vary between individuals in wild male chimpanzees: a reaction norm approach. *Front. Ecol. Evol.* **6**, 1–11. (doi:10.3389/fevo.2018.00085)
19. Angelier F, Wingfield JC. 2013 Importance of the glucocorticoid stress response in a changing world: theory, hypotheses and perspectives. *Gen. Comp. Endocrinol.* **190**, 118–128. (doi:10.1016/j.ygcen.2013.05.022)
20. Hau M, Casagrande S, Ouyang JQ, Baugh AT. 2016 Chapter two - glucocorticoid-mediated phenotypes in vertebrates: multilevel variation and evolution. In *Advances in the study of behavior* (eds JCM Marc Naguib, LW Simmons, L Barrett, S Healy, M Zuk), pp. 41–115. Cambridge, MA: Academic Press. (doi:10.1016/bs.asb.2016.01.002)
21. Schmidt KL, MacDougall-Shackleton EA, Soma KK, MacDougall-Shackleton SA. 2014 Developmental programming of the HPA and HPG axes by early-life stress in male and female song sparrows. *Gen. Comp. Endocrinol.* **196**, 72–80. (doi:10.1016/j.ygcen.2013.11.014)
22. Sonnweber R, Hohmann G, Stevens JMG, Deschner T, Fruth B, Fiedler AL, Nurmi NO, Behringer V. 2023 Average phenotype but not plasticity in two metabolic hormones covary in wild female bonobos (*Pan paniscus*). *Front. Ecol. Evol.* **11**, 1–12. (doi:10.3389/fevo.2023.1300003)
23. Crespi EJ, Williams TD, Jessop TS, Delehanty B. 2013 Life history and the ecology of stress: how do glucocorticoid hormones influence life-history variation in animals? *Funct. Ecol.* **27**, 93–106. (doi:10.1111/1365-2435.12009)
24. Anisman H, Zaharia MD, Meaney MJ, Merali Z. 1998 Do early-life events permanently alter behavioral and hormonal responses to stressors? *Int. J. Dev. Neurosci.* **16**, 149–164. (doi:10.1016/s0736-5748(98)00025-2)
25. Kentner AC, Cryan JF, Brummelte S. 2019 Resilience priming: translational models for understanding resiliency and adaptation to early life adversity. *Dev. Psychobiol.* **61**, 350–375. (doi:10.1002/dev.21775)
26. Berghänel A, Heistermann M, Schülke O, Ostner J. 2016 Prenatal stress effects in a wild, long-lived primate: predictive adaptive responses in an unpredictable environment. *Proc. R. Soc. B* **283**, 20161304. (doi:10.1098/rspb.2016.1304)
27. Berghänel A, Heistermann M, Schülke O, Ostner J. 2017 Prenatal stress accelerates offspring growth to compensate for reduced maternal investment across mammals. *Proc. Natl Acad. Sci. USA* **114**, E10658–E10666. (doi:10.1073/pnas.1707152114)
28. Eachus H, Ryu S. 2024 Glucocorticoid effects on the brain: from adaptive developmental plasticity to allostatic overload. *J. Exp. Biol.* **227**, 246128. (doi:10.1242/jeb.246128)
29. Moisiadis VG, Matthews SG. 2014 Glucocorticoids and fetal programming part 1: outcomes. *Nat. Rev. Endocrinol.* **10**, 391–402. (doi:10.1038/nrendo.2014.73)
30. Carrera SC, Godoy I, Gault CM, Mensing A, Damm J, Perry SE, Beehner JC. 2025 Stress responsiveness in a wild primate predicts survival across an extreme El Niño drought. *Sci. Adv.* **11**, q5020. (doi:10.1126/sciadv.adq5020)
31. Taff CC, Baldan D, Mentasana L, Ouyang JQ, Vitousek MN, Hau M. 2024 Endocrine flexibility can facilitate or constrain the ability to cope with global change. *Phil. Trans. R. Soc. B* **379**, 20220502. (doi:10.1098/rstb.2022.0502)
32. Anzà S, Heistermann M, Ostner J, Schülke O. 2025 Early prenatal but not postnatal glucocorticoid exposure is associated with enhanced HPA axis activity into adulthood in a wild primate. *Proc. R. Soc. B* **292**, 20242418. (doi:10.1098/rspb.2024.2418)
33. Girard-Buttoz C *et al.* 2021 Early maternal loss leads to short- but not long-term effects on diurnal cortisol slopes in wild chimpanzees. *eLife* **10**, e64134. (doi:10.7554/eLife.64134)
