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Late pregnancy glucocorticoid levels predict responsiveness in wild baboon mothers (*Papio cynocephalus*)

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Maternal care is the most significant measure of successful adaptation among female mammals. Understanding the predictors of individual differences in offspring care is a major objective of mammalian reproductive biology. Recent studies suggest that differences in caregiving motivation may be associated with variation in glucocorticoid (GC) hormones in new mothers. Despite these intriguing reports, questions remain about the stability of this association during a period of rapid change in both behaviour and physiology, about whether this relationship is dependent on other nonhormonal variables and about the generality of this pattern across species and in wild populations. Glucocorticoids modulate animals' responses to ongoing stressors and may also prepare animals for predictable future challenges. We evaluated evidence for both actions of GCs on maternal responsiveness towards infant cries during the first 2 months of infancy in 34 wild baboon mother–infant dyads in Amboseli, Kenya. We found that stable individual differences in faecal GCs during late pregnancy predicted stable individual differences in maternal responsiveness after birth, even after controlling for maternal rank and parity, and infant sex and distress rate. This study is among the first to provide evidence of preparative actions of GCs in wild animals and to show stability of behavioural and hormonal traits during a period of rapid changes in both hormones and behaviour. Because elevations in GCs during late pregnancy are probably primarily of fetal rather than maternal origin, our results raise the intriguing possibility that parent–offspring conflict may underlie the preparative actions of GCs on maternal responsiveness to infant distress.

Keywords: corticosteroid; hormone; hypothalamic–pituitary–adrenal axis; infancy; maternal care; modulating action; mother–infant relationship; parenting; *Papio cynocephalus*; preparative action

Maternal care is possibly the single most significant measure of successful adaptation by female mammals (Rosenblatt 1995). Mammalian infants are unusually dependent on mothers for growth and survival (Clutton-Brock 1991), and variation in offspring care can dramatically affect the fitness of both offspring and mothers (Fairbanks 1996). A major goal of mammalian reproductive biology is to understand the predictors and causes of individual differences in mothering behaviour. These differences may be associated with offspring sex (Lee & Moss 1986) or with variation in mothers' age and experience (Wang & Novak 1992), physical condition (Cameron & Linklater 2000) or social status (Clutton-Brock et al. 1986). More recently, studies of human mothers and of captive and laboratory populations of nonhuman mammals suggest that differences in mothering behaviour may be associated with variation among mothers in the hormones of pregnancy, parturition or lactation (Fleming et al. 1997b; Stallings et al. 2001; Bardi et al. 2003; Dwyer et al. 2004; Rees et al. 2004).

One class of hormones that has recently begun to receive attention for its potential role in mediating parent–offspring interactions is glucocorticoids (GCs).

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Glucocorticoids, steroid hormones secreted from the adrenal cortex, are released as part of the vertebrate stress response, a suite of behavioural and physiological events that help animals mobilize energetic resources to cope with and respond to challenging or stressful stimuli (Sapolsky et al. 2000). GCs also appear to be critical for the development of caregiving motivation in female mammals (Fleming et al. 1997b; Stallings et al. 2001; Rees et al. 2004) and are positively associated with pup feeding rates by males in one cooperatively breeding mammal species (Carlson et al. 2006).

Despite mounting evidence of the importance of GCs on infant–caretaker interactions, the specific actions of GCs on caregiving motivation remain poorly understood. To date, two classes of GC action have been described: modulating actions, or those that affect an organism's immediate response to a stressor, and preparative actions, or those that mediate the organism's response to a future challenge (Sapolsky et al. 2000). While the modulating actions of GCs on physiology and behaviour (e.g. pair bonding: DeVries et al. 1995; infant care: Rees et al. 2004) have been described, the preparative actions of GCs remain poorly understood (Sapolsky et al. 2000; Romero 2002). To our knowledge, no prior study has explored the dual nature of GC action on a single behavioural or physiological system, or found evidence for preparative actions of GCs in wild animals.

Elevations of GCs in anticipation of future challenges require that these conditions be predictable (Sapolsky et al. 2000). For example, avian migration is a predictable, energetically demanding process, and emerging evidence from studies of captive migrants suggest that GCs may play a role in preparing birds for migratory flight (Piersma et al. 2000; Landys et al. 2004). Less is known of the preparative actions of GCs on other processes and in other animals. For female mammals, parturition and the postpartum care of offspring represent predictable, energetically demanding challenges. The general increase in GCs in the fetal–maternal circulation near term in many mammals (Keller-Wood & Wood 2001) is typically thought to be important for promoting fetal growth and development (Pepe & Albrecht 1995; Gartner et al. 2002). This same increase may also function to prepare pregnant females for the challenges associated with motherhood and offspring care (Sapolsky et al. 2000). Support for this preparatory function could come from the demonstration that late-pregnancy elevations in GCs and not concurrent GCs are associated with variation in maternal responsiveness to infant-generated stimuli.

