Stress, Evolution, and Sociality in Wild Female Baboons

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I owe so much to so many.

To say that this dissertation is the result of a communal effort cannot do justice to the contributions made by my countless supporters. I have been blessed with a wealth of loved ones. When it seemed like I was attempting to accomplish the impossible—an education, a career, a marriage, a family, a life—someone would always surface from the crowd to assure me that I was on the most perfect path. I hope that each of you can recognize your efforts and will accept my gratitude.

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Six years have passed since my last visit, and I'm not sure whom I miss more—the baboons or my Tanzanian friends. By far the most important among many is Appolinaire Sindimwo, baboon researcher extraordinaire. I learned more about baboons in the time I spent with Appoly than I could ever hope to learn from those baboon researchers who publish regularly in scientific journals. He was patient as I struggled to communicate, first in French and later in Swahili. One of my proudest moments came when my Swahili was good enough that I could explain the concept of contact lenses to him, and I will always remember his surprise and fascination. I miss his stories about the Beach Troop baboons, about his daughter's close call with Frodo, and about his early days in the field with Craig.

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I was so fortunate to have been adopted by my Tanzanian mother, Tabitha Rulanyaga. Tabitha aptly filled in for my real mother by providing me with both personal and logistical support whenever I was in need. She met me and my visitors at the train station. She helped me acquire food, lodging, and transportation. It was my relationship with Tabitha that finally made Kigoma feel like a home. To this day, she sends me regular reports and pictures, and proudly claims my children as part of her family.

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I have also had the pleasure of assembling a large and devoted group of individuals who were so intrigued by the baboons and impressed with my own enthusiasm that they were willing to work hours and hours and hours . . . for nothing but the promise of good karma. Actually, as it turns out, baboons are addictive. One of the first and certainly most dedicated of the Gombe Baboon Project Volunteers was Jamie Shaver. Jamie toiled with the demographic data on a weekly basis for over two years, and she was so kind as to travel with me to Tanzania on two occasions—thus assuaging my higher-than-average social needs and saving me from too many lonely nights at Gombe. Caitlin Schmid also willingly spent many Sunday afternoons with me in the baboon office, sifting through a lot of less-than-exciting files and gathering data that has yet to be analyzed. She then, on

my advice, headed off to Carleton. Unable to bear the separation, I then followed her back to my alma mater and somehow convinced this English-Music double major to continue to work with the baboon data. She cannot escape me, and she should probably just stop trying.

Holly MacCormick can also attest to siren call of the baboons. She started off doing all sorts of basic work for me years and years ago, and now she is fully immersed in the injury data and willingly stays up until 4 am to try to uncover exciting patterns. She is hooked! Margaret Sarafiny is, because she is geography challenged, no longer able to assist me in my many endeavors, but she asks for regular updates and never forgets my birthday. My most heartfelt thanks also go out to all of the other volunteers who have worked with me: Lorelle Berkeley, Del Bosacker, Sharon Buley, Nathan Kass, Katryna Kerr, Bjorn Larson, Jacki Simonet, Charlie Soucheray, and Dawn Tanner. Much of their work was only tangentially related to the research reported here, but their efforts are nonetheless laudable. Special thanks go to the newest generation of baboon researchers: Dorothy van Oppen, and James Fuller. Your energy and insights make my continued work with the baboon data exciting and fresh on a daily basis. In fact, I *know* that I get more from teaching than I give. Without the energy I have drawn from the hundreds of incredible students with whom I have had the pleasure to work, I would never have gone to graduate school in the first place, and I would certainly never have finished. I will always appreciate every one of you.

Female baboons rely heavily on the friendships they develop with other females. As the results of this and many other studies suggest, these relationships are critical to a healthy, happy life. While female baboons rarely have more than the requisite number of female friendships, I have been blessed with an abundance of support. My friend Claire Geistfeld has been—however ironically—my touchstone of sanity. When the miles of rural roads stretch out before me and I begin to question every choice I have ever made, she is always there to tell me that I am all right. My friend Amy Blaubach has boundless optimism, and when my faith in myself waivers, she can fill me up with a sense of the possible. Shannon Murphy was my constant virtual companion through all of the ups and downs of graduate school. No matter what obstacle I faced, Shannon was always ready to give me sound advice and to let me know that there was always someone on my side no matter what the issue. Katie Kaehler, caregiver to my children, friend, and so much more—without her this dissertation would have been impossible. Jennifer Wolff has played so many critical roles during the last two years: colleague, friend, source of graduate school wisdom, and advocate. Alison Unger is the one person I can count on to remind me, in the midst of the chaos, that I need to take care of myself. Jennifer Theis helped me transition back into the world of research, after a year or two when I had totally given it up. I also thank Julia Rogers, Annika Lister Stroope, Rachel Lock, Christina Herzog, and many others I have mentioned already or who also happen to be family and will appear below.

Most of what I want to say about my incredible family I was able to say more succinctly in my dedications. My large and tight-knit extended family as well as the enormous family I had the good fortune to marry into all work to keep me well fed at holidays and well loved at all times. Uncles (Uncle Paul has requested special consideration), aunts (Aunt Sharonee certainly deserves special acknowledgement), cousins, great aunts, great uncles, my mother-in-law, my eight sisters-in-law, my nine brothers-in-law, my nieces, my nephews, my great nieces, and my great nephew. I have never once felt truly alone, even during those long, dark nights in that far away forest.

I have a tendency to be a bit independent and headstrong, and for some reason I cannot give up on anything—even when I find myself in harm's way. My mother once insisted that there must be a guardian angel who never leaves my side and is constantly orchestrating all of the countless successes in my life. I think that I have three: my bapa, my nana, and my uncle Scott. Losing each of these important people devastated me. Each and every time I think of them, I am almost crushed by the empty spaces they have left in my life, and time just doesn't seem to be healing these wounds. I can think of no better consolation than that they are in some small way responsible for everything that is good in my life. There is *so much good,* so they have been very busy.

My 95-year-old grandfather loves animals with the same ferocity that I do, and even though he let my baby fish die on the floor of his car decades ago, I will never deny that few people have shaped me as much as he has. He was, is, and will always be my greatest hero of all time.

When I think of my oma, I like to think of this *look* she gets when she is overcome with pride and gratitude. She gets it when she holds my tiny babies, or hears about my accomplishments. When I see that look, I know that she is thanking God for all that she has. As I write this, I hope that exactly that *look* is apparent on my own face.

My sister once selflessly took the blame for me, and in return I tolerate the fact that she has moved away and left me here in Minnesota to face all present and future consequences on my own. But really, I miss her every moment of every day. My brother never hides his feelings, and his passion for life is as deep and booming as his powerful voice. He, too, has moved away, and once again, my aching for him is overwhelming. I would not be who I am now had I had any other siblings.

I was once struck by an Emily Dickinson quote that sums up how I feel about my mother, and how I hope my own children will always feel about me: “To have *had* a mother—how mighty!” My father is the most amazing teacher that I have ever had or, I suspect, will ever have the good fortune to come across. If I can give my students just a small fraction of what he has given me over the years, I will have succeeded. Both of my parents deserve more thanks than mere words are capable of expressing.

Of all of my accomplishments, I am most proud of the fact that I was able to complete my graduate work while still being true to my ultimate priority: my family. Each has made some sacrifices, and for that I am also eternally grateful. Maura has spent much of the first four months of her life cradled in my lap as I typed with one hand. She may forever be haunted by the sound of clicking keys, or it may be the one thing most likely to comfort her throughout her life. It is a sound she must certainly associate with Mama. Maura also gave me a great gift when, just a few weeks before my defense, she committed to going to sleep each and every night at 8 pm, thus freeing up my evenings and *both* of my hands. I love this tiny girl.

When Charlotte was not even two, we stopped for a moment to watch the swamp monkeys at the zoo. She sat perched on my hips for what, in toddler time, is the equivalent of years. I can still close my eyes and feel the pride, the joy, the little girl clinging to me and staring out at the world with awe. Of all the traits to watch pass from generation to generation, this fascination with animals must run deep and strong. Charlotte's bright eyes and wise ways fill me up with a simple, warm kind of happiness. May she always know that her dreams—even monkey dreams—they *can* come true.

Liam, my Liam. If passion is the key to success in life (and if it is not, it should be), then oh, what Liam has in store for us all! I see so much of myself in this little boy of mine. Sometimes I ache for all of the tiny heartbreaks that he will face as he learns to weave his way through the expectations of an often unforgiving world. But I have such hope. One day, he too will learn to harness his passion, and it will bring him more and more happiness each day. Liam, when I think of how I want my students to feel when I lead them to a new idea or a new question, when I think of how I want them to experience learning, I think of your enthusiasm, your joy, and your unadulterated excitement when *you* discover something new. If I can help my students feel even a tiny fraction of that wonder, then I know they have learned something that will stay with them forever.

I can hardly imagine a time in my life when there was no Andrew by my side. We hadn't even met when I started graduate school. But when I went to Tanzania for the first time and it nearly killed me to wait for weeks to get word from him—I knew. I knew that he was the person who could be by my side, who could pick me up and dust me off and send me off looking for more. He understands my passions, and he celebrates them. He believes in me so deeply that even in my darkest moments, I know that he is just the person to help me sustain my belief in myself. He has made so many sacrifices helping me see this thing through, and even if I had failed, he would have loved me just the same. I look at my children and I know the aching, overwhelming, chest-splitting love my parents must feel for me—they cannot help but love me unconditionally. But I know that

Andrew *chooses* to love me. Knowing that is knowing something so powerful it could stop the world from spinning.

There are others—countless others—who have surfaced at just the right time to help me grow as a scientist, as a teacher, as a wife, as a mother. I thank you all. It is entirely possible that no one will ever read this tome of gratitude. But may it never be said that I am less than grateful for every person who has made my life so full.

DEDICATION

This dissertation is dedicated to my family.

To my grandfather, from whom I have inherited my fascination with animals.

To my oma, whose pride in me is a tangible thing.

To my mother and father, who have supported me in absolutely every endeavor I have undertaken. The knowledge that their love is unconditional and unlimited is a precious gift.

To my brother and sister, who have been my closest companions from the very beginning.

To my beloved husband, who so patiently stands by my side as I attempt to do one thousand things at once.

To Liam, my son, in honor of his fierce passion for all living things.

To Charlotte and Maura, my daughters, because I want nothing more nor less for them than that *they too* have every opportunity to follow *their* dreams.

ABSTRACT

Organisms that successfully respond to threats have higher fitness than those who fail to mount appropriate responses. The vertebrate stress response system has evolved as a suite of physiological and behavioral strategies for coping with such threats, but chronic activation of this system can carry long-term physiological costs. I studied a population of wild olive baboons (*Papio cynocephalus anubis*) and used demographic and hormonal data to investigate how social parameters influence exposure to stressors and also to consider the adaptive value of the chronic stress response. Social circumstances such as rank and access to social support have been associated with differential exposure to the physiological costs of stress including exposure to the stress hormone, cortisol. I found that neither rank nor social support correlated significantly with cortisol exposure in adult female baboons. In contrast, a juvenile female's social circumstances strongly influence her exposure to cortisol; maternal rank, a living mother, and the presence of multiple female kin were all significant predictors of cortisol exposure. I also considered the evolutionary history of the vertebrate stress response system. The system's major weakness has always been believed to be its enigmatic predisposition for chronic and pathological activation. I propose that this tendency for continued activation in the face of lasting challenges can carry its own adaptive benefits and is not, as has been long assumed, merely maladaptive. In reaction to a severe and prolonged stressor, the baboons implemented a number of cortisol-mediated coping strategies, suggesting that sustained activation of the stress response system may promote lasting changes in behavior and physiology that can promote survival. Thus, the long-term risk of pathology may be outweighed by more immediate survival benefits.

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CHAPTER 1:

FECAL STEROID SAMPLING FROM WILD BABOONS: CONTROLLING FOR PATTERNS OF DEGRADATION

Abstract

While fecal samples can be used to measure steroid hormones under natural conditions, proper interpretation of values from these samples has proven to be a challenge. Hormones can degrade quickly in feces, and this problem is exacerbated in samples collected under field conditions where immediate analysis and ideal preservation protocols are impossible. Furthermore, the patterns of degradation can vary significantly between preservation protocols, hormones, and species. In this study, samples were collected from female baboons at a remote field site and analyzed for cortisol metabolites. Because freezing was impractical, samples were stored for variable lengths of time in ethanol before analysis. In this chapter, I present evidence that cortisol's degradation in ethanol is more complex than previously known; the pattern of change can also vary with age and across social groups. By including preservation lag times as predictors in general linear models (GLM) and general linear mixed models (GLMM), I was able to control for the effects of these complex patterns of degradation.

Introduction

Steroid hormones are excreted in feces and urine and are known to reflect circulating levels in a variety of species (Whitten et al., 1998). As a result of continued advances in field endocrinology, we are able to study hormonal actions and patterns of release in an organism's natural environment. Fecal sampling has proven to be especially useful in a field setting, as samples can be quickly and unobtrusively obtained from an identified individual. Cortisol has been widely used as a measure of physiological costs (e.g. Sapolsky, 1982; Abbott et al., 2003; Wikelski & Cooke, 2006), and fecal sampling can be used to address specific evolutionary questions by gauging selective pressures under natural conditions (e.g. Creel, 2001; Koolhaas et al., 2006).

Studies in a variety of species have found that the identity of the excreted metabolites and the time course of their excretion are both species and hormone dependent, thus necessitating species-specific validation studies (Buchanan & Goldsmith, 2004; Touma & Palme, 2005). As baboons are a popular study organism in both captivity and the wild, much of the necessary validation work has been completed (Wasser et al., 1988; Wasser et al., 1991; Wasser et al., 1994; Stavisky et al., 1995; Wasser et al., 2000; Altmann et al., 2004). Cortisol excretion, for example, was found to correlate well with circulating levels and was shown to be accurately measurable in baboon feces (Wasser et al., 2000). In addition, cortisol is represented in feces by at least six different metabolites and is excreted approximately 26 hours after it is released into the circulatory system.

While fecal sampling makes non-invasive hormone sampling possible in the field, the remote location of many field sites poses a number of new challenges. Without expensive lab equipment—in many cases without a freezer, electricity, or running water—samples can not be analyzed immediately. Because samples are usually collected in one place and analyzed in another, there are often significant delays between collection and analysis. Steroid hormones in the intestines are accessible to bacteria that can rapidly metabolize them into a variety of forms both before and after elimination. Ethanol can counter this degradation for at least 21 hours (Wasser et al., 1988), and storage in a -20° C freezer can also decrease the activity of bacteria and minimize degradation.

At sites where samples can not be frozen, feces are often stored in ethanol at ambient temperature for long periods of time. Recent studies suggest that such samples may show significant changes in measurable steroid hormone concentrations in as little as two weeks, and even frozen samples can show significant changes in glucocorticoids and estrogens in as little as 30 days (Khan et al., 2002; Lynch et al., 2003). These changes are likely due in part to the increased extraction efficiency of prolonged exposure to ethanol. It is also likely that stored hormones continue to degrade over time. The patterns of hormone degradation are known to vary with the hormone under study, the method of preservation, and the species under study (Khan et al., 2002; Hunt & Wasser, 2003; Lynch et al., 2003; Pettitt et al., 2007; Yanai et al., 2007). New field preservation protocols appear to minimize this effect (Beehner & Whitten, 2004; Ziegler & Wittwer, 2005).

In the following chapters, I use fecal cortisol to address a number of questions pertaining to the behavior, physiology, and evolution of wild female primates. The samples used in these studies were collected before this problem of degradation was brought to light. As samples were exposed to ambient conditions for variable times both before (ethanol preservation lag) and after (freeze lag) preservation in ethanol, storage might adversely affect the reliability of cortisol levels measured from these samples. Because both ethanol preservation lag and freeze lag were known for each sample, I was able to control for the effect of storage time on measured cortisol. I was also able to elucidate related patterns of degradation for cortisol in this population of wild baboons under field conditions.

Methods

I. Study site and population

A. Gombe National Park, Tanzania.

Gombe National Park is located in Western Tanzania. Gombe is a small park of approximately 35km^2. The boundaries of the park are all ecologically discrete. The north and south are bounded by villages and deforested farm land. The eastern boundary is the rift escarpment (again bounded by farm land), and the western boundary is the shoreline of Lake Tanganyika. The park is characterized by a series of steep ridges rising from the lake to the escarpment; habitats range from thick riverine forests in the valleys to open grasslands on the ridges (Clutton-Brock & Gillett, 1979).

B. The Gombe Baboons

The subjects of this study are female olive baboons (*Papio cynocephalus anubis*). Numerous baboon troops reside within the boundaries of the park, and they can be found in any of its varied habitats. The baboons of nine of these troops have been continuously studied since 1967 (Packer, 1975; Ransom, 1981). The newest troop, BB, was excluded for lack of data. Thus, eight troops (AA, AB, AC, BA, C, DA, DB, and DC) are represented in this study. All baboons were habituated to human observers, and all study subjects have been followed throughout their lives.

All females over the age of three at the start of sample collection (n=106) were studied. As juveniles and adults represent significantly different life history stages, I analyzed these two datasets separately. Any female who was three or more and had not had a first swelling at the start of the study was considered a juvenile (n=22). All other females were considered adults (n=84).

II. Hormonal collection, preservation, and analysis

A. Sampling protocol and sample collection

Cortisol samples were collected in three discrete field seasons, between January 2000 and April 2002 (FS1: May & June 2000; FS2: February & March 2001; FS3: December 2001-April 2002). Fecal samples were collected opportunistically from all study subjects by field assistants who had been extensively trained in collection protocols. Each sample was collected only if the identified baboon was seen in the process of defecating. Any feces that could not be conclusively attributed to a female (e.g. more than one sample was found in close proximity) or were in poor condition (e.g. fell a great distance) were excluded. An average of 9.4 total samples were collected per female (adults: n=834; juveniles: n=160).

Samples were collected in small plastic zipper bags and were immediately mixed by manipulating them inside the plastic bag. Samples were collected at any time between 07:23 and 19:25; previous studies suggest that diurnal fluctuations in baboon fecal cortisol levels are minimal (Weingrill et al., 2004). Specimens were transported to the field lab at the end of each morning or afternoon shift.

B. Field preservation and transportation

Samples were remixed inside the plastic bag. Approximately 0.5g of the mixed samples were placed in pre-weighed 20 mL HDPE scintillation vials and covered with 5mL of 90% ethanol. Each sample was then shaken by hand for a minimum of two minutes until samples were adequately suspended in the preservative. The lag time between collection and preservation (ethanol preservation lag) was recorded for all samples. This lag ranged from 1 minute to a rare extreme of 8 hours, with an average time of 2:36 spent at ambient temperature without preservative. Once processed, samples were tightly sealed and wrapped in parafilm to prevent ethanol leaks. They were then kept in a cool, dark room for the duration of the field season.

Samples were transported under CDC permit (number 2001-05-246) to the St. Louis Zoo, St. Louis, Missouri. Upon arrival, samples were checked for signs of leaking and discarded if necessary. Samples were then analyzed immediately or frozen in the ethanol preservative in a -20˚C freezer. The lag between ethanol preservation and analysis or freezing (freeze lag) was also recorded for all samples. Samples were stored at ambient temperature in ethanol for an average of 76 days with a minimum of 15 days and a maximum of 181 days.

C. Steroid extraction

As samples had been stored in the extraction solute for a minimum of 15 days and experienced vigorous jostling during transport, I followed a simplified extraction protocol. Samples were placed on a shaker overnight at 200 RMP, after which they were allowed to settle for approximately one hour. The extract portion of the samples was then decanted into 12x75 polypropylene tubes, leaving behind all fecal material, and then centrifuged at 4,000 RPM for one hour. The supernatant was then poured into a cryovial and analyzed immediately.

The pellets remaining in the polypropylene tubes were resuspended in ethanol and poured back into the original scintillation vial. All vials were then left open overnight in a vented oven at 100ºC. When samples were completely dry, the vials were reweighed. By subtracting their initial weight from their weight with dry feces I could determine the dry weight of the feces.

D. Radioactive immunoassay

All samples were analyzed using radioactive immunoassay (RIA) in the Endocrinology Lab at the St. Louis Zoo. Standard cortisol RIA kits were used (Cortisol ACTIVE-RIA ^{125}I Kit, Diagnostic Systems Laboratories, Webster, Texas) and all samples were assayed in duplicate; paired samples with variation greater than 15% were reanalyzed. Hormone concentrations were corrected for volume of added solvent and also dry weight of feces in order to control for differences in moisture content (Wasser et al., 1993) and are therefore reported in ng/g. Although an antibody against cortisol was used in these assays, fecal glucocorticoids exist as a mixture of metabolites, and while "fecal cortisol" is used for convenience, these results should be understood to refer to fecal glucocorticoid metabolites.

E. Outlier analyses

Two samples were excluded due to lack of replicability either within or between batches. Paired aliquots of these two samples were run seven times at three different dilutions. Each batch showed extremely low replicability, and final cortisol values ranged from 4862-17250 ng/g for one and 5023-15188 ng/g for the other.

Fecal sampling following the protocol used in this study can show unreliably high or low values (Millspaugh & Washburn, 2004). On rare occasions, dry fecal weight can have very little to do with the actual weight of the feces. For example, samples may contain more or less indigestible material (such as seeds) which can significantly skew final hormone concentration measurements, resulting in extreme high or extreme low values. In order to remove any such values from the dataset, I created a set of exclusion criteria. I first identified any samples that were unusual for a given baboon and that were more than four standard deviations from the overall mean. I then looked at this subset of data points and excluded those that showed extreme high or low dry fecal weights as well as extreme high or low raw hormone values (prior to dry fecal weight correction). Four samples were identified as having extremely low fecal weights (<0.03 g) and extremely high cortisol and testosterone values (top 1%) and were therefore excluded based on these criteria.

III. Statistical analyses

I used SAS version 9.1 (SAS Institute, Inc, Cary, NC) and Proc Mixed to develop two models in order to identify predictors that correlate with cortisol levels for the adult and juvenile females. In order to control for the effects of storage delays and hormone degradation, I included the predictors ethanol preservation lag and freeze lag (for complete details on the predictors used and the development of these models, see Chapter 2). For both the adult and juvenile data sets, the models were initially developed as general linear mixed models (GLMM) with the random effect *baboon*. A random effect was considered in order to account for the correlation among repeated cortisol measures from the same baboon. If the model results were not significantly different under a general linear model (GLM), I subsequently dropped this random statement from the model.

Results

I. Model results: predictors of chronic stress

A. Wild adult female baboons

This model was initially developed as a general linear mixed model (GLMM), with the main effects and interactions detailed above. The random effect *baboon* was found to be insignificant and was subsequently dropped from the model indicating a low, non-significant correlation among samples from each baboon. Only the results of the general linear model (GLM) are reported below, and they can be seen in full in Table 1. I then developed a reduced model based on stepwise removal of insignificant interactions and main effects. The results of this reduced model's tests of fixed effects can be seen in Table 2. Those results central to sample degradation (shaded blue in the tables) will be discussed below.

B. Wild Juvenile Female Baboons

I developed a separate model for juvenile females. In this model, the random effect *baboon* was found to be significant indicating a significant correlation among samples from each baboon. Therefore, all results for juvenile females are from GLMM with the main effects and interactions detailed above. The results of this complete model can be found in Table 3. I developed a reduced juvenile cortisol model based on the same stepwise process described for the adults. The results of this reduced model's tests of fixed effects can be seen in Table 4. Again, results central to sample degradation (shaded blue in the tables) will be discussed below.

II. The effects of preservation on cortisol

Cortisol degradation could have occurred during two periods: the time samples were in the field before they were preserved in ethanol (ethanol preservation lag), or the time samples were in ethanol before they were analyzed or frozen (freeze lag). Differences in either lag time might affect levels of measurable cortisol. In order to control for this, I included both lag times as predictors in my models.

A. Ethanol preservation lag

There was no significant correlation between ethanol preservation lag and cortisol for unpreserved samples exposed to ambient conditions for less than eight hours (ethanol preservation lag: adults, GLM: $F_{1, 751}$=0.37, n=834, p=0.5458; juveniles, GLMM: $F_{1, 104}$ =1.68, n=160, p=0.1983). Furthermore, the amount of time between collection and preservation did not significantly affect post-preservation patterns of degradation (ethanol preservation lag*freeze lag: adults, GLM: $F_{1, 751}$=0.09, n=834, p=0.7645; juveniles, GLMM: $F_{1, 104}$ =0.61, n=160, p=4369).

B. Freeze lag

The amount of time samples remained unfrozen in ethanol significantly influenced cortisol levels in samples collected from adult females and juvenile females. The pattern of degradation, however, was not consistent across either troop or age class. For both adults and juveniles, the effect of freeze lag interacts significantly with troop (troop*freeze lag: adults, GLM: $F_{1, 796}$=2.73, n=834, p=0.0083; juveniles, GLMM: $F_{1, 110}$ =12.26, n=160, p<0.0001). The slopes that indicate the pattern of degradation for each troop vary considerably and suggest that samples collected from some troops increase over time while others show decreases. The only significant effect for adults is a negative correlation between freeze lag and cortisol in troop BA (Table 5); for every day of storage in ethanol these samples show a decrease in measurable cortisol of 37.8 ng/g. The differences between troops are more pronounced amongst samples collected from juvenile females. In contrast to the adults, the juveniles from troop BA show very little change in cortisol over time. Troop DC shows a strong negative correlation between freeze lag and cortisol, and AA shows a weaker yet significant downward trend. AB shows a significant increase in cortisol over time. It is also clear that slopes for each troop do not correlate well across age classes.

Discussion

By developing GLM and GLMM models that included predictors for both ethanol preservation and freeze lags, I was able to control for differential patterns of steroid hormone degradation during storage. The results of these models also show that exposure to ambient conditions without preservative for less than eight hours did not significantly affect cortisol levels in either adults or juveniles. In samples collected from adults, the length of time spent unfrozen in ethanol positively correlated with measurable cortisol; this finding is consistent with previous work with baboon samples (Khan et al., 2002; Lynch et al., 2003). This is likely because these samples underwent a prolonged extraction phase, which could increase the amount of cortisol and related metabolites in ethanol. It is also likely that metabolites of cortisol or related hormones degraded from forms that were not detected by this assay into forms that were (Khan et al., 2002). Storage time did not significantly affect measurable cortisol in samples collected from juvenile females; they showed no significant change in cortisol for up to 181 days. Patterns of degradation were significantly different across troops, and these troop-specific patterns also varied considerably across age classes.

These results suggest that field preservation and exposure to ambient conditions may have a more complex effect on measurable steroid hormones from fecal samples than previously assumed. While degradation patterns are known to differ across hormones and species (see above), it is now clear that they can also vary across age classes and within populations.

These significant differences in cortisol degradation were not the result of methodological bias. Samples from adults and juveniles were collected during three discrete field seasons, and no age class or troop was specifically targeted either early or late in the field season. All samples were preserved following the same protocol and without regard to the age class or troop membership of the individual sampled. As there was no significant interaction between the two predictors for lag, I can also rule out the possibility of a complex interaction between these two factors.

Many recent studies are highlighting new complexities associated with the use of feces for hormonal analysis. Males and females of the same species can show different profiles of excreted metabolites for the same hormone (Goymann, 2005), which might in turn lead to different patterns of degradation. Lynch et al. (2003) report overall increases in measured cortisol; however, as seen here, they also report considerable variation in the direction of change amongst individual samples.

As degradation of steroid hormones in feces is largely the result of the actions of intestinal microorganisms, it is possible that these effects are in part the result of different populations of intestinal bacteria. If different kinds or combinations of bacteria inhabit the digestive tract of baboons of different ages or different troops, then they might convert cortisol into different metabolites that may have more or less affinity for a given assay antibody. While ethanol effectively halts the activity of these bacteria, their actions before preservation may also create metabolites with variable stability or of differing extraction efficiencies. Differences in diet might also help account for both age class and troop differences. The composition and combination of foods eaten might influence the activity of the intestinal microorganisms and might affect the metabolites present in feces at the time of defecation, which might in turn influences patterns of degradation (Wasser et al., 1993; Goymann, 2005; Goymann et al., 2006). While no dietary data is available for this population during this time, juvenile baboons may have different food preferences than adult baboons. Each troop will also have access to different foods as determined by availability within their home range.

Those studies that first noted problems with hormone degradation suggested that validation studies specific to a given hormone in a given species should be completed in order to classify and control for these patterns of effect. If, as indicated here, these patterns are more complex than anticipated, then statistical control is a far more feasible approach. Future studies can avoid these problems altogether by using field preservation methods that are both more stable and more transportable (Beehner & Whitten, 2004; Ziegler & Wittwer, 2005). Data collected using previous techniques can, however, be successfully analyzed with proper statistical controls. Recent studies have begun to use

GLM and GLMM to analyze fecal hormone data (e.g. Beehner et al., 2005; Engh et al., 2006b), and including predictors for storage lags provides a simple and reliable solution to this problem and it may identify factors that influence patterns of steroid degradation in feces.

Table 1. Type 3 Tests of fixed effects: Full adult female model. Blue indicates predictors that might be impacted by complex patterns of degradation. Italics denote main effects and interactions that were found to be significant predictors of cortisol in this model.

Effect	**DF**	**F**	**P**
Ethanol Preservation Lag	1	0.37	0.5458
Freeze Lag	*1*	*5.01*	*0.0254*
Age	1	1.10	0.2956
Reproductive Status	5	0.68	0.6373
Number of Adult Female Relatives	1	0.37	0.5409
Rank	2	0.05	0.9485
Troop	*7*	*2.68*	*0.0095*
Field Season	*2*	*5.85*	*0.0030*
Ethanol Preservation Lag*Freeze Lag	1	0.09	0.7645
*Freeze Lag*Troop*	*7*	*2.70*	*0.0091*
Age*Field Season	2	0.74	0.4757
Age* Number of Adult Female Relatives	1	0.52	0.4707
Reproductive Status*Field Season	10	0.79	0.6380
Reproductive Status*Number of Adult Female Relatives	5	0.28	0.9232
Number of Adult Female Relatives*Field Season	2	0.37	0.6912
Number of Adult Female Relatives*Rank	2	0.85	0.4288
Rank*Troop	14	0.88	0.5788
*Rank*Field Season*	*4*	*2.67*	*0.0314*
*Troop*Field Season*	*14*	*2.08*	*0.0112*

Table 2. Type 3 tests of fixed effects: Reduced adult female model. Blue indicates potential degradation-related predictors. Italics denote main effects and interactions that were found to be significant predictors of cortisol in this model.

Effect	**DF**	**F**	**P**
Freeze Lag	*1*	*5.53*	*0.0189*
Rank	2	1.82	0.1634
Troop	*7*	*2.86*	*0.0059*
Field Season	*2*	*13.16*	*<0.0001*
*Freeze Lag*Troop*	*7*	*2.73*	*0.0083*
*Rank*Field Season*	*4*	*2.74*	*0.0279*
*Troop*Field Season*	*14*	*2.21*	*0.0063*

Table 3. Type 3 tests of fixed effects: Full juvenile female model. Blue indicates predictors that might be impacted by complex patterns of degradation. Italics denote main effects and interactions that were found to be significant predictors of cortisol in this model. †Denotes those that were insignificant in full model, but significant in reduced.

Effect	DF	F	P
Ethanol Preservation Lag	1	1.68	0.1983
Freeze Lag	1	1.82	0.1805
† Age	1	1.26	0.2645
† Mom Alive	1	2.06	0.1546
† Number of Adult Female Relatives	1	0.03	0.8597
Mom's Rank	*2*	*10.20*	*<0.0001*
Troop	*5*	*13.45*	*<0.0001*
Field Season	*2*	*8.43*	*0.0004*
Ethanol Preservation Lag*Freeze Lag	1	0.61	0.4369
*Freeze Lag*Troop*	*5*	*12.56*	*<0.0001*
Age*Mom Alive	1	0.28	0.5958
Age* Number of Adult Female Relatives	1	0.01	0.9190
*Age*Mom's Rank*	*2*	*7.08*	*0.0013*
*Mom Alive*Number of Adult Female Relatives*	*1*	*21.70*	*<0.0001*
Number of Adult Female Relatives*Field Season	2	1.01	0.3662
*Mom's Rank*Number of Adult Female Relatives*	*2*	*20.94*	*<0.0001*
*Mom's Rank*Field Season*	*4*	*2.92*	*0.0246*
*Troop*Field Season*	*10*	*7.35*	*<0.0001*

Table 4. Type 3 tests of fixed effects: Reduced juvenile female model. Blue indicates potential degradation-related predictors. Italics denote main effects and interactions that were found to be significant predictors of cortisol in this model.

Effect	DF	F	P
Freeze Lag	1	0.99	0.3215
Age	*1*	*8.03*	*0.0055*
Mom Alive	*1*	*13.95*	*0.0003*
Number of Adult Female Relatives	*1*	*10.69*	*0.0014*
Mom's Rank	*2*	*10.05*	*<0.0001*
Troop	*5*	*13.42*	*<0.0001*
Field Season	*2*	*10.90*	*<0.0001*
*Freeze Lag*Troop*	*5*	*12.26*	*<0.0001*
*Age*Mom's Rank*	*2*	*6.00*	*0.0034*
*Mom Alive*Number of Adult Female Relatives*	*1*	*38.65*	*<0.0001*
*Mom's Rank*Number of Adult Female Relatives*	*2*	*22.36*	*<0.0001*
*Mom's Rank*Field Season*	*4*	*2.50*	*0.00466*
*Troop*Field Season*	*10*	*7.32*	*<0.0001*

Table 5. The 95% confidence intervals for slopes for the effect of storage time on adult cortisol levels by troop. Red italics indicate those slopes that are significantly negative. Green italics indicate those that are significantly positive.

