BIOL PSYCHIATRY
1990;28:862–878

A.E. BENNETT AWARD PAPER

Adrenocortical Function, Social Rank, and Personality Among Wild Baboons

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Individual Differences and the Adrenocortical Axis

Biological psychiatrists have long been enamored with the hypothalamic-pituitary-adrenal (HPA) axis, and understandably so. It is a wonderously complex endocrine axis; consider the multifactorial hypothalamic control of adrenocorticotropic hormone (ACTH) release (Antoni 1986) or the differing time domains of glucocorticoid feedback inhibition (Keller-Wood and Dallman 1984). The axis is exquisitely sensitive to environmental perturbations whose effects can be extremely persistent. For example, 2 weeks of perinatal stimulation alters HPA activity for a rat's lifetime (Meaney et al 1988). The axis is hyperactive in depression and Alzheimer's disease, and this hyperactivity matters, given the pathogenic consequences of HPA hyperactivity (Munck et al 1984). How could anyone not be charmed by this neuroendocrine system?

For 12 years, I have indulged my own fondness for the HPA axis with a rather unique study population: I have studied the psychoendocrinology of wild baboons living in the Serengeti plains of East Africa. I have asked (1) How do personality and dominance rank in this primate society, as well as the *type* of society itself, influence HPA function? (2) What are the neuroendocrine mechanisms that mediate these differences in HPA function? (3) What are the consequences of these differences? The findings have been surprising, subtle, and, I believe, relevant to the psychoendocrinology of our own lives.

The HPA axis (along with the sympathetic nervous system) dominates the stress response. Stressors, perceived in the brain, cause hypothalamic release of cortisol-releasing factor (CRF), which triggers pituitary release of ACTH. This, in turn, causes adrenal secretion of glucocorticoids. CRF is the principal, but not sole releaser of ACTH. Instead, various ACTH "secretagogues" (including vasopressin, oxytocin, and catecholamines) stimulate ACTH release and/or augment CRF action (Antoni 1986). Evidence is also accumulating for the existence of inhibitors of ACTH release (Graf et al 1985; Grossman et al 1986). This multiplicity of regulators allows for neural coding such that different stressors have different orchestrations of secretagogues (Antoni 1986).

The extent of HPA activation is sensitive to stress in a surprisingly linear fashion. For example, increasing the magnitude of a somatic stressor (a greater magnitude of blood loss during hemorrhage) (Gann 1969) or a psychological stressor (increasing the degree

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Received April 18, 1990; revised June 11, 1990.

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0006-3223/90/$03.50

of novelty in a new environment (Levine et al 1989) increases glucocorticoid secretion linearly.

Once secreted, glucocorticoid actions typify the two-edged nature of the stress response. The steroids are vital for surviving physical stressors; they mobilize energy, increase cardiovascular tone, and suppress unessential anabolism such as growth, reproduction, and inflammation. However, glucocorticoid excess can cause steroid diabetes, myopathy, hypertension, and reproductive and immunosuppression (Munck et al 1984). Thus, optimal HPA function involves minimal secretion in the absence of stress, but robust secretion during stressors.

Like most endocrine axes, the HPA axis is under feedback control, such that elevated glucocorticoid concentrations inhibit subsequent HPA activity. This inhibition occurs at the brain and pituitary with varying time domains. Over the course of minutes, regulation is rate-sensitive; the rate of change of the circulating steroid signal determines the strength of feedback. This involves inhibition of release of preexisting pools of hormones. Over hours, the strength of feedback is determined by the level of steroid concentrations achieved, and this delayed feedback involves inhibition of synthesis of new hormones and of release of preexisting pools (Keller-Wood and Dallman 1984).

Given this complex framework, questions arise about the causes, correlates, and consequences of individual differences in HPA. For example, what accounts for differences in basal or stress-induced secretory rates, or in the speed of initiating or terminating a stress response? These mechanistic issues are the building blocks for studying larger issues: Why do bodies (and psyches) respond to stress differently? Why do individuals differ in their vulnerability to stress-related disease?

Olive Baboons and Social Dominance

These were questions I sought to answer in studying baboons. A wild population was preferable in that behavior would not be distorted by the spatial or demographic constraints of captivity. Moreover, wild animals experience normal stressors and pathogens, and can be followed over their life-spans. This allowed me to test whether findings emerging from study of stress physiology in the laboratory applied to a more natural setting.

Olive baboons (*Papio anubis*) were ideal for these studies. They are intelligent, long-lived, and reside in social troops of 50–200 individuals (Figure 1). They are large and thus easily observed in the open grassland. Their ecosystem is rich, and animals spend minimal time feeding; they have few worries about predators (cf. Smuts 1986; Strum 1982). Critically, this leaves them hours each day to devote to generating social stressors for each other, much as in our own ecologically buffered lives.

