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Review article

The next step for stress research in primates: To identify relationships between glucocorticoid secretion and fitness

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ABSTRACT

Glucocorticoids are hormones that mediate the energetic demands that accompany environmental challenges. It is therefore not surprising that these metabolic hormones have come to dominate endocrine research on the health and fitness of wild populations. Yet, several problems have been identified in the vertebrate research that also apply to the non-human primate research. First, glucocorticoids should not be used as a proxy for fitness (unless a link has previously been established between glucocorticoids and fitness for a particular population). Second, stress research in behavioral ecology has been overly focused on "chronic stress" despite little evidence that chronic stress hampers fitness in wild animals. Third, research effort has been disproportionately focused on the causes of glucocorticoid variation rather than the fitness consequences. With these problems in mind, we have three objectives for this review. We describe the conceptual framework behind the "stress concept", emphasizing that high glucocorticoids do not necessarily indicate a stress response, and that a stress response does not necessarily indicate an animal is in poor health. Then, we conduct a comprehensive review of all studies on "stress" in wild primates, including any study that examined environmental factors, the stress response, and/or fitness (or proxies for fitness). Remarkably, not a single primate study establishes a connection between all three. Finally, we provide several recommendations for future research in the field of primate behavioral endocrinology, primarily the need to move beyond identifying the factors that cause glucocorticoid secretion to additionally focus on the relationship between glucocorticoids and fitness. We believe that this is an important next step for research on stress physiology in primates.

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> Stress in addition to being itself, is also the cause of itself, and the result of itself.
>
> [(Roberts, 1950; a physician paraphrasing an address given by Hans Selye - , as quoted by Rosch 2010).]

1. Introduction

For nearly 40 years, researchers have sought to understand the causes and consequences of hormone fluctuations in wild animals (Wingfield, 1976). As a window into the energetic demands that accompany environmental challenges (Sapolsky et al., 2000), it is not surprising that glucocorticoids have come to dominate the majority of this research effort. However, it has been argued that this research effort has been disproportionately focused on the *causes* of glucocorticoid variation in wild vertebrate populations, rather than the fitness *consequences* (Bonier et al., 2009; Breuner et al., 2008; Dantzer et al., 2014). In this review, we argue that research on glucocorticoids in wild non-human primates suffers from a similar problem. Thus, we first outline some of the problems that unintentionally may have steered some of this research away from investigations into the adaptive nature of the "stress response". Second, to summarize the current state of glucocorticoid research in wild primate populations, we list all studies that have examined some aspect of stress physiology in wild primates. Our goal was to include any study that examined the relationship between environmental factors, the stress response, and performance/fitness variables. Remarkably, none of the primate research at the time of this review (Jan 2016) followed the connection between all three. Research efforts in the broader animal literature are increasingly being geared towards this end (e.g., Rivers et al., 2012; Marasco et al., 2015), and we suggest that primate research is ripe for this type of inquiry as well.

Centuries ago, the field of physics established the term "stress" as an external force that produces a proportional amount of "strain" (or deformation) on an object (Hooke's law). Importantly, the term "stress" describes the external cause, and the term "strain" describes the internal result. Today, in the mechanical engineering literature, there is little confusion about what constitutes stress versus strain, and we have many sound architectural structures as proof. By contrast, the adoption of the term "stress" for use in the physiological literature, has been more problematic (Levine, 2005). In 1936, Dr. Hans Selye introduced the term to physiology after he discovered that a noxious stimulus (e.g., injections) delivered repeatedly to laboratory rats produced peptic ulcers (Selye, 1936). He regrettably used the term "stress" interchangeably to describe both the cause (injections) and the effect (ulcers). (Selye later bemoaned that he would have rather gone down in history as the father of the "strain concept", Rosch, 2010). As exemplified in the opening quote to this review, early confusion surrounding the stress concept was due to the linguistic problem of using the same word to describe both the cause and effect (Romero et al., 2009) – and even more recent publications claim to "measure stress" without initially specifying whether this means they intend to measure the external stressor or the internal response (e.g., Moberg, 2000). One solution to this problem has been to use the term "stressor" to indicate the cause of stress (external or internal) and the term "stress response" to indicate the internal physiological response. However, by these definitions, the physiological reaction is deemed a "stress response" if it is triggered in response to a "stressor" – in what clearly becomes circular reasoning. To get around this, many researchers have successfully adopted an independent definition for a stressor – that is, any unpredictable and/or uncontrollable stimulus (Levine and Ursin, 1991), and this remains the most widely-used definition.

Yet, a deeper conceptual confusion remains. Perhaps because Hans Selye first discovered and reported the detrimental side of stress (i.e., chronic stress), the adaptive side to the stress response has never received the attention it deserves (i.e., the fitness-enhancing effects, with "fitness" defined as any proxy or direct measure of breeding success among individual animals in natural populations (Clutton-Brock, 1988)). To be clear, most studies on "stress" *do* explain the dual nature of the stress response. Nearly three quarters of the studies reviewed here include in their introductions a paragraph or two on the idea that although the initial physiological response to challenging stimuli is considered to be adaptive (typically with no empirical citations), a prolonged activation can cause severe reductions in health and longevity, with extensive empirical citations from the biomedical literature (e.g., Brotman et al., 2007; Juster et al., 2010; Lupien et al., 2009; Sapolsky, 2004a). The emphasis and focus is therefore on the latter, leaving a reader with the impression that this is a physiological response gone-wrong. The biomedical research has understandably focused on the negative health consequences that result from psychological stressors that impact the lives of humans. However, this "biomedical bias" has spilled over to non-biomedical literature and has unnecessarily influenced the direction and interpretation of research across many evolutionary and ecological disciplines (Boonstra, 2013).

With the exception of extreme situations, such as degraded habitats or captive environments, organisms in natural populations do not appear to experience net decreases in fitness from stress effects (Boonstra, 2013; Dantzer et al., 2014). Selection would not maintain a physiological response that routinely harmed an organism's fitness. This misunderstanding – mainly, that the stress response is harmful – could be alleviated if we had more evidence for the adaptive side of the stress response in natural populations (Fig. 1). As others have noted previously, research that seeks to link a stress response to an adaptive outcome has been scarce across behavioral ecology, more generally (Bonier et al., 2009; Breuner et al., 2008; Dantzer et al., 2014) and nearly entirely absent in primate behavioral ecology, specifically.

In this review, we start out with an attempt to clarify the conceptual framework behind the "stress concept", focusing on how this neuroendocrine response might mitigate the effects of a challenging environment. We focus on one particular physiological mediator, glucocorticoid secretion, because this class of hormones is the primary measure that is available from wild primate populations.

2. The stress concept – a conceptual framework

2.1. HPA activation

To understand the stress concept in an evolutionary conceptual framework, it is first necessary to outline the physiological mechanisms (see the following reviews for more comprehensive details: Charmandari et al., 2005; Johnstone et al., 2012; Romero and Wingfield, 2016; Sapolsky et al., 2000). First, sensory information from an unpredictable, uncontrollable, and/or aversive stimulus ("stressor")

arrives in the amygdala. These stressors derive from extrinsic (e.g., anthropogenic, ecological, or social in nature) or intrinsic sources (e.g., age, life history stage, reproductive condition, immune condition; Fig. 1). Second, the hypothalamus triggers a neuroendocrine cascade that includes the adrenomedullary (i.e., the fight-flight-freeze response) and the adrenocortical response (together, these two constitute the hypothalamic-pituitary-adrenal (HPA) axis). The adrenomedullary response activates the sympathetic nervous system causing the instantaneous release of catecholamines (e.g., epinephrine and norepinephrine) from the postganglionic fibers of the sympathetic