Associations between perinatal hormones and mothering behaviour, however, must be considered with caution for two reasons. First, the periods immediately before and after parturition involve rapid changes in physiology and behaviour (Krasnegor & Bridges 1990). Hormone–behaviour relationships observed during such periods may not represent biologically meaningful associations unless repeated measurement of both hormonal and behavioural traits indicate that their relationship is consistent within an individual over time. Although seldom considered, consistency or repeatability of traits and of the relationships between them provides insight into the extent to which these traits are representative of an individual (Boake 1989) as well as validation of the robustness of hormone–behaviour relationships during periods of change in behaviour and physiology. Second, associations between hormonal and behavioural traits may simply be by-products of both the hormones and the behaviour being influenced by other characteristics of an individual (e.g. mothers' age, experience or social status, or infant sex) (Carlson et al. 2006). Although often unavailable, data on these other factors are essential for assessing the extent to which hormone–behaviour relationships arise independently of other potentially confounding variables.

In this study, we evaluated the within-individual consistency of hormonal and behavioural traits during late pregnancy and early motherhood in a population of wild yellow baboon, *Papio cynocephalus*, mothers of known biographic histories in Amboseli, Kenya. We then evaluated the extent to which perinatal hormones were associated with variation in maternal motivation to respond to a major class of infant-generated stimuli: distress vocalizations. Offspring distress vocalizations elicit parental care in many animals (Clutton-Brock 1991) and are widely believed to represent honest indicators of offspring need or condition (Godfray 1991; Weary & Fraser 1995; Kilner & Johnstone 1997). Variation in the behaviour of mothers and other caretakers in response to distress signals may reflect individual differences in caregiving motivation (Saino et al. 2000; Farrell & Alberts 2002), and this variability can potentially affect infant growth, development or survival (Christe et al. 1996; Price et al. 1996).

Finally, we evaluated evidence for both the potential preparatory and the modulating actions of GCs on maternal responsiveness to infant distress cries in 34 mother–infant dyads. To do so, we measured naturally occurring variation in responsiveness to infant distress cries during the first 2 months of motherhood and related differences between mothers in response to infant cries to individual differences in faecal GC hormones during the 2 months before and after birth. We hypothesized that perinatal GC concentrations would be associated with variation in maternal attentiveness and responsiveness to infant stimuli. More specifically, we predicted that if late-pregnancy GCs help to 'prepare' females for responding to their future offspring, then mothers with higher GC levels before birth would be more responsive to infant distress cries after birth. In contrast, we predicted that if postpartum GCs modulate ongoing maternal responsiveness, then mothers with higher GC levels immediately after birth would be more responsive to ongoing infant distress.

METHODS

Subjects, Study Site and Behavioural Data

Thirty-four mother–infant pairs, members of five distinct social groups inhabiting the Amboseli basin, Kenya, were studied during July 2002–November 2003 by N.N. Baboons are nonseasonal breeders and are among the largest, most sexually dimorphic, omnivorous and terrestrial of the monkeys. The baboon population in Amboseli

has been under continuous observation for almost 4 decades, and data on life history are known since birth from near-daily records of demographic events and reproductive cycles for several hundred animals (Altmann 1980, 1998; Alberts & Altmann 2003; Altmann & Alberts 2003; see www.princeton.edu/~baboon for a complete bibliography and the Baboon Project Monitoring Guide, which outlines data collection protocols for this population).

For each mother–infant dyad, we determined (1) maternal parity (including current infant; Altmann et al. 1988), (2) maternal dominance rank (mother's ordinal rank number the month that she conceived the infant; Altmann 1980) and (3) infant sex. Maternal age was correlated with parity ($r^2 = 0.91$, $N = 34$, $P < 0.0005$), and parity, in turn, provided a measure of prior infant care experience. Subjects included five first-time mothers with no prior experience caring for offspring and 29 multiparous mothers with experience rearing one to eight prior infants.

Each mother–infant dyad was observed throughout the first 8 weeks of the infant's life, and particular effort was made to ensure that observations of each dyad were evenly distributed between morning and afternoon samples and between the earlier and the later of these weeks of infancy. Between 17 and 64 focal animal samples (Altmann 1974) of 20 min duration were collected for each mother–infant dyad, giving a mean ± SE of 11.6 ± 0.8 actual in-sight hours of observation per pair. Data were collected on a handheld computer, the Psion Workabout, using a custom program. During each sample, continuous data were collected on all changes in mother–infant contact (categorized as in or out of physical contact) and all occurrences of (1) affiliative and agonistic interactions between mothers, infants and other group members, (2) infant distress vocalizations and (3) changes in mothers' behaviour towards infants following infant distress.