Central to these social stressors is the dominance hierarchy and among the males that I study, social rank is the best predictor of quality of life; thus, my first studies concerned the relationship between rank and HPA function. Even in the richest ecosystem, resources and social perks are finite and are divided unevenly by rank. Fairly linear dominance hierarchies emerge, with rank determining ease of access to food, social grooming, resting sites and, to some extent, to sexual partners.

Attaining and maintaining high rank is a vastly complex task, part of the attraction of these animals. It can involve aggression or the threat of aggression. Male baboons fight frequently, often inflicting severe injuries, and rank can rest on the outcome of a fight. In addition, baboons have various conventionalized gestures that threaten aggression—for example, displaying canines close to the face of a rival with a "threat yawn"—and often substitute for aggression itself.

In addition, rank is determined by skill at handling situations that can only be defined as social stressors. For example, a male may be in a sexual consortship with an estrus female, attempting to maintain exclusive sexual access to her for days. Throughout, he may be harassed by another male, who, though not overtly threatening, may simply shadow so closely as to make it impossible for the first male to mate, feed, or rest. Often, the first male voluntarily relinquishes the consortship. As another example, forming a cooperative coalition with another male can be extremely advantageous in a fight; however, when the fight actually occurs, the male frequently fails to aid his coalitional partner, or even defects to the opposing side.

Thus, high rank can require more than mere strength, body mass, or canine length. At least as important, for example, is the ability to predict and control social circumstances and the capacity to form cooperative affiliations and to defect from them at advantageous times. In short, social skill, a taste for game theory, and the ability to endure social stress are highly rewarded. I pondered whether males succeeding in this enterprise had stress responses that differed from lower-ranking animals.

To study this, animals are observed in a manner to preclude bias (e.g., individuals are observed in a random sequence to avoid observing only those who are doing something interesting at that time) (Altmann 1971). Dominance hierarchies are generated from outcomes of approach–avoidance interactions; for example, who avoids whom or who is supplanted from a feeding or resting site by whom (Strum 1982; Packer 1977). For the less ethologically minded readers, it is worth noting that this is the basis for ranking systems used by most primatologists, rather than constructing hierarchies based on outcomes of overt fights; nevertheless, the two types of rankings correlate significantly (and ranking in neither correlates significantly with the extent of reproductive activity by adult males).

To obtain endocrine data, males are anesthetized with phencyclidine injected with a syringe fired from a blowgun (Figure 2). The anesthetic itself does not effect HPA function within this time span (Sapolsky 1982). Subjects are darted at the same time of day in order to eliminate circadian fluctuations in hormone values. Animals are not darted if they are ill, injured, have recently mated, or have had a fight to avoid distorting basal values. Animals must be unaware that they are targeted for darting to avoid anticipatory stress. Finally, a first blood sample must be taken within a few minutes of the onset of the anesthetic effects; at that time, glucocorticoid secretion has not yet increased in response to the stress of anesthetization; thus, the first blood sample still reflects basal values (in contrast, this lag time is sufficient to alter secretion of ACTH; thus basal ACTH concentrations can never be measured).

Socially Subordinate Baboons are Hypercortisolemic

Over the years, subordinate males consistently have higher basal concentrations of cortisol than do dominant males (Figure 3). Despite their lower basal concentrations of cortisol, dominant males mount as large and as rapid a cortisol stress response as subordinates due to a larger and faster rise in cortisol concentrations at the onset of a stressor (Sapolsky 1982).

The hypercortisolism of subordinate males is not surprising. Subordinate males are subject to far more stressors than are dominant males. The former have their feeding disrupted at the highest rates; a male may laboriously dig a tuber out of the ground, only to have it seized by a dominant male. Subordinates are the most likely to have

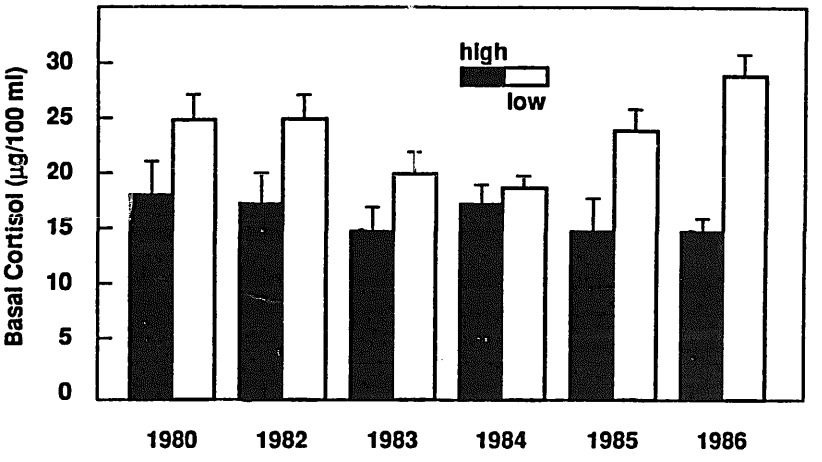


Figure 3. Basal cortisol concentrations of the 6 highest-ranking and 6 lowest-ranking males in each of 6 years of study. Total number of males under study/year ranged from 12 to 16. Values are derived from a single anesthetization/animal/season, as described in the text. Mean ± SEM. (From Sapolsky 1989.)