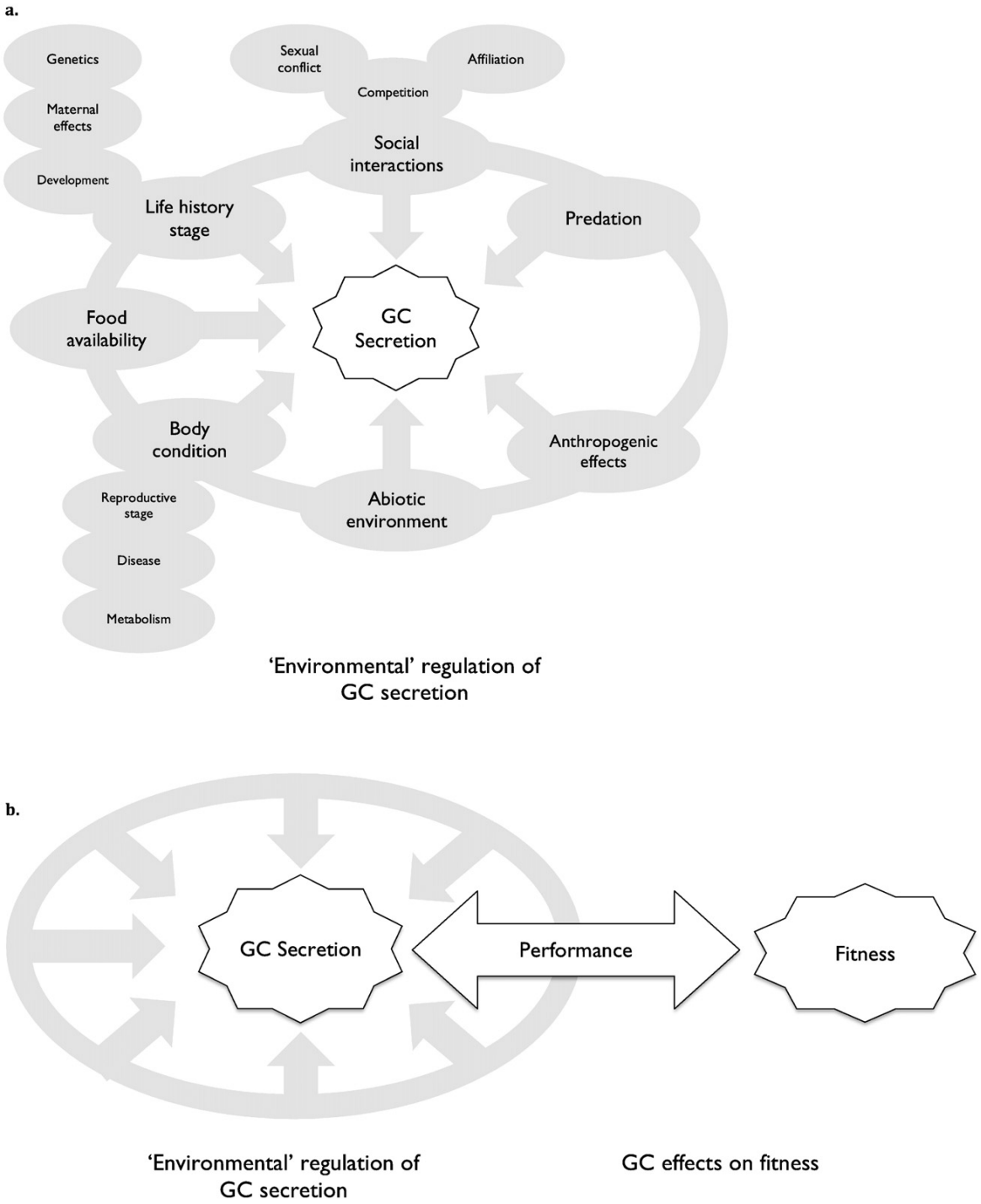

Fig. 1. Theoretical framework illustrating the relationship between (a) the 'environment' (gray arrows and circles) and GC secretion and (b) the relationship between GC secretion, intermediate performance, and fitness measures (modified from Breuner et al., 2008). Note that relationships among the gray circles are far more extensive than depicted here (and categories may overlap considerably). To date, there are no wild primate studies that examine both sides of this diagram: linking the environmental regulation of GC secretion to GC effects on fitness; and there is only one wild primate study that examined the relationship between GC secretion and fitness (Pride, 2005b).

nervous system (Morrison and Cao, 2000) and from the adrenal medulla of the adrenal gland (Wurtman and Axelrod, 1965). On a slightly slower timescale, the adrenocortical response causes the hypothalamus to synthesize and secrete corticotropin-releasing hormone (CRH), which stimulates the anterior pituitary to secrete adrenocorticotropic hormone (ACTH) into general circulation. ACTH travels to the adrenal cortex of the adrenal gland where it stimulates the synthesis and secretion of many adrenal hormones, including glucocorticoids (e.g., cortisol and corticosterone) as well as androgens (e.g., DHEA). Adrenal hormones enter systemic circulation via the bloodstream and travel to target receptors throughout the brain and body.

2.2. Negative feedback inhibition

Regulation of glucocorticoid (GC) secretion is subject to negative feedback control mediated by receptors primarily in the brain (Herman et al., 2012). Glucocorticoids bind to high-affinity mineralocorticoid receptors (MRs) or low-affinity glucocorticoid receptors (GRs), which function as transcription factors to positively or negatively regulate gene expression (De Kloet et al., 1998). At low levels, the high-affinity MRs are bound and facilitate daily metabolic signaling (Dallman et al., 1989). At high levels, such as during an environmental stressor, the low-affinity GRs are bound and mediate negative feedback inhibition of their own secretion (De Kloet et al., 1998).

Importantly, many of the physiological meditators that constitute the stress response – such as those involved in the hypothalamic-pituitary-adrenal (e.g., GCs) and cardiovascular (e.g., catecholamines) systems (Romero et al., 2009) – are the same mediators involved in routine metabolic functions (e.g., digestion, migration, reproduction, and circadian/circannual rhythms, (Wingfield, 2013b)). Indeed, their role in successful metabolism *is exactly why* these mediators are called upon to counteract stressful stimuli. For example, GCs (and many of the other physiological mediators involved in the stress response) mediate the effects of the stressors, recalibrate life history priorities, and/or restore homeostatic metabolic functions.

Because GCs mobilize energy when it is needed, we expect that more energy-demanding periods will be associated with more GC secretion. Specifically, organisms should exhibit extensive variation in GCs that correspond to circadian variation, circannual variation, and different developmental and reproductive stages. Variation in GC secretion across these categories constitute a normative response called "predictive homeostasis" (Fig. 2 modified from Romero et al., 2009) that has little to do with stressful stimuli. However, despite their routine role in metabolism, there remains a strong tendency in the literature to refer to GCs as "stress hormones" – a term that undermines the extent to which these hormones modulate energy balance on a day-to-day basis. Instead, a more appropriate descriptive term for GCs is "metabolic hormones" (e.g., Dantzer et al., 2016). This description helps broaden the scope of stress research to consider why these hormones fluctuate in response to social and ecological challenges and what they do to restore balance.

2.3. Initial stress response

Over and above physiological variation associated with predictive homeostasis, organisms are additionally expected to have the flexibility to respond to the cumulative "wear and tear" of challenging environmental stimuli that they experience day-to-day. This is what is typically called a stress response but (in moving away from terms that involve the word "stress") is increasingly known as "reactive homeostasis" (Fig. 2, Romero et al., 2009). An organism's adaptive response to a stressor leads to physiological and behavioral changes that increase chances for survival (Charmandari et al., 2005). Behavioral modifications include increased arousal and vigilance, improved cognition, and focused attention. Physiological responses include enhanced analgesia, elevations in core temperature, inhibition of non-essential metabolic

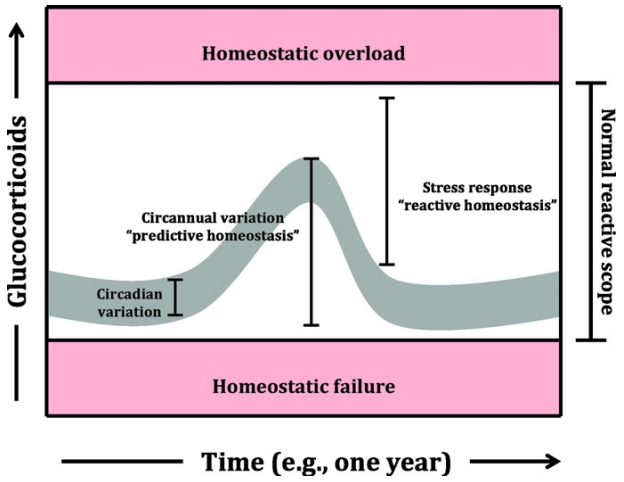

Fig. 2. Graphical model of how glucocorticoids (GCs) can vary across the day (circadian variation), the year (circannual variation), or in response to a challenging stimulus (stress response). All of these elevations in GCs combined fall within the "normal reactive scope" for an individual organism. That is, they are part of the adaptive variation in GCs that we should expect to observe in organisms through time. Although theoretically there is a "homeostatic overload" for GCs, this threshold is only crossed in extreme situations such as degraded habitats or captive environments. Figure and terminology modified from Romero et al., 2009.

functions (such as growth, appetite, and reproduction), increases in cardiovascular tone and respiratory rate, and a redirection of energy out of storage and into a form (glucose) that can be used immediately (Nelson, 2011). Together, the total variation in GC levels (or any other physiological mediator) that includes both the predictive and reactive homeostatic responses is called the "normal reactive scope" for an organism (Fig. 2, Romero et al., 2009). Importantly, an HPA response that remains within this normal reactive scope constitutes a protective, adaptive response by the organism to achieve allostasis (physiological stability in the face of environmental challenges; (McEwen and Wingfield, 2003)).