34. Malkoc K, Mentasana L, Casagrande S, Hau M. 2021 Quantifying glucocorticoid plasticity using reaction norm approaches: there still is so much to discover! *Integr. Comp. Biol.* **62**, 58–70. (doi:10.1093/icb/icab196)
35. Tkaczynski PJ *et al.* 2020 Patterns of urinary cortisol levels during ontogeny appear population specific rather than species specific in wild chimpanzees and bonobos. *J. Hum. Evol.* **147**, 102869. (doi:10.1016/j.jhevol.2020.102869)
36. Mentasana L, Hau M. 2022 Glucocorticoids in a warming world: do they help birds to cope with high environmental temperatures? *Horm. Behav.* **142**, 105178. (doi:10.1016/j.yhbeh.2022.105178)
37. Ouyang JQ, Hau M, Bonier F. 2011 Within seasons and among years: when are corticosterone levels repeatable? *Horm. Behav.* **60**, 559–564. (doi:10.1016/j.yhbeh.2011.08.004)

38. Araya-Ajoy YG, Mathot KJ, Dingemanse NJ. 2015 An approach to estimate short-term, long-term and reaction norm repeatability. *Methods Ecol. Evol.* **6**, 1462–1473. (doi:10.1111/2041-210x.12430)
39. Dingemanse NJ, Kazem AJN, Réale D, Wright J. 2010 Behavioural reaction norms: animal personality meets individual plasticity. *Trends Ecol. Evol.* **25**, 81–89. (doi:10.1016/j.tree.2009.07.013)
40. Dingemanse NJ, Dochtermann NA. 2013 Quantifying individual variation in behaviour: mixed-effect modelling approaches. *J. Anim. Ecol.* **82**, 39–54. (doi:10.1111/1365-2656.12013)
41. Bell AM, Hankinson SJ, Laskowski KL. 2009 The repeatability of behaviour: a meta-analysis. *Anim. Behav.* **77**, 771–783. (doi:10.1016/j.anbehav.2008.12.022)
42. Nader N, Chrousos GP, Kino T. Interactions of the circadian CLOCK system and the HPA axis. *Trends Endocrinol. Metab.* **21**, 277–286. (doi:10.1016/j.tem.2009.12.011)
43. Nicolaïdes NC, Charmandari E, Chrousos GP, Kino T. 2014 Circadian endocrine rhythms: the hypothalamic–pituitary–adrenal axis and its actions. *Ann. NY Acad. Sci.* **1318**, 71–80. (doi:10.1111/nyas.12464)
44. de Weerth C, van Hees Y, Buitelaar JK. 2003 Prenatal maternal cortisol levels and infant behavior during the first 5 months. *Early Hum. Dev.* **74**, 139–151. (doi:10.1016/s0378-3782(03)00088-4)
45. Santarelli S *et al.* 2017 An adverse early life environment can enhance stress resilience in adulthood. *Psychoneuroendocrinology* **78**, 213–221. (doi:10.1016/j.psyneuen.2017.01.021)
46. Petrucci LA, Mandalaywala TM, Parker KJ, Maestriperi D, Higham JP. 2016 Effects of early life adversity on cortisol/salivary alpha-amylase symmetry in free-ranging juvenile rhesus macaques. *Horm. Behav.* **86**, 78–84. (doi:10.1016/j.yhbeh.2016.05.004)
47. Behringer V, Deschner T, Deimel C, Stevens JMG, Hohmann G. 2014 Age-related changes in urinary testosterone levels suggest differences in puberty onset and divergent life history strategies in bonobos and chimpanzees. *Horm. Behav.* **66**, 525–533. (doi:10.1016/j.yhbeh.2014.07.011)
48. Behringer V, Deschner T, Murtagh R, Stevens JMG, Hohmann G. 2014 Age-related changes in thyroid hormone levels of bonobos and chimpanzees indicate heterochrony in development. *J. Hum. Evol.* **66**, 83–88. (doi:10.1016/j.jhevol.2013.09.008)
49. Berghänel A, Stevens JMG, Hohmann G, Deschner T, Behringer V. 2023 Adolescent length growth spurts in bonobos and other primates: mind the scale. *Elife* **12**, e86635. (doi:10.7554/eLife.86635)
50. Kreyer M, Behringer V, Deimel C, Fruth B. 2023 Neopterin levels in bonobos vary seasonally and reflect symptomatic respiratory infections. *EcoHealth* **20**, 93–104. (doi:10.1007/s10393-023-01633-y)