	Juvenile Females		**Adult Females**	
Troop	**Slope**	**95% CI**	**Slope**	**95% CI**
AA	***-50.36***	*-91.88, -20.84*	-1.30	-20.05, 17.44
AB	***107.19***	*51.30, 163.07*	-9.28	-33.26, 14.69
AC	-	-	-10.99	-30.16, 8.18
BA	-2.63	-49.89, 44.63	***-37.84***	*-56.75, -18.94*
C	-	-	-2.63	-14.25, 8.99
DA	11.79	-24.74, 48.31	4.80	-3.94, 13.55
DB	118.68	-42.86, 280.21	2.45	-4.79, 9.69
DC	***-274.03***	*-360.11, -187.96*	1.32	-6.81, 9.45

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CHAPTER 2:

ECOLOGICAL DISASTER AND THE ADAPTIVE VALUE OF THE CHRONIC STRESS RESPONSE

But pain or suffering of any kind, if long continued, causes depression and lessens the power of action, yet is well adapted to make a creature guard itself against any great or sudden evil.

The Autobiography of Charles Darwin
Charles Darwin, 1876

The first central concept of stress physiology is if one becomes stressed like a normal mammal, which is, for example, 30 seconds of screaming terror running across the Savannah, the stress response is perfectly designed to save your life. . . . The system did not evolve to be chronically activated.

Sapolsky, 2000

Abstract

Organisms that successfully respond to threats have higher fitness than those that fail to mount appropriate responses. The vertebrate stress response has evolved as a suite of physiological and behavioral strategies for coping with these threats, and the mechanisms of this response remain remarkably conserved across taxa. The system's major weakness has always been believed to be its enigmatic predisposition for chronic and pathological activation. In this chapter, I propose that this tendency for continued activation in the face of lasting challenges can carry its own adaptive benefits and is not, as has been long assumed, merely maladaptive. This is the first study to present evidence from a wild population in favor of an adaptive approach to the chronic stress response.

I studied a population of wild olive baboons (*Papio cynocephalus anubis*) and used demographic and hormonal data to investigate the adaptive value of the chronic stress response. I first identified a model chronic stressor—the sudden loss of a valuable resource—tantamount to an ecological disaster. I then examined the baboons' prolonged physiological and behavioral response to this stressor in order to identify practical coping strategies and examine their adaptive value. The results of this study provide strong evidence that the chronic stress response plays a crucial adaptive role. While acute stressors predominate, I found evidence that even chronic stressors of disastrous magnitude are by no means rare in an evolutionary sense. In reaction to such a stressor, the baboons implemented a number of cortisol-mediated coping strategies, indicating that sustained activation of the stress response system can promote lasting changes in behavior and physiology. Finally, the implementation of these long-term coping strategies can provide immediate benefits by optimizing condition and survival, outweighing any delayed risk of pathology.

I also discuss the widespread implications of this approach for our understanding of the vertebrate stress response system and its evolutionary history.

Introduction

I. The evolution of the chronic stress response

Long-term activation of the vertebrate stress response system presents an evolutionary conundrum for stress researchers. The stress response system, particularly the release of glucocorticoids, clearly increases fitness in the short term by allowing an organism to react to an acute stressor and maximize its chances of survival. Prolonged stress responses, however, significantly increase an individual's risk for a plethora of stress-related diseases.

Two main arguments have been offered to explain the puzzling persistence of a stress response system that is vulnerable to such a costly weakness as chronic activation. First, it has been assumed that chronic stressors are relatively rare in nature. Second, while short term activation is adaptive, the chronic activation of the system is simply a maladaptive side effect and does not represent the normal functioning of the system for which natural selection would have selected. This view continues to be prevalent in the few recent studies that consider the evolution of this system (e.g. Francis et al., 1999; Sapolsky et al., 2000; Glannon, 2002; McEwen, 2004; Sands & Creel, 2004; Bergman et al., 2005; Sapolsky, 2005; Wingfield, 2005).

I propose that the ability to respond to prolonged stressors with a chronic stress response may have profound fitness benefits as well. In response to a long-term stressor, the chronic release of glucocorticoids such as cortisol and corticosterone prompts continued metabolic and behavioral changes that allow the animal to maximize its chances of survival in "the best possible condition"(Wingfield & Kitaysky, 2002). Under this model, short-term survival benefits in response to stressors that last for more than the usual few moments will outweigh the delayed pathological costs such that we can explain the persistence of the chronic stress response throughout vertebrate evolution.

In nature, the daily stressors faced by animals are largely acute and fleeting, and brief bursts of glucocorticoids will be sufficiently beneficial in terms of securing survival and fitness. Many recent studies, largely tied to the advent of noninvasive hormonal sampling, aim to identify chronic stressors in wild populations over a variety of vertebrate taxa: fish (McBride et al., 1986), amphibians (Belden et al., 2005; Homan et al., 2003), reptiles (Romero & Wikelski, 2001; Woodley et al., 2003), birds (Kotrschal et al., 1998; Romero et al., 2000; Clinchy et al., 2004), and a variety of mammals including rodents (Hik et al., 2001), carnivores (Goymann et al., 2001), and primates (Weingrill et al., 2004). The results of these and other studies suggest that many animals face a barrage of chronic stressors during the course of their lives. If chronic stressors are *not* rare as presumed, and the chronic stress response does have an adaptive value, we can begin to explain the paradoxical maintenance of this aspect of the vertebrate stress response.

II. How can we study the adaptive value of the chronic stress response?

A. Understanding the basic physiology of the stress response

The stress response system is initially activated by the brain in the face of a stimulus that it perceives as a threat to the organism. The brain then initiates a cascade of physiological responses involving activation of the nervous and endocrine systems, including the hypothalamic-pituitary-adrenal (HPA) axis, and the eventual release of glucocorticoids from the adrenal glands. Glucocorticoids (primarily cortisol in humans and other primates, and henceforth I will use the two terms interchangeably) in turn provoke a number of metabolic and behavioral changes that, in sum, promote a shifting of energy resources from maintenance functions (i.e. anabolic functions) to those that will promote immediate survival (i.e. catabolic functions). For example, the proverbial zebra in the midst of a lion chase will divert resources from building energy reserves for the future (which he may never see) and instead convert energy into a form that is immediately useful for his active muscles. Along the same lines, energy normally devoted to "long-range" goals like immune or reproductive function is similarly diverted to meet his more immediate energetic needs.

The rationale for the body's acute responses to momentary stressors has always been apparent. But in the face of stressors that don't stop or dissipate after a few minutes or a few hours, these same physiological responses can become detrimental. Depleted energy reserves put an organism at risk in the future if for any reason it cannot meet its daily needs via immediately available resources. A reproductive or immune system that is not properly maintained may slowly deteriorate, leading to a host of negative consequences. Chronic stress has clear and profound consequences for every system in the vertebrate body.

It is hard to pinpoint a definition of "chronic" in terms of the stress response system. It has become clear that the duration of stressors can fall on a long continuum, from those that last for seconds to those that persist for years. Chronic has traditionally been used to designate any stressor that lasts longer than the "normal" acute stressor—anything that provokes a stress response for hours or days or longer (Creel, 2001). More recent approaches focus on chronic in terms of those stressors that tip the stress response to the side where pathological risks exist. A new set of terms has been introduced that involves the central concept of *allostasis* (McEwen, 1998; McEwen & Wingfield, 2003), and they are meant to focus on the fact that this tipping point may fall in different places along the continuum for different species, different stressors, and even the same animal facing the same stressor on different days. Allostasis is defined as the response to acute stressors, allostatic load as a response that has potentially pathological consequences, and allostatic overload as responses that carry assured negative physiological ramifications. For our purposes, I will return to the more simplified definition of chronic stressors as those that cause a prolonged or frequent stress response resulting in elevated glucocorticoid levels over days, months, or even years. Such continued exposure carries definite physiological risks.

B. The physiological and behavioral consequences of the chronic stress response

The ever-increasing prevalence of stress-related diseases has led to annual medical expenses in excess of $200 billion in the United States alone (McEwen & Lasley, 2002). In response to this continuing medical crisis, the pathological consequences of chronic cortisol exposure have been well studied in humans as well as captive and wild animals. Chronic cortisol exposure can significantly increase risk of physiological and behavioral diseases including heart disease, asthma, infertility, autoimmune disorders, eating disorders, and depression (Sapolsky, 1992; Sapolsky, 2005). Each of these diseases, and many others, has been studied in depth in the biomedical and psychiatric literature.

There has, however, been a relative paucity of research on the potential benefits of the physiological and behavioral changes elicited by cortisol and other glucocorticoids. The few studies that suggest that chronic responses could be beneficial usually say something perfunctory about how there may be some benefit up until a certain mystical level of "chronicness". In order to begin to consider the idea that chronic cortisol release may be an adaptive physiological response to chronic stressors, we need to think about how the associated physiological and behavioral changes might have short-term beneficial consequences and not just long-term pathological risks. The basic premise of this new approach assumes that, within an evolutionary framework, the immediate benefits of these changes promoted by chronically elevated cortisol will outweigh the delayed risks.

Consider the consequences of prolonged stress on reproductive physiology and behaviors. An enormous body of literature ties chronic glucocorticoid exposure to dozens of reproductive disorders afflicting both males and females in an extensive variety of vertebrate species. Continued cortisol exposure in males has been linked to decreases in testosterone, spermatogenesis, and sperm quality. In females, chronic cortisol release has been associated with lengthened and irregular cycles, anovulation, amenorrhea, miscarriage, premature parturition, and delayed menarche. Extreme cases for either sex can result in long-term and even permanent infertility. Chronic cortisol exposure can significantly impact reproductive behaviors as well. Elevated cortisol levels have been associated with overall decreases in sexual behaviors, including receptivity, arousal, and even the territorial and competitive behaviors associated with mating. Stress is anything but an aphrodisiac.

The negative impact of reduced fertility and reproductive behavior on an individual's lifetime reproductive success is obvious. What is less obvious, however, is that these physiological and behavioral "pathologies" may actually constitute successful strategies for coping with a stressful situation. What appears to be purely pathological may, under certain circumstances, have an overall benefit to reproductive success. One of life history theory's central tenants is that reproduction is energetically costly as well as risky, and it can be beneficial for organisms to avoid these costs and risks when conditions are appreciably suboptimal. Indeed, life history theory predicts a physiological response astoundingly similar to that induced by chronic cortisol exposure: amenorrhea, temporary infertility, delayed menarche. Chronic release of cortisol may represent a mechanism by which animals facing chronic stressors can postpone reproductive costs and survive in a better condition than they would otherwise, thereby maximizing future reproductive success and perhaps overall fitness. By shutting down the reproductive system during stressful times under the influence of cortisol, an individual may not only minimize energetic expenses, it may also curtail associated risks at a time when body condition is already compromised. In addition, when reproduction is delayed, a female will be less likely to bear offspring during a time when their chance of survival may still be low. This multipronged suppression of reproduction could effectively prevent a very risky investment. This would be particularly true for any organism with extended parental care—especially for female mammals.

Instead of discounting the long-term actions of cortisol we should assume that the widespread actions of the glucocorticoid stress response system—both acute and chronic—reflect the direct actions of natural selection. While this particular idea has been alluded to in the literature (Carr & Summers, 2002; Greenberg et al., 2002; Wingfield & Kitaysky, 2002; Wingfield & Sapolsky, 2003; Flinn, 2006; Koolhaas et al., 2006) and makes intuitive sense, this is the first field study to explore this approach.

C. Three critical questions

To study the possible adaptive value of the chronic stress response and suggest that it is more than an evolutionary side-effect, three critical questions must be answered: Are chronic stressors truly rare? What cortisol-mediated strategies might we see in response to chronic stressors? Are these strategies adaptive? I will briefly consider how each of these questions could be studied.

There is abundant literature on stress physiology for a variety of vertebrate taxa. I have chosen to focus on the primate stress response. Because of the complex nature of primate society, primates offer a unique opportunity to study a variety of physiological, ecological, and social stressors. Primates have also been studied extensively in the wild, and endocrinological and long-term demographic and behavioral data exist for a number of populations. In addition, research on the primate stress response is relevant to a vast number of disciplines including behavior, ecology, anthropology, medicine, conservation, psychology, and psychiatry.

ARE CHRONIC STRESSORS TRULY RARE?

To answer this question, we must first consider what factors constitute chronic stressors for primates. A brief perusal of current stress literature in wild or naturalistic settings suggests chronic stressors are a regular occurrence in the lives of most primates. There are a number of accepted ways to classify stressors; for example, Greenberg et all (2002) offers six types based on frequency and duration. I have chosen to divide stressors based on their causes: physiological, social, and ecological. Each will be presented here and discussed in greater detail below. Age (Sapolsky & Altmann, 1991; van Schaik et al., 1991; Robbins & Czekala, 1997) and reproductive state (van Schaik et al., 1991; Altmann et al., 2004; Weingrill et al., 2004; Bales et al., 2005) have been associated with chronically elevated cortisol in a number of primate species. Identified social stressors include either high or low rank (Sapolsky, 1982; van Schaik et al., 1991; Barrett et al., 2002; Cavigelli et al., 2003), social isolation or lack of social support (Sapolsky et al., 1997; Beehner et al., 2005), and social instability including hierarchy changes (Sapolsky, 1983) and immigration events (Alberts et al., 1992; Cristobal-Azkarate et al., 2007). Additional social stressors include suboptimal group size (Pride, 2005; Beehner et al., 2006; Chapman et al., 2007) and mating behaviors (Barrett et al., 2002; Lynch et al., 2002; Bergman et al., 2005). Known ecological stressors include seasonal changes (Strier et al., 1999; Lynch et al., 2002; Weingrill et al., 2004), resource loss (Chapman et al., 2007), and ecological disaster (Sapolsky, 1986; Pride, 2005). As this is a new and developing field, this list is certainly far from comprehensive. It will suffice to say that the typical wild primate faces many of these chronic stressors during the normal course of its life.

WHAT CORTISOL-MEDIATED STRATEGIES MIGHT WE SEE IN RESPONSE TO CHRONIC STRESSORS?

Addressing this question involves identifying potential coping strategies, such as the temporary shut-down of reproduction, that are known to be associated with chronically elevated cortisol. These strategies will probably involve maximizing likelihood of survival while minimizing energetic costs in the face of chronically stressful states and events. Some strategies may assist individuals in avoiding such stressors altogether. Once likely strategies have been identified, we will need to determine whether these strategies are seen in the wild in conjunction with chronic stressors.

Suppression of the reproductive and immune systems has been commonly understood to be a cost saving mechanism, allowing animals to shunt energy to systems that need immediate attention. Metabolic changes are amongst the most commonly discussed effects of chronic stress, so they play a likely role in the suite of successful coping strategies. Long-term changes include continued increases in available energy, improved efficiency, and changes in energy storage. Another commonly studied putative strategy is the increase in behaviors commonly associated with anxiety: increased vigilance, inability to focus, insomnia, and even a drive to escape or move to a safer environment. Other coping strategies may also involve changes in foraging behaviors and social behaviors.

ARE THESE CHRONIC STRESS RESPONSE STRATEGIES ADAPTIVE?

As most of the coping strategies we can expect to see have traditionally been considered deleterious and even pathological, the next step will be to determine whether such cortisol-mediated responses to chronic stressors can actually improve lifetime fitness? Do they improve chances of survival in optimal condition? Do they help prevent risky energetic investments during stressors? As a result, is an animal both in a better condition *and* better able to make safer fitness investments once the stressors pass?

There is a certain element of wisdom to suppressing reproduction, thereby physiologically preventing pregnancy and a risky investment in offspring during times of stress. Many of the other potential strategies also have conceivable fitness benefits in the face of a long, severe stressor. In many cases, the effects of chronic stress can vary depending on many factors including the nature of the stressor and the condition of the individual. For example, chronic stress can sometimes suppress and other times increase appetite (Dallman et al., 2004). This variability may represent a complex repertoire of available strategies that allow the chronic stress response the necessary plasticity to address the specific needs presented by a given situation, much as we see in the acute stress response.

Metabolic changes that control the availability of energy in the face of hardship would, in part, allow animals to adjust to continued variability in food availability while maintaining the minimal body systems necessary for survival. Anxiety has long been believed by evolutionary psychiatrists to have an adaptive purpose; it pays to be paranoid if the world really is out to get you. If a threat sparks the chronic release of cortisol, there is likely a continued risk, in which case it would be beneficial for an animal to increase vigilance, decrease attentiveness to non-survival based activities, and perhaps sleep less soundly when the danger persists at night. Whenever food is scare or energy is in great demand, both of which are common features of chronic stressors, increased foraging would be clearly beneficial. Any such change would result in significant changes in time budgets, perhaps leaving less time for behaviors not directly associated with immediate survival such as resting and social behavior (Dunbar, 1992).

Immune suppression in the face of a chronic stressor may present a less obvious benefit, allowing an animal to shunt energy to more crucial systems. It may also suppress inflammation and other immune responses, say in the case of an ankle injury, which might impair an animal’s ability to escape, forage, or otherwise cope with a continued stressor. In such a case, it might be better to risk further damage to the joint rather than face starvation and death. Cortisol-induced changes in immune function may also allow the body to counteract the increased risk of autoimmune diseases that often accompanies chronic stressors (Raberg et al., 1998).

III. Hypothesis: The chronic stress response has an adaptive value

A. Chronic stressors are not rare

There is considerable support in the primate literature to suggest that chronic stressors, as indicated by states and events that cause prolonged elevations in cortisol, are by no means rare in natural populations. Here, I will first define the focus of this review, briefly consider overarching factors that can be used to characterize chronic stressors, and then review the relevant primate literature.

In order to understand the evolution of the chronic stress response, it is imperative that we consider stressors impacting organisms that face the sort of historically relevant survival pressures that cannot be replicated in captivity. I therefore focus on research on wild primate populations in an attempt to identify and characterize naturally occurring chronic stressors. Primatologists have attempted to identify some stressors based on non-hormonal data (e.g. heart rate, Bowers et al., 1998), but I will focus on those studies for which cortisol has been found to correlate with chronic stressors. I make no attempt to distinguish between those that are sustained stressors and those that are intermittent but regular, as both can result in prolonged cortisol exposure. I have also chosen to consider both factors that provoke a stress response as well as those that appear to ameliorate chronic stress levels (e.g. social support)—the absence of which (e.g. social isolation) would likely constitute a chronic stressor.

Perhaps the most consistent finding in the primate stress literature—for that matter all vertebrate stress literature—is that anything that involves unpredictability and/or uncontrollability on the part of the individual can constitute a stressor. Any state or event that increases the frequency and/or duration with which an individual encounters one of these stressors can result in chronic stress. A chronic stressor will generally be some unpredictable circumstance over which the individual can assert little or no control, and that it must face with some frequency or for a prolonged duration.

PHYSIOLOGICAL STRESSORS

A handful of physiological factors have been associated with chronically elevated cortisol levels. The clearest case has been made for reproductive state, as studies report that pregnant and/or lactating females show elevated cortisol levels for a number of species including tamarins (Bales et al., 2005), macaques (van Schaik et al., 1991), and baboons (Altmann et al., 2004; Weingrill et al., 2004). This is likely a result of the increased energetic demands associated with gestation and lactation, although there is some indication that much of the gestational rise results from cortisol produced by the placenta and fetal adrenal gland (reviewed in Pepe & Albrecht, 1995) and may therefore represent some other adaptive purpose. Regardless, many female primates show chronic elevations of cortisol during these stages. Males often show similar signs of chronic stress during the breeding season (capuchins, Lynch et al., 2002; macaques, Barrett et al., 2002) or while on consortships (baboons, Bergman et al., 2005), but it is unclear whether these are the result of energetically costly physiological states or the associated social factors. Cortisol levels have also been shown to rise with age in captivity (Goncharova & Lapin, 2000; Goncharova & Lapin, 2002) and in the wild (baboons, Sapolsky & Altmann, 1991; macaques, van Schaik et al., 1991), perhaps as a result of dysregulation of the HPA system (Sapolsky et al., 1986; Gust et al., 2000). This result has been far from consistent in wild populations, and no correlation between age and cortisol has been found in the majority of recent studies (muriquis, Strier et al., 1999; capuchins, Lynch et al., 2002; female baboons, Beehner et al., 2005; male baboons, Bergman et al., 2005; tamarins, Bales et al., 2006). Cortisol has also been associated with adolescence in male gorillas (Robbins & Czekala, 1997)

SOCIAL STRESSORS

Social stimuli have long been identified as potent stressors (Mason, 1968) and have traditionally received disproportionate attention in the primate stress literature. Many assume that social interactions represent the bulk of a primate's stressors (e.g. Cristobal-Azkarate et al., 2007) and that social stressors are the predominant cause of stress-related pathologies (e.g. Wingfield, 2005). Some have gone so far as to suggest that in human society, non-social stressors are extremely rare (Flinn, 2006). Everyone agrees that social stressors are a relevant and regular source of stress in the lives of wild primates. In fact, some potential chronic stressors (e.g. subordinate status) would affect a significant proportion of primates throughout their entire lives.

Rank has been the most studied social stressor. The link between rank and stress is hardly clear. Even within a given species, the relationship between rank and stress can vary between sexes and between populations. Some studies have found that subordinates show chronically elevated cortisol (male baboons, Sapolsky, 1982; male macaques, van Schaik et al., 1991), others have found that high rank is more stressful (male macaques, Barrett et al., 2002; female lemurs, Cavigelli et al., 2003), and still others have failed to find any clear relationship (female lemurs, Pride, 2005; female tamarins, Bales et al., 2005; male capuchins, Lynch et al., 2002; female macaques, van Schaik et al., 1991; female baboons, Beehner et al., 2005; male baboons, Bergman et al., 2005; male gorillas, Robbins & Czekala, 1997). In some cases, the "styles" of dominant (male baboons, Sapolsky & Ray, 1989; Ray & Sapolsky, 1992) or subordinate (Virgin & Sapolsky, 1997) individuals are more important than the social position itself. Clearly, there is much variation both across and within primate taxa.

In a recent meta-analysis, Abbott et al (2003) found a number of major factors that predict whether rank will be associated with stress: little social control, few outlets for stress, no social support, and increased exposure to stressors. If one rank class is more closely associated with some combination of these four factors, there will likely be a correlation between rank and elevated cortisol. For example, subordinate male baboons have very little social control, have few individuals on which to displace frustration and aggression, have little to no social support, and are subject to a number of other rank-associated pressures (Sapolsky, 1982); they have been found to have chronically higher cortisol levels. What is clear, in spite of the inconsistencies, is that for many primates, social rank can constitute a chronic stressor, and a situation that would affect many individuals for a duration that is clearly associated with considerable pathological risks (as reviewed in Sapolsky, 2005).

When a hierarchy is volatile, primates often show the physiological signs of chronic stress (baboons, Sapolsky, 1983). As such, social instability has been presented as another clear predictor of prolonged cortisol exposure. Perturbations amongst high-ranking individuals are the most likely to cause chronic stress. Recent immigration events have also been shown to cause elevated cortisol, both for the immigrating animal and for the members of its new social group (howlers, Cristobal-Azkarate et al., 2007; baboons: Alberts et al., 1992; Beehner et al., 2005; Bergman et al., 2005). This is likely due to the fact that such events disturb the hierarchy and, when males transfer, often represent a significant increase in the risk of infanticide.

Social support has long been presented as a factor likely to ameliorate chronic stress in primates. The flip side, then, is that lack of social support and/or social isolation would be a clear chronic stressor. There is little research in wild populations that ties social support to cortisol, but there is some preliminary corroboration. Cortisol levels in chacma baboons rise significantly when social bonds are disrupted (Engh et al., 2006b). In the face of immigration events that present great risk of infanticide, female chacma baboons with friends show significantly smaller elevations in cortisol than those without (Beehner et al., 2005). Silk (2003) found that female baboons who form close relationships with other females have greater reproductive fitness, and she suggests that cortisol may play a role in this effect. In addition, Abbott (2003) cites the lack of social support as one key factor that would likely tie rank to stress. While few studies have considered stressors of any sort in wild juveniles (gorillas, Robbins & Czekala, 1997; baboons, Gesquiere et al., 2005), mother-infant separation in captive primates results in a clear and prolonged elevation of cortisol (Coe et al., 1978; Smotherman et al., 1979; Levine & Mody, 2003; Bardi et al., 2005). Isolation of gregarious animals has been one of the most widely used models of stress and depression (e.g. McKinney, 1984), further suggesting that lack of social support constitutes a potent chronic stressor in the wild where such support might be particularly valuable.

A few other social factors, including suboptimal group size (baboons, Beehner et al., 2006; lemurs, Pride, 2005; colobus, Chapman et al., 2007), breeding season conflicts (capuchins, Lynch et al., 2002; macaques, Barrett et al., 2002), and consortships (baboons, Bergman et al., 2005) have also been associated with chronic stress, although the body of evidence is not as yet extensive.

ECOLOGICAL STRESSORS

Because most studies have focused on social stressors, less is known about ecological factors that might represent chronic stressors. Interest has increased recently as concern rises over the wide-ranging impacts of global climate change and other human influences, as evidenced by the advent of the field of conservation physiology (Wikelski & Cooke, 2006). Seasonal changes have long been presumed to be a yearly stressor with chronic effects. Some studies have found that dry or cold seasons are correlated with elevated cortisol (Strier et al., 1999; Lynch et al., 2002; Weingrill et al., 2004; Pride, 2005), while others have found no significant relationship (Bergman et al., 2005). There is reason to believe that seasons directly related to notable variability in day length (as opposed to rainfall) may be more chronically stressful than those associated with changes in rainfall patterns (Weingrill et al., 2004). It has also been proposed that, as seasonal changes are predictable, most organisms have a set of coping strategies that minimize their physiological impact (McEwen & Lasley, 2002). Chronic resource loss in general has been associated with elevated cortisol in lemurs (Pride, 2005) and capuchins (Chapman et al., 2007), and is another popular "naturalistic" model of depression in animals (Bartolomucci, 2005). Little direct evidence has as yet been presented in the literature. However, the stressful effects of many stressors such as seasonal changes, low rank, and pregnancy may at some level reflect the effects of decreased resource availability.

Ecological disasters—drought, storms, habitat loss—are a particularly clear case of a potential chronic stressor, due at least in part to the long-lasting effect they can have on resource availability. Little cortisol data as yet exist for primates facing such a disaster, but the potentially prolonged and severe nature of this type of stressor is notable. Sapolsky (1986), for example, found a link between drought and chronically elevated cortisol in male baboons. There is glucocorticoid data for nonprimates, particularly birds, experiencing extreme storms (Wingfield et al., 1983; Smith et al., 1994; Astheimer et al., 1995) or human influences (Mullner et al., 2004; Ellenberg et al., 2007). There is also considerable primate demographic data following extended droughts, suggesting significant decreases in birth rates and increases in mortality (Hamilton, 1985; Dittus, 1988; Decker, 1994; Gould et al., 1999). These disaster-related demographic patterns have yet to be directly tied to elevations in cortisol. The long-lasting effects such a stressor might have can be seen in the dramatic example of how natural disasters affect human populations; resources remain diminished during prolonged recovery periods lasting months or even years. Such events are known to elevate cortisol and increase risk for depression and other stress-related disorders (e.g. Rotton et al., 1997; Sattler et al., 2006) The effects of such events on the physiology of primates and other wild animals will continue to be of primary concern as human actions create drastic consequences for threatened animal populations worldwide.

Ecological disasters such as drought or severe storms may also constitute just the sort of adaptive pressure for which the chronic stress response may have evolved: an entirely unpredictable stressor, beyond the control of the organism, with long-lasting effects. Any generalized pattern of coping strategies, as might logically be controlled by cortisol, that decreases energetic expenses and implements successful behavioral changes would be a clear adaptive boon (see Wingfield & Kitaysky, 2002). If in such a circumstance the chronic stress response promoted survival, it would clearly be well worth the long-term pathological costs.

It should already be quite clear that stressors that cause chronic elevations in cortisol are far from rare. In this chapter I will aim to address this matter with empirical evidence.

B. Cortisol mediates physiological and behavioral coping strategies in response to chronic stressors

Many of the well known physiological and behavioral responses to continued cortisol exposure can be re-envisioned as part of a set of beneficial coping strategies for animals facing adversity. Any of the fundamental components of the vertebrate chronic stress response that could conceivably minimize immediate energetic costs and maximize survival should be reexamined in this light. Ironically, the best place to look for potentially beneficial strategies is the literature on the pathological effects of chronic cortisol.

Once again, reproductive suppression makes a particularly likely candidate strategy for coping with prolonged stressors. Cortisol's short- and long-term effects on reproduction are well documented (Sapolsky et al., 2000; Wingfield & Sapolsky, 2003). Life history theory has long predicted that distressed animals will display exactly the reproductive responses promoted by cortisol, and adult female primates will likely do so by suppressing reproductive function as described above. A young female facing adversity might similarly avoid the costs of reproduction by delaying reproductive maturation, and cortisol is also known to mediate such an effect (Sapolsky, 1992; Sapolsky, 2005). Delays in the onset of menarche have been associated with known stressors in both monkeys (Bentley-Condit & Smith, 1997) and humans (Bale et al., 1996), and even moderate prenatal stress can significantly delay sexual maturation in rodents (Marchlewska-Koj et al., 2003). Cortisol has also been suggested as an underlying mechanism causing differential patterns in maturation for young female baboons (Bercovitch & Strum, 1993; Johnson, 2003). Such mechanisms of suppression could easily give an animal a survival advantage over another who continues to use valuable energy reserves for risky reproductive purposes.

The same explanation holds for cortisol's well known effects on the immune system (Sapolsky, 1992; Sapolsky et al., 2000). While the immune system is undeniably crucial in the fight against viruses, bacteria, and cancer, many of its long-term preventative functions may not be essential when short-term survival is at stake. Moreover, current research on the relationship between stress and the immune system suggests that our understanding of the relationship between cortisol and the immune system has been overly simplistic. What has long been believed to be general immune suppression in response to cortisol may in fact be a subtle yet efficient reallocation of resources within the immune system to components that focus on more immediate risks (e.g. Dhabhar, 2002). This finding actually lends itself to this new adaptive approach, as perhaps cortisol plays a role in coordinating a number of similarly subtle yet stunningly choreographed responses to continued stress in other body systems as well.

Perhaps the most fundamental role of cortisol in an acute stress response involves the effects it has on metabolism. Simply put, cortisol promotes a critical shift in metabolic priority toward a focused goal: the immediate availability of energy. Similarly, chronic exposure to cortisol causes long-term changes in metabolic priorities (see Sapolsky, 1992). Energy stores are continuously tapped and can become depleted. When energy is available, it is not converted to the most efficient storage forms—triglycerides in adipose tissue, or glycogen in the liver. Instead, it is more likely to be circulating in more useable forms, resulting in continuously elevated blood glucose and triglycerides, both of which can pose serious health risks (e.g. Vogelzangs et al., 2007). All of this can occur at the expense of the more catabolic pursuits of metabolism such as muscle maintenance. But if an animal is facing a chronic stressor—perhaps one that involves food shortages—putting money in the bank, as the analogy usually goes, is not the best idea. The animal will likely be withdrawing it tomorrow. An animal facing a chronic stressor, for example the aftermath of a destructive storm, has immediate energetic needs. In all likelihood, these needs will be the same tomorrow, so perhaps such long-lasting shifts in metabolic activity toward readily available resources will allow the animal to quickly meet the volatile energetic needs often brought on by chronic stressors.

Chronic cortisol exposure can affect appetite and foraging behaviors as well. Cortisol is generally understood to be an appetite suppressant, but the link between stress and obesity has long suggested a role in promoting ingestive behavior as well. In fact, it appears that the effect cortisol has on appetite is plastic, promoting increased appetite in response to some stressors and decreased appetite in response to others (Torres & Nowson, 2007). Elevated cortisol has been implicated in an increased time spent foraging and/or reduced time resting, rapid movement between foraging sites, and altered ranging patterns (Berenstain, 1986; Dunbar, 1992; Decker, 1994; Weingrill et al., 2004). Recent studies suggest that cortisol interacts with insulin to promote energy availability and increase foraging for nutritious food, while decreasing the disruptive impact of hunger when food is not readily available (Dallman et al., 1993; Dallman et al., 2004).

While it is hard to disentangle the effects of malnutrition from those of cortisol, a large body of evidence (including studies on long-term food-restricted diets) suggests that famine and cortisol are too inextricably intertwined to separate entirely (Sabatino et al., 1991; Bercovitch & Strum, 1993; Sapolsky, 1995; Kitaysky et al., 1999; Muller & Wrangham, 2004; Urbanski et al., 2004; Buck et al., 2007)

The most profoundly sensible response to a chronic stressor may be to attempt to escape its negative impacts whenever feasible. Birds facing an ecological disaster often leave affected habitats, a behavior that has been correlated with increased cortisol levels (Astheimer et al., 1995; Ellenberg et al., 2007). In primates an escape response might manifest as shifts in ranging behaviors to unaffected areas or transfers to unaffected social groups. Depression and anxiety, two stress-linked conditions (Joyce et al., 1994), have been associated with both a strong drive to escape and a profound feeling of entrapment when escape is impossible (Gilbert et al., 2002; Gilbert et al., 2004). Escape and abandonment have also been correlated with both ecological disaster and cortisol in a few recent avian studies (Astheimer et al., 1995; Ellenberg et al., 2007; for review, see Wingfield & Ramenofsky, 1997). General anxiety-related behavior has also been implicated as a successful coping strategy, particularly in the case of social stressors like subordinate rank (Gilbert et al., 2002). If dominant individuals are offsetting their own social stresses, it would be beneficial for a subordinate victim to withdraw, decrease activity, and increase vigilance.

DELAYED RESPONSES TO ECOLOGICAL CHANGE

This is not the first study to suggest that cortisol might invoke coping strategies in the face of more persistent stressors. Wingfield and Kitaysky (2002) suggest that cortisol may in fact have "anti-stress" properties, invoking adaptive physiological and behavioral responses to longer-term stressors caused by a capricious environment. Furthermore, they suggest that chronic stressors may activate different levels of strategic response, all mediated by cortisol. Initially, such stressors might trigger minimal initial increases in glucocorticoids that activate primary coping strategies (e.g. increases in foraging behavior or increases in escape behaviors) that actually ameliorate the effects of the stressor, promote immediate survival, and prevent further release of cortisol. In most cases, these primary coping strategies allow animals to successfully navigate unpredictable chronic stressors without drastically increasing the risk of long-term pathology. In extreme cases, these primary coping strategies will work temporarily, but may eventually fail to prevent a second, more marked, and perhaps pathological stress response. A sustained chronic stress response will only be seen when stressors are severe, sustained, and/or compounded by additional hardships. Even then, an extreme stress response would be expected only after initial coping strategies prove ineffective. As a result, the worst ramifications of a chronic stressor may only be apparent long after its initial onset.