2.4. Chronic HPA response

However, laboratory studies have demonstrated that if the magnitude or the duration of the stressor extends beyond what an organism can handle, the same HPA response can subsequently decrease an organism's health and longevity (Juster et al., 2010). This extended response is typically called a "chronic stress response" (also called "homeostatic overload", Fig. 2, Romero et al., 2009). The primary distinction from the initial stress response is that a chronic stress response leads to a state of HPA dysregulation, which hampers overall health and fitness (Sapolsky et al., 2000). For example, with respect to GC secretion in response to an immediate physical stressor, a "good" response is one that exhibits (1) a low GC baseline, (2) a fast GC increase, and (3) a rapid induction of negative feedback, usually measured experimentally by degree of dexamethasone resistance (Breuner et al., 2008). Chronically-stressed animals fail to exhibit one or more of these responses (although some species (e.g., voles) appear to be consistently dexamethasone resistant, Taymans et al., 1997). Across the biomedical literature, the chronic stress response has been linked to a number of adverse health effects and disorders (Chrousos and Gold, 1992; Sapolsky, 2004a), including decreases in growth rates, increases in cardiovascular disease, infertility, molecular ageing, and premature death (Epel et al., 2004; Homyack, 2010; Sapolsky et al., 2000). Despite support for the concept of chronic stress in laboratory animals and humans, it is doubtful whether this concept has any relevance for understanding the stress response in natural populations (Boonstra, 2013, Dantzer et al., 2016).

Table 1
Wild primate studies examining rank-related predictors of glucocorticoid levels (GCs).

Rank-related predictors								
Primate species	**Sex**	**GC predictor**	**Result**	**GC outcome**	**Result**	**Notes**	**Matrix[5]**	**Reference**
Pan troglodytes	F	Rank[1]	-			During pregnancy, lactation	*U*	Emery Thompson et al., 2010
Pan troglodytes	F	Rank[1]	-			In larger groups	*F*	Markham et al., 2014
Pan troglodytes	F	Rank[1]	-			During pregnancy	*F*	Murray et al., 2016
Papio ursinus	F	Rank[2]	-			With grooming metrics	*F*	Wittig et al., 2008
Papio ursinus	F	Rank[1]	-			Low rank "loners" have highest GCs	*F*	Seyfarth et al., 2012
Macaca fuscata	F	Rank[1]	-			High rank lower GCs than mid rank	*F*	MacIntosh et al., 2012
Cercopithecus mitis	F	Rank[1]	-				*F*	Foerster et al., 2011
Lemur catta	F	Rank[1]	+				*F*	Cavigelli 1999; Cavigelli et al., 2003
Macaca mulatta	F	Rank[2]	intx[3]			With proximity reach	*F*	Brent et al., 2011
Macaca sylvanus	F	Rank[2]	intx[3]			With grooming metrics	*U*	Sonnweber et al., 2015
Papio cynocephalus	F	Rank[1]	ns	HPA sensitivity	+		*S*	Sapolsky et al., 1997
Papio cynocephalus	F	Rank[2]	ns	Fetal loss	ns		*F*	Beehner et al., 2006a
Papio ursinus	F	Rank[2]	ns				*F*	Crockford et al., 2008
Papio ursinus	F	Rank[2]	ns	Short-term fertility	ns		*F*	Weingrill et al., 2004
Mandrillus sphinx	F	Rank[1]	ns	Short-term fertility	ns		*F*	Setchell et al., 2008
Cercopithecus mitis	F	Rank[2]	ns				*F*	Foerster & Monfort 2010
Macaca fascicularis	F	Rank[1]	ns				*U*	van Schaik et al., 1991
Macaca mulatta	F	Rank[1]	ns	Cytokines (IL-8)	+	No rank effect, but low rank increased GCs most during anaesthesia	*P*	Maestripieri et al., 2008; Hoffman et al., 2010; 2011; Hoffman & Maestripieri 2012
Macaca sylvanus	F	Rank[2]	ns				*F*	Shutt et al., 2007
Cebus capucinus	F	Rank[1]	ns				*F*	Carnegie et al., 2011
Leontopithecus rosalia	F	Rank[1]	ns				*F*	Bales et al., 2005
Callithrix jacchus	F	Rank[1]	ns				*F*	Sousa et al., 2005
Lemur catta	F	Rank[2]	ns	Mortality	+		*F*	Pride 2005a; 2005b
Lemur catta	F	Rank[2]	ns				*F*	Starliing et al., 2010
Pan troglodytes	M	Rank[1]	-				*U*	Wittig et al., 2015
Macaca assamensis	M	Rank[1]	-				*F*	Ostner et al., 2008a
Macaca fascicularis	M	Rank[1]	-				*U*	van Schaik et al., 1991
Pan paniscus	M	Rank[1]	+				*U*	Surbeck et al., 2012
Pan troglodytes	M	Rank[1]	+				*U*	Muller & Wrangham 2004
Papio anubis	M	Rank[1]	+			Central vs peripheral	*F*	Tkaczynski et al., 2014
Papio cynocephalus	M	Rank[1]	+			Alpha vs others	*F*	Gesquiere et al., 2011
Papio ursinus	M	Rank[1]	+				*F*	Kalbitzer et al., 2015
Lophocebus albigena	M	Rank[1]	+				*F*	Arlet et al., 2009
Macaca fascicularis	M	Rank[1]	+			Alpha vs others	*F*	Girard-Buttoz et al., 2014
Macaca fuscata	M	Rank[1]	+				*F*	Barrett et al., 2002
Cebus capucinus	M	Rank[1,2]	+			Alpha vs others	*F*	Schoof et al., 2011; 2013; 2014; 2016
Sapajus libidinosus	M	Rank[1]	+			Alpha vs others	*F*	Mendonça-Furtado et al., 2014
Alouatta pigra	M	Rank[1]	+			Central vs non-central	*F*	Van Belle et al., 2009
Propithecus verreauxi	M	Rank[1]	+				*F*	Fichtel et al., 2007
Papio anubis	M	Rank[1]	intx[4]	HPA sensitivity	+	Low rank more DEX resistant	*S*	Sapolsky 1982; 1983a; 1983b; 1985; 1990
Papio ursinus	M	Rank[1]	intx[4]				*F*	Bergman et al., 2005
Mandrillus sphinx	M	Rank[1]	intx[4]	Parasites	+		*F*	Setchell et al., 2010
Macaca mulatta	M	Rank[1]	intx[4]				*F*	Higham et al., 2013
Pan troglodytes	M	Rank[1]	ns				*F*	Muehlenbein & Watts 2010
Gorilla gorilla beringei	M	Rank[1]	ns				*U*	Robbins & Czekala 1997
Pongo pygmaues	M	Rank[1]	ns			Flanged vs unflanged	*F*	Marty et al., 2015
Hylobates lar	M	Rank[2]	ns	Parasites	ns		*F*	Gillespie et al., 2013
Papio cynocephalus	M	Rank[1]	ns	HPA sensitivity	+		*S*	Sapolsky et al., 1997
Papio papio	M	Rank[1]	ns				*F*	Kalbitzer et al., 2015
Macaca fascicularis	M	Rank[1]	ns				*F*	Girard-Buttoz et al., 2009
Macaca sylvanus	M	Rank[2]	ns				*F*	Young et al., 2014
Cebus apella	M	Rank[1]	ns			Alpha vs others	*F*	Lynch et al., 2002
Leontopithecus rosalia	M	Rank[1]	ns			Breeding vs non	*F*	Bales et al., 2006
Saguinus mystax	M	Rank[1]	ns			Breeding vs non	*F*	Huck et al., 2005
Lemur catta	M	Rank[1]	ns				*F*	Gould et al., 2005
Lemur catta	M	Rank[2]	ns				*F*	Pride 2005c
Lemur catta	M	Rank[2]	ns				*F*	Starliing et al., 2010
Eulemur rubrifrons	M	Rank[1]	ns			During breeding season	*F*	Ostner et al., 2008b
Eulemur rubrifrons	M	Rank[2]	ns				*F*	Clough et al., 2010
Propithecus verreauxi	M	Rank[2]	ns				*F*	Brockman et al., 2009

[1]Indicates studies where dominance rank was one of the primary factors examined.
[2]Indicates studies where dominance rank was only a secondary factor examined.
[3]Indicates an interaction between rank and a social variable (i.e., grooming or proximity).
[4]Indicates an interaction between rank and rank stability; STABILITY: high GCs in low-ranking animals, INSTABILITY: high GCs in high-ranking animals.
[5]For matrix, the following abbreviations were used: *U*=urine, F=fecal, *P*=plasma, *S*=serum, *H*=hair.