Maternal Response to Infant Distress Vocalizations

For each mother–infant pair, we determined (1) the infant distress rate, (2) the mother's responsiveness to infant distress and (3) her latency of response (how rapidly she responded to the distress). Infant distress rate was defined as the number of distress bouts given by the infant per hour observed. Nguyen (2006) found that the rate at which infants cried at Amboseli was correlated with the rate at which they received 'rough' handling by conspecifics, a finding consistent with the notion that crying reflects infant need or condition in this species. An infant distress bout was defined as beginning with the infant's first distress vocalization and ending when the infant no longer gave distress cries in relation to the original cause of distress. We categorized infant distress into two classes. Infant distress due to interactions between the mother and the infant (usually disputes over nipple access) were distinguished from those that were due to the behaviour or proximity of other group members or external stimuli, or that were otherwise clearly not arising from mother–infant interactions. The threats to infant safety and survival posed by other group members or other external sources were qualitatively greater and different from that posed by the infant's own mother. Moreover, maternal responses to infant cries caused by mother–infant interactions differed from responses to distress caused by other group members (i.e. mothers often embraced the infant protectively in the latter case but not in the former). Therefore, we limited our analysis to those distress bouts that were not associated with mother–infant conflict.

Maternal responsiveness was defined as the proportion of the total number of distress bouts given by an infant that were supported by its mother. Maternal support was defined by a change in the mother's behaviour that indicated that she was focused on identifying and alleviating the cause of the infant's distress, including embracing the infant to secure it against her body, avoiding or moving away from other group members and re-establishing mother–infant contact if the two were separated at the onset of distress. The speed of maternal response was defined as the delay between the onset of infant distress and the mother's first act of support. To estimate an individual's speed of response, we plotted, for each mother, the proportion of distress bouts that they had not yet responded to (on a logarithmic scale) against the time to response (in seconds on a linear scale). We used the slope of this survival function to describe how rapidly a mother responded to her infant's distress.

Consistency of individual differences in maternal responses to infant distress was evaluated using linear regression and Pearson correlation techniques. First, we examined whether an individual mother's responsiveness during the first postnatal month predicted her likelihood of responding during the second postnatal month. Second, we examined the correlation between the mothers' overall responsiveness and their speed of response using Pearson correlation analysis.

Faecal Hormone Sampling and Measurement across the Perinatal Period

Faecal samples were collected from each mother throughout the last 8 weeks of pregnancy and the first 8 weeks of motherhood. We collected 694 faecal samples from the 34 mothers, giving an average of 1.3 faecal samples per female per week. Faecal sample collection, storage, extraction and assay techniques for pregnant and lactating females in this population are described elsewhere (Altmann et al. 2004; Beehner et al. 2006).

Hormone data were then divided into days, weeks and months relative to parturition, with the date of birth considered the first day of week 1 and month 1 and the day before birth considered the last day of week −1 and month −1. Females varied in the number of faecal samples they contributed to the data set, and incomplete sampling can bias analyses of within-individual consistency and between-individual differences in hormone

values, especially during a period of rapid changes in hormones. Consequently, in 3 of the 4 months of sampling, mean monthly values were calculated only for those females for which samples were collected during at least 3 of the 4 weeks of each month. For the first postnatal month, however, mean values were calculated for all females for which we had samples from at least 2 of the 4 weeks, because values from the week after birth were excluded from our analyses since they partially reflect late-pregnancy values due to the lag time of 1–3 days between steroid hormone secretion and excretion in faeces (Wasser et al. 1993; Altmann et al. 2004). A total of 86 monthly means distributed across the 34 mothers were available for analyses.

Individual consistency of traits over time (i.e. over successive reproductive events) is useful for understanding how the measurement of these traits is representative of an individual (Boake 1989). Although our study focused on a single reproductive event within the life of a female baboon, repeatability of trait measurement over one perinatal period in a long-lived animal is useful for understanding individual consistency during a period of rapid changes in behaviour and physiology. Therefore, we assessed consistency of individual differences in faecal glucocorticoid (fGC) levels across late pregnancy and motherhood by comparing each individual's fGC values to the mean fGC value for all females across weeks. To control for time of sample collection relative to parturition, we generated residual hormone values using a locally weighted regression procedure (LOWESS; Cleveland & Devlin 1988) on the full set of raw fGC values (for similar analyses see Altmann & Alberts 2005; Beehner et al. 2006). Females with positive log residuals for any one week relative to parturition were considered to have high fGC values for that week, whereas females with negative log residuals were considered to have low values that week. We used linear regression analyses to examine whether an individual female showed a significant time trend up or down in log residuals across weeks and to examine whether a female's mean log residuals for any one month predicted her mean log residuals the following month (for details on this approach see Moses et al. 1992).