Such a system could explain previously reported delays in both the physiological and demographic responses to profound stressors. Gould *et al.* (1999) followed several lemur troops during a severe two-year drought. The demographic effects show a surprising one- to two-year delay between the onset of the drought and increased infant and adult mortality. The eventual recovery followed an additional two years later. Other studies suggest lasting effects of up to four years after the end of a disaster (Hamilton, 1985). Sapolsky (1986) followed a population of baboons responding to a one-year drought, and reported only a subtle increase in cortisol as well as an increase in foraging time, signs of male reproductive suppression, and decreased aggression. He saw neither significant changes in body weight nor notable demographic changes. In light of the work of

Wingfield and Kitaysky, this may represent a low-level first-tier chronic response and subtly successful coping strategies.

I further suggest that even the delayed and profound second-tier cortisol release suggested by Wingfield and Kitaysky could promote another line of advanced and sustained coping strategies in the face of the worst and least predictable stressors. It is in this more advanced response to the most unpredictable, unavoidable, and lasting stressors that we would most clearly expect to see the beneficial implementation of the suite of potential strategies outlined here. If conditions are particularly grave or remain suboptimal for considerable periods of time, animals may be driven to these more drastic measures. The pathological risks cannot be entirely discounted as a clear cost of the chronic stress response system, and one that has certainly played a significant role in the evolutionary history of this cortisol-mediated system. For such a system to have evolved, however, and to have been so remarkably conserved across vertebrate taxa, it seems likely that many of the potential strategies outlined above have been overwhelmingly beneficial throughout vertebrate history.

C. Cortisol-mediated coping strategies have an adaptive value

Acute cortisol release causes changes that are adaptive because they promote survival in the face of acute stressors. In much the same way, *chronic* cortisol release may cause longer-lasting changes that promote survival in the face of chronic stressors. These coping strategies may promote short-term benefits (e.g. decrease in costs associated with reproduction) and moderate-term benefits (e.g. a more rapid return to reproductive function when the stressor subsides) that under natural conditions outweigh any future costs associated with the increased risk of pathology. The chronic stress response and the sustained action of cortisol provide a ready mechanism for the implementation of such strategies. Any of the chronic responses discussed above, if they can be shown to fit this pattern, can be considered adaptive.

This re-envisioning of the chronic stress response and the role of cortisol is not wholly revolutionary, as the stress literature has been leaning this direction. Take, for example, McEwen’s recent focus on allostasis. His term *allostatic load* has itself evolved to take into consideration the idea that many of the potentially pathological responses to chronic stress may have short-term benefits (McEwen & Wingfield, 2003). Another recent study by Hadany *et al.* (2006) attempts to explain the evolution of the deleterious effects of cortisol using a vaguely group selectionist approach, suggesting that individuals possessing a gene that causes early death in less fit individuals can benefit close kin and proliferate in a population.

Others look toward the more classically individualist approach proposed here, and begin to question why natural selection may have favored a mechanism that has such deleterious consequences. They call for a more ecological approach (Carr & Summers, 2002; Greenberg et al., 2002; Korte et al., 2005; Koolhaas et al., 2006). This approach is less single-mindedly focused on the pathological costs associated with the chronic stress response, but also considers the possibility of distinct benefits. In doing so, these authors suggest, we can gain a more complete understanding of the evolutionary history of this complex system.

Wingfield and Kitaysky (2002) go so far as to offer a suite of potential cortisol-mediated coping strategies. They also suggest that the adaptive value of at least the primary tier of strategies is apparent in their ability to delay the onset of a more sustained release of cortisol. In other words, delays in the most drastic responses to chronic stressors may be evidence that cortisol has already prompted a suite of adaptive temporary strategies that persist for longer than we would expect for a typical acute stressor. Initial implementation of the chronic stress response leads to changes in physiology and behavior that, at least for a time, mediate the stressful effects of a harsh situation. If a more extreme stressor eventually leads to a more extreme chronic stress response, which in turn leads to more intense coping strategies that promote survival, then the prevalence of the chronic stress response system may no longer present such an untenable evolutionary problem.

There is another more subtle line of evidence for an adaptive angle developing in the literature. If the chronic components of the stress response system are truly maladaptive, then their prevalence can only be explained by an inability to turn the system off in times of chronic stress. Recent research, however, suggests that the system *can* be turned down or off when doing so provides unambiguous fitness benefits. For example, animals whose breeding system involves exceptionally stressful conditions such as extreme competition in an extreme environment (arctic ground squirrels, Boonstra et al., 2001) must reproduce even under the harshest of conditions. To have any reproductive success, they must effectively suppress the suppressive effects chronic glucocorticoid exposure has on reproduction. Wingfield and Sapolsky (2003) review this ability to selectively resist the effects of chronic stressors. They suggest that certain conditions, including semelparity in animals like salmon, will promote the evolution of this sort of glucocorticoid bypass. They also suggest a minimum of five possible mechanisms that would allow an animal to minimize the generally pathological effects of such profound and prolonged stress. Each would work at different points in a stress response, from decreasing the perception of stressfulness to initiating counteractive mechanisms when glucocorticoid levels are elevated. Some altricial avian neonates are also able to block chronic stress responses (Sims & Holberton, 2000; Love et al., 2003; Walker et al., 2005), likely because they are incapable of implementing strategies and will therefore only suffer the pathological consequences of this early exposure. Both of these scenarios suggest that the pathological consequences of the chronic stress response are not entirely unavoidable. If organisms are capable of turning a deleterious system down or off but they overwhelmingly do not, it seems a further indication that the system likely carries an adaptive benefit.

IV. The wild female baboons of Gombe

I have chosen to study the female baboons of Gombe National Park, Tanzania. These baboons are an excellent model organism for this research. This population has been studied extensively since 1967 (Packer, 1975; Ransom, 1981). There is sufficient data to address nearly all of the potential chronic stressors identified by the primate literature. The Gombe females represent not only a large sample population (n=106), but also one for which much is known about the lineage, development, and life history of each baboon. This study population can also provide the combination of long-term demographic and hormonal data vital to this research.

Baboons have been well studied in the stress literature, but the bulk of this research has been completed on male baboons. There is evidence that because there are profound differences in the social systems of male and female baboons, the chronic stressors females face may be quite different from those experienced by males. Female baboons are particularly interesting for the significant role social support may play. Female baboons are philopatric and matrilineal, and they form close associations with maternal kin; such support may be key in moderating negative life events, as has been indicated by non-hormonal data (Silk et al., 2003).

V. A sudden resource loss represents an ecological disaster and a model chronic stressor

Ecological disasters, while an extreme case, offer a superb opportunity to consider whether the chronic stress response system with its concomitant rise in cortisol might promote strategies that improve fitness. The majority of the potential chronic stressors discussed above are predictable in some way. Seasonal changes happen with extreme regularity, and specific sets of strategies have likely evolved to help individuals weather such changes—migration, for example. Social stressors, while not necessarily on a predictable schedule, are something any social animal is likely to face in some form during their life, and thus fitting strategies also likely exist—social withdrawal, subordinate behaviors, and other "social damage limitation strategies"—perhaps in part explaining an animal parallel for depression (Gilbert, 2000). But ecological disasters are just the sort of rare, unpredictable, yet devastating stressors which the chronic stress response might have helped our vertebrate ancestors survive.

For the Gombe baboons, one recent event represents just such a profound, unexpected, and sudden loss of resources. Until 1998, local fishermen entered the park for a few days each month in order to fish for *dagaa*—small fish that they caught and then dried on the beach. The baboons had become very adept at stealing these drying fish, and the fish represented a ready, regular, and valuable protein source (Ransom, 1981). In addition, the fishermen and their families kept small temporary huts that were vacant when they were not fishing. The baboons also scoured these huts and the surrounding areas for food. All but one of the study troops (Troop C) regularly came down to the beach in search of *dagaa*-related refuse. In 1998, Tanzanian National Parks (TANAPA) banned *dagaa* fishing within the borders of the park.

While not a *natural* disaster, this loss represents a clear ecological challenge of similar scope. Again, resource loss plays a likely role in many stressors, and has been associated with increased cortisol, stress-related pathologies, and depression. While predictable resource loss is common—for example, during the dry season—those losses that are unpredictable (e.g. ecological disasters) are known to lead to significant increases in glucocorticoid exposure in a number of species. This difference may be in part due to the evolution of successful coping strategies for common events and the ability of animals to perceive a coming change (changing day length, rainfall patterns) and provoke the necessary responses without necessarily invoking a physiological stress response. I propose that the chronic release of glucocorticoids may be adaptive in that it allows animals to respond to the sort of general and unpredictable stressor represented by natural disasters with a set of similarly general yet effective coping strategies.

For a majority of the Gombe study troops, the *dagaa* were a valuable and available source of protein, and one that had become a significant part of their diet. Once a month, the baboons spent their days on the beach, and during these times most agonistic interactions involved these drying fish (Ransom, 1981). Even during the remainder of the month, the baboons made frequent trips to the beach in search of scraps of fish and human refuse. The rewards for these behavior patterns likely included a diet richer in protein and fat, much like the diets reported for "junk food" monkeys with access to garbage pits. Such human-influenced diets have not only been found to alter dominance and ranging patterns (Altmann & Muruthi, 1988; Eley et al., 1989; Singh & Vinathe, 1990; Kemnitz et al., 2002), they have known physiological costs such as increased weight and its related risks (Altmann et al., 1993; Kemnitz et al., 2002).

Certainly the loss of this resource represented a challenge to these baboons, exacerbated by the fact that the baboons could not have anticipated the loss. Nor could they have had any idea how long this change would last. An analogous loss for members of Western society might be the sudden disappearance of grocery stores and all their fat and protein-laden goodies. The loss might, in time, lead to a more natural and healthful approach to our dietary needs. But if the loss were sudden and unpredictable, it would certainly evoke uncertainty and stress. Such a change would clearly require significant physiological and behavioral responses, and certain coping strategies would likely prove to be more effective than others. If no change was made, either physiological or behavioral, the long-term impact of this loss would be particularly drastic: failure to shift strategies could mean certain death. Perhaps the same is true for the baboons and the loss of the *dagaa*, and perhaps a chronic release of cortisol helped the baboons navigate this profound, unpredictable ecological disaster.

VI. Predictions: The chronic stress response elicits adaptive coping strategies

A. Activation of the chronic stress response occurs frequently in the lives of wild female baboons

If the chronic stress response does represent a successful coping strategy in the face of chronic stressors, I expect that the activation of this response and its associated elevation in cortisol will occur with some frequency in the lives of wild female baboons. A number of related predictions logically follow. First, numerous factors will invoke the chronic stress response in female baboons. Second, those chronic stressors that represent candidates for further study will cause predictable physiological and behavioral changes. Any extreme stressors will also cause patterned changes in demographic parameters. Finally, the chronic stress response and concomitant elevations in cortisol will be a common part of the life history of wild female baboons.

B. Ecological disaster and elevated cortisol will be correlated with predictable physiological and behavioral coping strategies

When chronic stressors do occur—especially unpredictable, uncontrollable chronic stressors like ecological disasters—I expect to see an increased incidence of behavioral and physiological strategies known to be correlated with extensive cortisol exposure. As there are levels of chronic stress response (e.g. mild and severe: Wingfield et al., 1997; Wingfield et al., 1998), I expect that the magnitude and/or frequency of such coping strategies will increase or decrease in concert with cortisol levels. These strategies will likely involve the same metabolic, foraging, and reproductive strategies discussed above.

The data available for the Gombe baboons will illustrate changes in male transfer behavior, foraging and ranging behavior, and female reproductive suppression. The male baboons will show increased rates of transfer immediately following the loss of the *dagaa.* Because the effects of this loss were widespread, the males who transferred likely found conditions to be similar in their new troops, thus I expect short term immigrations to be particularly prevalent. Once it became clear that this strategy was not effective in the face of this particular stressor, the number of transfers should return to normal as the period of stress continued.

Migration strategies are not an option for philopatric female baboons—or for animals facing a chronic stressor that encompasses an entire habitat. In such instances, coping must involve strategies that minimize costs and risks and maximize available energy. I therefore expect to see shifts in foraging and ranging patterns that include preferential movement to areas higher in the hills with richer, natural resources. As initial home range sizes were sufficient to meet the nutritional needs of the baboons when *dagaa* was included in their diets, I also expect to see expanded home ranges as the baboon troops compensate for this loss.

Adult females will show evidence of reproductive suppression, including decreased birthrates, increased inter-birth rates, and increased miscarriage rates. I also expect to see juvenile females delaying menarche in response to the stressful conditions and elevated cortisol of this post-dagaa period

C. These cortisol-mediated strategies will promote survival and optimize body condition in the face of chronic stress

As the baboons attempt to adapt to the *dagaa* loss, an initial suite of cortisol-mediated coping strategies will likely delay the most obvious demographic response. Eventually, a marked increase in cortisol will mediate more dramatic coping strategies that will effectively minimize energetic costs and risks in the face of this ecological disaster, allowing animals to survive even this extreme stressor. In addition, animals that incorporate these coping strategies will be more likely to survive and subsequently regain optimal physical condition. Thus, the immediate survival benefits of chronic cortisol release will outweigh future pathological risks.

Methods

I. Study site and population

A. Gombe National Park, Tanzania.

Gombe National Park is located in Western Tanzania. Gombe is a small park of approximately 35km^2. The boundaries of the park are all ecologically discrete. The north and south are bounded by villages and deforested farm land. The eastern boundary is the rift escarpment (again bounded by farm land), and the western boundary is the shoreline of Lake Tanganyika. The park is characterized by a series of steep ridges rising from the lake to the escarpment; habitats range from thick riverine forests in the valleys to open grasslands on the ridges (Clutton-Brock & Gillett, 1979).

B. The Gombe Baboons

The subjects of this study are female olive baboons (*Papio cynocephalus anubis*). Numerous baboon troops reside within the boundaries of the park, and they can be found in any of its varied habitats. The baboons of nine of these troops have been continuously studied since 1967 (Packer, 1975; Ransom, 1981). The original two troops, Beach troop (also known as BA) and Camp troop (C), lived on the beach and forested areas of Kakombe Valley. B troop and its descendant troops have fissioned seven times, resulting in a total of 9 study troops. The overall range of this study population expanded considerably with the troop splits (personal communication, D. A. Collins). However, they are still centralized in the Kasakela and Kakombe valleys where they maintain flexible and overlapping territories and do not range near the park's boundaries. Two additional troops, LA and LC, range farther north in the Linda Valley; however, these troops were excluded from the present study because of unreliable data. The newest troop, BB, was also excluded for lack of data. Thus, eight troops (AA, AB, AC, BA, C, DA, DB, and DC) are represented in this study. All baboons were habituated to human observers, and all study subjects have been followed throughout their lives.

All females over the age of three at the start of sample collection (n=106) were studied. As juveniles and adults represent significantly different life history stages with vastly different stressors, I analyzed these two datasets separately. Any female who was three or more and had not had a first swelling at the start of the study was considered a juvenile (n=22). All other females were considered adults (n=84).

II. Demographic and behavioral data collection

Trained Tanzanian field assistants follow these baboons and collect data on a near-daily basis (data collection through the time of this study varied between 5-7 days/week) with rare breaks in data collection of more than a few days. All baboons were individually recognizable and could be identified by at least two field assistants. Assistants collected daily attendance data and compiled monthly reports. Attendance data included births, deaths, reproductive status, consort pairs, wounds, and male transfers. Because of the completeness of data collected, major demographic and reproductive parameters were known: age, age at first swelling or first transfer, number of offspring, inter-birth intervals, etc. As all females were born during the long-term study, maternal relationships are also known (daughter, mother, sister, grandmother, aunt, cousin, non-kin).

Monthly reports provide details on inter-troop interactions, illnesses, injuries, shifts in dominance hierarchies, and details of major events (e.g. troop splits). In addition, check-sheets summarize dominance data based on daily ad lib sampling of supplantations and dyadic aggressive interactions. Yearly ranks are based on the number of individuals dominated. Confounding rank reversals were resolved by incorporating the weighted ranks of each opponent. Monthly ranging maps are also provided for each troop.

III. Hormonal collection, preservation, and analysis

A. Sampling protocol and sample collection

Cortisol samples were collected in three discrete field seasons, between January 2000 and April 2002 (FS1: May & June 2000; FS2: February & March 2001; FS3: December 2001-April 2002). Fecal samples were collected opportunistically from all study subjects by field assistants who had been extensively trained in collection protocols. Each sample was collected only if the identified baboon was seen in the process of defecating. Any feces that could not be conclusively attributed to a female (e.g. more than one sample was found in close proximity) or were in poor condition (e.g. fell a great distance) were excluded. Three or more fecal samples were collected from most subjects during each field season with an average of 9.4 total samples per female (adults: n=834; juveniles: n=160). Only 7 females were sampled five or fewer times; four of these were females who died during the course of this study and three were juveniles. As few samples were collected from the juveniles of C troop, this troop was excluded from the juvenile dataset.

Samples were collected in small plastic zipper bags and were immediately mixed by manipulating them inside the plastic bag. Samples were collected at any time between 07:23 and 19:25; previous studies suggest that diurnal fluctuations in baboon fecal cortisol levels are minimal (Weingrill et al., 2004). Specimens were transported to the field lab at the end of each morning or afternoon shift.

B. Field preservation and transportation

Samples were remixed inside the plastic bag. Approximately 0.5g of the mixed samples were placed in pre-weighed 20 mL HDPE scintillation vials and covered with 5mL of 90% ethanol. Each sample was then shaken by hand for a minimum of two minutes until samples were adequately suspended in the preservative. The lag time between collection and preservation (ethanol preservation lag) was recorded for all samples. This lag ranged from 1 minute to a rare extreme of 8 hours, with an average time of 2:36 spent at ambient temperature without preservative. Once processed, samples were tightly sealed and wrapped in parafilm to prevent ethanol leaks. They were then kept in a cool, dark room for the duration of the field season.

Samples were transported under CDC permit (number 2001-05-246) to the St. Louis Zoo, St. Louis, Missouri. Upon arrival, samples were checked for signs of leaking and discarded if necessary. Samples were then analyzed immediately or frozen in the ethanol preservative in a -20˚C freezer. The lag between ethanol preservation and analysis or freezing (freeze lag) was also recorded for all samples. Samples were stored at ambient temperature in ethanol for an average of 76 days with a minimum of 15 days and a maximum of 181 days.

C. Steroid extraction

As samples had been stored in the extraction solute for a minimum of 15 days and experienced vigorous jostling during transport, I followed a simplified extraction protocol. Samples were placed on a shaker overnight at 200 RMP, after which they were allowed to settle for approximately one hour. The extract portion of the samples was then decanted into 12x75 polypropylene tubes, leaving behind all fecal material, and then centrifuged at 4,000 RPM for one hour. The supernatant was then poured into a cryovial and analyzed immediately.

The pellets remaining in the polypropylene tubes were resuspended in ethanol and poured back into the original scintillation vial. All vials were then left open overnight in a vented oven at 100ºC. When samples were completely dry, the vials were reweighed. By subtracting their initial weight from their weight with dry feces I could determine the dry weight of the feces.

D. Radioactive immunoassay

All samples were analyzed using radioactive immunoassay (RIA) in the Endocrinology Lab at the St. Louis Zoo. Standard cortisol RIA kits were used (Cortisol ACTIVE-RIA ^{125}I Kit, Diagnostic Systems Laboratories, Webster, Texas) and all samples were assayed in duplicate; paired samples with variation greater than 15% were reanalyzed. Hormone concentrations were corrected for volume of added solvent and also dry weight of feces in order to control for differences in moisture content (Wasser et al., 1993) and are therefore reported in ng/g. Although an antibody against cortisol was used in these assays, fecal glucocorticoids exist as a mixture of metabolites, and while "fecal cortisol" is used for convenience, these results should be understood to refer to fecal glucocorticoid metabolites.

E. Outlier analyses

Two samples were excluded due to lack of replicability either within or between batches. Paired aliquots of these two samples were run seven times at three different dilutions. Each batch showed extremely low replicability, and final cortisol values ranged from 4862-17250 ng/g for one and 5023-15188 ng/g for the other.

Fecal sampling following the protocol used in this study can show unreliably high or low values (Millspaugh & Washburn, 2004). On rare occasions, dry fecal weight can have very little to do with the actual weight of the feces. For example, samples may contain more or less indigestible material (such as seeds) which can significantly skew final hormone concentration measurements, resulting in extreme high or extreme low values. In order to remove any such values from the dataset, I created a set of exclusion criteria. I first identified any samples that were unusual for a given baboon and that were more than four standard deviations from the overall mean. I then looked at this subset of data points and excluded those that showed extreme high or low dry fecal weights as well as extreme high or low raw hormone values (prior to dry fecal weight correction). Four samples were identified as having extremely low fecal weights (<0.03 g) and extremely high cortisol and testosterone values (top 1%) and were therefore excluded based on these criteria.

IV. Model development

Two statistical models were developed to investigate a variety of potential chronic stressors, including the loss of the *dagaa*. The first model considered factors likely to affect cortisol levels of adult females and the second model focused on juvenile females.

A. Adult female predictors

All main effects and interactions tested in this model are identified in Table 1. As all females were born during the 35-year study period, age was accurate to within a few days and was treated as a linear variable. Reproductive status was determined for each individual sample based on observation data for the date of collection. The status classes were pregnant, lactating, flat (indicating a non-fertile phase), swelling, on consortship, or menstruating.

Adult female relatives provide most of the known social support for female baboons (Silk et al., 2006a; Silk et al., 2006b). I included a count of the female's mother, adult daughters, maternal grandmother, maternal adult sisters, aunts, and nieces who were alive and members of the same troop on the day a given sample was collected. This predictor was treated as a linear variable.

The rank of each female was determined as both a continuous variable (based on proportion of females dominated) and as a number of possible categorical variables (each with slight adjustments to the "breaks" between high, medium, and low, none of which affected the basic results of the model). Whenever possible, a rank specific to the year of sample collection was included for each female; however, there were gaps in the data. For example, rank analyses were not collected for troop C for any of the three study years. Because of the overwhelming stability of female baboon hierarchies (Hausfater et al., 1982; Packer et al., 1995), I recreated the hierarchy during this period by considering each female's last known rank, correcting for females who had died, and adjusting for maturing females. As monthly reports accurately report most reversals in the female hierarchy, I was able to use this information to verify that there were no unusual changes in rank not represented in available rank data. As the significant results of the model were the same for both continuous and categorical ranks, I chose to focus analyses on the categorical data. This, in part, allowed me to control for minor rank changes that may have been missed in the extrapolated troop rank determinations. Categorical ranks are also easier to interpret graphically and are more widely used in the baboon literature.

Each female was also identified by the troop in which she resided at the time of sampling, and each sample was classified according to the field season in which it was collected. I also considered season as a preliminary predictor, and each sample was categorized as collected in either the dry or wet season as determined by annual rainfall patterns at the time of sampling (generally Oct-May, wet; Jun-Oct dry; Packer et al, 1995). All biologically relevant two-way interaction terms were also considered in the initial model.

B. Juvenile female predictors

Most of the parameters for the juvenile females were determined following the same methods presented here for the adult females (see Table 1 for a complete list of predictors). In addition, the status of each female's mother (alive or dead) was determined for each sample at the time of collection. Maternal rank was also considered as a categorical variable (mother in the top third of the hierarchy=high; middle third=middle; bottom third=low) as outlined above. Again, all relevant two-way interactions were included in the development of the initial juvenile model.

V. Statistical analyses

I used SAS version 9.1 (SAS Institute, Inc, Cary, NC) and Proc Mixed to develop models in order to identify predictors that correlate with cortisol levels. For both the adult and juvenile data sets, the models were initially developed as general linear mixed models (GLMM) with the random effect *baboon* and the predictors identified in Table 1. A random effect was considered in order to account for the correlation among repeated cortisol measures from the same baboon. If the model results were not significantly different under a general linear model (GLM), I subsequently dropped this random statement from the model. LS Means and Tukey-Kramer adjusted group mean comparisons were also calculated using SAS 9.1; for both, the statement “by level” was included to account for the unbalanced nature of this dataset.

Results

Ecological disaster and the frequency of chronic stressors

I. How does this disaster affect the Gombe baboons?

A. Reproductive parameters

BIRTH RATES

In the 30 years preceding the removal of the fishermen, the study populations had grown dramatically from 2 troops to 9 (8 of which are included in this study) and from 86 individuals to 270. Birthrates (# of live births per adult female) remained high and relatively steady during this period (Figure 1), with an average annual birth rate of 0.40 births per adult female. In a comparison of birthrates in the last full year with *dagaa* access (1997: 0.42 births per adult female) to the first full year without (1999: 0.29 births per adult female), birthrates dropped more than 30%. This decline was not unique. The long-term data show three similar declines (in 1979, 1981, and 1985) in thirty years. What makes the 1997/99 drop different is the duration of the decline. In each previous drop, birthrates quickly rebounded. Following the loss of the *dagaa*, birthrates stayed at or below 0.30 for three consecutive years. To look at this drop in terms of the number of annual births, 42 infants were born in 1997, the final full year of fishing. In 1999, only 23 infants were born in a population of roughly the same size. Birthrates dropped, perhaps as the baboons reacted to this severe ecological change. The baboons did eventually recover demographically in 2002 with a rise in annual birthrate to 0.46 births per adult female.

INFANT MORTALITY

Infant mortality (# infant deaths/# at-risk infants in the previous year) has been a particularly volatile parameter since 1968 (Figure 2). The average infant mortality rate from 1968-2002 was 0.34 deaths/infant, although it ranged from 0.04 in 1985 to 0.86 in 2001. A comparison of the pre- and post-loss statistics suggests that infant mortality rose by 24%: 0.19 deaths/infant in 1997 to 0.25 deaths/infant in 1999. This is by no means the worst increase on record. In fact, the most significant rise in infant mortality in over thirty years occurs in the third year after the removal. In 2001, infant mortality rises for the fourth straight year, from 0.35 deaths/infant to an astounding and all-time high of 0.86 deaths/infant. This constitutes a more than two-fold increase in the space of one year, with 86% of at-risk infants dying. This seems to suggest a delayed demographic response to a profound ecological challenge. The baboons finally showed signs of a population-wide recovery in mid- to late-2002. While infant mortality remained elevated for the duration of my field season, it dropped to zero for much of the remainder of the year resulting in an annual infant mortality rate of 0.29 deaths/infant.

MISCARRIAGE RATES

Miscarriage rates are also a valuable demographic parameter in times of ecological change. The long-term pattern suggests that miscarriage rates (# known pregnancy losses/# known pregnancies) can vary widely and change from year to year (Figure 3). The loss of the *dagaa* does not appear to have immediately affected miscarriage rates, as the rate of loss in 1997 was 0.16 miscarriages per pregnancy and the rate in 1999 was essentially unchanged at 0.15 miscarriages per pregnancy. As with infant mortality, the most striking increase in over 30 years is the rise in miscarriage rates in 2001. During this year, 29% of detectable pregnancies end in loss—a more than two-fold increase representing the highest miscarriage rate ever observed in Gombe baboons.

JUVENILE FEMALE MORTALITY

The long-term data suggest that Gombe females rarely die between three years of age and maturity. From 1968-2002, the average adolescent mortality rate for the eight study troops is 0.03, a figure based on only seven such deaths in 35 years. In fact, no juvenile female deaths have been recorded since 1990 (Figure 4). What is striking about this data is that even in the face of this extreme stressor, juvenile females did not die as a result of the lost resources.

ADULT FEMALE MORTALITY

Adult female mortality averages 0.06 deaths/female per year, with annual rates ranging between 0 and an unusually high 0.19 (Figure 5). There was an apparent increase in adult female mortality immediately following the fishing ban, when mortality rates increased from 0.02 in 1997 to 0.09 in 1999. This increase is by no means unusual in light of the long term data, as increases of similar magnitude can be seen at least three times in the preceding 30 years. The three years following the removal do not show any obvious pattern of increased mortality, as the average rate for the following three years are only slightly elevated at 0.07. It appears that the loss of the fish did not have a significant impact on adult female mortality.

C. Demographic effects of the dagaa removal

The demographic data suggests a clear picture. The fishermen were removed from the park and the baboons lost a valuable resource of protein upon which they had depended for decades. The baboons soon began to suffer. Birthrates dropped and infant mortality increased within the first year. Two years later, circumstances become dire. Birthrates remained low, infant mortality reached nearly catastrophic heights, and the adult females suffered record-high miscarriage rates. By the latter half of 2002, the baboons finally returned to their pre-loss normal as birthrates, infant mortality, and miscarriage rates all approached average levels.

II. Does the dagaa loss cause a chronic stress response?

A. Model results: predictors of chronic stress

WILD ADULT FEMALE BABOONS

The statistical model identified variables associated with elevated exposure to (or ability to ameliorate) glucocorticoid levels in adult female baboons. This model was initially developed as a general linear mixed model (GLMM), with the main effects and interactions detailed above. The random effect *baboon* was found to be insignificant and was subsequently dropped from the model indicating a low, non-significant correlation among samples from each baboon. Only the results of the general linear model (GLM) are reported below, and they can be seen in full in Table 2. I then developed a reduced model based on stepwise removal of insignificant interactions and main effects.

The results of this reduced model's tests of fixed effects can be seen in Table 3. Those results central to the loss of the *dagaa* will be discussed below; those results pertaining to social factors such as rank and social support will be presented in Chapter 3.

WILD JUVENILE FEMALE BABOONS

As adolescence represents a vastly different life history stage for female baboons, with different states and stressors, I developed a separate model for juvenile females. In this model, the random effect *baboon* was found to be significant indicating a significant correlation among samples from each baboon. Therefore, all results for juvenile females are from GLMM with the main effects and interactions detailed above. The results of this complete model can be found in Table 4. I developed a reduced juvenile cortisol model based on the same stepwise process described for the adults. The results of this reduced model's tests of fixed effects can be seen in Table 5. Again, some of these results will be discussed below and others will be discussed in Chapter 3.

B. Evidence of a profound chronic stress response

The final models show that the field season in which a sample was collected is a significant predictor of mean cortisol levels for both adults (GLM: $F_{2, 796}$=13.16, n=834, p<0.0001) and juveniles (GLMM: $F_{2, 110}$=10.90, n=160, p<0.0001).

WILD ADULT FEMALE BABOONS

Starting in late 2001, in parallel with the devastating demographic changes, the adult females experienced an immense population-wide increase in cortisol levels that continued at least through April 2002 (Figure 6; Tukey-Kramer: p<0.0001 compared to both mid 2000 [FS1] and early 2001 [FS2]). This change clearly indicates a huge, widespread, and prolonged stress response. Samples collected during this period (FS3) show significantly elevated predicted mean cortisol. This profound rise in cortisol levels affects nearly every baboon, as the vast majority of baboon-specific cortisol trajectories show consistently higher cortisol levels during this time.

This significant rise in cortisol levels is also independent of state. No reproductive status ameliorates this effect (reproductive status*field season, GLM: $F_{10, 751}$=0.79, n=834, p=0.6380). Nor is any particular age group spared (age*field season, GLM: $F_{2, 751}$=0.74, n=834, p=0.4757). To verify, I ran an additional model with age classes (young: maturity-11.5 yrs; middle: >11.5-16.5, aged >16.5+; Beehner et al, 2005) with virtually identical negative results. No amount of social support is protective (number of adult female relatives*field season, GLM: $F_{2, 751}$=0.37, n=834, p=0.6912), even when the number of adult female relatives is treated as a categorical variable in order to better identify state-dependent differences (number of adult female relatives* field season, GLM: $F_{7, 732}$=0.26, n=834, p=0.9777). Not even the presence of a mother (mom=1, no mom=0, living mother* field season, GLM: $F_{1, 751}$=0.00, n=834, p=0.9996) or the absence of any adult female relative (1+ adult female relatives=1, none=0, adult female relative*field season, GLM: $F_{1, 751}$=0.31, n=834, p=0.5751) is enough to significantly ameliorate the effects of this period of post-*dagaa* stress.

The dynamics of social rank as a predictor change significantly in late 2001 (rank*field season, GLM: $F_{4, 796}$=2.74, n=834, p=0.0279, see also Chapter 3), but each rank class still shows a significant rise in predicted mean cortisol during this time (Figure 7; Tukey-Kramer: for high and medium rank classes, FS3 is significantly greater than FS1 or FS2, p<0.0001; for low rank class, FS3 is significantly greater than FS2, p=0.0089).

The interaction between troop and field season is significant (GLM: $F_{14, 796}$=2.21, n=834, p=0.0063), and a graphical depiction of LSmeans shows a clear increase in predicted mean cortisol during this period for all troops except DB (Figure 8a). The decrease in cortisol in DB is insignificant (LSmeans, slice=troop, DB: p=0.1636). For all other troops, FS3 is significantly greater than both FS1 and FS2 (LSmeans, slice=troop, AA: p=0.0013, AB: p=0.0370, AC: p=0.0019, BA: p<0.0001, C: p=0.0002, DA: p=0.0024 DC: p<0.0001). Thus, the vast majority of the troops suffered as a result of the complex effects surrounding the loss of the *dagaa.*

WILD JUVENILE FEMALE BABOONS

Field season is also a significant predictor of cortisol levels in juvenile females (GLMM: $F_{2, 110}$=10.90, n=160, p<0.0001), and cortisol rises significantly during this same late 2001 to mid 2002 period (Figure 9; Tukey-Kramer: p<0.0001 compared to both mid 2000 [FS1] and early 2001 [FS2]).

As with the adults, this rise in cortisol is independent of age, maternal rank, or social support. Further analyses show that a juvenile's age does not interact significantly with field season. Designating age as a categorical variable is similarly non-significant. While social support is clearly important to cortisol levels in juvenile females (mom alive, GLMM: $F_{1, 110}$=13.95, n=160, p=0.0003; number of adult female relatives, GLMM: $F_{1, 110}$=10.69, n=160, p=0.0014; see also Chapter 3), no amount significantly mitigates the effects of field season. The dynamics of maternal rank also changed significantly during this period (GLMM: $F_{4, 110}$=2.50, n=160, p=0.0046). While all maternal rank classes show a pattern of increasing cortisol in FS3, this increase is only significant in daughters of low-ranking females (Figure 10; Tukey-Kramer: FS3 is significantly greater than FS1 or FS2, $p<0.0001$).