2.5. The paradox of the stress response

Given that the stress response largely functions to prioritize *present energy usage* over *future energy storage* (a classic life history tradeoff, (Wingfield and Sapolsky, 2003)), the dual nature of the stress response is intuitively appealing; it is adaptive over the short-term but increasingly unsustainable over time. In support of this, there is solid evidence for the energy-mobilizing actions of the HPA axis in response to a stressor, making it easy to predict how HPA activation might facilitate, say, an escape from a predator. Likewise, there is equally solid evidence for attributing adverse health outcomes to these effects when chronically activated. The eloquent writings of Robert Sapolsky have popularized the concept of chronic stress across the biomedical, behavioral, and evolutionary sciences (Sapolsky, 1993a; Sapolsky, 1990b, 1993b; Sapolsky et al., 2000). Consequently, nearly three quarters of the stress studies we reviewed here paid at least some tribute to the dual nature of the stress response in their introductions. But, this places the focus on the wrong side of the equation.

In the biomedical literature, the vast majority of research on stress has been devoted to the non-adaptive, chronic side. This bias in research effort understandably stems from an applied, biomedical interest in preventing stress-related disease in humans. However, this bias may also underlie three interrelated but unwarranted assumptions in the non-biomedical stress literature: (1) that because the stress response is "bad", it can be used as a proxy for fitness, (2) that wild populations routinely experience chronic stress, and (3) that the adaptive side of the stress response has been well-supported and, therefore, is not in need of our research effort. Our purpose here is not to demonstrate that these assumptions are always wrong, but rather to alert researchers that the applied problems of the biomedical world rarely translate to the evolutionary and ecological problems of the natural world (Boonstra, 2013; Orchinik, 1998; Romero, 2004).

Table 2
Wild primate studies examining anthropogenic predictors of glucocorticoid levels (GCs).

		Anthropogenic predictors					
Primate species	**Sex**	**GC predictor**	**Result**	**GC outcome**	**Result**	**Matrix[1]**	**Reference**
Papio anubis	F	Crop-raiding	-	PdG	+	*F*	Lodge et al., 2013
Macaca mulatta	F	Capture stress	+			*P*	Maestripieri et al., 2008
Macaca sylvanus	M	Human impact	+			*F*	Maréchal et al., 2011
Cercopithecus aethiops	M	Capture stress	+			*S*	Suleman et al., 2004
Cercopithecus ascanius	M/F	Habitat disturbance	-			*U*	Aronsen et al., 2015
Procolobus gordonorum	M/F	Human impact	-			*F*	Barelli et al., 2015
Procolobus rufomitratus	M/F	Capture stress	-			*F*	Wasserman et al., 2013
Procolobus rufomitratus	M/F	Habitat disturbance	-			*U*	Aronsen et al., 2015
Eulemur rubriventer	M/F	Habitat disturbance	-			*F*	Tecot 2013
Gorilla gorilla	M/F	Human impact	+			*F*	Shutt et al., 2014
Pongo pygmaues	M/F	Human impact	+			*F*	Muehlenbein et al., 2012
Lophocebus albigena	M/F	Habitat disturbance	+			*U*	Jaimez et al., 2012
Macaca mulatta	M/F	Capture stress	+			*P*	Suomi et al., 1989
Chlorocebus aethiops	M/F	Human impact	+			*H*	Fourie et al., 2015b
Procolobus rufomitratus	M/F	Environment fragmentation	+			*F*	Chapman et al., 2006
Alouatta palliata	M/F	Capture stress	+			*F*	Aguilar-Cucurachi et al., 2010
Alouatta palliata	M/F	Environment fragmentation	+			*F*	Dunn et al., 2013
Alouatta palliata	M/F	Environment fragmentation	+			*F*	Gómez-Espinosa et al., 2014
Alouatta pigra	M/F	Environment fragmentation	+			*F*	Martínez-Mota et al., 2007
Alouatta pigra	M/F	Human impact	+			*F*	Behie et al., 2010
Alouatta pigra	M/F	Habitat disturbance	+			*F*	Rangel-Negrin et al., 2014
Ateles geoffroyi	M/F	Habitat disturbance / captivity	+			*F*	Rangel-Negrin et al., 2009
Eulemur collaris	M/F	Habitat disturbance	+			*F*	Balestri et al., 2014
Microcebus murinus	M/F	Capture stress	+			*F*	Hämäläinen et al., 2014
Galago moholi	M/F	Human impact	+			*F*	Scheun et al., 2015
Alouatta seniculus	M/F	Human impact / Environment fragmentation	+ / ns			*F*	Rimbach et al., 2013
Alouatta belzebul	M/F	Human impact	ns			*F*	Monteiro et al., 2013
Ateles hybridus	M/F	Human impact / Environment fragmentation	ns			*F*	Rimbach et al., 2013

[1]For matrix, the following abbreviations were used: *F*=fecal, *U*=urine, *P*=plasma, *S*=serum, *H*=hair.

Table 3
Wild primate studies examining ecological predictors of glucocorticoid levels (GCs).

Ecological predictors							
Primate species	**Sex**	**GC predictor**	**Result**	**GC outcome**	**Result**	**Matrix[1]**	**Reference**
Papio anubis	F	Temperature / food availability	+ / -			*F*	MacLarnon et al., 2015
Papio cynocephalus	F	Group size	Low in mid-sized			*F*	Markham et al., 2015
Papio cynocephalus	F	Seasonality	High in dry season			*F*	Gesquiere et al., 2008
Papio ursinus	F	Predation	+			*F*	Engh et al., 2006
Papio ursinus	F	Seasonality	High in winter	Short-term fertility	ns	*F*	Weingrill et al., 2004
Macaca assamensis	F	Food availability	- (pregnant females only)	Offspring growth / motor skill acquisition / immune function	+ / - / -	*F*	Berghänel et al., 2016
Papio cynocephalus	M	Seasonality	High in dry season			*F*	Gesquiere et al., 2011b
Theropithecus gelada	M	Seasonality / altitude	High when cold and high altitude			*F*	Beehner & McCann 2008
Macaca assamensis	M	Food availability / physical condition	ns			*F*	Schülke et al., 2014
Macaca sylvanus	M	Temperature (and male bonds)	High when cold, but lowered by male bonds			*F*	Young et al., 2014
Procolobus rufomitratus	M/F	Diet quality	-	Population size	ns	*F*	Chapman et al., 2007; 2016
Procolobus rufomitratus	M/F	Group size	ns			*F*	Snaith et al., 2008
Alouatta pigra	M/F	Fruit consumption	-	Parasites / population size	+ / -	*F*	Behie et al., 2013

[1]For matrix, the following abbreviations were used: *F*=fecal, *U*=urine, *P*=plasma, *S*=serum, *H*=hair.

2.6. Glucocorticoids are not a proxy for fitness

With the explosion of non-invasive and field-friendly methods for hormone monitoring in recent decades, GCs are increasingly being measured in natural populations (Anestis, 2010; Dantzer et al., 2014; Sheriff et al., 2011a). This body of research seeks to interpret the biological significance of variation in hormone levels in wild organisms; and elevations in GCs are commonly interpreted as an index of environmental stress. However, across these studies, an individual (or population) with higher GCs are routinely assumed to be in worse condition compared to an individual (or population) with lower GCs (Bonier et al., 2009). To what degree is this assumption warranted?