Predictors of Individual Differences in GCs and Responsiveness

We evaluated the contributions of maternal rank, parity, infant sex and infant distress rate to explaining individual variation in maternal responsiveness and fGC hormones by using general linear model (GLM) procedures. Because associations between perinatal hormones and mothering behaviour may depend on these maternal and infant traits, this approach enabled us to evaluate the extent to which these factors might influence responsiveness and the pattern of secretion of perinatal fGC. To evaluate the hypothesis that GCs function to 'prepare' mothers for responding to their future offspring, we measured the extent to which individual mothers' late-pregnancy GC levels predicted their subsequent likelihood of supporting their own infants in distress. To evaluate the hypothesis that GCs modulate ongoing offspring care, we measured the extent to which individual GC levels during the postnatal period explained variation in ongoing maternal responsiveness. All analyses were performed in SPSS 12.0 (SPSS 2003, Chicago, IL, U.S.A.) and were two tailed with $P \leq 0.05$.

RESULTS

Individual Differences in Responsiveness and fGCs during the Perinatal Period

Overall, mothers supported a mean ± SD of 67 ± 14% of infants' distress bouts (range 33–92%, $N = 34$). Mothers were moderately consistent in their response to infant distress during the first 2 months of motherhood. The probability that a mother supported her infant during the first postnatal month significantly predicted the probability that she supported the infant during the second ($r^2_{33} = 0.140$, $N = 34$, $P = 0.029$). In addition, more responsive mothers (i.e. those who supported a greater proportion of their infant's distress bouts) also tended to respond faster ($r^2 = 0.138$, $P = 0.030$), indicating that these two measures of response were coordinated in responsive mothers.

Females also tended to have consistent fGC values throughout the perinatal period. Specifically, more than 82% of subject females showed no significant time trend up or down in residual fGC concentrations over the entire 16-week period. Furthermore, mean values for the last month of pregnancy were significantly predicted by the mean values for the previous month ($r^2 = 0.269$, $P = 0.001$), and mean monthly residual fGC values for each of the 2 postnatal months showed a (nonsignificant) tendency to be correlated with the mean monthly fGC value from the previous month. In Amboseli baboons, fGC levels are elevated from mid-gestation onwards and change little across the last 8 weeks of pregnancy, but fGCs decline sharply after birth, returning to prepregnancy levels within 2–3 weeks postpartum (Altmann et al. 2004; Beehner et al. 2006). Despite the rapid pace of change in fGCs during this period, the results of our analyses indicate that most females showed a moderate degree of consistency in fGC values across the perinatal weeks, with some individuals characterized by consistently higher levels and others characterized by consistently lower levels.

Predicting Maternal Responsiveness to Offspring Distress

Neither an individual female's mean responsiveness during the first 2 months of infancy (overall model $r^2_{adj,4} = 0.058$, $P = 0.701$) nor her mean fGC concentrations during each of the 4 perinatal months were predictable from maternal rank, parity, infant sex or infant distress rate (Appendix, Table A1).

However, consistent with the hypothesized preparatory function of fGCs, mothers with higher overall fGC

concentrations during the month before birth were significantly more responsive to infant distress cries than were mothers with lower fGC values ($r^2_{26} = 0.169$, $N = 27$, $P = 0.033$; Fig. 1). Moreover, when we constructed a multivariate model incorporating prenatal fGCs and other potential predictors of maternal responsiveness, we found no significant effects of maternal rank, parity, infant sex or distress rate on this pattern (Table 1). The predictive association between prenatal fGCs and maternal responsiveness became more pronounced as females neared parturition. Faecal GC concentrations for weeks −4 and −3 were not predictive of future response to offspring distress ($r^2_{17} = 0.062$, $N = 18$, $P = 0.320$). However, mothers with higher fGC concentrations during the last 2 weeks of pregnancy (weeks −2 and −1) had significantly higher responsiveness scores than did mothers with lower fGC values ($r^2_{23} = 0.345$, $N = 24$, $P = 0.003$). Again, this predictive relationship held true even after we constructed a multivariate model incorporating the potential effects of maternal rank, parity, infant sex and distress rate on maternal responsiveness. Our results are consistent with the hypothesis that late pregnancy fGCs 'prepare' mothers for responding to stimuli from their future offspring.

In contrast to the predictive power of prepartum fGCs, the hypothesis of modulating effects of postpartum fGCs was not supported. Postpartum fGCs were not predictive of individual variation in maternal responsiveness. Specifically, mean fGC concentrations during the first and second months of infancy were not associated with individual variation in ongoing maternal responsiveness (month 1: $r^2 = 0.075$, $P = 0.195$; month 2: $r^2 = 0.097$, $P = 0.193$). (For the purposes of comparison, results for the first month postpartum are presented in Fig. 2 and Table 2.) Furthermore, a multivariate model incorporating the effects of maternal rank, parity, infant sex, distress rate and mean postpartum fGCs (for 18 females with faecal samples from at least 5 of the first 8 weeks of life) found that none of the above variables explained a significant proportion of the variance in responsiveness (overall model $r^2 = 0.047$, $P = 0.542$). Our results therefore do not support the hypothesis that fGC hormones during the postnatal period modulate ongoing maternal behaviour towards offspring.