The interaction between troop and field season is once again significant (Figure 8b; GLMM: $F_{10, 110}=7.32$, n=160, $p<0.0001$). All six troops show an increase in predicted mean cortisol during this period, and these increases are significant in troops AB and DC (FS3>FS2 & FS1; AB, Tukey-Kramer: $p<0.003$; DC, Tukey-Kramer: $p<0.001$).

C. The profound chronic stress response to the dagaa loss

The worst of the chronic stress response clearly mirrors the worst of the demographic response. In fact, a closer look at the demographic data suggests an even closer connection. The infant deaths in 2001 are concentrated during the six-month period prior to the third field season. The demographic crisis of 2001 clearly represents a change in the practical severity of the stressor that began when the fishermen were removed in 1998. No hormonal samples were collected during this mid-2001 period; however, it is physiologically reasonable to assume that the significant increase in cortisol levels that immediately follows represents a physiological change that began during this period in mid-2001 and extended into 2002.

To further test this demography-physiology connection, I compared the infant mortality data preceding each of the three field seasons. In order to get a clearer picture of what was happening within these periods, I also looked at the ratio of infant deaths to infant births. This gave me a sense for how many infants were being born in comparison to how many were dying. In the period before FS3 young baboons were dying at nearly twice the rate before FS2 and four times the rate before FS1 (Figure 11). The deaths-per-birth rates for the six months leading into FS1 and FS2 were 0.29 and 0.22 respectively. The rate leading into FS3 was 0.85—higher than any annual rate in the previous 35 years (Figure 2)—meaning that for nearly every birth in this six month span an infant baboon died. Together, these data suggest that this increase in infant mortality is connected to the marked increase in measured stress levels in FS3. Cortisol levels likely rose during this mid-2001 period and then remained high well into 2002.

A closer look at the miscarriage data shows a similar pattern. These results further suggest that both the demographic and physiological changes are a response to the loss of the *dagaa*. The marked increase in miscarriages during 2001 is concentrated in the same six months preceding FS3. An analysis of the six months preceding each field season reveals a pattern that is strikingly similar to that of infant mortality, with a profound increase in both the demographic and physiological response to the stressor during and immediately preceding FS3. In the period immediately preceding FS3, miscarriage rates were more than three times greater than before FS2 and nearly twice as high as the months preceding FS1 (Figure 12). There were 6 known pregnancy losses between June and November 2001—only one less than the previous annual record in 1997 when the number of adult females was roughly the same.

That the miscarriage data so closely follows infant mortality data suggests a number of conclusions. First, both followed a significant delay after the initial stressor event, the loss of the *dagaa*. Second, these parallel demographic changes support the idea that the measured increase in cortisol in late 2001 likely began earlier that year. And finally, the implicated stressor not only affects infant condition and likelihood of survival, but also adult female physiology during this period.

Since there are no hormonal data available from before the loss of the *dagaa* it is impossible to know whether cortisol levels immediately increased in the years following this ecological perturbation. It does seem quite likely that an initial rise in cortisol would have paralleled the initial changes in demography, signifying two levels of chronic stress response. Whether there was an initial increase or not, cortisol levels remained essentially constant from 2000 until at least June 2001. By December of 2001, cortisol levels increased significantly in parallel with both infant mortality and miscarriage. After the onset of this severe ecological stressor, an immediate decrease in birth rates was followed by a delayed response in infant mortality, miscarriage rates, and cortisol levels.

Even in the face of this extreme ecological stressor, neither the juvenile nor adult females showed increased mortality rates either as a result of the initial loss of resources or as a result of the significant physiological changes in 2001 (Figures 4 & 5). The demographic data also show that the baboons recovered in mid-2002. While there are no hormonal data to show whether there was a physiological recovery as well, the baboons not only weathered this disastrous interlude, they survived, and they recovered. Any physiological response that allows them to accomplish such a feat in the face of such a severe stressor would have a clear fitness advantage.

III. Is the loss of the dagaa the most likely cause of these changes?

A. The disastrous nature of resource loss

Dagaa had become such a prominent part of the diet of these baboons that many of their daily activity and foraging patterns centered on maximizing their intake. Anecdotal evidence from the baboon followers suggests that the baboons did not immediately respond to the loss. In the grocery analogy, if the store is closed once, you assume it is inventory day and you plan to try again tomorrow. Once the fishermen did not return for several months, the baboons began to return to the forests. Their ranging patterns changed so much that the followers had difficulty finding them on any given day (personal communication, A. Sindimwo). The baboons also moved a lot faster and higher in the hills. The population-wide impact of this stressor is also important: there was no "substitutable resource" to match the high-protein crop of dried fish. Few events other than a true natural disaster would be expected to have such a widespread impact upon a population.

B. Cortisol results closely parallel demographic response

The cortisol data closely parallels the demographic data during the same time period; cortisol likely mirrored demography both during the initial onset of the stressor when demographic effects were less dramatic and during the recovery.

C. Delayed response to ecological change

The initial response of the Gombe baboons likely reflects the relatively effective implementation of first-tier behavioral and physiological coping strategies. As predicted, because of the severity and prolonged nature of this stress, we eventually see an even more profound chronic stress response. The apparent temporal disconnect between the initial stressor and the demographic is as predicted above. Furthermore, the demographic effects almost directly mirror those of Gould et al (1999), with a one- to two-year delay in onset and another two-year delay to recovery and is similar to responses reported by both Sapolsky (1986) and Hamilton (1985).

D. Could some other cause more directly explain the rise in cortisol?

Initially, it seemed surprising for the physiological effects to be so temporally disconnected from the loss of the *dagaa*. I therefore looked at a number of factors that might represent a more direct cause for this significant change in the female baboon's hormonal profiles.

METHODOLOGICAL FACTORS:

BATCH EFFECT. In order to rule out any batch effects from the RIA analyses, I ran the model with categorical groupings of samples analyzed at the same time. Batch, however, was collinear with field season; the two are too correlated to enter the model together. Samples were batched chronologically, and samples collected during roughly the same time period were analyzed together. All samples from FS3 were analyzed in one of three batches, each of which had average cortisol levels considerably above the overall mean. It is unlikely to see such a profound batch-related increase for all batches within one field season when all protocols were held constant and quality control results were within normal ranges. Thus, field season, rather than batch, is the key variable.

ECOLOGICAL FACTORS:

SEASONALITY. Initially, my model included season (dry or wet) as a predictor. FS1 was categorized as dry and FS2 and FS3 were considered wet (Packer et al, 1995). Season, however, was also collinear with field season. Elevated cortisol levels in FS3 clearly drove any perceived seasonal effect. When I ran the model with season and excluded field season, season's effect was insignificant. In the absence of data from FS3, the average predicted mean cortisol levels for the dry season (FS1: 460.1 ng/g) and the wet season (FS2: 354.2 ng/g) are nearly identical.

RAINFALL. I looked at Gombe rainfall data collected by Tanzania National Parks (TANAPA) staff from 1993-2002 (1998 data were misplaced by TANAPA) in order to determine whether the baboons were responding to a change in rainfall patterns. The average monthly rainfalls for each field season are predictably commensurate with season (FS1, dry, 14mm/month; FS2, wet, 239.8mm/month; FS3, wet, 169.2mm/month) but do not show any striking pattern that would explain heightened stress in FS3. Rainfall measures were also available from 1978-1997, but represent a seasonal year (wet season + dry season, October-September); these data were collected by Gombe Stream Research Center (GSRC) staff. A look at yearly data suggests that rainfall in 2000 was low compared to 2001 or 2002, but not abnormally so in light of long-term data (Figure 13). During the period from 1994-1996, rainfall was lower (less than 900mm) for three consecutive years. Infant mortality rates fluctuated but were roughly average during this time, miscarriage rates were normal, and birthrates remained relatively high. It is therefore unlikely that the slightly reduced rainfall in 2000 accounts for the demographic and physiological signs of stress documented for late 2001.

ADDITIONAL HUMAN INFLUENCES. In 2000, two young chimpanzees died of a pathogen caused by contact between the chimpanzees and humans. Recognizing baboons as a possible vector of a similar future infection, TANAPA and the Gombe research staff made a number of significant changes to minimize disease transmission between humans and primates. Until 2000, the trash pits were largely open, and many baboons frequented these in search of food. The trash pits began to be more restricted just prior to FS1, though most of these changes were short-term fixes that many of the baboons quickly learned to bypass. The pits were largely baboon proof by FS2. The baboons were still a regular presence in the village during FS3, but human refuse was no longer an easily attainable food source. Even during the years of open trash pits, only two of the study troops (DA and DC) were seen at the trash pits with great frequency. It is therefore unlikely that the increased inaccessibility of trash pits could account for the population-wide rise in cortisol levels, but it could certainly be one of many contributing factors.

SOCIAL FACTORS:

RANK STABILITY. Considerable change in the hierarchy, particularly amongst high-ranking males, is another well documented stressor for baboons (Sapolsky, 1983). An overall increase in rank instability might explain the rise in cortisol levels seen in 2001. I identified changes in either male or female ranks for each of the 8 troops from 1998-2002. Female ranks were predictably stable with the exception of two troops during 2000. BA underwent major changes (Table 6), presumably as a result of a troop split in September 1999. In spite of these remarkable changes in female ranks, measured cortisol levels in BA are low during this time. DC also shows notable changes in female ranks in 2000, with the rise of a low-ranking mother-daughter pair to the middle of the hierarchy and the precipitous fall of one member of a large high-ranking family (Table 7), again with no measurable effect on cortisol. Neither of these examples of female rank instability have a clear correlation to the population-wide demographic and physiological changes of 2001.

Male ranks were volatile in 1999 and 2000, with significant changes in roughly half of the troops for which there are rank data. In contrast, 2001 was strikingly stable, with all troops showing either no change or minor changes in the male hierarchy. Neither the female nor male rank data suggest a discernable direct influence of rank stability on the sudden rise of cortisol levels in FS3.

TROOP SIZE. Actual troop size varies widely among populations of wild baboons (genus *Papio*), with averages ranging from 101 individuals per group in Lakipia, Kenya to only 19 in Giants Castle, South Africa (Dunbar, 1992). Optimal troop size has been shown to be influenced by such factors as rainfall, temperature, vegetative density, and ecologically-influenced time constraints on foraging behavior. Gombe troop sizes are consistently lower than predicted maximums. Nonetheless, significant changes in troop size—particularly widespread increases—could constitute a stressor as a direct result of increased intra-troop competition (Hill et al., 2000) and might affect population-wide cortisol levels.

However, population-wide average troop size remained almost constant from 1998-2002, ranging between 30 and 34 total baboons per troop (Figure 14). The average annual numbers for adult females or males remained similarly stable, with approximately 5 males and 10 females per troop from 1998-2002.

I also calculated the same yearly parameters for each troop. All three measures of troop size changed very little in any of the troops over the study period. Overall troop size changes noticeably in troop BA in 1999 as a result of a troop split immediately preceding FS1 (Figure 15), then remains stable throughout the three collection periods. The overall size of DA dropped noticeably from 1998-2002 in spite of consistent numbers of adult males and females, suggesting particularly high infant mortality in this troop. None of these troop-specific changes, however, show a clear pattern that would explain the observed effects of 2001.

TRANSFER RATES. Male transfers can cause considerable upheaval within a troop and can also significantly affect female cortisol (Alberts et al., 1992; Sapolsky, 1996; Beehner et al., 2005; Bergman et al., 2005). I looked at a number of measures of yearly male transfer rates to ascertain whether changes in population-wide transfer patterns might account for the increased cortisol of 2001. These measures included total number of transferring males (all immigrants with a tenure of ≥1 day), number of medium-term transfers (residing for ≥1 mo), and number of long-term transfers (≥6 mos). All of these measures can be seen in Figure 16. 1999 shows a noticeable increase in short-term transfers, with 21 short-term immigrant males. The influence of such events on female cortisol levels is known to be almost immediate (Alberts et al., 1992; Beehner et al., 2005), so this rise in transfers cannot explain the events of 2001. Transfer rates for males who stay for a month or more are extremely consistent from 1999-2002, with either 21 or 22 male transfers across the population. Long-term transfer rates vary subtly, but not in a pattern that would explain the change in stress in FS3. I also determined immigration rates (# new males in a given period/number of males) for all definitions of transfers.

Overall immigration rates suggest the same peak in 1999 (Figure 17), but no patterns correlated with FS3. None of these measures indicates any population-level rise in male movement that would explain the increase in cortisol levels in FS3.

E. Dagaa loss as a complex ecological disaster

No other cause can account for what we see in late 2001 to mid 2002. The most likely primary stressor remains the loss of the valuable resource represented by the *dagaa*, though a number of smaller scale factors such as decreased rainfall and more troop-specific changes such as rank instability and garbage regulations might have had a synergistic effect that sent cortisol levels over the edge in late 2001. Together, these factors constitute a truly complex ecological disaster for these baboons.

IV. Does this disaster affect all study troops?

A. Home range data

I used monthly ranging maps from before the *dagaa* loss (1997) and after (1999) to compare population ranging patterns. According to baboon followers, soon after the fishermen were evicted, the baboons began to spend proportionally more time in the forests and higher in the hills. They also reported that the baboons became much more difficult to track and were more likely to be dispersed. The maps suggest that the population-wide range changed very little over this period. However, many of the troops did extend their home range, resulting in much more inter-troop overlap (Figure 18) and suggesting that there was a considerable increase in inter-troop competition as a result of this resource loss. Troop C, the one troop that did not suffer directly from the loss of the *dagaa,* shows the most noteworthy shift in home range (Figure 19). They shifted their range both to the north and up higher into the hills, most likely as a result of increased competition from the troops returning to the forest. They are the only troop that relinquished a significant portion of their usual home range, and so were indirectly affected by the ecological change. As these maps do not indicate any measure of occupancy in each part of their home range, there is no way to test whether baboons indeed spent more of their time up higher in the hills.

Home range patterns in 2001 indicate even more overlap. Once again, the overall range of the population changes very little, but the proportion of overlap is much greater, suggesting continued inter-troop competition amongst the study troops as the effects of the resource loss continued.

B. Inter-troop interactions

I looked at reported inter-troop interactions per month—events in which two troops come into close contact, corrected for the number of observation days. This measure of interactions (# interactions/observation days) was calculated for each troop and population-wide. Overall patterns indicate increased competition in the form of steadily increased interactions both immediately following the 1998 removal and during the increased stress of 2001 (Figure 20). This suggests that the baboons' paths crossed more frequently in response to the removal of the fishermen. Furthermore, even troop C—the only troop not directly affected by the loss—had a steep increase in inter-troop competition immediately following the loss (Figure 21), as the other troops ranged further into the forest and began to compete for resources with "inland" troops.

V. Historical view on the frequency of severe ecological stressors

To estimate the frequency of this kind of ecological perturbation, I used demographic patterns to identify past periods which might indicate a similar chronic stress response in the Gombe baboons. I looked for periods showing at least two out of three of the demographic patterns observed after the loss of the *dagaa* from 1968-1998. I looked for any two year period with ≥25% decline in birth rate, ≥75% increase in infant mortality and/or a 100% increase in miscarriage rates.

I was able to identify five periods that met the criteria for two or more of these parameters (Table 8), including two periods that involved all three parameters. Even with the more conservative approach, these results suggest that there have been three ecological perturbations affecting population-wide demographics from 1968-2002: the first occurred in 1978-1980, the second in 1985-1987, while the *dagaa*-related disaster occurred from 1999-2001 (see Figures 1, 2, & 3).

Even if we exclude the perturbation caused by human activities, a major perturbation occurred every 17 years. The average lifespan of the Gombe females who reach three years of age is approximately 16 years, meaning most females will have to endure this sort of stressor sometime during their lifetimes. Saying such chronic stressors are rare is like saying economic recessions and severe droughts are rare. Chances are we all face both in our lives.

Each event affected the baboons for at least three years. It is impossible to say whether the baboons faced chronically elevated cortisol during the two earlier perturbations, but it is likely that they were significantly stressed long enough to be considered chronic and potentially pathological.

One additional noteworthy result of this analysis was that in all cases, infant mortality followed a decrease in birthrate by at least one year. The pattern for the 1985-1987 stressor parallels the demographic patterns observed after the removal of the fishermen almost exactly, with an early decrease in birthrate followed by an increase in infant mortality and miscarriages. The demographic data suggest no increase in either juvenile or adult female mortality after the loss of the *dagaa.* However, as shown in Figure 4, each of the six periods with juvenile mortality corresponded to one of the crisis periods—although there were only seven juvenile deaths in the entire 35 year period. Adult female mortality rates spike in 1979 (Figure 5) in correspondence with the 1978-1980 event (note again the delay), and again during the event in 1989-1990.

The Adaptive Value of the Chronic Stress Response

Survival in the face of any life-threatening stressor entails successfully implementing behavioral and physiological coping strategies that improve survival. In an evolutionary sense, any such strategy that favors short-term survival will be favored, even at the potential cost of long-term health. After all, reproductive success becomes a moot point when an organism is dead. A number of the known effects of chronic cortisol exposure may, if considered in this light, have had significant adaptive value over the course of vertebrate evolution. Wingfield and Kitaysky (2002) suggest that many such cortisol-mediated coping strategies might allow animals to quickly avert or avoid the effects of a chronic stressor, thereby avoiding the pathological effects of chronic cortisol exposure. I'd like to take this one step further and suggest that in the face of some chronic stressors, continued implementation of these same cortisol-mediated strategies may also be immediately adaptive.

I. Does the chronic stress response elicit adaptive behavioral coping strategies?

A. Transfer behavior

The Gombe males closely follow the pattern of transfer behaviors predicted by this adaptive approach. In the year following the loss of the *dagaa*, immigration rates hit 98%, with 43 transfer events for an average study population of 43.6 males. The five-year pattern of transfer events also follows as expected, with a sudden increase in short-term transfers but little change in long-term transfers (Figure 16). Transfers are known to be stressful (Alberts et al., 1992; Beehner et al., 2005; Bergman et al., 2005), so we would also expect males to avoid them unless the costs of staying were higher.

B. Foraging and ranging behavior

While no direct foraging data exists for the Gombe baboons, anecdotal evidence suggests that the baboons spent more time searching for food and traveled farther and faster on any given day. While the population-wide range of the baboons changed very little, many troops expanded their ranges with the removal of the fishermen, but intensified in 2001 as cortisol levels were at their highest (Figure 18).

II. Does the chronic stress response elicit adaptive physiological coping strategies in the adult females?

Pregnant and lactating females did not show higher levels of cortisol than females in other stages of reproductive cycling (but see also Weingrill et al., 2004). I was also unable to find any difference between non-reproductive females and those that reproduced at any time during the entire study period or during the cortisol peak in 2001. Even if reproduction is not a classic cortisol-related stressor, there is ample evidence that female primates experience associated costs and risks.

A. Reproductive suppression and cortisol

In the face of a severe chronic stressor, cortisol might be instrumental in bringing about physiological changes that allow females to postpone reproduction until conditions improve, thereby maximizing survival in the short-term. From this perspective, cortisol is a mechanism by which a female can avoid a costly pregnancy. Note that I use the term "avoid" in an evolutionary sense and do not mean to imply conscious choice on the part of the individual.

In the years following the loss of the *dagaa*, there was a clear pattern of reproductive decline that closely mirrored the changes in cortisol. Inter-birth intervals also increased, following roughly the same pattern as cortisol release and suggesting that cortisol might have mediated reproductive suppression. Any female that did become pregnant during this period suffered higher than average miscarriage rates (Figure 3) and infant mortality (Figure 2). However, females directly affected by infant mortality do not appear to have been more or less stressed than other females. When infant loss was added to the original statistical model along with all potentially relevant interactions, the loss was not correlated with increased cortisol in either the full (GLM: $F_{1, 737}$=0.28, n=834, p=0.5936) or the final reduced model (GLM: $F_{1, 793}$=0.29, n=834, p=0.5922).

The few cases of successful reproduction suggest another relationship between stress and reproduction. Adding successful reproduction (defined as raising an infant to 12 months of age) to the original statistical model revealed that successful females had significantly lower cortisol than those who either avoided reproduction or experienced miscarriage or infant loss (GLM: $F_{1,\,793}$=4.73, n=834, p=0.0300). This result suggests that those females who were best able to respond to this severe stressor or those that suffered the fewest consequences of this stressor were best able to reproduce successfully.

Cortisol may have played an important role in allowing some females to avoid further investment in risky pregnancies. In one final model, I further subdivided the reproductive state of each female based on whether she had successfully carried a pregnancy to term, miscarried, made multiple reproductive attempts, or avoided pregnancy during the study period. The results of this model suggested that those females that miscarried had higher cortisol levels than any other subgroup. I therefore condensed these categories and added the predictor "miscarriage" and all biologically relevant interactions to the original model. After stepwise reduction, the final model included the significant predictors miscarriage (GLM: $F_{1,\,792}$=5.07, n=834, p=0.0246) and an additional interaction term number of adult female relatives * rank class (GLM: $F_{2,\,792}$=, n=834, p=0.0369); the remaining terms in the model are nearly identical to those presented in Table 3. Females who conceived during the post-*dagaa* period and then miscarried had higher predicted mean cortisol than those who either avoided reproduction or carried pregnancies to term (Figure 22). These results suggest that elevated cortisol might have allowed female baboons to terminate a costly investment during a stressful time; such a mechanism has been previously observed in female baboons (Beehner et al., 2006).

B. Reproduction and survival

Perhaps most significantly, adults who successfully avoided reproduction—in other words, failed to get pregnant—were less likely to die during this period of considerable hardship. For this analysis, I included all females who were cycling, pregnant, or lactating throughout the study and were "at risk" of becoming pregnant, except for four extremely old females (average age=21.78; average lifespan for females who reach adulthood=17.23) whose amenorrhea and deaths were presumably age related. I divided the remaining females into two classes—those who avoided reproduction following the loss of the *dagaa* and those who had detectable pregnancies. The three-year mortality rate for reproductives (0.125 deaths/female) was over twice as high as for non-reproductives (0.048 deaths/female), suggesting that reproduction was especially costly during this period. I found no significant relationship between cortisol and females who died during the course of study, indicating that cortisol itself was not a likely factor in these deaths. Rather, avoiding reproduction during an extreme chronic stressor may improve short-term survivorship and thus be a very beneficial effect of the chronic stress response.

It also deserves restating that even during this time of extreme stress, adult mortality rates do not increase dramatically, as one would expect. It instead seems likely that cortisol itself was instrumental in instigating many of the behavioral and physiological coping strategies that allowed the vast majority of the female baboons to weather the long-term effects of this severe ecological disaster.

III. Does the chronic stress response elicit adaptive physiological coping strategies in the juvenile females?

As expected, the juvenile females delay menarche in response to the stressful conditions and elevated cortisol of the post-*dagaa* period. Age at first swelling is known for 168 baboons from 1968 to 2002. I compared the pre-stress average to that of the post-*dagaa*-loss juveniles. Two of these females died before cycling. All but three of the remaining females had begun cycling by the end of March 2003. I assigned each of the remaining three females the lowest possible age at first cycling (age as of April 1, 2003); as a result, any measured difference in age at first swelling is conservative. The pre-stress average age of first swelling was 4.88 years, and the post-stress average was 6.12 years ($p<0.0001$). Juvenile females during this time of great hardship clearly show a significant delay in the onset of reproductive maturity.

As the daughters of low-ranking females have higher cortisol than daughters of high-ranking females in times of stress (see Chapter 3), any cortisol-mediated delay should be greater in these low-ranking females as the baboons adjust to the loss. In a comparison of study females, daughters of high-ranking females (n=4) had an average age of onset of

5.68 years during this stressful period, significantly different than that of daughters of low-ranking females (n=10) who began cycling at 6.37 years (p=0.044). All of the three non-cycling females at the end of the study were daughters of low ranking females, so once again the significance of this difference is likely greater than reported. A correlation between rank and delayed onset of swelling has been previously reported for this population (Packer et al., 1995).

As the effects of the stressor set in and then worsened in 2001, cortisol levels rose. Juvenile females experienced a significant delay in maturation until the worst of the stressor had passed; all but three of the 20 surviving females were cycling by mid-2002. By delaying cycling and reproduction, especially in the face of such a severe chronic stressor, juvenile females likely avoid excess energetic demands as well as costly and risky pregnancies and may improve their condition both during and immediately following an ecological disaster. They survive and are better able to maximize lifetime reproductive success during more auspicious times.

Discussion

My overarching goal in this study has been to promote a more complete understanding of and appreciation for the full scope of the stress response system. To be more specific, I believe the results of this study can expand our limited understanding of the evolution of the vertebrate stress response. While the field of stress research is over 70 years old, this is the first study to use empirical evidence gathered under natural conditions to consider the adaptive benefits of a highly conserved aspect of the chronic stress response system: the continued release of cortisol in response to a lasting stressor.

I. The Evolution of the Chronic Stress Response

In this study, I have begun to address a long-overlooked yet crucial evolutionary question: Why has the chronic stress response system—so clearly prone to deleterious and even deadly consequences—continued to persist throughout the history of vertebrates? I have also posed a subsequent question: might a persistent stress response in the face of a chronic stressor actually be adaptive? I have proposed that the vertebrate stress response system is not only useful in helping animals survive short-term life threatening circumstances, but also in promoting and prompting successful strategies in the face of long-term life threatening circumstances. I focused on three integral questions: 1.) Are chronic stressors truly rare in nature? 2.) What cortisol-mediated strategies might we see in response to a chronic stressor? 3.) Do these cortisol-mediated strategies have adaptive value?

A. Sudden resource loss as a model chronic stressor

For the Gombe baboons, the expulsion of the fishermen and the loss of the *dagaa* were tantamount to an ecological disaster. The loss was abrupt, unexpected, and unavoidable. More importantly, its effect on the population was clear and long-lasting, following a pattern strikingly similar to that reported for more classic, more natural ecological disasters. Within a year, birthrates began to drop and infant mortality began to rise. After two years, birthrates remained low, and infant mortality and miscarriage rates became extreme—a clear indication of the ecological significance of this lost resource.

Hormonal data suggest a close parallel between these demographic effects and the activation of the chronic stress response. Samples collected during the period with the worst demographic effects show significant elevations in cortisol in both adult and juvenile baboons. Furthermore, this response is independent of reproductive state, age, and social status, and is not ameliorated by social support. I investigated a number of other putative causes for this marked increase in cortisol, and no other cause can account for such a profound and widespread response.

B. Are chronic stressors truly rare?

Many factors have been associated with the prolonged release of cortisol in primates. I therefore hypothesized that events and states that cause the chronic release of cortisol would be fairly commonplace in a natural population. Indeed, the results of my model suggest that such ubiquitous phenomena as adolescence can invoke a chronic stress response. These results will be discussed in more detail in Chapter 3, but they clearly suggest that chronic stressors are indeed part of the normal life history of the vast majority of wild female baboons.

Using conservative demographic criteria based on the baboon's response to the *dagaa* crisis, I was able to identify between two and five probable ecological crises in the previous 35 years. The results of this analysis suggest that the average female baboon will face at least one protracted ecological disaster, inflicting devastating demographic effects and elevated cortisol levels for up to three years.

C. What cortisol-mediated strategies might we see in response to chronic stressors?

If cortisol does carry adaptive benefits in times of great stress, we can expect that it plays a role in prompting behavioral and physiological changes. Two clear behavioral strategies, transfer behaviors and changes in ranging patterns, correlated closely with the baboons' response to this ecological disaster, and both have been previously linked to cortisol.

TRANSFER BEHAVIOR

In what appears to be an attempt to escape this hardship, immigration rates rose to 98% in the year following the fishing ban, indicating an average of nearly one transfer per male baboon in the study population. The majority of these transfers were short-term—rates for transfers that lasted more than one month were virtually unchanged—suggesting that the baboons were transferring in search of better circumstances but failing to find them. As cortisol levels rose significantly in late 2001, we might also expect another rise in short-term transfers. Although there was a slight rise in 2001 and 2002, it was not commensurate with the cortisol data. As so many male baboons had attempted to transfer at the beginning of the disaster–and experienced ubiquitously compromised conditions–it is possible that many had already gained a cognitive understanding of the widespread nature of the resource loss and therefore did not attempt further transfers–especially since transfers are intrinsically stressful (Alberts et al., 1992; Bergman et al., 2005).

FORAGING AND RANGING BEHAVIOR

Animals responding to ecological stressors will often face significant changes in food availability. As a result, they can be expected to respond by changing both time budgets and ranging patterns in order to compensate for lost resources. While we have no data on time budgets, the monthly ranging patterns of the baboons suggest that the majority of troops expanded their ranges in an effort to fill the dietary hole left by the *dagaa*. Ranges expand most noticeably in close parallel with cortisol levels in 2001, suggesting that the chronic stress response may play a role in prompting changes in ranging behaviors. While it is difficult to pinpoint a single cause for this pattern of range expansion, cortisol is known to affect both metabolism and foraging behaviors, and these changes are certainly influenced by increased foraging pressures. The baboons reportedly spent less time on the beach, moved faster and ranged farther on any given day, and spent more time searching for foods that were a part of their more natural diet.

REPRODUCTIVE SUPPRESSION

While previous work has indicated that reproductive states such as pregnancy and lactation are themselves chronic stressors for female baboons (van Schaik et al., 1991; Altmann et al., 2004; Weingrill et al., 2004; Bales et al., 2005), my data indicated no such correlation, although there was a slight trend toward elevated stress in pregnancy (A. Bosacker, unpublished data). Breeding-age females showed >20% increase in inter-birth intervals during the years following the loss of the *dagaa*. Finer analyses further suggest that every indicator of reproductive suppression commenced in the year following the *dagaa* loss and worsened at the height of cortisol exposure, suggesting direct connections between stress, cortisol and reproductive suppression. As cortisol is known to have multiple suppressive effects on reproductive function in adult females, this tight correlation suggests that cortisol promotes physiological strategies that allow females to postpone the costs and risks of reproduction until conditions improve.

The juvenile females also show signs of reproductive suppression, as evidenced by a significantly decreased rate of reproductive maturation. The average age at first swelling in the years following the resource loss increases by over a full year, from 4.88 to 6.12 years of age. Daughters of low-ranking females, who face higher cortisol levels during this period, show even more prolonged reproductive delays during this time (5.68 vs. 6.37), providing further evidence of a coordinating role for cortisol. The literature also offers substantial support for such a link, as high stress situations and high cortisol levels are often associated with similar reproductive delays (Bentley-Condit & Smith, 1997; Bale et al., 1996; Marchlewska-Koj et al., 2003). Continued data on these study females may reveal whether the females who delayed reproduction longer during this stressor had greater reproductive success during the years following the recovery.

D. Are These Chronic Stress Response Strategies Adaptive?

If the chronic stress response and its key hormonal integrator, cortisol, do indeed offer adaptive benefits to animals facing chronic stressors, then they must provide immediate benefits that offset the risk of long-term pathological consequences. While it is easy to infer benefits for the cortisol-mediated strategies identified above, it is more difficult to provide empirical evidence of their benefits for condition and survival. For example, if ecological conditions deteriorate, an animal may move to an unaffected area, and under many circumstances it would benefit from doing so. If cortisol is instrumental in prompting this behavior, its actions can then be considered adaptive. As few of the male baboons appear to have found improved conditions elsewhere, this was likely an ill-matched strategy for such a ubiquitous stressor as this. But on an evolutionary timescale, do males prompted to move by a cortisol-mediated mechanism survive such stressors in better condition than those who remain? If, in a chronic stress event, food becomes more difficult to find, then successful foraging will likely require more time and energy. If cortisol prompts shifts in behavior that promote increased foraging-related ranging behaviors, such an action would certainly be beneficial. The changes in ranging patterns exhibited by the Gombe baboons are suggestive of such a mechanism, but quantification of related benefits remains a challenge.

Because of the vast reproductive information embedded in the long-term demographic data, I chose to focus on reproductive suppression in order to address this third question of adaptive value. I uncovered two lines of evidence that chronic cortisol exposure and reproductive suppression carried immediate selective benefits: high cortisol levels promoted the early termination of risky pregnancies, and females with more complete reproductive suppression had higher rates of survival.

MINIMIZING RISKY INVESTMENTS

Cortisol may coordinate a mechanism that prevents females from making continued investments in particularly risky pregnancies, an idea which has previous support in the literature (Beehner et al., 2006). Females who conceived did not show significantly different cortisol levels from those who did not conceive. However, those who conceived and subsequently miscarried did exhibit higher predicted mean cortisol than either females who carried babies to term or showed complete reproductive suppression. Continuing a risky pregnancy under the pressures of a chronic stressor imposes considerable costs. For example, if energy is already at a premium, the added energetic expense of a pregnancy may decrease an animal's chance of survival and may also cause continued physiological consequences that delay recovery or compromise future reproduction. Also, even if a female successfully completes a pregnancy in spite of a compromised state, if the infant is then exposed to conditions that entail high infant mortality, her continued investment will likely prove to have been unsound. Again, she will have lost her energetic investment, suffer compromised condition and survival, and she will have no fitness benefit to show for it. Based on the overwhelming effects of this particular chronic stressor on infant survival, the adaptive benefits of a cortisol-mediated mechanism that mediates such a risky investment are clear. It is a mechanism that likely served many of the Gombe females well, as miscarriage rates during this stressor soar to roughly 30% of detectable pregnancies. As the same ecological stressor carried an infant mortality rate of 86%, these early terminations were an adaptive means of making the best of a bad situation.

It is also interesting to note that females who successfully carried infants to term had lower cortisol levels; this could indicate a better initial condition or other individual characteristics (e.g. membership in a more successful troop) that made the resource loss inherently less stressful and a reproductive investment therefore less risky. None of these results is surprising, and in fact each supports the general adaptive value model for the chronic stress response presented here. Individuals respond to the same stressor to varying degrees, and while this stressor clearly affects all of the baboons, its effects were certainly not uniform. Those females who fare the best are expected to have better general condition, lower cortisol levels, and less reproductive suppression.