First, let's consider where this assumption comes from. A challenging environment can lead directly to a reduction in fitness. For example, if we compare animals in an environment with high predator pressure to one with low predator pressure, animals in the high-predator environment are more likely to exhibit an elevated stress response during the months before some of them get eaten (due to increased predator encounters). A researcher comparing animals in the two environments might report that animals with elevated GC levels have higher mortality than those with lower GC levels. Even though the higher number of predators is separately causing both the elevation in GCs and the mortality, the assumption is that elevated GCs can be used to successfully predict mortality (and, indeed, in this case, they can). This has been termed the "Cort-Fitness Hypothesis" (Bonier et al., 2009). Proponents of this hypothesis have argued that elevated GCs are the canary-in-the-coal-mine with respect to fitness (MacDougall-Shackleton et al., 2013).

But, this hypothesis is problematic for several reasons that have remained underappreciated, despite several excellent reviews on the topic (Bonier et al., 2009; Boonstra, 2013; Breuner et al., 2008; Dickens and Romero, 2013). First, testing this hypothesis often leads

Table 4
Wild primate studies examining (non-rank-related) social predictors of glucocorticoid levels (GCs).

Social predictors (non-rank-related)							
Primate species	**Sex**	**GC predictor**	**Result**	**GC outcome**	**Result**	**Matrix[1]**	**Reference**
Papio cynocephalus	F	Immigrant male	+	Lymphocytes	-	*B*	Alberts et al., 1992
Papio ursinus	F	Death of kin / social bonds	+ / -			*F*	Engh et al., 2006a
Papio ursinus	F	Focused groom networks	-			*F*	Wittig et al., 2008
Papio ursinus	F	Predictability of social interactions	-			*F*	Crockford et al., 2008
Papio ursinus	F	Immigrant male	+			*F*	Beehner et al., 2005a
Macaca assamensis	F	Same-sex sociality / opposite-sex sociality	- (non-mating season) / - (mating season)			*F*	Fürtbauer et al., 2014
Macaca mulatta	F	Aggression	+			*P*	Westergaard et al., 2003
Macaca mulatta	F	Self-directed behaviors	ns			*F*	Higham et al., 2009
Macaca sylvanus	F	Grooming metrics	Lower GCs giving than receiving			*F*	Shutt et al., 2007
Macaca sylvanus	F	Grooming metrics	See full result in Table 7			*U*	Sonnweber et al., 2015
Papio anubis	M	Self-directed behaviors / aggression / grooming	ns			*F*	Ellis et al., 2011
Papio anubis	M	Personality	See full result in Table 7			*S*	Ray & Sapolsky 1992
Papio anubis	M	Personality	See full result in Table 7			*S*	Virgin & Sapolsky 1997
Papio cynocephalus	M	Immigrant male	+	Lymphocytes	-	*B*	Alberts et al., 1992
Macaca assamensis	M	Mate guarding / consort duration	ns			*F*	Schülke et al., 2014
Macaca fascicularis	M	Mate guarding low-rank, nulliparous females	+			*F*	Girard-Buttoz et al., 2014b
Macaca sylvanus	M	Aggression / male bonds	High with high aggression, but lowered by male bonds			*F*	Young et al., 2014
Cebus capucinus	M	Presence of periovulatory females	+			*F*	Schoof et al., 2014
Propithecus verreauxi	M	Presence of infants	+			*F*	Brockman et al., 2009

[1]For matrix, the following abbreviations were used: *F*=fecal, *U*=urine, *P*=plasma, *S*=serum, *H*=hair.

to an apples-to-oranges comparisons. Most agree that, in response to an environmental challenge, elevated GCs should increase an individual's chance of surviving the challenge. For example, in an environment with increased predator pressure, prey animals with higher GC secretion may have heightened vigilance behavior, which could increase the likelihood of detecting a predator. In other words, under the same environmental challenge, individuals with a *more* effective stress response should have higher fitness than an individual with a *less* effective one. But, this is rarely the comparison that is made in stress research. Rather, researchers measure GCs across different populations under different environmental challenges, or within the same population across different years, and then report (more often than not) that the population with higher GCs must be doing worse than the population with lower GCs. Yet, this relationship would emerge simply because GCs are a proxy for environmental challenges that *themselves* are related to reduced fitness. That is, the fitness-reducing effects of the stressor are not distinguished from the fitness-reducing effects of the stress response (Johnstone et al., 2012).

Comparing GCs across environments is, of course, entirely warranted if the goal is to identify the environmental variables that affect GC secretion; and this approach has produced the wealth of information that is described in Tables 1–6. Indeed, this approach is perhaps the most useful in conservation studies (if validated, see Madliger and Love, 2016), where researchers intentionally use increased GCs as a proxy for environmental challenges.

Thus, if GC levels are to be used as a proxy for fitness, these assumptions must be validated with empirical data – that is, either fitness itself or a proxy for fitness must be measured while controlling for environmental challenges. Yet, this is rarely done. Because most research has been focused on the wrong comparisons, the adaptive nature of the stress response remains poorly understood.

2.7. Chronic stress is probably not a selective force in natural populations

In his popular book, *Why Zebras Don't Get Ulcers*, Sapolsky argued that wild animals cannot suffer from chronic stress (Sapolsky, 2004b), since the stress axis evolved as an adaptive response to challenges and chronic stress is maladaptive and pathological. He reasoned that "sustained psychological stress is a recent invention, mostly limited to humans and other social primates" (p. 4–7). A recent review followed up on this idea claiming that any stress response in a wild population must be adaptive and therefore promote survival (Boonstra, 2013). Certainly, evidence for a chronic stress response (and homeostatic overload) is found in captive animals in laboratories and zoos, and evidence is on the rise for chronic stress effects in wild animals living in anthropogenically-disturbed environments (Dantzer et al., 2016). Chronic stress in laboratory animals is typically produced by repeatedly restraining, shocking, or (for social species) isolating individuals from the rest of the group. But, do we have evidence for non-adaptive stress effects ("homeostatic overload", Fig. 2) for animals living in undisturbed environments? The debate here is not whether wild animals are exposed to long-term stressors (indeed, often they are, Clinchy et al., 2013); but rather whether wild animals suffer homeostatic overload as a result – that is, whether the HPA axis falls into dysregulation and leads to additional fitness costs beyond those imposed by the environmental challenge.

Table 5
Wild primate studies examining intrinsic predictors of and outcomes for glucocorticoid levels (GCs).

Intrinsic predictors							
Primate species	**Sex**	**GC predictor**	**Result**	**GC outcome**	**Result**	**Matrix[1]**	**Reference**
Pan troglodytes	F			Maternal behavior	+	*F*	Stanton et al., 2015
Pan troglodytes	F	Rank of mother, reproductive stage	ns			*F*	Murray et al., 2016
Papio cynocephalus	F	Age	+			*S*	Sapolsky & Altmann 1991
Papio cynocephalus	F	Age / maturation	See full result in Table 7			*F*	Gesquiere et al., 2005
Papio cynocephalus	F	Gestation stage	ns	Fetal loss	ns	*F*	Beehner et al., 2006a
Papio cynocephalus	F			Maternal behavior	+	*F*	Nguyen et al., 2008
Macaca mulatta	F	Reproductive stage	See full result in Table 7			*P*	Maestripieri et al., 2008; Hoffman & Maestripieri 2012
Cercopithecus mitis	F	Parasites	+			*F*	Foerester et al., 2015
Microcebus murinus	F	Age / body mass	+ / ns (dry season only)			*F*	Hämäläinen et al., 2015
Pan troglodytes	M			Parasites	+	*U*	Muehlenbein 2006
Pan troglodytes	M	Age	+			*F*	Seraphin et al., 2008
Pan troglodytes	M	Rank of mother, reproductive stage	See full result in Table 7	Offspring GCs	see full result in Table 7	*F*	Murray et al., 2016
Papio anubis	M			Testosterone	-	*S*	Sapolsky 1985
Papio cynocephalus	M	Age	+			*S*	Sapolsky & Altmann 1991
Papio cynocephalus	M	Age / maturation	See full result in Table 7			*F*	Gesquiere et al., 2005
Papio cynocephalus	M	Rank of mother	-			*F*	Onyango et al., 2008
Papio cynocephalus	M			Wound healing	+	*F*	Archie et al., 2012
Lophocebus albigena	M			Parasites	+	*F*	Arlet et al. 2015
Cebus capucinus	M	Age	ns			*F*	Jack et al., 2014
Microcebus murinus	M	Age / body mass	+/ + (dry season only)			*F*	Hämäläinen et al., 2015
Papio anubs x hamadryas hybrids	M	Age / hybridity	U-shaped / +			*H*	Fourie et al., 2015a
Macaca mulatta	M/F	Maternal rejection	+			*F*	Mandalaywala et al., 2014
Procolobus rufomitratus	M/F	Parasites	+			*F*	Chapman et al., 2007; Chapman et al., 2016
Cebus apella nigritus	M/F			Deceptive alarm calling	ns	*F*	Wheeler et al., 2014
Eulemur rubrifrons	M/F	Sex / season	+ (Males) / + (mating season)	Parasite species richness, parasite infection intensity	-	*F*	Clough et al., 2010

For matrix, the following abbreviations were used: *F*=fecal, *U*=urine, *P*=plasma, *S*=serum, *H*=hair.