Table 1. Effects of faecal GC concentrations the month before birth, maternal rank, parity, infant sex and distress rate on individual variation in maternal responsiveness to infant distress cries in yellow baboons

	Responsiveness		
Overall model	$r^2_{adj,5}$=0.062, N=27		
	MS	F	P
Model	0.026	1.341	0.286
Error	0.019		
Predictor variables			
Dominance rank	0.015	0.759	0.393
Parity	0.000	0.022	0.884
Infant sex	0.002	0.119	0.734
Infant distress rate	0.028	1.478	0.238
fGCs the month before parturition	**0.108**	**5.623**	**0.027**

Results of the GLM indicate that observed differences between mothers in maternal responsiveness were significantly predicted only by prenatal GC hormones (in bold).

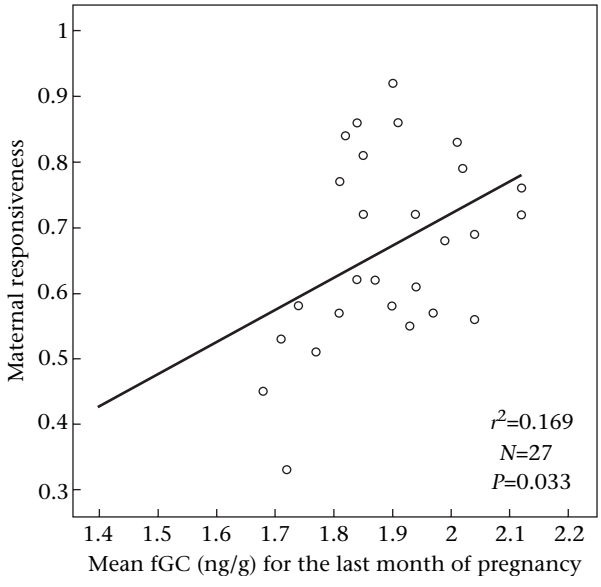

Figure 1. Faecal glucocorticoid (fGC) concentrations during the last month of pregnancy as a predictor of maternal responsiveness to infant distress in yellow baboons. Each point represents the values for an individual mother, her maternal responsiveness and mean log fGC values for the last month of pregnancy ($N = 27$, the subset of females with data for ≥3 of the 4 prepartum weeks; see Methods).

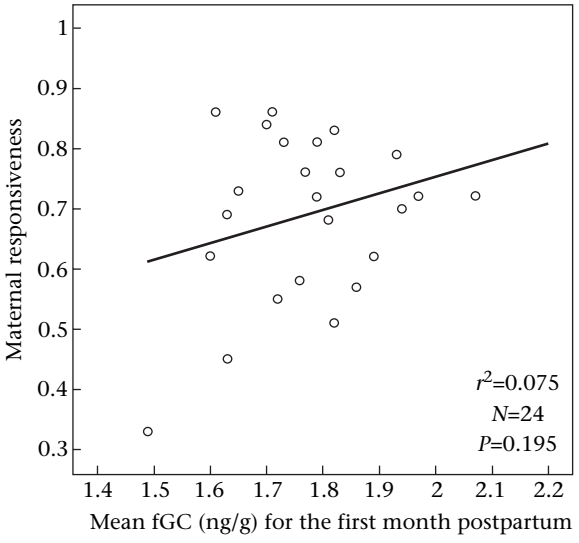

Figure 2. Faecal glucocorticoid (fGC) concentrations during the first month postpartum as a predictor of maternal responsiveness to infant distress in yellow baboons. Each point represents the values for an individual mother, her maternal responsiveness and mean log fGC values for the first month after birth ($N = 24$, the subset of females with data for ≥2 of the 3 postpartum weeks; see Methods).

Table 2. Effects of mean faecal GC concentrations for the first month postpartum, maternal rank and parity and infant sex and distress rate on individual variation in maternal responsiveness to infant distress cries in yellow baboons

	Responsiveness		
Overall model	$r^2_{adj,5}$=0.033, N=24		
	MS	F	P
Model	0.017	0.852	0.532
Error	0.020		
Predictor variables			
Dominance rank	0.003	0.134	0.718
Parity	0.017	0.863	0.365
Infant sex	0.029	1.491	0.238
Infant distress rate	0.002	0.123	0.730
fGCs the first month postpartum	0.012	0.607	0.446

Results of the GLM indicate that fGCs for the first postpartum month did not predict observed differences between mothers in responsiveness to distress cries.