51. Bessone M, Booto L, Santos AR, Kühl HS, Fruth B. 2021 No time to rest: how the effects of climate change on nest decay threaten the conservation of apes in the wild. *PLoS One* **16**, e0252527. (doi:10.1371/journal.pone.0252527)
52. Moscovice LR, Douglas PH, Martínez-Iñigo L, Surbeck M, Vigilant L, Hohmann G. 2017 Stable and fluctuating social preferences and implications for cooperation among female bonobos at LuiKotale, Salonga National Park, DRC. *Am. J. Phys. Anthropol.* **163**, 158–172. (doi:10.1002/ajpa.23197)
53. Oelze VM, Douglas PH, Stephens CR, Surbeck M, Behringer V, Richards MP, Fruth B, Hohmann G. 2016 The steady state great ape? Long term isotopic records reveal the effects of season, social rank and reproductive status on bonobo feeding behavior. *PLoS One* **11**, e0162091. (doi:10.1371/journal.pone.0162091)
54. Surbeck M, Deschner T, Weltring A, Hohmann G. 2012 Social correlates of variation in urinary cortisol in wild male bonobos (*Pan paniscus*). *Horm. Behav.* **62**, 27–35. (doi:10.1016/j.yhbeh.2012.04.013)
55. Nurmi NO, Sonnweber R, Schülke O, Moscovice LR, Deschner T, Hohmann G. 2023 Bonobo mothers have elevated urinary cortisol levels during early but not mid or late lactation. *Primates* **64**, 215–225. (doi:10.1007/s10329-022-01044-7)
56. Behringer V, Berghänel A, Deschner T, Lee SM, Fruth B, Hohmann G. 2022 Transition to siblinghood causes a substantial and long-lasting increase in urinary cortisol levels in wild bonobos. *eLife* **11**, e77227. (doi:10.7554/eLife.77227)
57. Hauser B, Mugisha L, Preis A, Deschner T. 2011 LC-MS analysis of androgen metabolites in serum and urine from east African chimpanzees (*Pan troglodytes schweinfurthii*). *Gen. Comp. Endocrinol.* **170**, 92–98. (doi:10.1016/j.ygcen.2010.09.012)
58. Hauser B, Deschner T, Boesch C. 2008 Development of a liquid chromatography–tandem mass spectrometry method for the determination of 23 endogenous steroids in small quantities of primate urine. *J. Chromatogr. B* **862**, 100–112. (doi:10.1016/j.jchromb.2007.11.009)
59. Wessling EG, Kühl HS, Mundry R, Deschner T, Pruetz JD. 2018 The costs of living at the edge: seasonal stress in wild savanna-dwelling chimpanzees. *J. Hum. Evol.* **121**, 1–11. (doi:10.1016/j.jhevol.2018.03.001)
60. Miller RC, Brindle E, Holman DJ, Shofer J, Klein NA, Soules MR, O'Connor KA. 2004 Comparison of specific gravity and creatinine for normalizing urinary reproductive hormone concentrations. *Clin. Chem.* **50**, 924–932. (doi:10.1373/clinchem.2004.032292)
61. Hanneman SK, Cox CD, Green KE, Kang DH. 2011 Estimating intra- and inter-assay variability in salivary cortisol. *Biol. Res. Nurs.* **13**, 243–250. (doi:10.1177/1099800411404061)
62. Schaebs FS. 2017 *Social and ecological correlates of male capuchin endocrinology*. Leipzig, Germany: Universität Leipzig. See [https://pure.mpg.de/pubman/faces/ViewItemOverviewPage.jsp?itemId=item\\_2491819](https://pure.mpg.de/pubman/faces/ViewItemOverviewPage.jsp?itemId=item_2491819).
63. Mastorakos G, Ilias I. 2009 Maternal and fetal hypothalamic-pituitary-adrenal axes during pregnancy and postpartum. *Ann. NY Acad. Sci.* **997**, 136–149. (doi:10.1196/annals.1290.016)
64. Gelman A. 2008 Scaling regression inputs by dividing by two standard deviations. *Stat. Med.* **27**, 2865–2873. (doi:10.1002/sim.3107)
65. R Core Team. 2023 R: A language and environment for statistical computing. Vienna, Austria: R Foundation for Statistical Computing. See <https://www.R-project.org/>.