Table 6
Wild primate studies examining performance (light green) and fitness (dark green) outcomes of GCs.

Associated outcomes of GCs

Primate species	Sex	GC outcome	Result	Matrix [1]	Reference
Pan troglodytes	F	Maternal behavior	+	*F*	Stanton et al., 2015
Pan troglodytes	M	Parasites	+	*U*	Muehlenbein 2006
Hylobates lar	M	Parasites	ns	*F*	Gillespie et al., 2013
Papio anubis	F	PdG	+	*F*	Lodge et al., 2013
Papio anubis	M	Testosterone	-	*S*	Sapolsky 1985
Papio anubis	M	HPA sensitivity	+	*S*	Sapolsky 1983
Papio cynocephalus	F	Maternal behavior	+	*F*	Nguyen et al., 2008
Papio cynocephalus	M	Wound healing	+	*F*	Archie et al., 2012
Papio cynocephalus	M/F	Lymphocytes	-	*B*	Alberts et al., 1992
Papio cynocephalus	M/F	HPA sensitivity	+	*S*	Sapolsky et al., 1997
Mandrillus sphinx	M	Parasites	+	*F*	Setchell et al., 2010
Lophocebus albegena	F	Parasites	+	*F*	Arlet et al., 2015
Macaca assamensis	F	Offspring growth / motor skill acquisition / immune function	+ / - / -	*F*	Berghänel et al., 2016
Macaca mulatta	F	Cytokines (IL-8)	+	*P*	Maestripieri et al., 2008; Hoffman et al., 2010; 2011; Hoffman & Maestripieri 2012
Cebus apella nigritus	M/F	Deceptive alarm calling	ns	*F*	Wheeler et al., 2014
Eulemur rubrifrons	M/F	Parasite species richness, parasite infection intensity	-	*F*	Clough et al., 2010
Papio cynocephalus	F	Fetal loss	ns	*F*	Beehner et al., 2006a
Papio ursinus	F	Short-term fertility	ns	*F*	Weingrill et al., 2004
Mandrillus sphinx	F	Short-term fertility	ns	*F*	Setchell et al., 2008
Alouatta pigra	M/F	Parasites / population size	+ / -	*F*	Behie et al., 2013
Procolobus rufomitratus	M/F	Population size	ns	*F*	Chapman et al., 2007; 2016
Lemur catta	M/F	Mortality	+	*F*	Pride 2005a; 2005b

[1] For matrix, the following abbreviations were used: *F*=fecal, *U*=urine, *P*=plasma, *S*=serum, *H*=hair

Although zebras are not supposed to get ulcers, Sapolsky surmised that social primates might nevertheless suffer the harmful effects of chronic psychological stressors. In the early 1980s, he found evidence that wild male olive baboons (*Papio anubis*) experienced what appeared to be HPA dysregulation. He demonstrated that low-ranking males exhibited hypercortisolism compared to high-ranking baboons, and – more importantly – that these low-ranking males were less responsive to negative feedback (Sapolsky, 1983b). This result was later replicated in wild yellow baboons (*Papio cynocephalus*) and included both males and females (Sapolsky et al., 1997). The authors attributed resistance in subordinates to the high rate of unpredictable and uncontrollable aggression that low-ranking animals routinely receive (Sapolsky et al., 1997). Since these studies, a few others have reported HPA dysregulation in wild populations due to long-term stressors such as El Niño (Romero and Wikelski, 2010), perceived predation risk (Zanette et al., 2011), and a combination of high predation and low food (Clinchy et al., 2004).

However, many studies have not demonstrated dysregulation in populations under long-term stressors. For example, snowshoe hares (*Lepus americanus*) are exposed to repeated stressors during the cyclic population declines associated with high predation risk (Sheriff et al., 2011b). Yet, during these periods they do not exhibit any signs of pathology or dysfunction of the HPA axis (Cary and Keith, 1979). A similar result was reported for arctic ground squirrels (*Urocitellus parryii*), who demonstrated a prolonged stress response to increased predator prevalence but with no evidence of corresponding pathology or an unresponsive HPA axis (Boonstra, 2013). Even in invertebrates, experimentally-induced chronic stress conditions in four anuran species did not result in decreased immunity or increased mortality (Haislip et al., 2012).

So, can natural populations suffer from homeostatic overload? One problem is that it is difficult to identify a clear endocrine profile for chronically-stressed organisms (Dickens and Romero, 2013). Documenting similar responses at multiple levels of GC regulation (e.g., high baselines, reduced reaction to a stressor, and/or resistant to negative feedback) would be an ideal approach (Dickens and Romero, 2013), but the logistical challenges involved in conducting such experimental studies in the wild are demonstrated by the modest number of field experiments that have actually done so. If indeed chronic stress plays a negligible role in natural populations, then (unless evidence is offered otherwise) the assumption should be that GC secretion serves an adaptive function (is within the normal reactive scope). Chronic stress would be better left unmentioned. As an analogy, a study on feeding ecology would not feel it necessary to describe the risks of obesity (a problem generally found only in humans or animals in captivity) as a potential negative outcome of feeding.

2.8. We need more studies on the adaptive nature of the stress response

Ever since Selye's initial publications, we have taken for granted that the "acute" stress response is adaptive (we avoid the term "acute" because it conflates the magnitude of the stress response with the

duration, Johnstone et al., 2012). Yet, surprisingly few studies have actually demonstrated the adaptive side. Many studies have examined GC secretion under stressful and other metabolic demands, but very few of these studies have taken this research to the next level – identifying how differential GC secretion produces differential performance or survival (Bonier et al., 2009; Breuner et al., 2008). We know that organisms need at least some GCs to survive: the complete removal of GCs through adrenalectomy causes increased mortality (Darlington et al., 1990) and patients with Addison's Disease (who present with primary adrenal insufficiency) suffer premature mortality (Bergthorsdottir et al., 2006). Nevertheless, we have remarkably little evidence from natural populations that differential GC secretion can lead to more (or less) adaptive outcomes.

Seven years ago, a comprehensive review of the entire biological literature found only 53 studies that examined the relationship between GCs and fitness (Bonier et al., 2009). Of the studies that measured GCs and fitness, only half of them found support for the Cort-Fitness Hypothesis (i.e., that individuals with higher GCs had reduced fitness). And, more than a third of these studies demonstrated the exact opposite – that high GCs under stressful conditions led to increased fitness (Bonier et al., 2009). In a recent example, zebra finches (*Taeniopygia guttata*) with prolonged exposure to stressful conditions (i.e., repeated and unpredictable activation of the HPA axis through food deprivation) actually lived longer than those with no deprivation (Marasco et al., 2015). Increasingly, studies are able to demonstrate that GCs released during stressful events can aid an organism in survival.

Thus, although the Cort-Fitness Hypothesis is not well-supported, it continues to influence the direction and interpretation of research across many evolutionary disciplines. As we describe in what follows, primate behavioral ecology, in particular, is in need of studies that examine the fitness consequences of inter-individual differences in the stress response.

3. Review of wild primate research on stress

Our second objective is to identify how studies on stress in wild primates fit into the conceptual framework illustrated above. We reviewed all published studies that measured some aspect of physiological stress in a natural primate population. We conducted an exhaustive search of the literature (1980–Jan 2016) to identify any study that measured (1) at least one aspect of the environment in relation to GC secretion, or (2) the relationship between GC secretion and at least one aspect of performance (Arnold, 1983) or fitness. Our purpose here was not to provide a thorough summary of each study but rather to summarize where research efforts have been concentrated and where they are needed.