DISCUSSION

We found that fGC hormone levels during the last 2 weeks of pregnancy predicted variation among individuals in responsiveness to infant distress cries after parturition, a pattern that persisted after we controlled for maternal rank, parity, infant sex and distress rate. Our results suggest that differences between baboon mothers in responsiveness to infant distress were, in part, caused by differences between mothers in late-pregnancy fGC concentrations.

Pregnancy in mammals results in an activation of the hypothalamic–pituitary–adrenal (HPA) axis, causing an increase in GC concentrations in fetal–maternal circulation, especially as females near parturition (Pepe & Albrecht 1995; Gartner et al. 2002). An increase in GC secretion near term is essential for promoting normal fetal growth and development (Pepe & Albrecht 1995; Keller-Wood & Wood 2001; Gartner et al. 2002), for coordinating parturition (Challis & Lye 1994; Keller-Wood & Wood 2001) and for the initiation and maintenance of lactation at the end of pregnancy (Stern et al. 1973; van der Schoot & de Greef 1983). As such, hypercortilism during pregnancy is considered an adaptive physiological condition (Beehner et al. 2006).

Our results suggest that increased GC secretion during late pregnancy may also facilitate an increased awareness or attentiveness to infant cues, possibly by mobilizing the energy reserves needed to cope with these anticipated future challenges (Sapolsky et al. 2000). To date, elevations of GCs before the onset of predictable future challenges have been documented only for a few taxa (reviewed in Sapolsky et al. 2000; Romero 2002), including laboratory populations of food-deprived rats presented with challenges before feeding (e.g. Levine et al. 1989) and captive populations of birds before the onset of migratory flight (Piersma et al. 2000; Landys et al. 2004). Elevations of GCs may also occur in anticipation of other predictable challenges in naturalistic contexts, from seasonal fluctuations in food availability to temporal variation in male–male competition for mates (Sapolsky et al. 2000). However, data from the wild are scarce (Romero 2002) and no prior study, in captivity or the wild, has examined intraspecific variability in GC-associated preparations to anticipated future challenges. The high predictability of the challenges associated with offspring care make late pregnancy a particularly potent stimulus for preparative GC secretion in female mammals.

How might GCs 'prepare' mothers for responding to their future offspring? GCs may act to guide offspring care by 'activating' existing maternal neural circuitry for responding to infant stimuli. Results of a recent experimental study of adrenalectomized rat mothers given replacement doses of GCs suggest that while GCs are not essential for organizing or initiating offspring care, they appear to be important in activating or stimulating previously organized mechanisms of infant care (Rees et al 2004). GCs may function to enhance the general state of arousal and reactivity in mothers of newborn infants (Mason 1968). This heightened arousal may assist mothers in identifying and remembering the salient and unique characteristics of their infants, and this rapid learning of infant cues may hasten the development of responsiveness to infant stimuli (Fleming 1990).

An alternative explanation for the relationship between individual variation in prenatal fGCs and maternal responsiveness is that infants who experienced higher levels of GCs prenatally may, after birth, require more care and, as a result of their greater demand for attention (see de Weerth et al. 2003; Davis et al. 2005), receive more care than infants who experienced lower levels of GCs prenatally. If this explanation is correct for Amboseli baboons, one would expect that infants who cried more often experienced higher GC levels prenatally and received more care after birth than infants who cried less often. However, our results provide no support for a relationship between infant variation in distress rates and maternal variation in either prenatal fGCs or responsiveness to infant cries.

The apparent preparatory influence of GCs on maternal responsiveness in Amboseli baboons may instead reflect changes in other hormones or neurotransmitters as a result of activation of the HPA axis (Fleming et al. 1997a). Indeed, oxytocin, corticotropin-releasing hormone (CRH) and opioids are important in the regulation of infant care in captive populations of a few species of mammals (reviewed in Numan 1994). Nonexperimental studies that use noninvasive hormone sampling such as this study cannot rule out this possibility, both because they cannot definitely distinguish between causation and other sources of correlation and because noninvasive hormone sampling, particularly from faeces, does not permit measurement of some of the relevant compounds.

How do the relationships between perinatal GCs and responsiveness to infant distress in baboon mothers at Amboseli compare to those reported in other studies? A summary of studies that explored the relationships between GCs and infant care behaviour in 12 studies of eight species of mammals is presented in Table 3.