PROMOTING SURVIVAL

The energetic costs of reproduction can negatively affect an individual's overall condition. In the face of a severe chronic stressor, even slight changes in condition may push an individual over the tipping point between survival and death. I looked at reproductive status and mortality data to determine whether cortisol-mediated reproductive suppression improves survival. Indeed, females with more complete reproductive suppression suffered markedly lower mortality rates than those who had one or more detectable pregnancies. Therefore, reproductive suppression can increase survival under conditions of chronic stress that produce prolonged periods of elevated cortisol.

In times of unremitting stress, the chronic stress response and extended cortisol release can promote reproductive suppression, thereby improving an individual's chance to survive until conditions eventually improve. Thus the health risks from prolonged cortisol release are overcome by the improved chances for survival.

DELAYS AND MORTALITY DATA

Two additional results provide further evidence of the adaptive nature of the chronic stress response. Many animals show long delays between the onset of a severe ecological stressor and their eventual demographic and physiological response. According to Wingfield and Kitaysky (2002), such delays are the result of cortisol's early activation of a primary suite of adaptive behavioral and physiological response that allow the animal to react optimally to the stressor and thereby decrease its overall impact. This response is likely coordinated either by brief or low-level increases in cortisol. However, if this initial response delays or avoids a more extreme reaction, then this suite of cortisol-induced strategies has clear fitness benefits. The most drastic rise in cortisol in the Gombe baboons was delayed by over two years, perhaps in part as a result of the initial effectiveness of behavioral strategies.

The pervasive and devastating impact of this stressor is clear. The loss of such a valuable resource as the *dagaa* prompted the baboons to exhibit a number of costly behaviors such as troop transfers and home-range adjustments, signifying that current selective pressures were so strong that the benefits of abating this stressor were less than the considerable costs of these behaviors. Nevertheless, almost all of the affected animals successfully navigated this disaster, perhaps as a direct result of the adaptive nature of the chronic stress response.

E. Are these apparent strategies simply a matter of malnutrition?

Could widespread malnutrition following the loss of the *dagaa*, independent of cortisol, have caused the observed demographic, behavioral, and reproductive patterns? Food restriction and malnutrition can lead to many of the same reproductive changes reported for cortisol: cycle irregularities, anovulation, amenorrhea, etc. Incidentally, reproductive suppression in food restriction research has long been understood to have an adaptive value in that it promotes survival when survival is most crucial. In fact, long-term food restriction can significantly extend lifespan in a number of taxa. While research is ongoing, there is mounting evidence that the same effects will be identified in primates (Mattison et al., 2003; Bodkin et al., 2003).

Whether malnutrition or cortisol prompted the responses observed in this study, the loss of the *dagaa* likely affected the local carrying capacity. The overall range of the study population did not change from 1998-2002 (Figure 18), and thus the population did not compensate for lost resources by expanding their ranges to include new areas.

If the *dagaa* had allowed the baboons to overreach the “normal” carrying capacity, malnutrition should have led to a swift drop in the size of the population. While the overall population size dropped by about 12% in the four years following the resource loss, there was virtually no change in the first full year following the removal of the fishermen. Instead, the population declined by about 7% in the second year, stabilized for a year, then fell by another 4%. As there was no further decline in food availability from 1999 to 2000 or 2001 to 2002, the initial delay and the subsequent secondary drop are difficult to reconcile with malnutrition alone. There were no reports of an overall change in body composition and no anecdotal evidence that the baboons suffered from profound hunger. Furthermore, while some reproductive parameters do indicate rapid suppression as predicted by malnutrition, the strongest effect occurred a full three years after the loss.

The observed patterns of demographic and physiological change closely follow what Wingfield and Kitaysky (2002) predict from cortisol-mediated coping strategies and delays in response. The striking parallel between the profound increase in cortisol and the period of most marked reproductive suppression (Figures 11 & 12) is also strongly suggestive of a cortisol-mediated mechanism. In this case, the evidence suggests that cortisol plays an important role in mitigating the responses to hardship long noted by life history theory.

Malnutrition may certainly have played a role in prompting cortisol-mediated strategies. There is a well documented relationship between caloric restriction and cortisol, suggesting that hunger itself can constitute a chronic stressor. It is possible that at various times during the post-*dagaa* adjustment periods the baboons suffered the effects of malnutrition, exacerbating the cortisol response, which in turn instigated coping strategies that alleviated hunger and malnutrition.

While the link between food restriction, reproductive suppression, and lifespan has been studied extensively, the complex mechanisms that underlie this process are poorly understood (Masoro, 2006). There is, however, a possible link between cortisol and food-restricted longevity. Although the significance of endocrine mechanisms such as cortisol has long been noted (Nelson et al., 1995), little research has been conducted to determine the exact role that cortisol might play. Initially, glucocorticoids were believed to play a purely degenerative role in aging, and it was assumed that the mechanisms of food restriction might alleviate these effects in some way. While the full picture is as yet unclear, more recent research suggests that the benefits of food restriction may in part act through cortisol. There is mounting evidence that regular mild stressors may be beneficial, promoting mechanisms of maintenance and repair and perhaps even retarding the aging process (Sabatino et al., 1991; Nelson et al., 1995). Food-restricted macaques show additional diurnal peaks in cortisol release that may play a role in the mechanisms affecting longevity (Urbanski et al., 2004). Recent cellular-level research also suggests that while chronic cortisol exposure can have negative short-term effects, prior exposure to such stressors can actually have lasting beneficial effects on cellular physiology (Kletsas et al., 2007). While much controversy still surrounds food restriction research and the role of cortisol, it is increasingly likely that cortisol plays a significant–and complex–role (Landfield et al., 2007)

II. Reinterpreting the Chronic Stress Response

Virtually all previous research on chronic stress exposure in either the wild or in captivity has focused solely on the costly and potentially pathological consequences of cortisol. Cortisol has come to be widely used as a proxy measure for the physiological costs associated with natural states such as low rank and reproduction as well as the impacts of human activities such as habitat destruction and tourism. This remains a valuable approach for stress research, especially for studies on wild populations. In fact, the results presented here further support numerous previous studies that use cortisol to validate putative stressors. What my research suggests, though, is the need for a more complete interpretation of the results of such studies that also considers what animals might have to gain from the continued activation of the chronic stress response. To refocus on a more holistic interpretation of stress may better inform us about the function and evolution of the stress response. Perhaps more importantly, it may also facilitate a more complete understanding of the true costs and benefits associated with some of the more complex questions of vertebrate evolution. I have chosen to briefly revisit two popular topics in stress physiology under this more adaptive vision of chronic stress.

A. Rethinking stress and hierarchies

Animal behaviorists have long been fascinated by hierarchical behavior and the evolution of this complex social dynamic. What historical forces have shaped this system of interactions? If low-ranking animals suffer disproportionate costs, why do they appear to accept their position so readily? What do high-ranking animals have to gain at the top, and what selective forces might limit seemingly adaptive aggressive behaviors that would help individuals secure and maintain dominance? Stress physiologists have been using glucocorticoids for decades in order to quantify some of the costs associated with various positions in the hierarchy. While very early work focused on the rigors of high rank and the "executive stress syndrome," the majority of studies assume that low rank carries greater physiological costs. The relationship between rank and stress has actually proven to be extraordinarily variable; high rank confers certain stress-related physiological costs in some cases and low rank in others (for review, see Abbott et al., 2003). In many cases, no particular position exposes an animal to more stressors than another. While the relationships identified by such studies may vary, the interpretation of such correlations has been remarkably consistent: when rank is correlated with cortisol, animals face unavoidable pathological consequences.

Consider, for example, those hierarchies for which low rank constitutes a chronically stressful state. Male baboons exemplify this pattern (Sapolsky, 1982), and low-ranking males have been shown to suffer a host of the physiological effects of chronic stress. The traditional view has been that the increased exposure to cortisol is simply a cost and consequence of an unfortunate position. The evolution of the formidable physical features and the notoriously aggressive behaviors of the male baboon can, at least in part, be explained as attempts to effectively avoid these costs. There is undeniable merit to this approach. When low rank is associated with high stress, selective pressures will certainly promote traits and behaviors that can minimize an individual's chance of being relegated to an inferior position.

But might the chronic stress response of subordinate males carry more immediate adaptive benefits? If low rank carries costs—reduced food, more harassment—then perhaps prolonged cortisol exposure promotes a set of strategies that will optimize the animal's response to his suboptimal rank. Under this model, an individual who responds to low rank via the activation of the chronic stress response will then be more likely to respond with a set of behaviors that will actually minimize many of the risks associated with his status.

Low rank and high cortisol have each been associated with depression and anxiety in a number of animal models including primates. In turn, depression and anxiety are linked to the presentation of suites of behaviors including lethargy, increased vigilance, and avoidance behaviors. While research on these conditions is overwhelmingly driven by an interest in their pathological consequences, evolutionary psychiatrists have taken a less conventional approach by recognizing that the prevalence of depression and anxiety are suggestive of a historically adaptive role (as reviewed in Nesse, 2000; see also below). They suggest that the display of depression-like and anxiety-like behaviors have long benefited animals facing a number of ecological and social challenges, although their focus is often on subordinate individuals. A male baboon at the bottom of the hierarchy, for example, might implement avoidance strategies. By actively avoiding close contact with higher ranking individuals, as depressed individuals often do, he may minimize the risk of bearing the brunt of their aggression. In other words, animals facing the stress of low rank may benefit from the same cortisol-mediated suite of behavioral strategies associated with depression—an evolutionary defense of sorts—that can actually improve overall fitness. A similar scenario could apply to females coping with the stress of a new and potentially infanticidal male—her best move might be to maintain a low profile and avoid social contact; if cortisol helps drive this response, then her elevated cortisol may also serve an important purpose.

While a full consideration of the evolution of depression and anxiety is outside the scope of this discussion, the basic approach presented by the evolutionary psychiatrists is illustrative of how a more adaptive consideration of the chronic stress response might inform our understanding of hierarchies and rank-related behaviors. In our evolutionary history, precursors to depression and anxiety may have played a role in allowing low-ranking individuals to make the best of a bad situation. The chronic stress response and elevated cortisol that we see in male baboons are certainly indicative of a suboptimal position in the hierarchy with associated long-term physiological costs. It may also be part of a subtle yet adaptive response that minimizes the negative repercussions of a less than ideal social status.

B. Rethinking the plasticity of the stress response system

One of the most exciting developments in stress research involves an advanced appreciation for the plasticity of the vertebrate stress response system. While the physiological response to stress is in many ways extremely consistent across vertebrate taxa, the finer functions of the system are extremely responsive to the specific characteristics of the individual and the stressor. Mounting evidence suggests that the brain and body are able to interpret the specific circumstances of many stressors and then to further modulate responses based on the condition of the individual: sex, social state, reproductive state, age. As a result, an individual can mount tailored responses to two distinct stressors, or a single stressor might evoke a different response in the same individual at different life stages.

Previous experiences can also significantly affect the way an individual responds to a given stressor. In fact, even minor exposure to stressful events either in utero or early in life can permanently reshape the stress response system. In many cases, animals who experience—or whose pregnant mothers experience—a given stressor will become more responsive to future stressors than individuals whose early lives were less eventful (e.g. Clarke & Schneider, 1993). They mount significantly different responses to new stressors, often involving greater increases in cortisol as well as more dramatic changes in behavior. This reorganization of the stress response system and its heightened sensitivity are often persistent and, in many circumstances, permanent. Much of the related research focuses on the long-term consequences of this adjusted stress response system, specifically a higher lifetime exposure to cortisol and ultimately a higher risk for stress-related pathologies. Early stress experiences are almost universally considered to be deleterious, if not dangerous. For example, pregnant women are strongly encouraged to avoid stress in pregnancy for fear of the lasting damage it might cause her unborn child—advice that ironically often only serves to send women into a frenzied panic of spiraling psychosomatic stress.

What adaptive benefit might there be to permanently adjusting an individual's stress response system in response to its early experiences? Perhaps, for example, individuals who suffer early stressors are those whose environment will expose them to more frequent lifelong exposure to stress. The early reshaping of the stress response system could represent an adaptive "priming" of the system that actually promotes long-term coping strategies that ultimately benefit an animal that is likely to face a lifetime of relative hardship.

Young daughters of low-ranking female baboons have higher cortisol levels than daughters of mothers with high rank (see Chapter 3). Because rank is matrilineally inherited, these daughters will almost certainly spend their entire lives near the bottom of the hierarchy. Perhaps early exposure to stress prompts permanent adjustments in her stress response system that will promote strategies for coping with her low status. In adulthood, successful strategies triggered in infancy may significantly attenuate her exposure to cortisol and actually minimize her rank-related exposure. This could also help explain the lack of rank-related cortisol differences in adulthood.

This more adaptive approach to the effects of early stress has antecedents in the literature (for review, see Badyaev, 2005). Recent studies propose a "that which doesn't kill you makes you stronger" approach, suggesting that prior exposure to stressors can promote specific behavioral and physiological responses that minimize the need for full-blown chronic stress responses. Similar studies also suggest that individuals that have experienced minor chronic stressors exhibit a more efficient response to more substantial threats, minimizing both cortisol and the detrimental effects of the stressor. An emerging pattern strongly suggests that chronic activation of the stress response system can promote stress-minimizing strategies.

III. Implications of an adaptive approach to the chronic stress response

Evolutionary psychiatrists, in many ways beating evolutionary biologists at our own game, have been contemplating the evolutionary history of stress-related behaviors for decades (for recent work see Gilbert, 2000; Nesse, 2000; Gilbert et al., 2002; Martin, 2002; Gilbert et al., 2004; Gilbert, 2006). How and why, they wonder, do presumably adaptive behavioral coping strategies elicit debilitating disorders like depression, anxiety, and post-traumatic stress disorder? Modern society, with all of its psychological rigors and physical demands, is easy to blame. Yet in order to truly understand these disorders, they argue, we need to understand their evolutionary roots. In doing so, evolutionary psychiatrists hope to gain a broader understanding of the symptoms these disorders present and the circumstances that trigger them. Perhaps this more holistic approach can lead to more effective, more efficient treatments and, just as importantly for millions of people, encourage a broader societal respect for the specter of mental illness. This exemplifies the approach I would like to see embraced more fully throughout the field of stress research. We can certainly agree that chronic stressors are threats to our well being, and any animal that suffers a constant barrage of these threats is also at a greater risk for the more indirect stress-related pathologies. But the stress response system is constantly proving to be far more effective and eloquently coordinated than we have ever imagined.

Overall Conclusions

The results of this study strongly suggest that even the chronic components of the stress response system can offer significant adaptive benefits for animals facing chronic stressors. The sudden removal of a valuable resource brought about marked demographic and physiological changes in this wild population of baboons, including a significant population-wide increase in cortisol exposure. The baboons also showed evidence of behavioral and physiological cortisol-mediated coping strategies, including increased transfer behavior, changes in ranging patterns, and reproductive suppression. This study also provides additional evidence that cortisol may represent a useful mechanism for minimizing investment in risky pregnancies and thereby salvaging overall condition during a severe stressor. Furthermore, those females who implemented a more complete strategy of reproductive suppression showed increased likelihood of survival. These and other coping strategies mediated by the continued release of cortisol may provide short-term fitness benefits by reducing mortality under dire circumstances. These more immediate benefits, especially in the face of a life-threatening stressor, would certainly outweigh the eventual physiological costs long attributed to chronic stress.

Suggesting that the chronic stress response has an adaptive advantage runs counter to decades of research that focused on “the double-edged sword” nature of the stress response system: the acute stress response is adaptive, chronic activation is merely maladaptive and pathological. It instead seems clear that the vertebrate stress response system is more complex than we had anticipated. Accepting this fact can provide a better understanding of how the system can promote survival under a wide variety of stressful conditions presented by a continuously unpredictable natural environment.

Table 1. Potential chronic stressors considered as predictors of cortisol.

Adult Model Predictors	Juvenile Model Predictors
Ethanol Preservation Lag	Ethanol Preservation Lag
Freeze Lag	Freeze Lag
Age	Age
Reproductive Status	Mom Alive
Number of Adult Female Relatives	Number of Adult Female Relatives
Rank	Mom's Rank
Troop	Troop
Field Season	Field Season
Ethanol Preservation Lag*Freeze Lag	Ethanol Preservation Lag*Freeze Lag
Freeze Lag*Troop	Freeze Lag*Troop
Age*Field Season	Age*Mom Alive
Age* Number of Adult Female Relatives	Age* Number of Adult Female Relatives
Reproductive Status*Field Season	Age*Mom's Rank
Reproductive Status*Number of Adult Female Relatives	Mom Alive*Number of Adult Female Relatives
Number of Adult Female Relatives*Field Season	Number of Adult Female Relatives*Field Season
Number of Adult Female Relatives*Rank	Mom's Rank*Number of Adult Female Relatives
Rank*Troop	Mom's Rank*Field Season
Rank*Field Season	Troop*Field Season
Troop*Field Season	

Table 2. Type 3 tests of fixed effects: Full adult female model. Italics denote main effects and interactions that were found to be significant predictors of cortisol in this model.

Effect	DF	F	P
Ethanol Preservation Lag	1	0.37	0.5458
Freeze Lag	*1*	*5.01*	*0.0254*
Age	1	1.10	0.2956
Reproductive Status	5	0.68	0.6373
Number of Adult Female Relatives	1	0.37	0.5409
Rank	2	0.05	0.9485
Troop	*7*	*2.68*	*0.0095*
Field Season	*2*	*5.85*	*0.0030*
Ethanol Preservation Lag*Freeze Lag	1	0.09	0.7645
*Freeze Lag*Troop*	*7*	*2.70*	*0.0091*
Age*Field Season	2	0.74	0.4757
Age* Number of Adult Female Relatives	1	0.52	0.4707
Reproductive Status*Field Season	10	0.79	0.6380
Reproductive Status*Number of Adult Female Relatives	5	0.28	0.9232
Number of Adult Female Relatives*Field Season	2	0.37	0.6912
Number of Adult Female Relatives*Rank	2	0.85	0.4288
Rank*Troop	14	0.88	0.5788
*Rank*Field Season*	*4*	*2.67*	*0.0314*
*Troop*Field Season*	*14*	*2.08*	*0.0112*

Table 3. Type 3 tests of fixed effects: Reduced adult female model. Italics denote main effects and interactions that were found to be significant predictors of cortisol in this model.

Effect	DF	F	P
Freeze Lag	*1*	*5.53*	*0.0189*
Rank	2	1.82	0.1634
Troop	*7*	*2.86*	*0.0059*
Field Season	*2*	*13.16*	*<0.0001*
*Freeze Lag*Troop*	*7*	*2.73*	*0.0083*
*Rank*Field Season*	*4*	*2.74*	*0.0279*
*Troop*Field Season*	*14*	*2.21*	*0.0063*

Table 4. Type 3 tests of fixed effects: Full juvenile female model. Italics denote main effects and interactions that were found to be significant predictors of cortisol in this model. †Denotes those that were insignificant in full model, but significant in reduced.

Effect	DF	F	P
Ethanol Preservation Lag	1	1.68	0.1983
Freeze Lag	1	1.82	0.1805
† Age	1	1.26	0.2645
† Mom Alive	1	2.06	0.1546
† Number of Adult Female Relatives	1	0.03	0.8597
Mom's Rank	*2*	*10.20*	*<0.0001*
Troop	*5*	*13.45*	*<0.0001*
Field Season	*2*	*8.43*	*0.0004*
Ethanol Preservation Lag*Freeze Lag	1	0.61	0.4369
*Freeze Lag*Troop*	*5*	*12.56*	*<0.0001*
Age*Mom Alive	1	0.28	0.5958
Age* Number of Adult Female Relatives	1	0.01	0.9190
*Age*Mom's Rank*	*2*	*7.08*	*0.0013*
*Mom Alive*Number of Adult Female Relatives*	*1*	*21.70*	*<0.0001*
Number of Adult Female Relatives*Field Season	2	1.01	0.3662
*Mom's Rank*Number of Adult Female Relatives*	*2*	*20.94*	*<0.0001*
*Mom's Rank*Field Season*	*4*	*2.92*	*0.0246*
*Troop*Field Season*	*10*	*7.35*	*<0.0001*

Table 5. Type 3 tests of fixed effects: Reduced juvenile female model. Italics denote main effects and interactions that were found to be significant predictors of cortisol in this model.

Effect	DF	F	P
Freeze Lag	1	0.99	0.3215
Age	*1*	*8.03*	*0.0055*
Mom Alive	*1*	*13.95*	*0.0003*
Number of Adult Female Relatives	*1*	*10.69*	*0.0014*
Mom's Rank	*2*	*10.05*	*<0.0001*
Troop	*5*	*13.42*	*<0.0001*
Field Season	*2*	*10.90*	*<0.0001*
*Freeze Lag*Troop*	*5*	*12.26*	*<0.0001*
*Age*Mom's Rank*	*2*	*6.00*	*0.0034*
*Mom Alive*Number of Adult Female Relatives*	*1*	*38.65*	*<0.0001*
*Mom's Rank*Number of Adult Female Relatives*	*2*	*22.36*	*<0.0001*
*Mom's Rank*Field Season*	*4*	*2.50*	*0.00466*
*Troop*Field Season*	*10*	*7.32*	*<0.0001*

Table 6. Rank changes in Troop BA between 1999 and 2000. Colors indicate family groups. The Afu family (green) left in September 1999 to form a new troop.

Rank	1999 Female Hierarchy	2000 Female Hierarchy
1	**Afu**	**WhiteThorn**
2	**Araceae**	**Whitlow**
3	**Azanza**	**WhiteRose**
4	**Acida**	**W.Lychee**
5	**Azima**	**Mikania (menarche 8/99)**
6	**Amara**	**Mimosa**
7	**Annona**	**Morinda**
8	**Canna**	**Webbia**
9	**Whitlow**	**Canna**
10	**WhiteRose**	**Amara**
11	**Webbia**	**Annona**
12	**W.Lychee**	
13	**WhiteThorn**	
14	**Mimosa**	
15	**Morinda**	

Table 7. Rank changes in Troop DC between 1999 and 2000. Colors indicate family groups.

Rank	1999 Female Hierarchy	2000 Female Hierarchy
1	**Sipoty**	**Sipoty**
2	**Salimia**	**Salimia**
3	**Snowdrop**	**Snowdrop**
4	**Soda**	**Soda**
5	**Saladi**	**Saladi**
6	**Sangrilla**	**Harina**
7	**Semolina**	**Hag**
8	**Spice**	**Sopapila (menarche 5/00)**
9	**Harina**	**Semolina**
10	**Hag**	**Spice**
11	**Halisi**	**Halisi**
12		**Sangrilla**

Table 8. Previous episodes of probable ecological stress. The periods presented below are those that show two or more of the extreme demographic patters characteristic of a severe ecological stressor. Each episode of presumed stress is identified by color (e.g. pink indicates a triumvirate of demographic indicators of profound stress between 1978-1980).

Low Birthrate	**High Infant Mortality**	**High Miscarriage Rate**
1970	1972	1970
1978-1979	1979-1980	1973
1981	1986	1978-1979
1985	1990	1982-1983
1989		1986-1987
		1994-1995

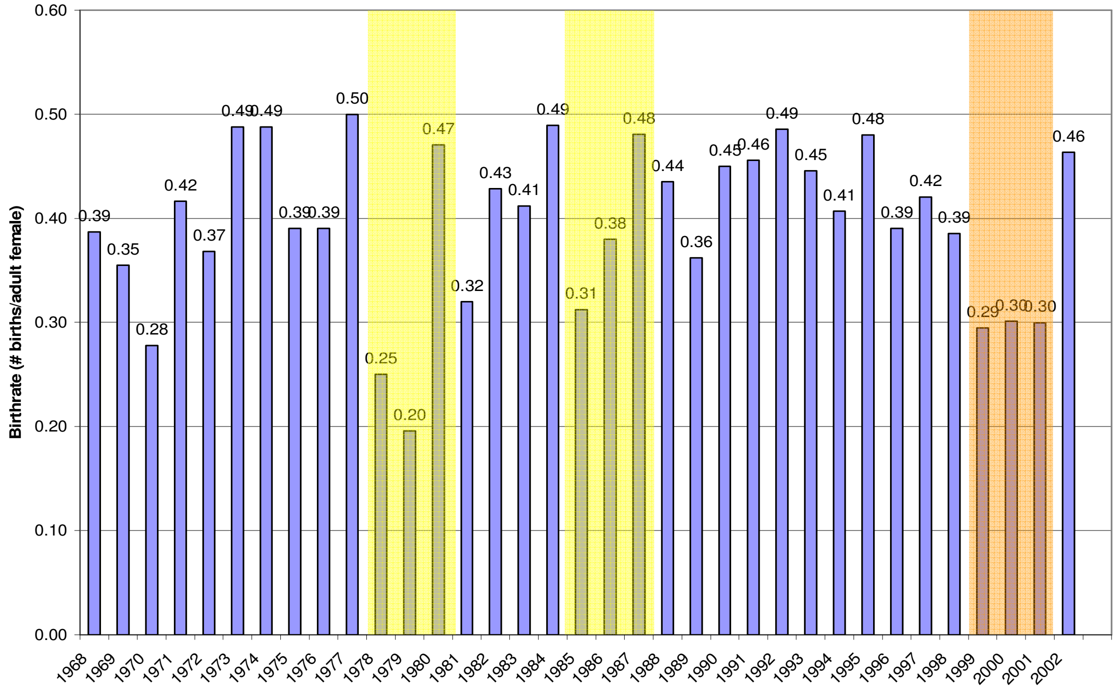


Figure 1. Annual birthrates (1968-2002). The orange shaded section highlights the 3-year post-*dagaa* period. Areas shaded in yellow highlight previous 3-year periods of ecological perturbation (defined on p. 68).

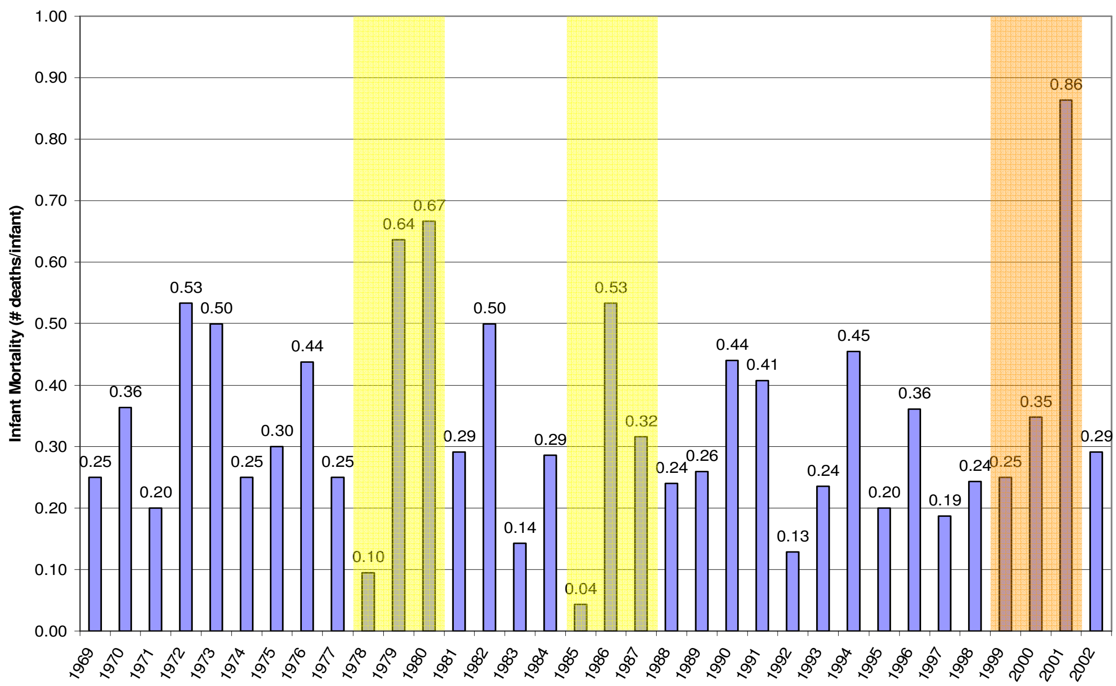


Figure 2. Mortality rates for infants under 1 year of age (1968-2002). The orange shaded section highlights the 3-year post-*dagaa* period. Areas shaded in yellow highlight previous 3-year periods of ecological perturbation (defined on p. 68).

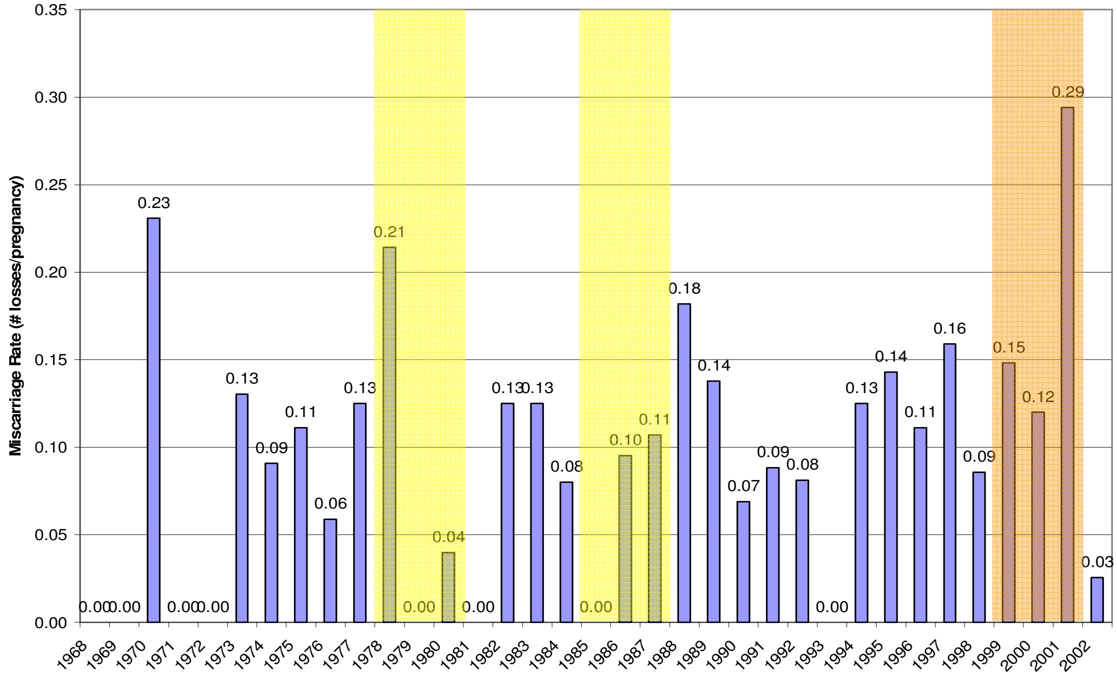


Figure 3. Miscarriage rates (1968-2002). The orange shaded section highlights the 3-year post-*dagaa* period. Areas shaded in yellow highlight previous 3-year periods of ecological perturbation (defined on p.68).

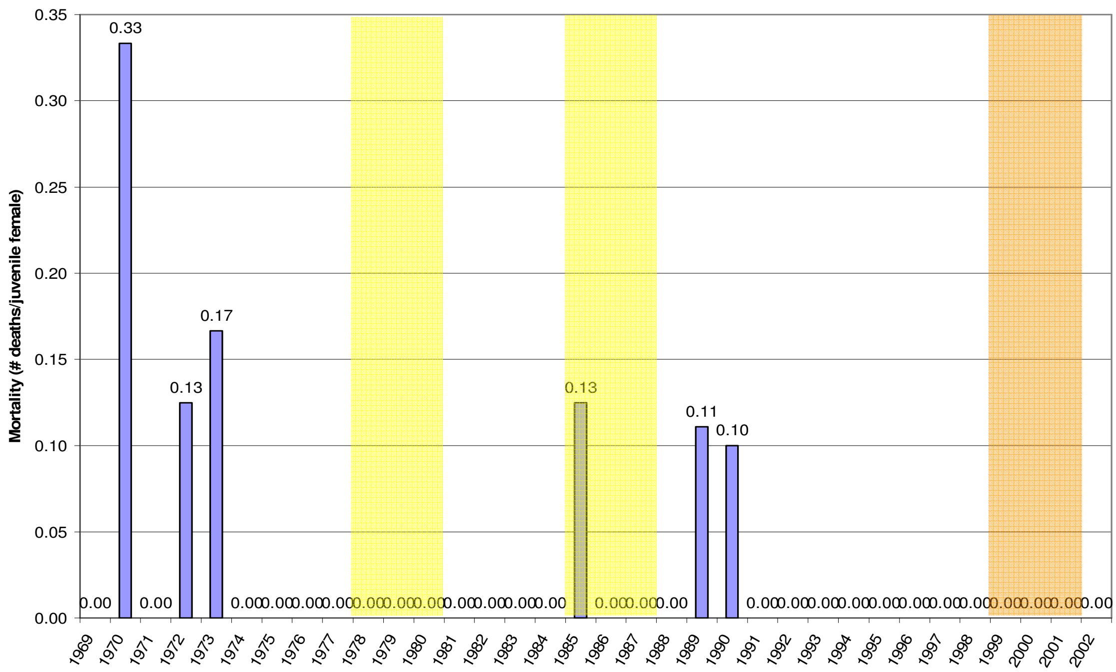


Figure 4. Mortality rates for juvenile females (1968-2002). The orange shaded section highlights the 3-year post-*dagaa* period. Areas shaded in yellow highlight previous 3-year periods of ecological perturbation (defined on p.68).