66. RStudio Team. 2020 RStudio: integrated development for R. Boston, MA, USA: RStudio. See <http://www.rstudio.com/>.
67. Nussey DH, Wilson AJ, Brommer JE. 2007 The evolutionary ecology of individual phenotypic plasticity in wild populations. *J. Evol. Biol.* **20**, 831–844. (doi:10.1111/j.1420-9101.2007.01300.x)
68. Bates D, Mächler M, Bolker B, Walker S. 2014 Fitting linear mixed-effects models using lme4. *J. Stat. Softw.* **67**, 01. (doi:10.18637/jss.v067.i01)
69. Bolker BM, Brooks ME, Clark CJ, Geange SW, Poulsen S JR, Stevens MH, White JS. 2009 Generalized linear mixed models: a practical guide for ecology and evolution. *Trends Ecol. Evol.* **24**, 127–135. (doi:10.1016/j.tree.2008.10.008)
70. Fox GA, Negrete-Yankelevich S, Sosa VJ (eds). 2015 *Ecological statistics: contemporary theory and application*, Illustrated. Oxford, UK: Oxford University Press.
71. West BT, Welch KB, Galecki AT. 2022 *Linear mixed models: a practical guide using statistical software*. New York, NY: CRC Press.
72. Huelsenbeck JP, Rannala B. 1997 Phylogenetic methods come of age: testing hypotheses in an evolutionary context. *Science* **276**, 227–232. (doi:10.1126/science.276.5310.227)
73. Fox J, Weisberg S. 2018 *An R companion to applied regression*. Thousand Oaks, CA: SAGE Publications.
74. Nakagawa S, Schielzeth H. 2010 Repeatability for Gaussian and non-Gaussian data: a practical guide for biologists. *Biol. Rev.* **85**, 935–956. (doi:10.1111/j.1469-185x.2010.00141.x)
75. Araya-Ajoy YG, Dingemanse NJ. 2017 Repeatability, heritability, and age-dependence of seasonal plasticity in aggressiveness in a wild passerine bird. *J. Anim. Ecol.* **86**, 227–238. (doi:10.1111/1365-2656.12621)

76. Schoenemann KL, Bonier F. 2018 Repeatability of glucocorticoid hormones in vertebrates: a meta-analysis. *PeerJ* **6**, 4398. (doi:10.7717/peerj.4398)
77. McEwen BS, Wingfield JC. 2010 What is in a name? Integrating homeostasis, allostasis and stress. *Horm. Behav.* **57**, 105–111. (doi:10.1016/j.yhbeh.2009.09.011)
78. Romero LM, Dickens MJ, Cyr NE. 2009 The reactive scope model — a new model integrating homeostasis, allostasis, and stress. *Horm. Behav.* **55**, 375–389. (doi:10.1016/j.yhbeh.2008.12.009)
79. Platje E, Vermeiren RRJM, Branje SJT, Doreleijers TAH, Meeus WHJ, Koot HM, Frijns T, van Lier PAC, Jansen LMC. 2013 Long-term stability of the cortisol awakening response over adolescence. *Psychoneuroendocrinology* **38**, 271–280. (doi:10.1016/j.psyneuen.2012.06.007)
80. Rotenberg S, McGrath JJ, Roy-Gagnon MH, Tu MT. 2012 Stability of the diurnal cortisol profile in children and adolescents. *Psychoneuroendocrinology* **37**, 1981–1989. (doi:10.1016/j.psyneuen.2012.04.014)
81. Shirtcliff EA, Allison AL, Armstrong JM, Slattery MJ, Kalin NH, Essex MJ. 2012 Longitudinal stability and developmental properties of salivary cortisol levels and circadian rhythms from childhood to adolescence. *Dev. Psychobiol.* **54**, 493–502. (doi:10.1002/dev.20607)
82. Dupont L, Thierry M, Zinger L, Legrand D, Jacob S. 2024 Beyond reaction norms: the temporal dynamics of phenotypic plasticity. *Trends Ecol. Evol.* **39**, 2023. (doi:10.1016/j.tree.2023.08.014)
83. Behringer V, Stevens JMG, Sonnweber R. 2022 Salivary cortisol reaction norms in zoo-housed great apes: diurnal slopes and intercepts as indicators of stress response quality. *Anim. (Basel)* **12**, 522. (doi:10.3390/ani12040522)
84. Baldan D, Negash M, Ouyang JQ. 2021 Are individuals consistent? Endocrine reaction norms under different ecological challenges. *J. Exp. Biol.* **224**, b240499. (doi:10.1242/jeb.240499)
85. Behringer V. 2025 Replication data for: ontogeny of cortisol reaction norms in wild bonobos (*Pan paniscus*). *GRO* (doi:10.25625/HHE8V7)
86. Sonnweber R, Hohmann G, Deschner T, Fruth B, Heistermann M, Behringer V. 2026 Supplementary material from: Ontogeny of cortisol reaction norms in wild bonobos (*Pan paniscus*). Figshare. (doi:10.6084/m9.figshare.c.8243462)