An examination of the seven studies that provided strong evidence for their reported relationship between GCs and infant care behaviours (for criteria used to

Table 3. Relationships between glucocorticoids (GCs) and infant caregiving behaviour in mammals: only the first seven studies provided strong evidence for their reported relationships

Species	Condition	Subjects	Hormone measured*	Prepartum GCs†	Postpartum GCs†	Strong evidence for reported relationship?‡	Associated behaviours§	Reference
Humans, *Homo sapiens*		Mothers (N=30)	Plasma ***cortisol*** (N=60)		+	Y	Of 2 classes of behaviours examined, ***affectionate infant directed behaviours***	Fleming et al. 1987
		Mothers (N=58)	Salivary ***cortisol*** ($N \leq 58$)		+	Y	***Maternal recognition of own infant's body odour***	Fleming et al. 1997b
		Mothers (N=86)	Salivary ***cortisol*** ($N \geq 86$)		+	Y	Mothers who ***reported feeling greater sympathy*** in response to infant cries had higher baseline heart rates and cortisol levels	Stallings et al. 2001
Rats, *Rattus norvegicus*	Captivity	Adrenalectomized mothers (N=32)	Varying doses of replacement ***corticosterone***	na	+	Y	Females given higher doses of corticosterone spent more ***time over pups*** and ***in the nest***	Rees et al. 2004
	Captivity	Adrenalectomized mothers (N=36)	Replacement ***corticosterone***	na	+	Y	*Pup licking* occurred at higher rates and ***latency to show maternal behaviour*** upon pup exposure was shorter in females given replacement corticosterone	Graham et al. 2006
Meerkats, *Suricata suricatta*	Wild	Male helpers (N=36)	Plasma ***cortisol*** (N=47)	na	+††	Y	***Rate of pup feeding*** by males in a cooperatively breeding species; ***playback experiments*** that used pup begging calls to increase feeding rates also gave rise to increases in cortisol in male helpers	Carlson et al. 2006
Yellow baboons, *Papio cynocephalus*	Wild	Mothers (N=34)	Faecal ***glucocorticoids*** (N=694)	+	=	Y	***Maternal responsiveness*** to infant distress cries	This study
Gorillas, *Gorilla gorilla*	Captivity	Mothers (N=8)	Urinary ***cortisol*** (N=55)	=	−	N	Of 4 behaviours, ***% of time in ventral–ventral contact during locomotion***	Bahr et al. 1998
Japanese macaques, *Macaca fuscata*	Captivity	Mothers (N=8)	Urinary ***cortisol*** (N=128)	=	−	N	Of 4 behaviours examined, ***maternal rejection*** of infant approaches or contacts attempted	Bardi et al. 2003
Sheep, *Ovis aries*	Captivity	Mothers (N=21)	Plasma ***cortisol*** ($N = \sim 300$)	=	−‡‡	N	Of 2 axes of behaviour examined, ***infant grooming***	Dwyer et al. 2004
Baboons, *Papio* sp.	Captivity	Mothers (N=89)	Urinary ***cortisol*** (N=1600)	+‡‡	=§§	N	Of 4 axes of behaviour examined, ***(1) affiliative behaviours directed at infant*** (prepartum); ***(2) maternal anxiety*** (postpartum)§§	Bardi et al. 2004

*N = total number of samples assayed for hormone of interest.

†+ Indicates that the study found evidence that GCs were positively associated with caregiving behaviour; − indicates that the study found evidence that GCs were negatively associated with caregiving behaviour; = indicates that the study found no evidence of a relationship between the hormones and the behaviours measured; blank indicates that the relationship was not examined; na indicates that it was not possible to examine this relationship.

‡Defined as those studies that were conducted on more than 30 animals and were either (1) experimental in nature (i.e. involved removal of the adrenal glands, the source of GCs, and subsequent GC replacement) or (2) designed to test the relationship between GCs and specific infant care behaviours (i.e. those behaviours, based on previous research on GC–behaviour interactions, most likely to be under the influence of GCs).

§Caregiving behaviours that showed a significant association with GCs (indicated in bold italics) as reported by authors when $P < 0.05$, regardless of the number of hormone–behaviour relationships examined.

††Blood sampling of male helpers was conducted during pup-feeding episodes when pups were 35–75 days old (a period of sampling comparable to postpartum sampling of mothers).

‡‡Effect considered less well supported because the study explored relationships between multiple hormonal and behavioural variables without correcting for the multitude of analyses conducted.

§§Although 'maternal anxiety' (the rate of displacement behaviours by mothers) was correlated with postpartum cortisol levels, these behaviours were not directed at infants or performed in association with the care of infants; we therefore concluded that postpartum GCs were not associated with maternal behaviour in this study.