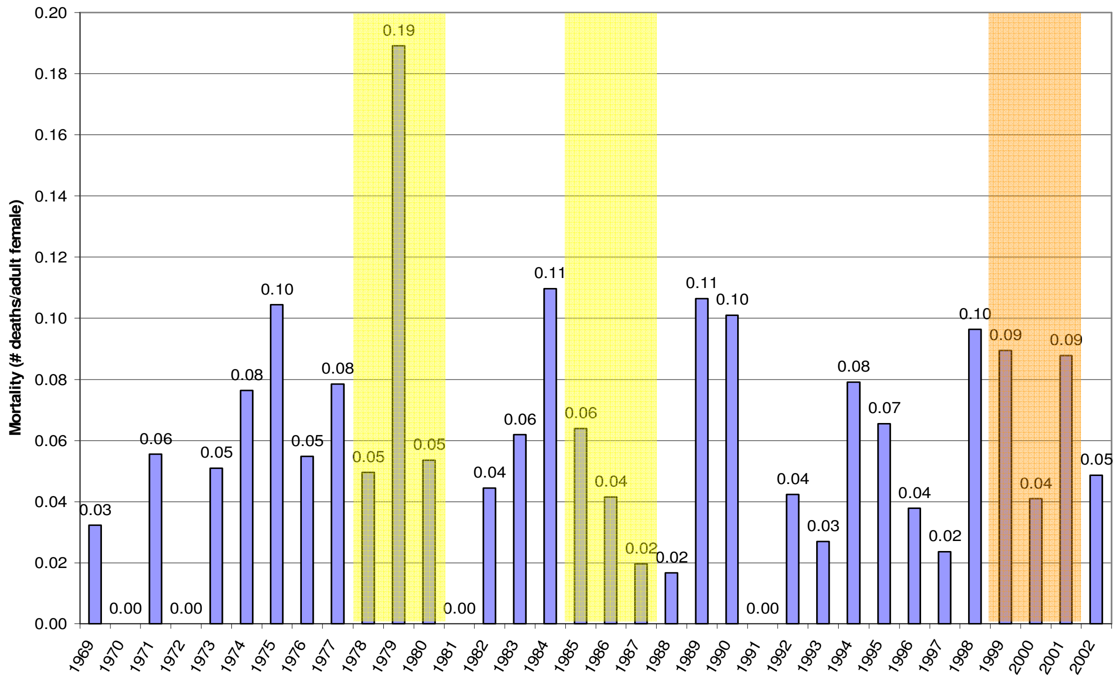


Figure 5. Mortality rates for adult females (1968-2002). The orange shaded section highlights the 3-year post-*dagaa* period. Areas shaded in yellow highlight previous 3-year periods of ecological perturbation (defined on p. 68).

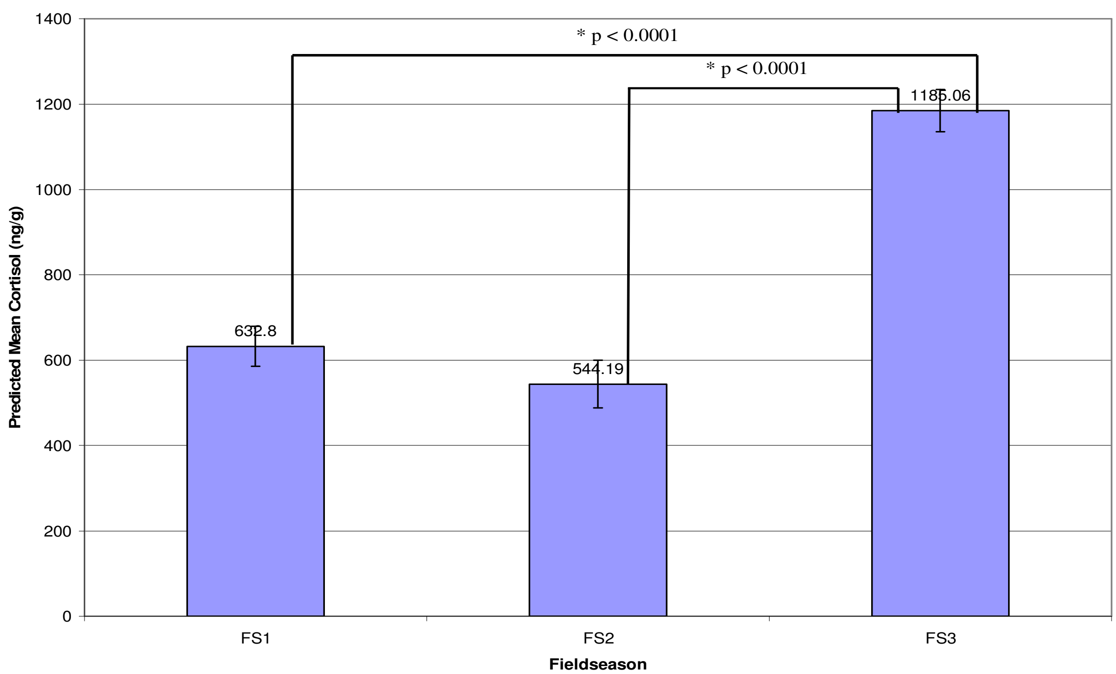


Figure 6. Effect of field season on predicted mean cortisol in adult female baboons. *(FS1: May & June 2000; FS2: February & March 2001; FS3: December 2001-April 2002)*

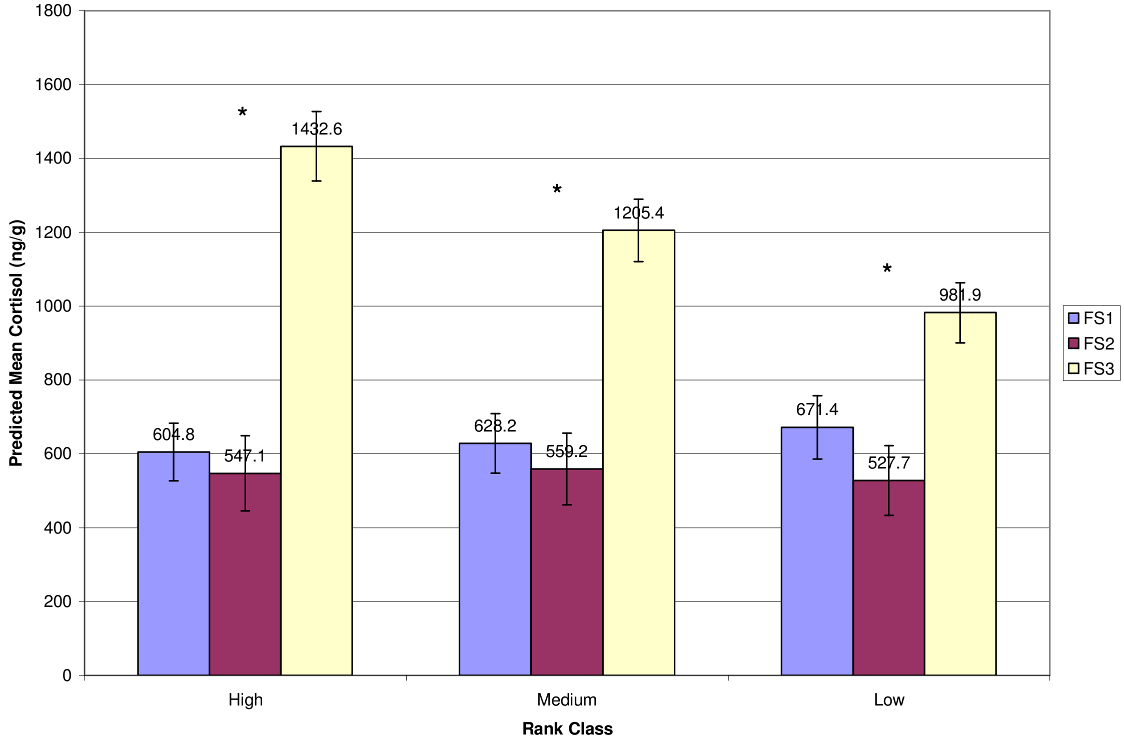
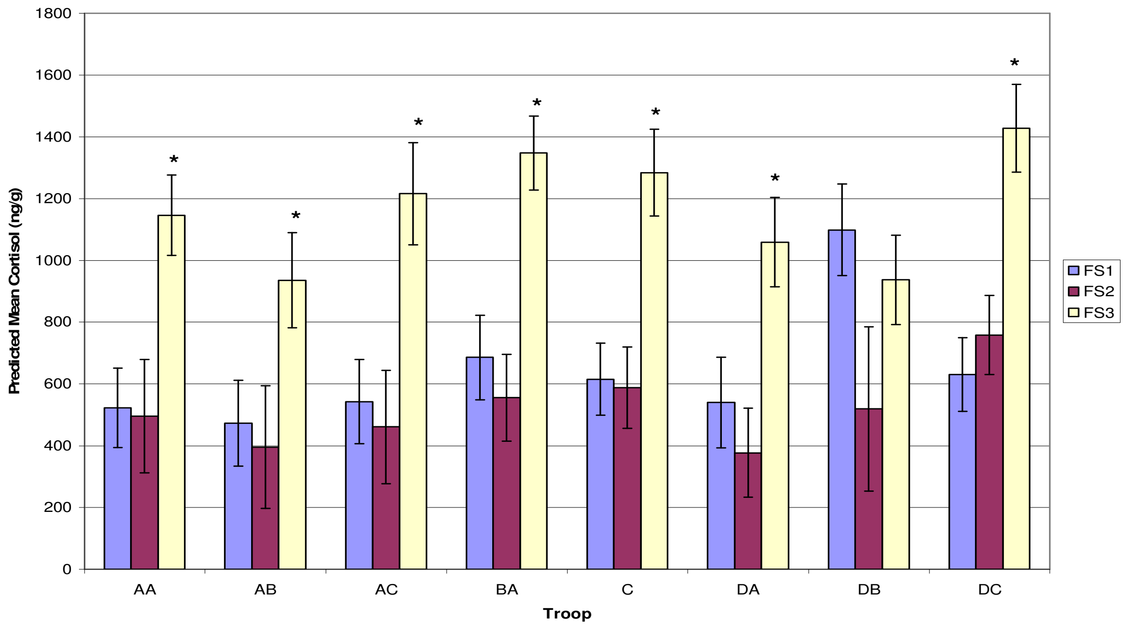


Figure 7. The interaction of rank and field season and its effect on predicted mean cortisol in adult female baboons. For the high and medium rank classes, FS3 is significantly higher than FS2 and FS1 ($p < 0.0001$). For the low rank class, FS3 is significantly greater than FS2 ($p=0.0089$) but not FS1 ($p=0.1797$). *(FS1: May & June 2000; FS2: February & March 2001; FS3: December 2001-April 2002)*

a.)



b.)

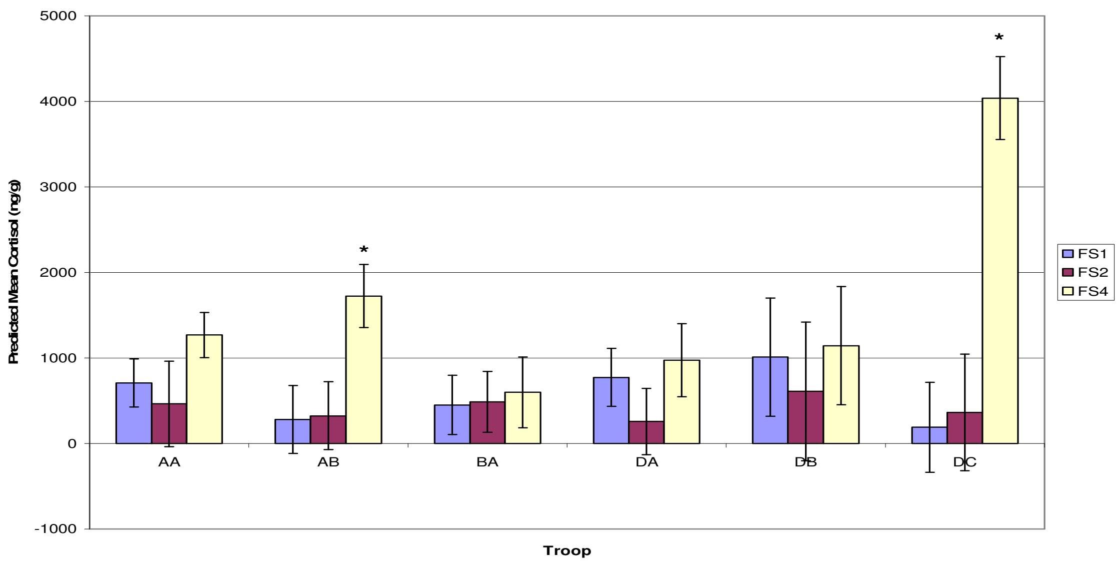


Figure 8. The interaction of troop and field season and its effect on predicted mean cortisol in female baboons. These graphs show predicted mean cortisol for each field season for **a.)** the adult females and **b.)** the juvenile females. **a.)** Seven of eight troops shows a clear increase in FS3, and Tukey-Kramer comparisons show that this difference is significant for five of these troops (FS1 vs. FS3 and FS2 vs. FS3, $p<0.05$) **b.)** All of the juvenile troops show an increase in predicted mean cortisol during this period, and two of these significant (AB, Tukey-Kramer: $p<0.003$; DC, Tukey-Kramer: $p<0.001$). *(FS1: May & June 2000; FS2: February & March 2001; FS3: December 2001-April 2002)*

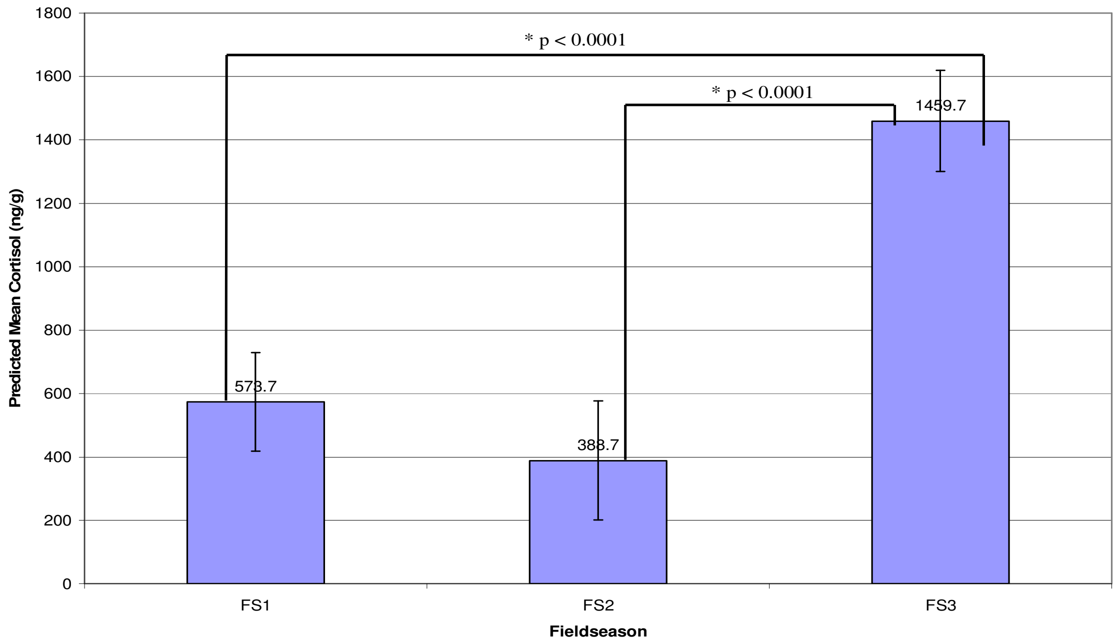


Figure 9. Effect of field season on predicted mean cortisol in juvenile female baboons. *(FS1: May & June 2000; FS2: February & March 2001; FS3: December 2001-April 2002)*

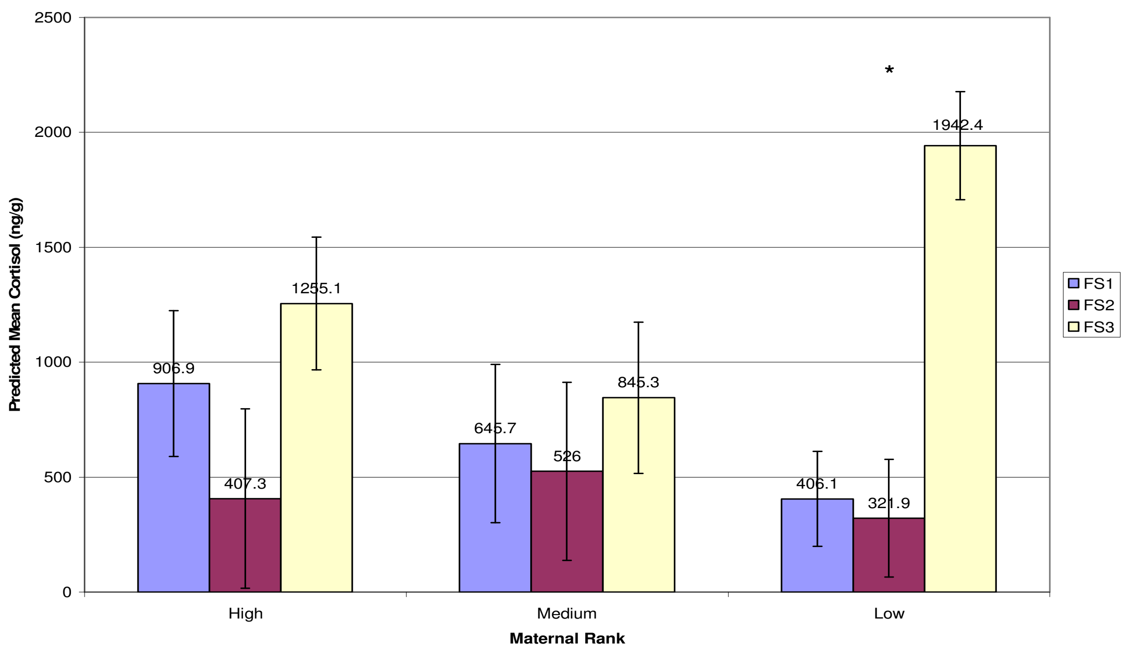


Figure 10. The interaction of mother's rank and field season and its effect on predicted mean cortisol in juvenile female baboons. Each rank class shows increased cortisol in FS3, but this difference is only significant in daughters of low ranking females (FS3>FS1 & FS2, $p < 0.0001$). *(FS1: May & June 2000; FS2: February & March 2001; FS3: December 2001-April 2002)*

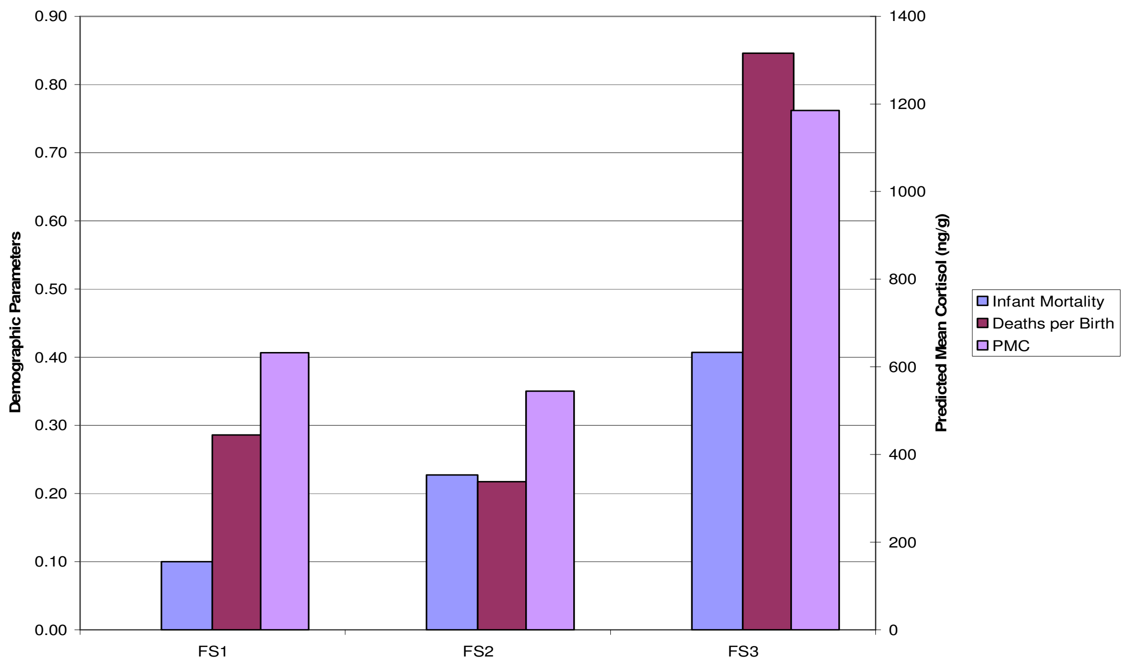


Figure 11. Infant mortality parameters for the six months preceding each field season. Predicted mean cortisol (PMC) for adult females closely follows increases in infant mortality in FS3. *(FS1: May & June 2000; FS2: February & March 2001; FS3: December 2001-April 2002)*

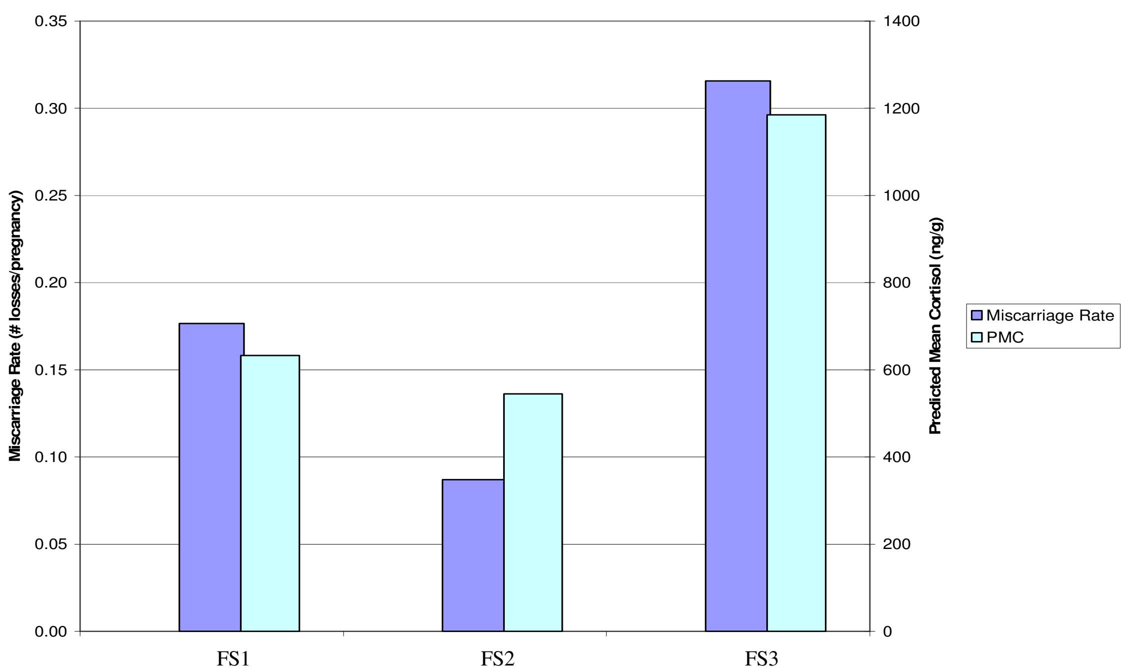


Figure 12. Miscarriage rates for the six months preceding each field season. Predicted mean cortisol (PMC) for adult females closely follows increases in miscarriage rates in FS3. *(FS1: May & June 2000; FS2: February & March 2001; FS3: December 2001-April 2002)*

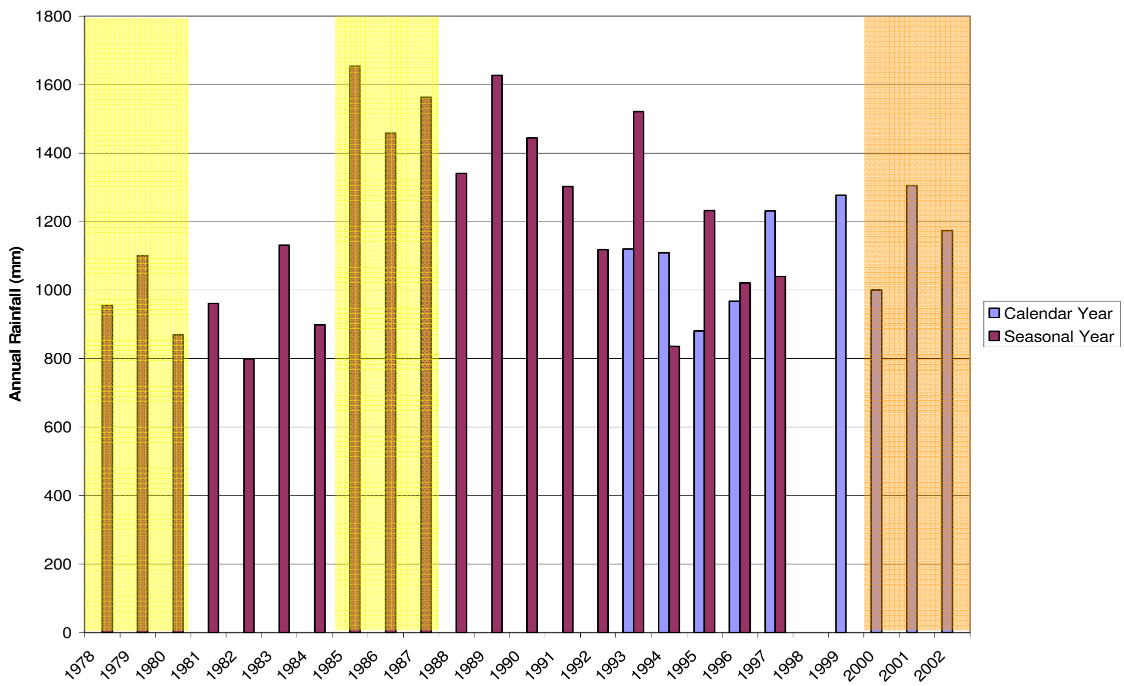


Figure 13. Long-term annual rainfall from Gombe National Park from 1978-2002. No data are available for 1998. Blue bars (1993-2002) represent annual rainfall for the calendar year, as discussed above. Purple bars (1978-1997) represent annual rainfall for the seasonal year (wet+dry, Oct-Sept); these data were collected by Gombe Stream Research Centre staff. Areas shaded in yellow highlight previous 3-year periods of ecological perturbation (defined on p. 67).

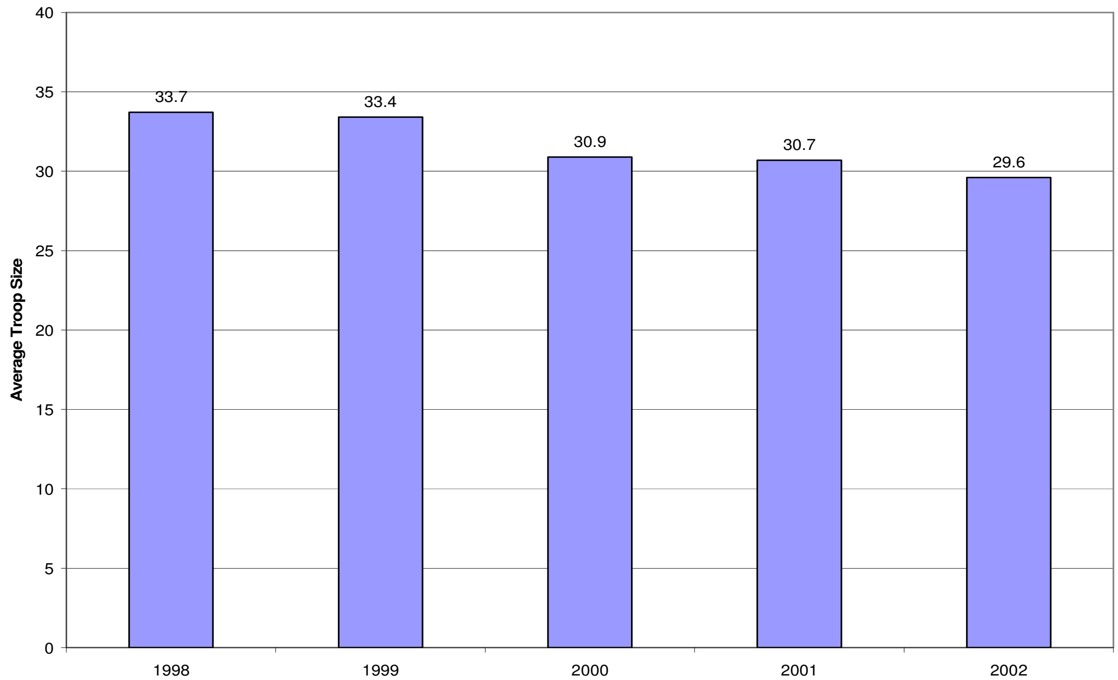


Figure 14. Population-wide yearly average troop size from 1998-2002.

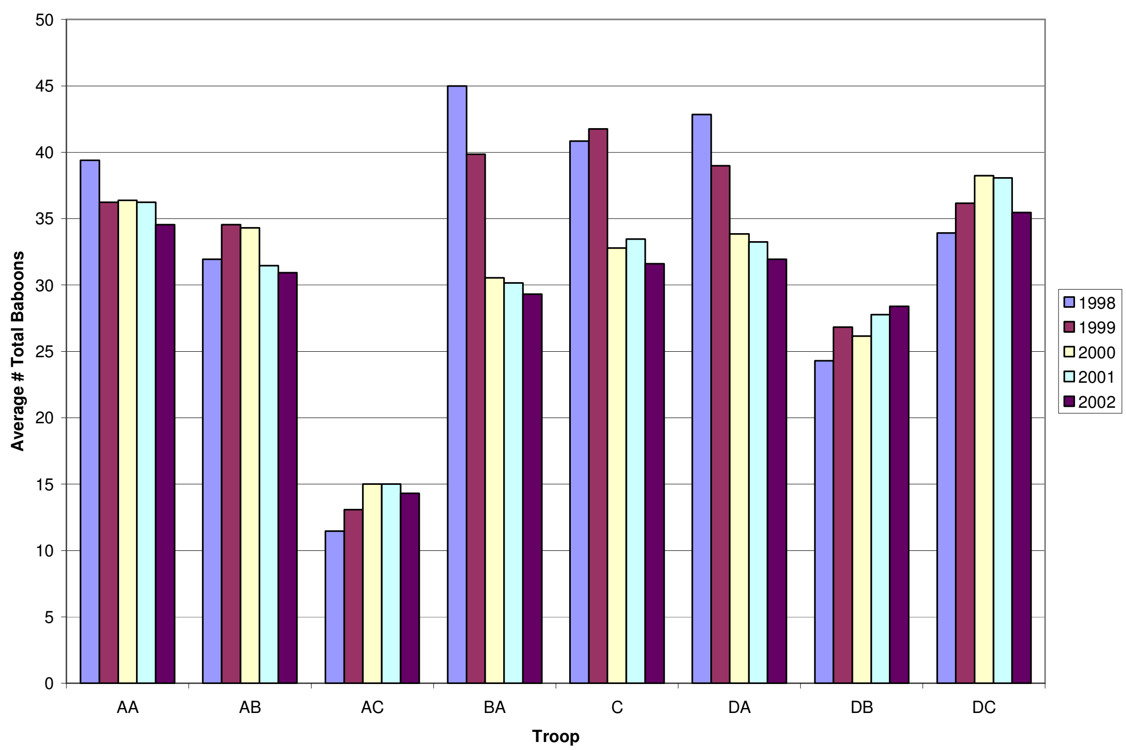


Figure 15. Average yearly troop size for the eight study troops from 1998-2002.

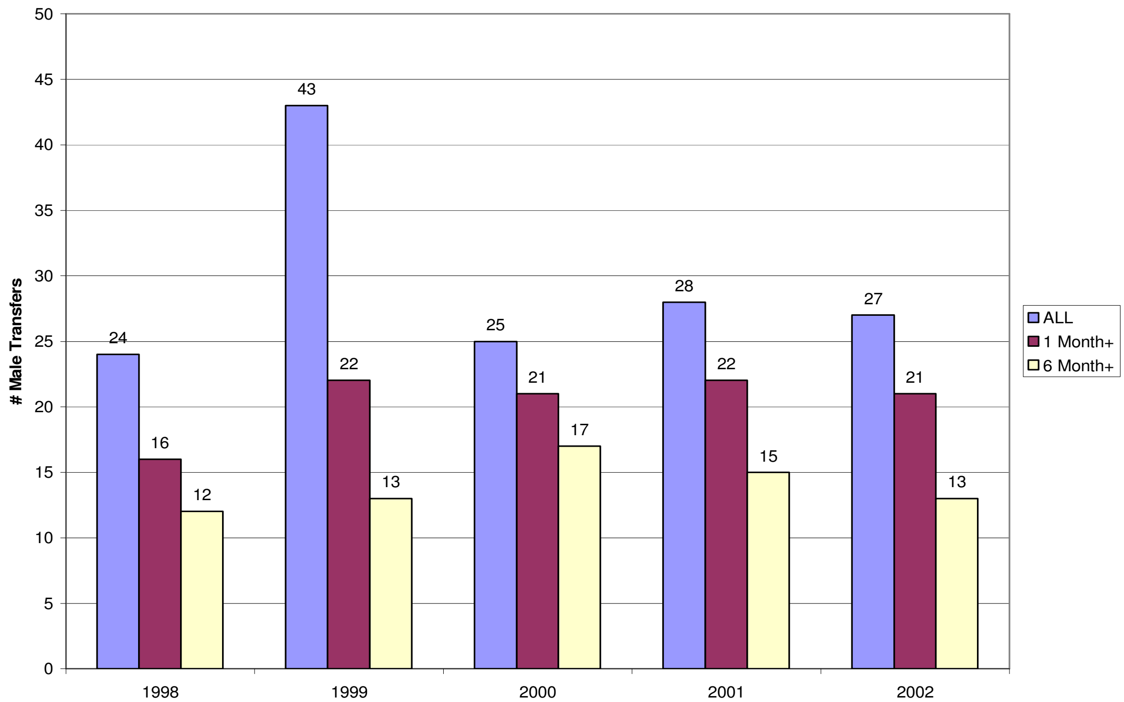


Figure 16. Total yearly transfers for the eight study troops. Transfers are separated by the tenure of the transferring males. The blue columns indicate all transfers, the red those that stay for 1 month or more, and the white those that stay for six months or more. The difference between blue and red indicates all short-term transfers.