3.1. Literature review

We conducted our literature search using Scopus, Web of Science, and Google Scholar search engines. We cross-referenced search terms including: *stress, glucocorticoid, corticosterone, cortisol, natural, wild, primate, ape, chimpanzee, gorilla, orangutan, gibbon, siamang, monkey, baboon, guenon, vervet, macaque, langur, capuchin, callitrichid, marmoset, tamarin, tarsier, lemur, loris, galago*. We also searched using many primate genus names – particularly for species where one of the previous terms is not included in the common name. We excluded all captive studies as well as studies that simply served as a validation for a particular antibody (regardless if the subjects were captive or wild). We did include data derived from semi-natural and provisioned populations. Finally, although there are several excellent reviews on hormones and behavior in primates (Abbott et al., 2003; Anestis, 2010; Bercovitch and Ziegler, 2002; Cheney and Seyfarth, 2009; Maestripieri and Hoffman, 2011; Saltzman and Maestripieri, 2011), we excluded all reviews since we are focused here on identifying gaps in our research efforts.

We identified a total of 138 separate studies on GCs and stress in wild primates. Many of these studies used the same (or overlapping) datasets from the same population, and thus from this point forward we lump these publications as one "study" so as not to bias our descriptive summary of research trends due to one well-studied population (we do not attempt a meta-analysis here, but we list separately each study in the Supplemental Materials for others to use). We organized these studies according to five broad categories of variables known to be associated with GC secretion in primates: rank-related variables (Table 1), anthropogenic variables (Table 2), ecological variables (Table 3), non-rank-related social variables (Table 4), and intrinsic variables (Table 5). Many studies were included in more than one table when the data included more than one species, sex, and/or category of variable. Conversely, when many studies from the same site on the same taxa report similar results, we combined these into one entry. The left side of each table lists the variables that were hypothesized to influence GC secretion (*GC predictors*); the right side of each table lists the variables that were hypothesized to be affected by GCs (*GC outcome)* – although many of these relationships are likely bidirectional. To highlight the few primate studies that examined the outcomes of GC secretion, we compiled these together into an additional table (Table 6). Tables include *all variables examined* and not just the variables that emerged as significant. Some variables (like parasites) could potentially be either a predictor or an outcome of GC secretion, and in these cases we used the researchers' designations (in their statistical models) as the deciding factor for placement. Finally, although Fig. 1 suggests that the potential "GC predictors" are the *causes* of GC secretion, the vast majority of the studies reviewed here only demonstrated a correlation between the two.

3.2. Overall results

An examination of Tables 1–6 reveals two striking trends. First, two variables (rank-effects, anthropogenic effects) have provided the impetus for the vast majority of studies on GC secretion. This comes as no surprise given that dominance rank is a salient feature in many primate societies (de Ruiter and van Hooff, 1993; van Schaik, 1989) and that a third to half of all primate species are endangered due to human activities and habitat destruction (IUCN, 2012; Mittermeier and Oates, 1985; Schipper et al., 2008). Second, perhaps the most conspicuous trend for all six tables *is the near absence of investigations on the "GC outcome" side*. Of 138 studies on GCs in wild primates, only 22 of them examined how GC secretion was associated with some aspect of performance and/or fitness (Table 6). We will return to this point shortly.

3.3. GCs and dominance rank

Dominance rank was the most predominant variable analyzed with respect to GCs, appearing in 60 studies. There was no consensus endocrine profile for either high- or low-ranking individuals across primate taxa, but one trend was that high GCs accompany positions where energetic/metabolic demands are high (due to excess expenditure or reduced intake, e.g., Emery Thompson et al., 2010; Fichtel et al., 2007; Gesquiere et al., 2011a) or where reproductive success is threatened (due to losing mates or offspring, e.g., Bergman et al., 2005). Another trend was that more of the studies on females produced a negative relationship between baseline GCs and dominance rank, while more of the studies on males produced a positive one (Table 1). For both trends, it may be that the secretion of GCs is not tied to high- or low-rank per se but rather to the process by which dominance rank is achieved (Abbott et al., 2003; Sapolsky, 1992).

Without exception, all primate studies took the view that dominance rank "caused" differences in GC levels and not the reverse (although in practice, these studies were correlational). Yet, we found that elevations in GC levels received different explanations depending on whether they accompanied high- or low-rank positions. For example, if high-ranking (i.e., presumably successful) individuals had high GC levels, then the elevation in GCs was considered a necessary cost of

high rank. But, if low-ranking (i.e., presumably unsuccessful) individuals had high GC levels, then the elevation in GCs was considered to have negative consequences for health and fitness. However, none of these studies linked individual endocrine differences to fitness outcomes. Currently, we have no data to indicate whether elevated GCs serve to improve or hamper the situation for individuals at these different ranks. What should future studies of dominance rank focus on?

First, the starting point for research inquiry should be the assumption that the measured endocrine profile is adaptive. Both high and low rank have direct costs associated with them, and the elevation in GCs associated with these costs should be viewed as the organism's attempt to ameliorate them. For high rank, the costs are likely due to the increased energetic demands related to achieving and maintaining high rank (e.g., Gesquiere et al., 2011a; Muller and Wrangham, 2004) – particularly when the dominance hierarchy is unstable (e.g., Sapolsky, 1992; Bergman et al., 2005); while for low ranking individuals the costs are likely due to limited energy intake or frequent social stress (e.g., Abbott et al., 2003). It is more difficult to understand the adaptive nature of GCs in low-ranking individuals. An increase in GCs can temporarily mobilize energy to deal with shortages of food, fight competitors, or perhaps even facilitate a rise in dominance status (where possible), an alternative mating strategy (e.g., coalitions, sneak copulations, cross-sex bonds), or the solicitation of social support. For example, some evidence from captive primates demonstrated that individual GC concentrations predicted future dominance rank (Johnson et al., 1996; McGuire et al., 1983).

Second, we need to generate data on the consequences of GCs in relation to rank. For example, it would be productive to examine how GCs help individuals achieve or keep high rank or how they deal with the demands associated with a given dominance rank. If we identify an association between dominance rank and GC levels, we should then examine the longitudinal pattern of GCs with respect to rank. Do low-rank individuals with high GCs climb the hierarchy? Do high-rank individuals with high GCs maintain their tenure for longer? Conceivably, individual HPA regulation could cause an individual to rise or fall in rank depending on their ability (or inability) to mobilize energy for a given contest. Researchers could do this by examining how current GCs affect future rank/fitness. Another promising approach might be to compare animals under a similar (and severe) stressor. For example, if we examine all high rank males during periods of instability, might individual GC levels predict future rank changes?

Third, we should consider the two hypotheses laid out in the "stress dynamics of dominance" scenario (Cavigelli and Caruso, 2015). (1) Where dominants have higher GC levels than subordinates, they should exhibit more frequent GC elevations than subordinates due to short-lived but intense periods of fighting. Thus, maximum but not mean GC levels are the important metric for testing this hypothesis. (2) Where subordinates have higher GC levels than dominants, they should exhibit long-term elevations in GC production with higher basal levels compared to dominants due to more metabolic demands like exclusion from food resources. Mean GC levels are the important metric for testing this hypothesis.

3.4. GCs and anthropogenic disturbance

Most data from anthropogenic disturbance studies support the idea that human activities and habitat destruction cause an increase in GCs for the disturbed populations (Table 2). Much of this research is applied, but it too has the potential to be evolutionarily informative if we can separate the effects of the environment from the effects of the GCs. In many cases, animals will be doing poorly because the environment is killing them. But, there will also be cases where animals are doing poorly because the stress response is killing them (they have entered homeostatic overload area). So, how do we distinguish between these two? First, researchers can compare individuals with comparable disturbance. Are individuals with higher GC levels more or less likely to survive? To reproduce? Second, researchers can check for signs of HPA dysregulation. In cases where animals are captured, it may be possible to measure individual GC responses to an induced stressor (e.g., capture) and/or check for DEX resistance. When capture is not possible, researchers can use natural experiments (e.g., short-term environmental disasters or increased predation events) as acute stressors and measure individual responses non-invasively (e.g., fecal or urinary hormones). Significant elevations in GCs should be expected for individuals with a properly functioning HPA axis. Finally, if researchers have access to deceased corpses, they can look for signs of enlarged adrenal glands.