identify strong evidence from less well supported evidence, see Table 3) yields some insight into the role of GCs in mediating infant care across taxa. The proposition that GCs may be important for mediating individual differences in infant care is supported by all seven investigations (Table 3). In addition, evidence from the majority of these studies suggests that GCs are important in mediating caretaker responsiveness to infant-generated stimuli (from odours to cries), a pattern consistent with GCs' role in helping animals cope with and respond to challenging stimuli. Of the seven studies that provided strong evidence of GC–infant care interactions, our study is the first to evaluate and find strong evidence consistent with the hypothesis that, during late pregnancy, GCs act to 'prepare' mothers for responding to stimuli of future offspring. However, in contrast to the patterns found in previous studies of human and rat mothers, we did not find evidence that postnatal concentrations of GCs contribute to variation in ongoing responsiveness to offspring cries despite a degree of continuity between pre- and postnatal fGCs. Our results suggest the possibility that, in baboons, GCs may become less important as mothers gain experience with the current offspring. Given the role of the HPA axis in learning and memory formation (McCormick et al. 1997), GCs may assist in the formation of maternal memory of current infant stimuli or experiences (Rees et al. 2004). Data from a recent experimental study with rats are consistent with this hypothesis (Graham et al. 2006). Because chronic activation of the HPA axis can lead to pathology (Sapolsky 1993), maternal memory of infant stimuli and experiences may help ensure that mothers continue to respond appropriately to offspring stimuli without requiring prolonged activation of the HPA axis. The absence of an effect of maternal experience with previous offspring on maternal responsiveness to the current offspring suggests that the purported GC-enhanced maternal memory of infant stimuli may be specific to the current infant. If this pattern is true, offspring may be under selective pressure to coopt this process for their own benefit.

Indeed, the apparent preparative influence of prenatal GCs on maternal responsiveness in Amboseli baboons may be a reflection of parent–offspring conflict. Haig (1993) hypothesized that pregnancy may give rise to an evolutionary 'tug-of-war' over the amount of resources that an individual offspring can obtain from its mother at the expense of the mother's other offspring. Evidence from recent experimental studies with mice and humans suggests that, consistent with Haig's hypothesis, evolutionary conflicts underlie a range of complications and disorders in pregnancy and child development (Haig 2004). These conflicts may also contribute to the high levels of circulating GCs as mothers near term. Studies of rodent and human pregnancies have demonstrated that most of the circulating GCs during late gestation are produced by the fetus (Chatelain et al. 1980; Fencl et al. 1980). If baboon fetuses, like their rodent and human counterparts, are also largely responsible for the increase in GCs in fetal–maternal circulation during late gestation, they may, therefore, be responsible for the apparent GC-mediated changes in maternal arousal and responsiveness to infant stimuli postpartum. The results of this study raise the intriguing possibility that the fetus may 'manipulate' its mother into providing more care than she would otherwise by modulating the production of chemical messengers that influence maternal arousal and responsiveness. The exact mechanisms of this putative 'manipulation' of mothers and the counterstrategies mothers may use to better meet the demands of the current offspring with those of future offspring remain to be elucidated.

In conclusion, this study provides the first test of hypotheses regarding the dual nature of GC action on behaviour as well as one of the first sets of evidence for preparative secretions of GCs in anticipation of a predictable future challenge in wild animals. In addition, this study provides the first investigation into intraspecific variability in GC-associated preparations to anticipated future challenges. Finally, this study provides one of the first examinations of the relative stability of behavioural and hormonal traits during a single reproductive event, a period of rapid changes in hormones and behaviour. Despite recent advances in our understanding of the influence of GCs on offspring care, additional research is needed to fully understand the exact mechanisms through which GCs may influence the behaviour of caretakers towards infants.

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Appendix

Table A1. Effects of maternal dominance rank and parity and of infant sex and distress rate on individual variation in perinatal faecal glucocorticoid concentrations (fGCs) of adult female yellow baboons

	Glucocorticoid concentrations											
	Month −2 (N=21)			Month −1 (N=27)			Month +1 (N=24)			Month +2 (N=20)		
Overall model	$r^2_{adj,4}$=0.115			$r^2_{adj,4}$=0.052			$r^2_{adj,4}$=0.028			$r^2_{adj,4}$=0.039		
	MS	F	P	MS	F	P	MS	F	P	MS	F	P
Model	0.006	0.483	0.748	0.010	0.680	0.613	0.021	1.168	0.356	0.018	0.820	0.532
Error	0.012			0.015			0.018			0.021		
Predictor variables												
Dominance rank	0.004	0.311	0.585	0.008	0.523	0.477	0.013	0.754	0.396	0.043	2.019	0.176
Parity	0.009	0.742	0.402	0.013	0.891	0.355	0.034	1.925	0.181	0.000	0.010	0.920
Infant sex	0.001	0.108	0.747	0.002	0.133	0.791	0.003	0.168	0.687	0.009	0.418	0.528
Infant distress rate	0.001	0.116	0.738	0.033	2.174	0.155	0.013	0.712	0.409	0.001	0.024	0.880

Results of the GLMs indicate that an individual female's mean fGC concentrations during each of the 4 perinatal months were not predictable from maternal rank, parity, infant sex or infant distress rate. MS = mean square.