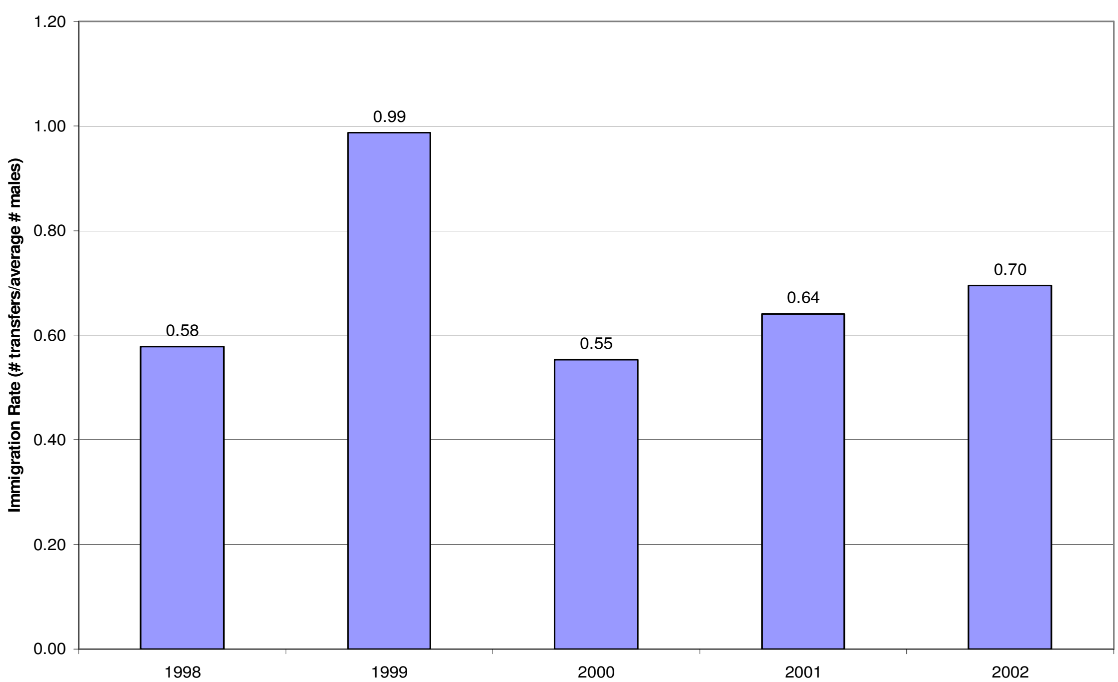
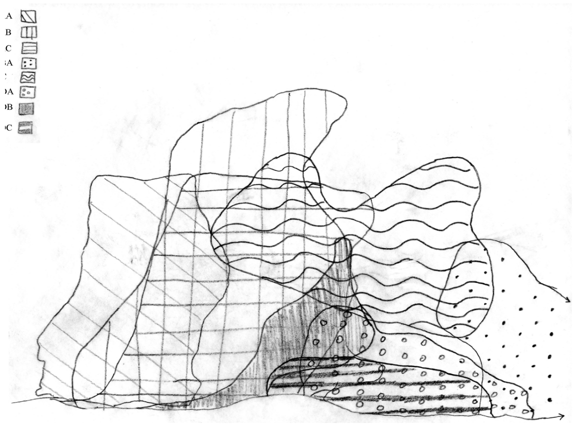
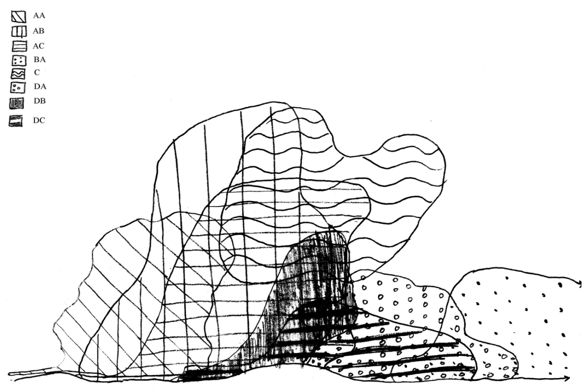


Figure 17. Population-wide immigration rates for 1998-2002.

a.)



b.)



c.)

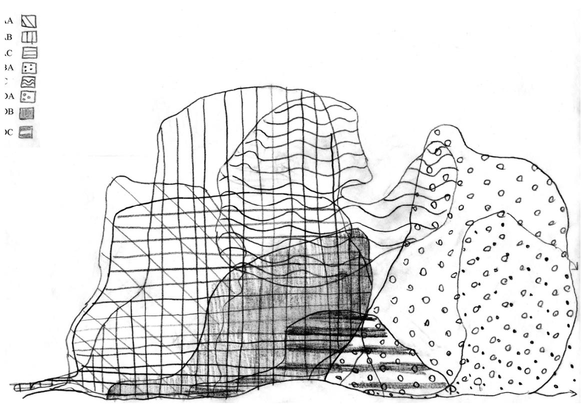


Figure 18. Monthly home range maps before and after resource loss. These maps represent one month's observed ranging patterns for each of the eight study troops **a.)** before the *dagaa* removal (1997), **b.)** after the *dagaa* removal (1999), and **c.)** at the approximate onset of the profound stress response (2001). The majority of maps are from July; however, due to a change in data collection, the most recent available maps were used for troops AA, DB, and DC. Even when these three maps are excluded, the increase in overlap is clear.

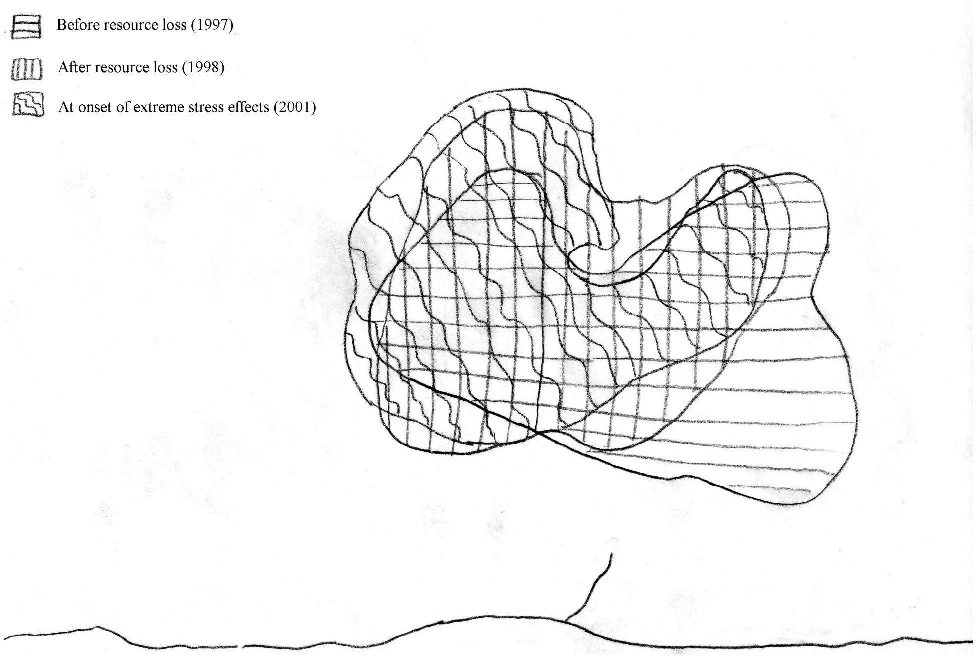


Figure 19. Monthly home range maps before and after resource loss for C troop. This map\ represent observed ranging patterns for C troop, the only troop not directly affected by the *dagaa* removal, in 1997, 1999, and 2001.

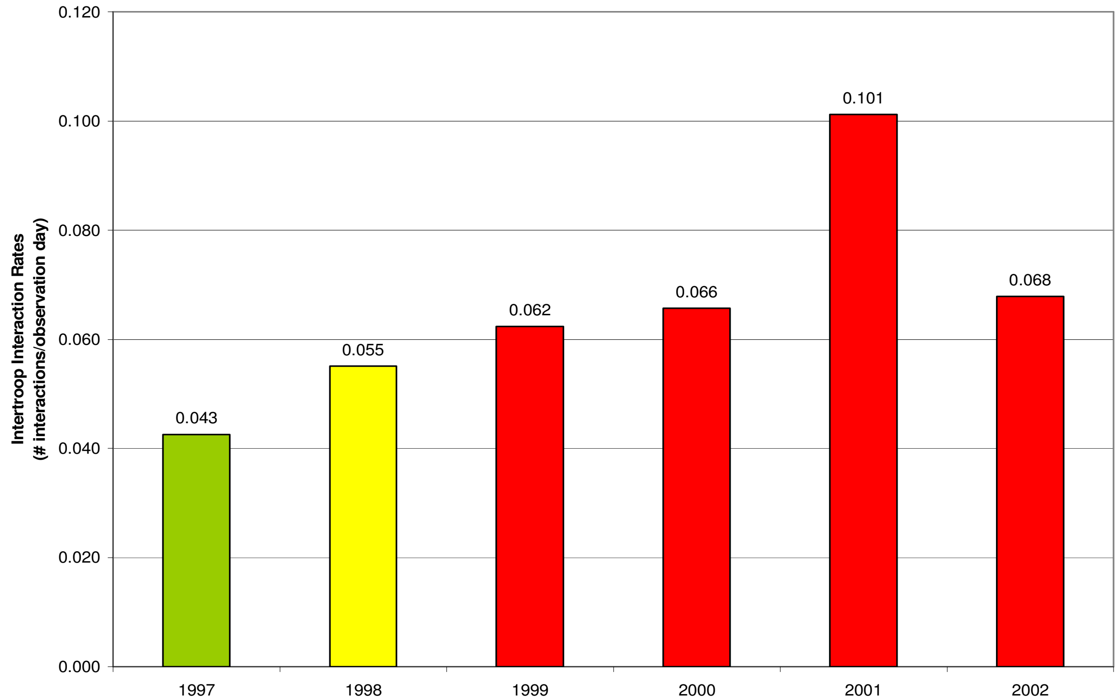


Figure 20. Population-wide inter-troop interaction rates (1997-2002). Green indicates the last full year before the *dagaa* removal, yellow the year of the removal, and red the years following.

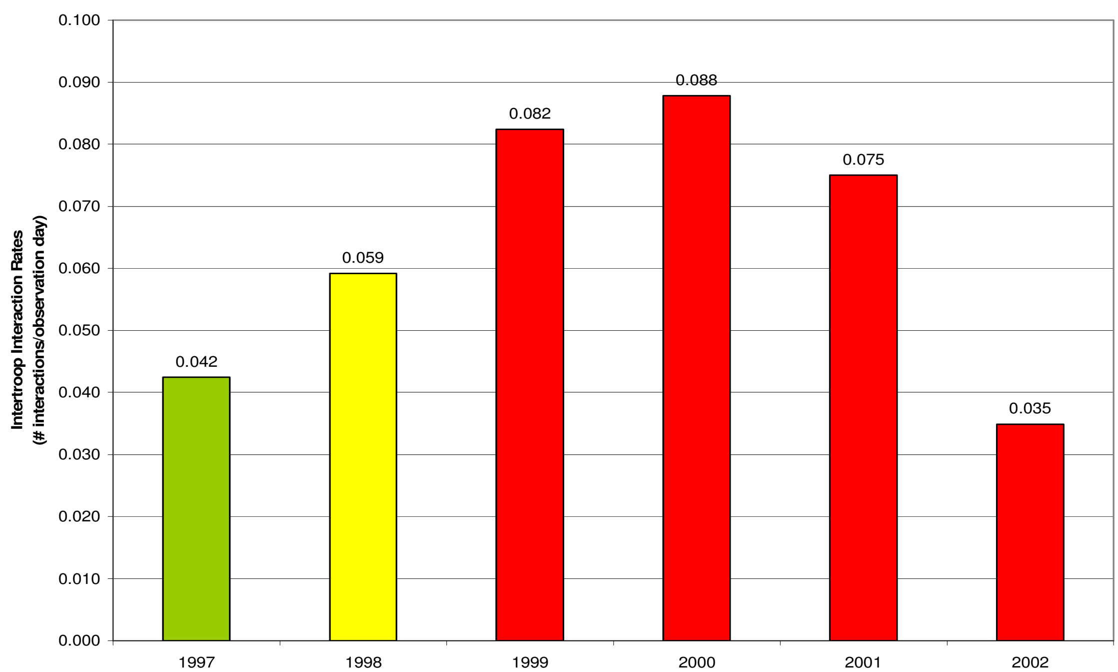


Figure 21. Troop C inter-troop interaction rates. Green indicates the last full year before the *dagaa* removal, yellow the year of the removal, and red the years following.

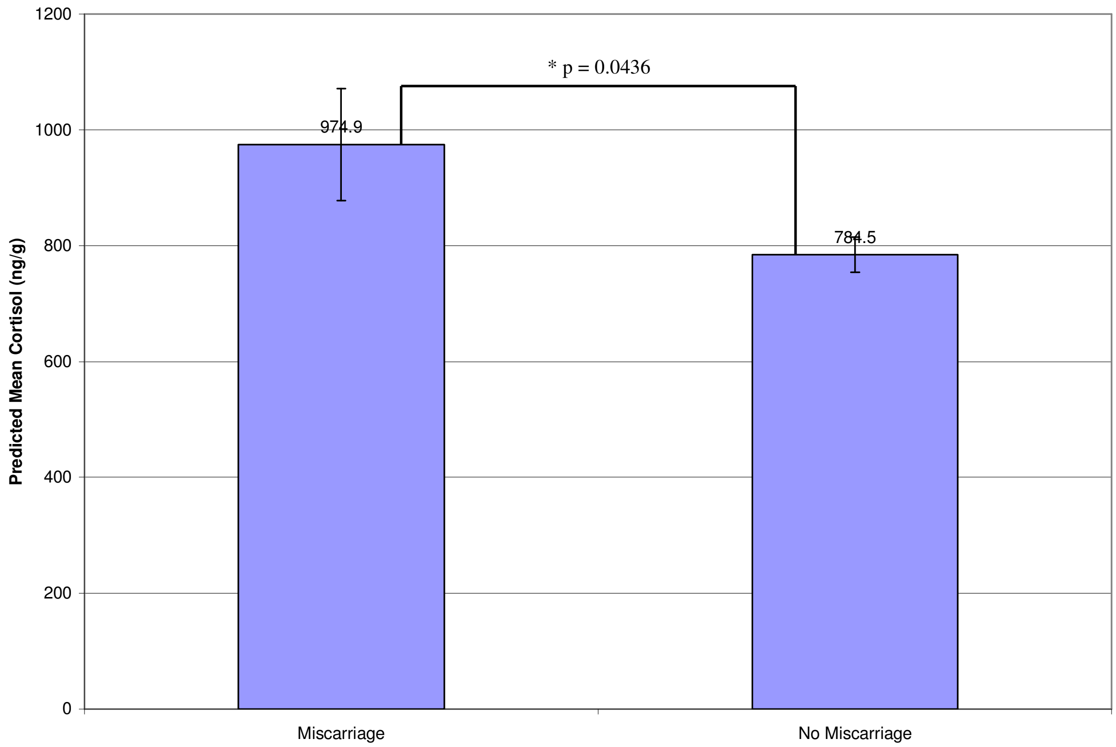


Figure 22. Predicted mean cortisol and incidents of miscarriage. Females who suffered one or more miscarriage during a period of severe ecological stress are compared to those who did not miscarry.

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CHAPTER 3:

STRESSORS, COPING STRATEGIES, AND THE EVOLUTION OF PRIMATE SOCIALITY

Abstract

Social circumstances such as rank and access to social support have been associated with differential exposure to the physiological costs of stress. In this chapter, I investigated the social costs and coping strategies associated with the philopatric and matrilineal society characteristic of female Old World monkeys. I used cortisol to assess how the facets of a wild female baboon's social landscape affect her exposure to stressors. I also looked for evidence of coping strategies that female primates might implement in order to minimize these social costs. In adult female baboons, neither rank nor access to social support is correlated with exposure to cortisol under normal circumstances. Further analyses suggest that exposure to stress in adolescence may promote the development of rank-related coping strategies that minimize rank-related stress in adulthood. While there is no direct relationship between more close female kin and lower stress, this is likely in part because friendships are so valuable that females without kin will cultivate friendships with unrelated females. Maternal rank and access to social support do significantly influence an adolescent female's exposure to cortisol. The daughters of high-ranking females are at lower risk for stress-inducing social conditions than the daughters of low-ranking females, and a number of indicators of social support can also notably decrease exposure to the physiological costs of stress. Even under the auspices of female philopatry and the presumed benefits of proximity to kin, the presence of female relatives can carry physiological costs for orphaned females and the daughters of low-ranking females.

Introduction

In Chapter 2, I developed a new approach to understanding the vertebrate stress response system, its adaptive role in times of crisis, and its evolutionary history. I also suggested that high cortisol levels can serve to identify and quantify stressors, and cortisol may also be useful for identifying coping strategies. By measuring cortisol to elucidate costs and potential coping strategies, we may be able to develop a more complete picture of the selective pressures and adaptive traits that have driven vertebrate evolution. In this chapter, I will use this approach to investigate the social evolution of primates. I use cortisol to determine the physiological costs associated with an individual's demographic status. I also consider social factors that may mitigate an animal's physiological response to stressors.

I. Understanding the evolution of primate sociality

Primates offer a fascinating opportunity for evolutionary biologists to expand our understanding of the processes and circumstances that have led to the evolution of a diverse set of social systems and social behaviors. In order to unravel the complex evolutionary history of primate sociality, we must strive to fully understand how the facets of a given individual's social landscape shape her experiences and ultimately influence her lifetime reproductive success. In this study, I focus on the philopatric and matrilineal society characteristic of female Old World monkeys.

II. Using cortisol to identify social stressors

Because animals facing suboptimal social conditions will activate the physiological stress response system, in turn elevating cortisol levels and increasing long-term risk of stress-related pathologies, many studies have used cortisol as a proxy measure for identifying physiological costs. The most widely studied characteristic is social status, and a variety of studies have attempted to quantify the relative costs associated with individuals with high or low rank (e.g. Sapolsky, 1982; Sapolsky & Ray, 1989; van Shaik et al., 1991; Sapolsky et al., 1997; Barrett et al., 2002; Lynch et al., 2002; Cavigelli et al., 2003;

Weingrill et al., 2004; Beehner et al., 2005; Bergman et al., 2005; Engh et al., 2006b). Other social conditions that carry known stress-related costs for primates include social isolation (Sapolsky et al., 1997; Beehner et al., 2005), destabilized social status (Sapolsky, 1983), exposure to novel social partners (Alberts et al., 1992; Cristobal-Azkarate et al., 2007), suboptimal group size or composition (Pride, 2005; Beehner et al., 2006; Chapman et al., 2007), and mating behaviors (Barrett et al., 2002; Lynch et al., 2002; Bergman et al., 2005). Cortisol has also been used to study trajectories of stress-related costs across development (e.g. adolescence, Gesquiere et al., 2005) or when social conditions change (e.g. after the loss of a social companion, Engh et al., 2006a).

Such studies assess the physiological costs associated with an animal's social experiences. Traditionally, correlation with cortisol has been considered to be an indicator of considerable selective pressure. Low rank, for example, can carry such physiological costs (Sapolsky, 1982; van Shaik et al., 1991; for review, see Abbott et al., 2003); these costs may then promote the development of agonistic traits such as aggressive behavior or body size that might improve an individual's social status. This traditional approach often focused primarily on avoiding stressful states. A more complete approach, however, can also use differential patterns of cortisol exposure to consider adaptive strategies that an individual might employ in the face of unavoidable social stressors.

III. Using cortisol to identify social coping strategies

A lack of identifiable physiological costs in the face of a known challenge may indicate two things: the challenge may not provoke a classic stress response (i.e. difficulty is not always indicative of a physiological stressor; Cherel et al., 1988), or the animal has access to coping strategies that effectively negate the physiological response prompted by the stressor (Wingfield & Kitaysky, 2002). For example, many of the anxiety behaviors associated with a subordinate status may help an individual avoid the need to initiate a stress response, thereby decreasing cortisol exposure (e.g. Nesse, 2000; see also discussion in Chapter 2). In male primates, certain behavioral patterns such as a propensity to displace aggression on a third party can significantly decrease a low-ranking individual's exposure to cortisol (Virgin & Sapolsky, 1997). Similar strategies have been identified for high-ranking males (Sapolsky & Ray, 1989). Comparably little work has been done to elucidate cortisol's ability to promote the type of adaptive strategies that may have evolved to minimize exposure to social and non-social stressors.

Social relationships play an important role in the lives of many primates, and strong social bonds have even been shown to improve lifetime fitness (Silk et al., 2003). Such relationships have also been shown to ameliorate an individual's response to an acute stressor (Biondi et al., 1986; Saltzman et al., 1991; Gust et al., 1996; Biondi & Picardi, 1999). Perhaps by developing strong social bonds, an animal can decrease the long-term risk of pathology. If so, the stress-relieving nature of social relationships may have played a significant role in promoting the evolution of primate sociality.

IV. What factors define a female baboon's social experiences?

In Old World primate societies, females are philopatric and form matrilineal hierarchies in which a female inherits the rank position directly below her mother. In this study, I measured cortisol levels to estimate the manifold ways that rank and alliances can affect the social experiences of a female baboon. By considering how an individual's social status and alliances correlate with this physiological variable, I investigated both socio-physiological costs and potentially protective strategies against social stress.

A. Rank

High-ranking females can experience shorter inter-birth intervals (Packer et al., 1995; Wasser et al., 1998; Cheney et al., 2004), increased infant survival (Packer et al., 1995; Cheney et al., 2004), and increased lifetime reproductive success (Johnson, 2003; Altmann & Alberts, 2005). Many of these effects are likely the result of nutritional benefits associated with priority access to limited foods (Lee, 1987; Abbott, 1987; Barton, 1993). The daughters of high-ranking females grow faster (Johnson, 2003; Cheney et al., 2004; Altmann & Alberts, 2005) and mature more quickly (Bercovitch & Strum, 1993; Packer et al., 2000; Altmann & Alberts, 2005; see also Chapter 2) than the daughters of low-ranking females, probably in part because the mother's rank secures access to superior resources (Altmann & Alberts, 2005). High rank also confers social benefits, as dominant females face little harassment (Abbott, 1987) and are more socially integrated than their low-ranking counterparts (Silk et al., 2003).

A low-ranking female and her daughters, on the other hand, face the opposite: extended inter-birth intervals, decreased infant survival, slower growth, and delayed maturation. Perhaps most notably, they will be more likely to face the costs associated with food shortages and nutritional insufficiencies. The only identified costs of high rank are an increased risk of miscarriage and compromised fertility (Packer et al., 1995) and higher rates of infanticide (Cheney et al., 2004). Because low-rank is associated with so many negative consequences, it seems logical to assume that it will result in elevated cortisol.

Previous research has uncovered no relationship between rank and cortisol exposure in female baboons (Weingrill et al., 2004; Beehner et al., 2005), and the work of Abbott et al. (2003) suggests that this is in part because of their access to social support. It is also possible that female baboons lack identifiable physiological costs because low-ranking females implement a suite of coping strategies (see Chapter 2).

B. Social Support

One of the most effective coping strategies available to social animals is their relationships with conspecifics. Social support is well known to reduce stress in humans and other primates (e.g. Biondi et al., 1986; Levine, 1993; Biondi & Picardi, 1999; Thorsteinsson & James, 1999). The presence of a peer can significantly decrease an individual's response to a stressor (Gust et al., 1996). In male baboons, those with more developed social relationships show decreased exposure to cortisol (Sapolsky et al., 1997).

Female baboons invest considerable time and energy in maintaining strong and durable affiliative relationships with other females. Females bias their attentions toward close kin, especially mothers, daughters and maternal sisters (Silk et al., 2003; Silk et al., 2006a; Silk et al., 2006b). Relationships with kin are stronger, more equitable, and more stable than those with non-kin (Silk et al., 2006a). Mothers and maternal kin play an instrumental role in the rank acquisition process and can significantly decrease age at first birth (Cheney et al., 2004). A more general measure of social integration has been shown to positively influence infant survival (Silk et al., 2003).

A female who loses an important ally will show a marked increase in cortisol (Engh et al., 2006a). At the same time, she will significantly increase affiliative behaviors and develop new relationships. As she does so, cortisol quickly drops to normal. Not only is the loss of a valuable social partner stressful (thus implying their presence reduced stress), new affiliations seem to abate this surge in cortisol and promote recovery from the loss

V. The social circumstances of a female baboon will influence her exposure to cortisol

During the juvenile stage, a female undergoes significant physiological and social changes as she reaches reproductive maturity and enters the adult hierarchy. The adult stage, in contrast, is one of relative social stability, punctuated only by the cycles of reproduction and motherhood. I used the same stage-differentiated models developed in Chapter 2 in order to investigate how rank and social support influence physiological costs in each major life history stage. By looking at how social factors correlate with cortisol for both adult and juvenile baboons, I can address a variety of questions pertaining to the social experiences of female baboons.

A. Wild adult female baboons

Aside from slight shifts caused by births and deaths, an adult female baboon holds the rank she acquired in adolescence (Hausfater et al., 1982; Gombe baboons, Packer et al., 1995). Her familial alliances are also extremely stable (Silk et al., 2006a). Because her social circumstances are largely constant, any social factor that might increase her exposure to cortisol can certainly be considered a chronic stressor.

RANK

In light of all the ways that low rank can make a female baboon's life more difficult, it is surprising that no study has yet identified a relationship between rank and cortisol (Weingrill et al., 2004; Beehner et al., 2005). Perhaps low-ranking females have developed a repertoire of coping strategies that allows them to alleviate and minimize the physiological implications of their suboptimal status. Because their adult rank is predictable from birth, they may even be able to anticipate low status and implement appropriate behavioral strategies prior to adulthood (see Chapter 2). If so, the Gombe females should also show no significant correlation between rank and cortisol.

In 2001, the entire Gombe baboon population faced an extreme food shortage. When resources become more limited, the benefits of high rank should amplify any rank-related difference in cortisol (Bercovitch & Strum, 1993; Cheney et al., 2004). A dominant female will have priority access to what little resources are available, and low-ranking females will be left with even less.

If low-ranking females have instead had greater experience with rank-related coping strategies, we would expect an entirely different pattern. When an entire population faces an extreme food shortage, low-ranking females should show fewer physiological repercussions than high-ranking females. In the case of the Gombe baboons, the loss of the *dagaa* would thus be expected to disproportionately raise cortisol levels in females with high social status.

SOCIAL SUPPORT

High-ranking females rely on close relatives to assist in attaining and maintaining rank, and their relationships with female kin have been shown to be of higher quality than those between low-ranking kin (Silk et al., 2003). The absence of such relationships might therefore be expected to disproportionately increase the cortisol release of high-ranking females. On the other hand, if social support can decrease stress, low-ranking females might have more to gain from relationships with their kin.

B. Wild juvenile female baboons

AGE

As a developing female baboon faces substantial physiological and social change, she is likely exposed to an increasing number of novel stressors. I therefore expect that maturing females will show rising cortisol levels. This pattern could be driven primarily by her physiological maturation (Dirks et al., 2002) and its associated stressors: the metabolic demands of reproduction, increased harassment by males. If physiological changes drive a positive cortisol trend, any age effect should be consistent across ranks, and there will be no significant interaction between age and maternal rank.

Alternatively, an identifiable age trend could be more directly tied to a young female's changing social circumstances. At roughly the age of physiological maturation, female baboons enter the adult hierarchy. A female who is destined to hold high rank will undergo a series of rank reversals until she reaches the position immediately below her mother. The daughters of low-ranking females will simply join their family at the bottom of the hierarchy. If this process of social change is inherently stressful, a female's maternal rank should affect her age-related trajectory of cortisol exposure; there will be a significant interaction between age and maternal rank.

MATERNAL RANK

Many of the known benefits of high rank are one generation removed. The daughter of a dominant female is more likely to survive infancy; she grows faster, grows larger, and reaches maturity at an earlier age. She enjoys the same priority access to superior resources as her mother, and she will likely face few food-related stressors. She is also likely to benefit from her mother's high-quality social alliances with close kin. Because of all of these potentially stress-reducing benefits, it seems probable that daughters of high ranking females will have decreased exposure to cortisol compared to daughters of low ranking females. As with the adult females, juvenile baboons facing ecological hardship would be expected to show disproportionately elevated cortisol.

SOCIAL SUPPORT

Social support is particularly valuable during periods of change and uncertainty (e.g. Gust et al., 1996). If social support can ameliorate stress, then an adolescent female baboon facing constant change might have much to gain from her relatives. The most critical stress-reducing role is that played by a mother (Silk et al., 2006b); her influence likely helps daughters avoid some stressors and mitigates the effects of others. Her absence leaves an immature female with few protections against stress. Maternal kin may also provide crucial support to a juvenile baboon; if so, a large social network could protect a young female against the physiological costs of stress.

Certainly these presumed benefits of kin are to be expected in a philopatric society, and the focus in the literature has been on identifying and classifying the benefits of living in a social group with one's kin. Little research has considered the negative side of a life lived with one's family, leaving one critical question yet to be asked. Is there ever an instance when one's family can become one's burden? This model will address this question by looking for instances in which a large network of kin is actually correlated with increased exposure to stressors and elevated levels of cortisol.

Methods

I. Study site and population

A. Gombe National Park, Tanzania.

Gombe National Park is located in Western Tanzania. Gombe is a small park of approximately 35km^2. The boundaries of the park are all ecologically discrete. The north and south are bounded by villages and deforested farm land. The eastern boundary is the rift escarpment (again bounded by farm land), and the western boundary is the shoreline of Lake Tanganyika. The park is characterized by a series of steep ridges rising from the lake to the escarpment; habitats range from thick riverine forests in the valleys to open grasslands on the ridges (Clutton-Brock & Gillett, 1979).

B. The Gombe Baboons

The subjects of this study are female olive baboons (*Papio cynocephalus anubis*). Numerous baboon troops reside within the boundaries of the park, and they can be found in any of its varied habitats. The baboons of nine of these troops have been continuously studied since 1967 (Packer, 1975; Ransom, 1981). The original two troops, Beach troop (also known as BA) and Camp troop (C), lived on the beach and forested areas of Kakombe Valley. B troop and its descendant troops have fissioned seven times, resulting in a total of 9 study troops. The overall range of this study population expanded considerably with the troop splits (personal communication, D. A. Collins). However, they are still centralized in the Kasakela and Kakombe valleys where they maintain flexible and overlapping territories and do not range near the park's boundaries. Two additional troops, LA and LC, range farther north in the Linda Valley; however, these troops were excluded from the present study because of unreliable data. The newest troop, BB, was also excluded for lack of data. Thus, eight troops (AA, AB, AC, BA, C, DA, DB, and DC) are represented in this study. All baboons were habituated to human observers, and all study subjects have been followed throughout their lives.

All females over the age of three at the start of sample collection (n=106) were studied. As juveniles and adults represent significantly different life history stages with vastly different stressors, I analyzed these two datasets separately. Any female who was three or more and had not had a first swelling at the start of the study was considered a juvenile (n=22). All other females were considered adults (n=84).

II. Demographic and behavioral data collection

Trained Tanzanian field assistants follow these baboons and collect data on a near-daily basis (data collection through the time of this study varied between 5-7 days/week) with rare breaks in data collection of more than a few days. All baboons were individually recognizable and could be identified by at least two field assistants. Assistants collected daily attendance data and compiled monthly reports. Attendance data included births, deaths, reproductive status, consort pairs, wounds, and male transfers. Because of the completeness of data collected, major demographic and reproductive parameters were known: age, age at first swelling or first transfer, number of offspring, inter-birth intervals, etc. As all females were born during the long-term study, maternal relationships are also known (daughter, mother, sister, grandmother, aunt, cousin, non-kin).

Monthly reports provide details on inter-troop interactions, illnesses, injuries, shifts in dominance hierarchies, and details of major events (e.g. troop splits). In addition, check-sheets summarize dominance data based on daily ad lib sampling of supplantations and dyadic aggressive interactions. Yearly ranks are based on the number of individuals dominated. Confounding rank reversals were resolved by incorporating the weighted ranks of each opponent. Monthly ranging maps are also provided for each troop.

III. Hormonal collection, preservation, and analysis

A. Sampling protocol and sample collection

Cortisol samples were collected in three discrete field seasons, between January 2000 and April 2002 (FS1: May & June 2000; FS2: February & March 2001; FS3: December 2001-April 2002). Fecal samples were collected opportunistically from all study subjects by field assistants who had been extensively trained in collection protocols. Each sample was collected only if the identified baboon was seen in the process of defecating. Any feces that could not be conclusively attributed to a female (e.g. more than one sample was found in close proximity) or were in poor condition (e.g. fell a great distance) were excluded. Three or more fecal samples were collected from most subjects during each field season with an average of 9.4 total samples per female (adults: n=834; juveniles: n=160). Only 7 females were sampled five or fewer times; four of these were females who died during the course of this study and three were juveniles. As few samples were collected from the juveniles of C troop, this troop was excluded from the juvenile dataset.

Samples were collected in small plastic zipper bags and were immediately mixed by manipulating them inside the plastic bag. Samples were collected at any time between 07:23 and 19:25; previous studies suggest that diurnal fluctuations in baboon fecal cortisol levels are minimal (Weingrill et al., 2004). Specimens were transported to the field lab at the end of each morning or afternoon shift.

B. Field preservation and transportation

Samples were remixed inside the plastic bag. Approximately 0.5g of the mixed samples were placed in pre-weighed 20 mL HDPE scintillation vials and covered with 5mL of 90% ethanol. Each sample was then shaken by hand for a minimum of two minutes until samples were adequately suspended in the preservative. The lag time between collection and preservation (ethanol preservation lag) was recorded for all samples. This lag ranged from 1 minute to a rare extreme of 8 hours, with an average time of 2:36 spent at ambient temperature without preservative. Once processed, samples were tightly sealed and wrapped in parafilm to prevent ethanol leaks. They were then kept in a cool, dark room for the duration of the field season.