3.5. GCs and maternal effects

Although, there were only two studies on the effects of maternal GCs on developing offspring in wild primates (Nguyen et al., 2008; Onyango et al., 2008; Berghänel et al., 2016), this is an extremely promising area of research in captive primates (Parker and Maestripieri, 2011) and wild, non-primate taxa (Dantzer et al., 2013). Because the HPA axis can be permanently altered during development due to stressors that impact the mother (and fetus), this type of research has the potential to help us understand why there might be inter-individual variance in HPA responsiveness to the same stressor. For example, in rats, a mother's GC levels during gestation can down- or up-regulate transcription of GRs in offspring to be less or more responsive to stressful stimuli, respectively (Weaver et al., 2004). One hypothesis posits that changes in GC responsiveness are a predictive adaptive response that can help offspring adapt to new conditions they may experience once they are born (Sheriff and Love, 2013; but see argument against maternal effects in Uller et al., 2013). Another hypothesis posits that these changes are simply the result of developmental constraints (Hayward et al., 2013). We expect that distinguishing between these hypotheses will be an active area of research for primate studies in the future.

3.6. GC effects on performance and/or fitness

Perhaps the most striking feature about each table is the near absence of investigations on the "effect" side. Only 22 primate studies examined how GC secretion affected some aspect of performance or fitness (Table 6). Most of these outcome variables were related to an individual's infection status (e.g. parasite richness, abundance (Arlet et al., 2015; Behie and Pavelka, 2013; Berghänel et al., 2016; Clough et al., 2010; Gillespie et al., 2013; Muehlenbein, 2006; Setchell et al., 2010)) or other immune parameters (e.g., wound healing, lymphocytes, or cytokines (Alberts et al., 1992; Archie et al., 2012; Hoffman et al., 2010; Hoffman et al., 2011; Hoffman and Maestripieri, 2012; Maestripieri et al., 2008)). Although far from consensus, the majority of these studies demonstrated a negative relationship between GC secretion and one or more immunity measures (Table 6).

Of all 22 studies, only 6 of them investigated fitness components related to reproduction (3 studies: Beehner et al., 2006; Setchell et al., 2008; Weingrill et al., 2004) or survival (3 studies: Behie and Pavelka, 2013; Chapman et al., 2007; Chapman et al., 2016; Pride, 2005b; Pride, 2005c). Two of the studies on survival were at the population level (Behie and Pavelka, 2013; Chapman et al., 2007; Chapman et al., 2016; Chapman et al., 2006) and therefore were only *indirect* tests of GCs on fitness. Therefore, out of more than one hundred studies on wild primates, *only one study directly examined individual glucocorticoid profiles in conjunction with fitness* (Pride, 2005b). In this 2-year study, researchers measured GCs across a population of ring-tailed lemurs (*Lemur catta*) and found a weak (non-linear) relationship between higher GCs and increased mortality. The author concluded that "…stress can be considered a mortality risk factor in wild populations as it is in humans". Although our purpose is to promote more studies that directly measure fitness, we caution against the language used in the author's final conclusion. If our science focuses only on the relationship between the endocrine trait and fitness outcomes, we (once again) fail to identify

the very phenotype by which endocrine traits cause variation in fitness. We need both sides of the equation. This is becoming increasingly known as "the missing trait" problem (Dantzer et al., 2016). Indeed, identifying this "missing trait" (or missing variable) should be a primary objective of stress research. One roadblock has been the focus on the stress response itself as the variable affecting poor fitness outcomes.

3.7. Why do we have so few primate studies that measure the effects of GCs?

First, primates are long-lived organisms that require decades of research to obtain true measures of fitness –that is, lifetime reproductive success (and, even estimates of lifetime reproductive success require many years of observation). Second, given the logistics and expense of hormone research, most detailed hormone studies tend to be labor-intensive "snapshots" in time – that is, cross-sectional studies to identify the variables that affect GC secretion, with a necessarily vague connection to what these hormone profiles might mean for performance and fitness. Even for long-term studies with a hormonal component, the hormone sampling is generally targeted to a particular age/sex/status category and not often longitudinal in nature (but see Emery Thompson et al., 2010; Gesquiere et al., 2005, 2008, 2011a, 2011b; Wittig et al., 2015). Despite these obstacles, we argue that primate studies should be at the forefront of research on how GCs mediate the relationship between the environment and fitness because many long-term primate studies actually *do have* very good estimates of survival and reproductive success in a large sample of known individuals. Moreover, primate studies are able to draw on a wealth of data on proxies for fitness (e.g., alpha tenure, interbirth intervals, infant survival to weaning) that may be easier to measure in coordination with short-term hormone samples. Finally, many long-term primate projects are adopting the "gold standard" of longitudinal hormone sampling from known individuals – projects that are sure to produce many upcoming and exciting publications. These are exactly the circumstances required for constructing a detailed picture of the relationship between the environment, endocrine traits (in conjunction with other known individual differences), and differential fitness.

4. The next step for research efforts

We know that GC secretion can and does directly influence fitness. However, despite the large number of studies that focus on measuring GC levels in wild primates, almost none of this research has been devoted to uncovering how GC production serves as a mediator between behavior, reproduction, and survival. Although we need to continue to identify the factors that cause GC secretion in new populations, for many populations where these studies have been done, we would encourage researchers to take an additional step. Therefore, the final objective for this review is to provide several recommendations for future research in the field of primate behavioral endocrinology with respect to stress physiology.

- **Focus on how GCs affect performance.** For populations where GCs are routinely measured, researchers can begin to examine the relationship between GC levels and performance. Performance measures can be internal measures related to health and condition (e.g., parasites, cytokines, wound healing, other hormones), behaviors known to be related to reproductive success (e.g., mate acquisition, rank acquisition, mate choice, parental behavior), and/or behaviors known to be related to survival and longevity (e.g., alarm calling, foraging, vigilance, sociality).
- **Focus on how GCs affect fitness.** Similar to the above, researchers can begin to identify the relationship between GC levels and fitness measures (estimated or actual) in their populations. These fitness measures need not be lifetime reproductive success, but can be proxies for fitness or estimates of fitness (e.g., tenure as an alpha male, interbirth interval, fertility rate, infant survival). For example, a study on mountain white-crowned sparrows (*Zonotrichia leucophrys oriantha*) found higher survival for individuals with higher baseline and stress-induced GC levels, but higher fecundity for individuals with higher baseline but lower stress-induced GC levels (Patterson et al., 2014).
- **For both of the above, control for environmental challenges.** Whenever researchers compare hormones from individuals across different environments, they are making an apples-to-oranges comparison (see examples in Dantzer et al., 2016). Rather, researchers should focus research efforts on measuring individual GC differences *within* stress-inducing categories (e.g., dry season, drought, low rank, elevation in predation pressure) or by including environmental challenges in a statistical model.
- **Remember that GCs are metabolic hormones first** (and stress hormones second). Researchers should continue to consider and control for differences in metabolic activity due to reproductive stages, life-history stages, and other seasonal variables. Some animals will be more metabolically active than others (e.g., pregnant females) and thus will have higher GCs. This should not be interpreted as a "stress response" for these individuals. Adopting a metabolic framework (as the Reactive Scope Model promoted by Romero et al., 2009) will enhance our interpretation of how GCs link the environment of an individual with their fitness.
- **Use acute disasters as "natural experiments"**. Researchers should try to take advantage of natural and/or anthropogenic disasters when a population is suddenly faced with a singular, but severe, environmental challenge (e.g., El Niño, an increase in predators, a decrease in prey, bush fires, anthropogenic effects). The idea here is to determine if some individuals are more or less likely to survive (and/or reproduce) than others, and whether these differences can be attributed to an individual's stress response.
- **When possible, check for evidence of HPA dysregulation.** Researchers should try to take advantage of more invasive opportunities to examine whether individual animals exhibit dysregulation of the HPA axis. Recall that a "good" response is one that exhibits a low GC baseline (under no stressors), a fast GC increase, and a rapid induction of negative feedback. Try to obtain some of these measures when possible.

In a review of primate field endocrinology, Higham (2016) notes that the only appropriate way to consider hormones is in an evolutionary context – that hormones such as GCs provide a set of physiological instructions for the body to respond (in terms of physiological, behavioral, and life history adjustments) to the inputs from a particular environment (Higham, 2016). Taking this next step towards identifying the adaptive significance of the stress response in primate studies will not only fill in this long-standing gap in our understanding of this widely-studied phenomena, but it will also help us to make predictions about how particular primate populations might respond to new environmental challenges.

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