Samples were transported under CDC permit (number 2001-05-246) to the St. Louis Zoo, St. Louis, Missouri. Upon arrival, samples were checked for signs of leaking and discarded if necessary. Samples were then analyzed immediately or frozen in the ethanol preservative in a -20˚C freezer. The lag between ethanol preservation and analysis or freezing (freeze lag) was also recorded for all samples. Samples were stored at ambient temperature in ethanol for an average of 76 days with a minimum of 15 days and a maximum of 181 days.

C. Steroid extraction

As samples had been stored in the extraction solute for a minimum of 15 days and experienced vigorous jostling during transport, I followed a simplified extraction protocol. Samples were placed on a shaker overnight at 200 RPM, after which they were allowed to settle for approximately one hour. The extract portion of the samples was then decanted into 12x75 polypropylene tubes, leaving behind all fecal material, and then centrifuged at 4,000 RPM for one hour. The supernatant was then poured into a cryovial and analyzed immediately.

The pellets remaining in the polypropylene tubes were resuspended in ethanol and poured back into the original scintillation vial. All vials were then left open overnight in a vented oven at 100ºC. When samples were completely dry, the vials were reweighed. By subtracting their initial weight from their weight with dry feces I could determine the dry weight of the feces.

D. Radioactive immunoassay

All samples were analyzed using radioactive immunoassay (RIA) in the Endocrinology Lab at the St. Louis Zoo. Standard cortisol RIA kits were used (Cortisol ACTIVE-RIA ^{125}I Kit, Diagnostic Systems Laboratories, Webster, Texas) and all samples were assayed in duplicate; paired samples with variation greater than 15% were reanalyzed. Hormone concentrations were corrected for volume of added solvent and also dry weight of feces in order to control for differences in moisture content (Wasser et al., 1993) and are therefore reported in ng/g. Although an antibody against cortisol was used in these assays, fecal glucocorticoids exist as a mixture of metabolites, and while "fecal cortisol" is used for convenience, these results should be understood to refer to fecal glucocorticoid metabolites.

E. Outlier analyses

Two samples were excluded due to lack of replicability either within or between batches. Paired aliquots of these two samples were run seven times at three different dilutions. Each batch showed extremely low replicability, and final cortisol values ranged from 4862-17250 ng/g for one and 5023-15188 ng/g for the other.

Fecal sampling following the protocol used in this study can show unreliably high or low values (Millspaugh & Washburn, 2004). On rare occasions, dry fecal weight can have very little to do with the actual weight of the feces. For example, samples may contain more or less indigestible material (such as seeds) which can significantly skew final hormone concentration measurements, resulting in extreme high or extreme low values. In order to remove any such values from the dataset, I created a set of exclusion criteria. I first identified any samples that were unusual for a given baboon and that were more than four standard deviations from the overall mean. I then looked at this subset of data points and excluded those that showed extreme high or low dry fecal weights as well as extreme high or low raw hormone values (prior to dry fecal weight correction). Four samples were identified as having extremely low fecal weights (<0.03 g) and extremely high cortisol and testosterone values (top 1%) and were therefore excluded based on these criteria.

IV. Model development

Two statistical models were developed to investigate a variety of potential chronic stressors, including the loss of the *dagaa*. The first model considered factors likely to affect cortisol levels of adult females and the second model focused on juvenile females.

A. Adult female predictors

All main effects and interactions tested in this model are identified in Table 1. As all females were born during the 35-year study period, age was accurate to within a few days and was treated as a linear variable. Reproductive status was determined for each individual sample based on observation data for the date of collection. The status classes were pregnant, lactating, flat (indicating a non-fertile phase), swelling, on consortship, or menstruating.

Adult female relatives provide most of the known social support for female baboons (Silk et al., 2006a; Silk et al., 2006b). I included a count of the female's mother, adult daughters, maternal grandmother, maternal adult sisters, aunts, and nieces who were alive and members of the same troop on the day a given sample was collected. This predictor was treated as a linear variable.

The rank of each female was determined as both a continuous variable (based on proportion of females dominated) and as a number of possible categorical variables (each with slight adjustments to the "breaks" between high, medium, and low, none of which affected the basic results of the model). Whenever possible, a rank specific to the year of sample collection was included for each female; however, there were gaps in the data. For example, rank analyses were not collected for troop C for any of the three study years. Because of the overwhelming stability of female baboon hierarchies (Hausfater et al., 1982; Packer et al., 1995), I recreated the hierarchy during this period by considering each female's last known rank, correcting for females who had died, and adjusting for maturing females. As monthly reports accurately report most reversals in the female hierarchy, I was able to use this information to verify that there were no unusual changes in rank not represented in available rank data. As the significant results of the model were the same for both continuous and categorical ranks, I chose to focus analyses on the categorical data. This, in part, allowed me to control for minor rank changes that may have been missed in the extrapolated troop rank determinations. Categorical ranks are also easier to interpret graphically and are more widely used in the baboon literature.

Each female was also identified by the troop in which she resided at the time of sampling, and each sample was classified according to the field season in which it was collected. I also considered season as a preliminary predictor, and each sample was categorized as collected in either the dry or wet season as determined by annual rainfall patterns at the time of sampling (generally Oct-May, wet; Jun-Oct dry; Packer et al, 1995). All biologically relevant two-way interaction terms were also considered in the initial model.

B. Juvenile female predictors

Most of the parameters for the juvenile females were determined following the same methods presented here for the adult females (see Table 1 for a complete list of predictors). In addition, the status of each female's mother (alive or dead) was determined for each sample at the time of collection. Maternal rank was also considered as a categorical variable (mother in the top third of the hierarchy=high; middle third=middle; bottom third=low) as outlined above. Again, all relevant two-way interactions were included in the development of the initial juvenile model.

V. Statistical analyses

I used SAS version 9.1 (SAS Institute, Inc, Cary, NC) and Proc Mixed to develop models in order to identify predictors that correlate with cortisol levels. For both the adult and juvenile data sets, the models were initially developed as general linear mixed models (GLMM) with the random effect *baboon* and the predictors identified in Table 1. A random effect was considered in order to account for the correlation among repeated cortisol measures from the same baboon. If the model results were not significantly different under a general linear model (GLM), I subsequently dropped this random statement from the model. The same statistical program was used for all further analyses including LS Means and Tukey-Kramer adjusted group mean comparisons.

Results

I. Model development: predictors of chronic stress

WILD ADULT FEMALE BABOONS

The statistical model identified variables associated with elevated exposure to (or ability to ameliorate) glucocorticoid levels in adult female baboons. This model was initially developed as a general linear mixed model (GLMM), with the main effects and interactions detailed above. The random effect *baboon* was found to be insignificant and was subsequently dropped from the model indicating a low, non-significant correlation among samples from each baboon. Only the results of the general linear model (GLM) are reported below, and they can be seen in full in Table 2. I then developed a reduced model based on stepwise removal of insignificant interactions and main effects. The results of this reduced model's tests of fixed effects can be seen in Table 3.

WILD JUVENILE FEMALE BABOONS

As adolescence represents a vastly different life history stage for female baboons, with different states and stressors, I developed a separate model for juvenile females. In this model, the random effect *baboon* was found to be significant, indicating a significant correlation among samples from each baboon. Therefore, all results for juvenile females are from GLMM with the main effects and interactions detailed above. The results of this complete model can be found in Table 4. I developed a reduced juvenile cortisol model based on the same stepwise process described for the adults. The results of this reduced model's tests of fixed effects can be seen in Table 5.

II. How do the social characteristics of a female baboon influence her exposure to cortisol?

A. Which social factors influence cortisol levels in adult females?

RANK

This model shows that rank does not significantly affect cortisol levels in the adult females of Gombe (GLM: $F_{2, 751}$=0.05, n=834, p=0.9485). Treating rank as a continuous variable gives a nearly identical model, and flat LOESS fit plots for cortisol and continuous rank further support this negative result. The lack of a rank effect was also consistent across all eight study troops (GLM: $F_{14, 751}$=0.88, n=834, p=0. 5788). There was also no significant interaction with family size (GLM: $F_{2, 751}$=0.74, n=834, p=0.4288) indicating, for example, that a large family did not mitigate any stress that might be associated with low rank.

There was, however, a notable change during the period when the baboons were suffering the worst effects of the *dagaa* loss (FS3: December 2001-April 2002; GLM: $F_{4, 796}$=2.74, n=834, p=0.0279). During this period of extreme stress, high-ranking females showed significantly higher cortisol levels than low-ranking females (Figure 1; Tukey-Kramer: p = 0.0094). The effect of this stressor appears to have caused an overall rise in cortisol that disproportionately affects baboons at the top of the hierarchy.

Rank instability could constitute a stressor with a profound effect on females at the top of the hierarchy (male baboons, Sapolsky, 1983). Since two of the troops had gone through recent upheavals in female ranks (see Chapter 2), it was possible that the rank effect observed in FS3 might have been driven by the localized cortisol levels of one or a few of the troops. In my initial model, however, the interaction between rank and troop was insignificant (GLM: $F_{14, 751}$=0.88, n=834, p=0. 5788), indicating that the effect of rank was not significantly different from troop to troop. To be certain, I ran the full model with a three-way interaction between rank, troop, and field season to identify whether there was any statistical evidence that these effects were specific to a subset of troops. This interaction was also found to be insignificant (GLM: $F_{42, 723}$=1.15, n=834, p=0.2711). Therefore, the increased stress of high-ranking females appears to be a general phenomenon in FS3 and not specific to any one of the troops.

SOCIAL SUPPORT

The degree of social support available to an adult female baboon had no effect on her cortisol levels (GLM: $F_{1, 751}$=0.37, n=834, p=0.5409). There was no interaction with rank (GLM: $F_{2, 751}$=0.85, n=834, p=0.4288), and family support was not significantly more valuable to females facing a severe chronic stressor (GLM: $F_{2, 751}$=0.37, n=834, p=0.6912). I ran a number of additional analyses that considered total adult female relatives as a categorical variable, the presence of adult female relatives as a yes/no variable, and only the number of adult sisters; none of these was significantly correlated with cortisol levels. Only the stress-reducing power of a mother approached significance; those adult females with living mothers showing lower predicted mean cortisol than those without (GLM: $F_{1, 795}$=0.05, n=834, p=0.0549).

B. Which social factors influence cortisol levels in juvenile females?

AGE

As the juvenile baboons progressed through the crucial adolescent stage they showed a significant trend of increasing cortisol with age (GLMM: $F_{1, 110}$=8.03, n=160, p=0.0055). More importantly, age interacted significantly with maternal rank, and this interaction will be addressed below. During the juvenile period, young females undergo physiological and social maturation as they reach menarche and enter the hierarchy. These study females reached sexual maturity (indicated by onset of sexual swellings) at approximately 6.12 years, an age at which cortisol levels had increased substantially. Young females generally begin rank ascension at roughly the time they reach sexual maturity.

MATERNAL RANK

Maternal rank was found to significantly affect a young female's exposure to cortisol via its interactions with field season, age, and number of female relatives, all of which carry disproportionately adverse effects for the daughters of low ranking females.

While no maternal-rank-related differences were observed in FS1 or FS2, an effect was apparent in FS3 (Figure 2). In this time of ecological crisis, there were significant differences between ranks (LSmeans, slice=field season, FS3: p=0.0189), with low-ranking females experiencing markedly elevated cortisol levels. This pattern is notably the opposite of that observed for adult females.

The stress-relieving effects of high maternal rank also affect the relationship between age and cortisol levels (GLMM: $F_{2, 110}$=6.00, n=160, p=0.0034). Daughters of high-ranking females show a dramatic and significant decrease in cortisol levels as they approach maturity; cortisol levels drop approximately 3088 ng/g per year of age (95% CI: -4902.2, -1272.3). In contrast, daughters of low- and middle-ranking females show no significant relationship between age and cortisol (middle, slope=-26.68, 95% CI: -843.86, 790.49; low, slope=164.30, 95% CI: -291.53, 620.12). As a result of this interaction, high-ranking females show decreased exposure to cortisol during the juvenile period and are essentially protected from any stress-inducing effects related to maturation.

SOCIAL SUPPORT

This model offers substantial evidence that social support can significantly influence a juvenile female baboon's exposure to cortisol. A number of key factors that represent familial support create a fairly complex picture of the role social relationships can play during this critical adolescent stage.

Mothers can provide significant stress relief for their daughters, as the presence of a living mother interacts with the number of adult female relatives. When a mother is present, each additional female relative can decrease a juvenile's exposure to cortisol by approximately 2978 ng/g (95% CI: -3914.7, -2041.12). . The benefits of female relatives do not, however, extend to young baboons that have lost their mothers. For an orphaned female, additional kin is correlated with significantly elevated stress by a factor of 1350 ng/g per kin (95% CI: 600.59, 2098.60). It appears that mothers provide critical social support by coordinating the beneficial effects of aunts and sisters.

The influence that family has on the cortisol levels of a young baboon is also dependent upon her mother's rank (GLMM: $F_{2, 110}=22.36$, $n=160$, $p<0.0001$). The predicted relationship—more social support, less cortisol—fits for females who will likely be high- or middle-ranking during adulthood. The slopes for both high and middle ranking females are significantly negative; for each additional female relative, cortisol drops approximately 2490 ng/g (95% CI: -3222.11, -1758.49) or 1953 ng/g (95% CI: -3104.34, -802.34) respectively. However, there is a striking positive correlation between number of adult female relatives and cortisol levels for those likely to inherit places at the bottom of the hierarchy, with an increase of 2001 ng/g (95% CI: 1162.31, 2840.02) per additional female relative. Social support for high- and middle-ranking baboons clearly helps mitigate chronic stress during this crucial maturation and rank-acquisition phase. However, adult female relatives appear to be a stress-inducing burden for low ranking baboons. This further suggests that social support can play a critical role during this adolescent phase by minimizing stress exposure.

Discussion

I. Social rank and the evolution of coping strategies

High rank is generally neither a physiological boon nor burden for an adult female baboon, as rank did not correlate with cortisol exposure. This finding parallels studies completed on female baboons in other populations (Weingrill et al., 2004; Beehner et al., 2005), thus this relationship appears to be the norm for wild females. Both previous studies cite the work of Abbott et al. (2003), which identified key factors that might decrease the stress related to low rank. They suggest that because female baboons have access to social support and because rank's effects are relatively subtle, no correlation is expected. Male baboons, in contrast, differ from females on both of these criteria and show a profound physiological cost for low status (Sapolsky, 1982).

This result is still difficult to reconcile when so many ecological and social benefits have been associated with high rank. Two additional explanations have been proposed: high rank has physiological costs that approximately equal those incurred by low-ranking females; or none of the negative repercussions of low rank cause physiological stress. It has been suggested that high rank does carry physiological costs (Packer et al., 1995). While this effect is not consistent across all populations (Altmann et al., 1995), it is strong at Gombe. Perhaps, as is the case here, the costs associated with dominance are not consistent across time, in which case they might also vary considerably across populations. A large-scale comparative approach which aims to classify differences in a variety of physiological costs across baboon populations could be extremely revealing. It might allow us to identify ecological and social factors that cause differences in socio-physiological costs and could provide valuable insight into the evolution of baboon sociality.

In this case, however, it seems unlikely that low and high-ranking females experience distinct classes of stressors that, under normal conditions, result in indistinguishable cortisol profiles. No studies have specifically pinpointed increased cortisol as a cost of dominance in female baboons. In contrast, many of the detrimental effects of low rank are known to result in increased cortisol levels—food deprivation, for example (e.g. Sabatino et al., 1991; Urbanski et al., 2004). It therefore seems unlikely that low-ranking females who suffer such challenges would fail to show elevated cortisol unless they are able to effectively bypass these obstacles.

The loss of the *dagaa* provided an opportunity to test whether low-ranking females implement coping strategies that, under normal circumstances, allow them to overcome their rank-related food challenges. According to the traditional approach, high-ranking females should be less affected by food deprivation than low-ranking females, and they should show a concomitantly smaller rise in cortisol. Alternatively, high-ranking females might be more affected because they lack appropriate coping strategies. The results presented here clearly support the latter, as high-ranking females have significantly higher cortisol than low-ranking females during times of ecological crisis. These results suggest that when hardship strikes, a life of relative privilege leaves an individual with few defenses. In contrast, a life of small adversities may more adequately prepare her to handle a dramatic challenge.

It is also notable that juvenile females show the opposite effect. Again, under normal conditions there are no rank-related differences; however, during this period of hardship the daughters of low-ranking females experienced significantly elevated cortisol. Daughters of low-ranking females were also more likely to face physiological costs associated with their social circumstances. The daughters of low- and middle-ranking females show elevated stress with age, and in particular they experience significantly high levels of cortisol during the critical rank acquisition stage. In addition, the daughters of low-ranking females do not benefit from the social support provided by relatives; instead, female kin actually increase their exposure to physiological costs (see discussion below). Low-ranking adults show no such rank-related costs.

It is unlikely that all the factors that cause elevated cortisol in young low-ranking females promptly disappear in adulthood; rather, it is more likely that adults can better alleviate these effects. A growing body of evidence suggests that early exposure to cortisol can significantly change behavioral patterns expressed in adulthood (e.g. Clarke & Schneider, 1993; Badyaev, 2005). Perhaps early social and physiological cues are a forecast of future experiences, and the activation of appropriate coping strategies is enough to nullify the physiological costs of low rank. This finding hints at an epigenetic interpretation which deserves more thorough treatment than can be offered here.

II. The critical role of female affiliations in baboon society

The size of an adult female's familial network does not significantly decrease her exposure to cortisol. This is particularly surprising, as both female kin and social affiliations in general are known to be integral components of a female baboon's life. If any animal *should* show social buffering, it would be a philopatric female, and physiological benefits of close associations with female kin would help explain the evolution of this system.

The results of this study indicate that *more* relatives do not necessarily mean *more* stress relief, and that females do not face serious costs of social isolation. Recent research on female affiliations supports this interpretation. First, while female baboons do show a preference for female relatives, in their absence they will develop successful relationships with non-kin (Silk et al., 2003; Silk et al., 2006a; Silk et al., 2006b). Furthermore, it appears that a modest number of affiliations will suffice, as few females actively maintain a very large network and few if any females are truly isolated (Silk et al., 2006b). The literature also suggests that a female's relationships do alleviate physiological stress, as the loss of a close female relative results in a significant increase in cortisol (Engh et al., 2006a). However, this increase in cortisol is only transient, as females quickly increase affiliative behaviors and form new relationships that appear to replace the physiological and social benefits of their lost friend. In sum, female bonds do provide a social buffer to stress; in fact, they are so critical that female-female relationships are both ubiquitous and constant in the life of a female baboon. Any female who finds herself without adequate social support will quickly fill that gap.

III. A young female baboon's social circumstances strongly influence her exposure to physiological costs

The average female baboon faces increasing exposure to cortisol as she approaches maturity. This result contrasts with a previous study that found no such effect (Gesquiere et al., 2005); however, this may be the result of a very different statistical approach. Cortisol appears to increase because of an increased rate of social stressors as females reach maturity and begin to enter the hierarchy. If this effect were driven by stressors associated with physiological maturation, there should be only minimal rank-related differences in the age slopes. Instead, we see obvious differences; while the age trajectory for low- and mid-ranking daughters is flat or increasing, stress drops dramatically as high-ranking daughters reach maturity.

A mother's rank can influence her daughter's pattern of cortisol release in a few key ways. In each case, daughters of high ranking females are more likely to be protected from the negative effects of cortisol. As noted above, daughters of high-ranking females show decreasing exposure to stress as they progress through the juvenile stage and enter the adult hierarchy. Daughters of low-ranking females instead face constant or increasing exposure. Maternal rank can determine whether a young female's family represents a significant source of stress relief. For a young female destined to achieve high rank, a large social network can decrease her cortisol levels. However, additional relatives represent increased competition for young females at the bottom of the hierarchy; perhaps because low-ranking kin have no social influence to wield. While low-ranking mothers can still minimize their daughter's exposure to stressors, more distant kin have little to offer. During times of ecological hardship, as in the loss of the dagaa, the daughters of high-ranking females once again benefit by way of decreased cortisol exposure.

While low rank was often associated with increased cortisol in juvenile females, adult females showed no such effects. In fact, as discussed above, low-ranking females actually fared better following the loss of the *dagaa*. It appears that many of the physiological costs of an adult female's low rank are actually passed on to her developing daughters. These results also suggest that low-ranking daughters may have reduced access to social resources that would attenuate their response to stressors. In fact, rank's interactions with indicators of social support show that low maternal rank can actually reverse the stress-relieving nature of some social traits. High maternal rank may protect young females from undue physiological costs. The improved growth and faster development observed in daughters of high-ranking females may be in part the result of reduced exposure to cortisol during adolescence (Sapolsky, 1992; Johnson, 2003).

Mothers also appear to influence the physiological costs incurred by daughters by coordinating the benefits offered by kin. In her absence, close kin actually increase physiological costs, suggesting that they become something more akin to competition than social buffer. Again, recent behavioral studies have uncovered a parallel effect (Silk et al., 2006b). Females with living mothers form close affiliations with maternal sisters and other maternal kin. When their mothers die, they strengthen bonds with sisters, but bonds with more distantly related kin deteriorate. Silk et al. further suggest that these were relationships of convenience—a simple side-effect of a daughter's bond with her mother and a mother's bonds with *her* sisters.

The social and physiological experiences of juvenile females can vary vastly, and the most fortunate female appears to be one who comes from a large and high-ranking family. She is born to a mother who is able to devote more time to her daughters—a mother better equipped to provide optimal care (Bardi et al., 2005). This high-ranking mother's priority access to resources provides her daughter with adequate food and ideal nutrition. Her strong relationships with kin provide her daughter with a network of social support that can significantly decrease her exposure to stressors while simultaneously decreasing the severity of unavoidable stressors. This network becomes especially valuable during the rank acquisition process, when a larger network means an easier climb up the hierarchy. As this daughter ascends the hierarchy, her exposure to cortisol drops as a result of her own improved status. Eventually, she settles into the rank immediately below her mother and is in a position to offer her own daughters the benefits associated with her rank and kin.

The other extreme appears to be the orphaned daughter of a low-ranking female with a large extended family. In this case, the young female suffers all of the physiological costs of low rank and lacks the potent social buffer provided by a mother. As she reaches maturity, she finds herself at the bottom of the hierarchy, competing with numerous relatives for limited resources. She is particularly at risk during times of ecological adversity. Eventually, however, she develops effective coping strategies that allow her to circumvent many chronic stressors.

What might this suggest about the selective pressures that have shaped the social systems of female Old World monkeys? First, any cortisol-related physiological costs associated with a given rank appear to affect juveniles more than adults. This increased exposure to cortisol may contribute to the slower growth and delayed maturation we see in the daughters of low-ranking females, and in turn could lead to a decreased rate of reproduction as well as an increased risk of future pathology. If proximity to kin is a stress-reducing strategy promoted by female philopatry, these benefits are strongest for juvenile females. Also, female philopatry is not without physiological costs, as proximity to kin actually increases cortisol in low-ranking and motherless females.

Overall Conclusions

For adult female baboons, neither high rank nor maternal kin appear to offer benefits by way of reduced cortisol exposure. It appears that low-ranking females are able to avoid the physiological ramifications of a more difficult life by developing a repertoire of effective coping strategies which serves them well during times of ecological change. The social circumstances of rank and family size appear to exert their greatest physiological effects during adolescence. The orphaned daughter of a low-ranking female faces considerable physiological costs, and a maturing female with a living high-ranking mother and a large matriline enjoys an improved physiological outlook.

Table 1. Potential chronic stressors considered as predictors of cortisol.

Adult Model Predictors	Juvenile Model Predictors
Ethanol Preservation Lag	Ethanol Preservation Lag
Freeze Lag	Freeze Lag
Age	Age
Reproductive Status	Mom Alive
Number of Adult Female Relatives	Number of Adult Female Relatives
Rank	Mom's Rank
Troop	Troop
Field Season	Field Season
Ethanol Preservation Lag*Freeze Lag	Ethanol Preservation Lag*Freeze Lag
Freeze Lag*Troop	Freeze Lag*Troop
Age*Field Season	Age*Mom Alive
Age* Number of Adult Female Relatives	Age* Number of Adult Female Relatives
Reproductive Status*Field Season	Age*Mom's Rank
Reproductive Status*Number of Adult Female Relatives	Mom Alive*Number of Adult Female Relatives
Number of Adult Female Relatives*Field Season	Number of Adult Female Relatives*Field Season
Number of Adult Female Relatives*Rank	Mom's Rank*Number of Adult Female Relatives
Rank*Troop	Mom's Rank*Field Season
Rank*Field Season	Troop*Field Season
Troop*Field Season	

Table 2. Type 3 tests of fixed effects: Full adult female model. Italics denote main effects and interactions that were found to be significant predictors of cortisol in this model.

Effect	**DF**	**F**	**P**
Ethanol Preservation Lag	1	0.37	0.5458
Freeze Lag	*1*	*5.01*	*0.0254*
Age	1	1.10	0.2956
Reproductive Status	5	0.68	0.6373
Number of Adult Female Relatives	1	0.37	0.5409
Rank	2	0.05	0.9485
Troop	*7*	*2.68*	*0.0095*
Field Season	*2*	*5.85*	*0.0030*
Ethanol Preservation Lag*Freeze Lag	1	0.09	0.7645
*Freeze Lag*Troop*	*7*	*2.70*	*0.0091*
Age*Field Season	2	0.74	0.4757
Age* Number of Adult Female Relatives	1	0.52	0.4707
Reproductive Status*Field Season	10	0.79	0.6380
Reproductive Status*Number of Adult Female Relatives	5	0.28	0.9232
Number of Adult Female Relatives*Field Season	2	0.37	0.6912
Number of Adult Female Relatives*Rank	2	0.85	0.4288
Rank*Troop	14	0.88	0.5788
*Rank*Field Season*	*4*	*2.67*	*0.0314*
*Troop*Field Season*	*14*	*2.08*	*0.0112*

Table 3. Type 3 tests of fixed effects: Reduced adult female model. Italics denote main effects and interactions that were found to be significant predictors of cortisol in this model.

Effect	**DF**	**F**	**P**
Freeze Lag	*1*	*5.53*	*0.0189*
Rank	2	1.82	0.1634
Troop	*7*	*2.86*	*0.0059*
Field Season	*2*	*13.16*	*<0.0001*
*Freeze Lag*Troop*	*7*	*2.73*	*0.0083*
*Rank*Field Season*	*4*	*2.74*	*0.0279*
*Troop*Field Season*	*14*	*2.21*	*0.0063*

Table 4. Type 3 tests of fixed effects: Full juvenile female model. Italics denote main effects and interactions that were found to be significant predictors of cortisol in this model. †Denotes those that were insignificant in full model, but significant in reduced.

Effect	**DF**	**F**	**P**
Ethanol Preservation Lag	1	1.68	0.1983
Freeze Lag	1	1.82	0.1805
† Age	1	1.26	0.2645
† Mom Alive	1	2.06	0.1546
† Number of Adult Female Relatives	1	0.03	0.8597
Mom's Rank	*2*	*10.20*	*<0.0001*
Troop	*5*	*13.45*	*<0.0001*
Field Season	*2*	*8.43*	*0.0004*
Ethanol Preservation Lag*Freeze Lag	1	0.61	0.4369
*Freeze Lag*Troop*	*5*	*12.56*	*<0.0001*
Age*Mom Alive	1	0.28	0.5958
Age* Number of Adult Female Relatives	1	0.01	0.9190
*Age*Mom's Rank*	*2*	*7.08*	*0.0013*
*Mom Alive*Number of Adult Female Relatives*	*1*	*21.70*	*<0.0001*
Number of Adult Female Relatives*Field Season	2	1.01	0.3662
*Mom's Rank*Number of Adult Female Relatives*	*2*	*20.94*	*<0.0001*
*Mom's Rank*Field Season*	*4*	*2.92*	*0.0246*
*Troop*Field Season*	*10*	*7.35*	*<0.0001*

Table 5. Type 3 tests of fixed effects: Reduced juvenile female model. Italics denote main effects and interactions that were found to be significant predictors of cortisol in this model.

Effect	**DF**	**F**	**P**
Freeze Lag	1	0.99	0.3215
Age	*1*	*8.03*	*0.0055*
Mom Alive	*1*	*13.95*	*0.0003*
Number of Adult Female Relatives	*1*	*10.69*	*0.0014*
Mom's Rank	*2*	*10.05*	*<0.0001*
Troop	*5*	*13.42*	*<0.0001*
Field Season	*2*	*10.90*	*<0.0001*
*Freeze Lag*Troop*	*5*	*12.26*	*<0.0001*
*Age*Mom's Rank*	*2*	*6.00*	*0.0034*
*Mom Alive*Number of Adult Female Relatives*	*1*	*38.65*	*<0.0001*
*Mom's Rank*Number of Adult Female Relatives*	*2*	*22.36*	*<0.0001*
*Mom's Rank*Field Season*	*4*	*2.50*	*0.00466*
*Troop*Field Season*	*10*	*7.32*	*<0.0001*

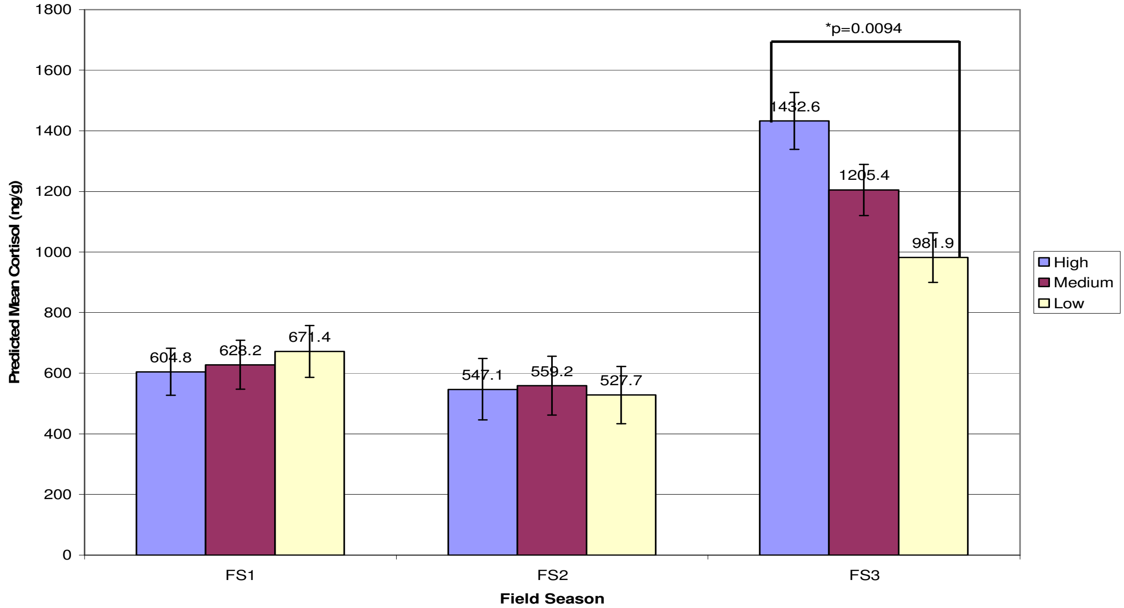

Figure 1. The interaction of rank and field season and its effect on predicted mean cortisol in adult female baboons. In FS3 there is a significant difference between high and low ranking adults. *(FS1: May & June 2000; FS2: February & March 2001; FS3: December 2001-April 2002)*

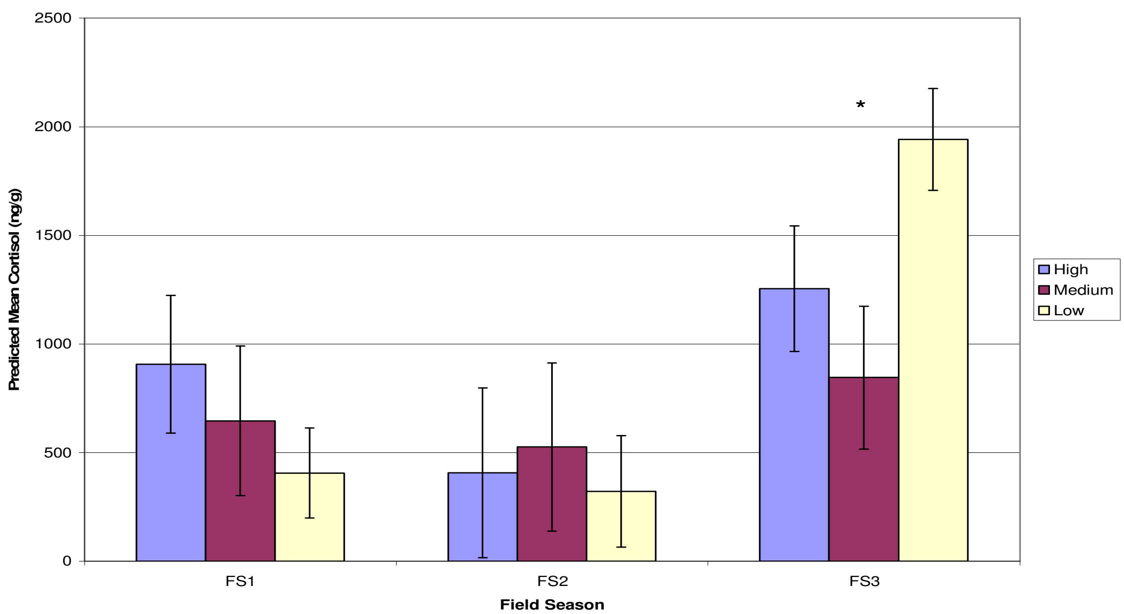

Figure 2. The interaction of maternal rank and field season and its effect on predicted mean cortisol in juvenile female baboons. While young females show no significant differences between maternal rank classes in FS1 and FS2, there is a significant difference between rank classes in FS3 (LSmeans, slice=field season, FS3: p=0.0189). *(FS1: May & June 2000; FS2: February & March 2001; FS3: December 2001-April 2002)*

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