grooming and sex disrupted. Probably of greatest stressfulness, subordinates are subject to the highest rates of displaced aggression—a dominant male when losing a fight will often attack a subordinate bystander without warning (Strum 1982; Sapolsky 1983a; Smuts 1986). For a subordinate male, life is filled with lack of control, predictability, or outlets for frustration, all potent psychogenic stressors (Weiss 1970, 1984; Levine et al 1989). Supporting these data, social subordinance is associated with HPA hyperactivity among rodents, other primate species in stable dominance hierarchies, and carnivores (Archer 1970; Barnett 1955; Bronson and Eleftheriou 1964; Davis and Christian 1957; Fox and Andrews 1973; Golub et al 1979; Louch and Higginbotham 1967; Manogue et al 1975; Popova and Naumenko 1972; Sassenrath 1970; Southwich and Bland 1959).

An interesting exception to the pattern occurred in 1984, when subordinate males had the lowest basal cortisol values of any year. That was the year of the tragic East African drought when grassland biomass decreased 75%. Body weight did not decline, but baboons spent significantly more time foraging each day (Sapolsky 1986). The most striking behavioral effect of this difficult period was a 78% reduction in the rate of aggression, including displaced aggression. Such drought-induced reduction in aggression has been noted previously, suggesting that much of the aggression occurring among social species during more affluent times represents "behavioral fat." Given that subordinates are the most frequent victims of displaced aggression, the 1984 data suggest, ironically, that the drought and its behavioral consequences were a hidden blessing for subordinate individuals.

The Hypercortisolism of Subordinate Males is Probably Neural in Origin

If, as I suggest, the hypercortisolism of subordinate males reflects the stressfulness of their lives, such hypersecretion should be driven at the central nervous system (CNS) level. Alternatively, the excessive secretion could reflect a change in the HPA peripheral dynamics. My work suggests that the hypersecretion is of CNS origin.

The hypercortisolism of subordinate males does not appear to arise from a slower cortisol clearance rate. As evidence, the half-life of circulating 3H-cortisol in baboons was independent of basal cortisol concentrations (Sapolsky 1983b).

The hypercortisolism does not appear to arise from enhanced adrenal sensitivity to ACTH. Over a broad and relatively physiological range of cortisol values elicited by differing doses of ACTH, high-ranking and low-ranking males did not differ in their responsiveness to the peptide (Figure 4). The hypersecretion also is unlikely to be pituitary in origin. Subordinate males not only did not have enhanced pituitary sensitivity to CRF, but even had blunted responsiveness to the secretagogue (Figure 5) (Sapolsky 1989). This could arise at least two different ways. First, the elevated cortisol concentrations could exert an enhanced negative feedback signal at the pituitary, damping its responsiveness to CRF. Alternatively or additionally, the pituitary may lose intrinsic responsiveness to CRF. The latter mechanism appears to underly the damped pituitary responsiveness to CRF in subordinate baboons. As evidence, baboons were injected with metyrapone, which rapidly inhibits adrenal steroidogenesis and markedly decreases circulating cortisol concentrations. In the absence of such a steroid feedback signal, subordinate males were still hyporesponsive to CRF (Sapolsky 1989), showing that the pituitaries were intrinsically less responsive. Whether they were also less responsive because of the enhanced cortisol feedback signal that normally occurs was not determined.

Regardless of which mechanism causes the damped pituitary responsiveness to CRF, the interpretation is the same: given the preeminant role of CRF as an ACTH secretagogue and the normal adrenal sensitivity to ACTH in subordinates, if the pituitary of subordinates is less sensitive to CRF—yet these males normally hypersecrete cortisol—then there must be hypersecretion from the hypothalamus. The onus of dysfunction shifts to the brain.

Hypercortisolism Among Baboons and Feedback Resistance

If subordinate males are hypercortisolemic because they are stressed frequently, then their hypercortisolism should be driven by the brain, where the stressors are sensed. The data thus support this "psychosocial" explanation for the hypercortisolism. However, additional data support a more "neuroendocrine" explanation for the hypersecretion, in that baboons with elevated basal cortisol concentrations are also dexamethasone resistant.

In these studies, it was not possible to administer the classic Dexamethasone Suppression Test (DST) (Carroll 1972); baboons were anesthetized throughout, dexamethasone could not be administered the evening before, and animals were only monitored for 6 hr. Nevertheless, striking differences emerged. The higher the basal cortisol concentrations, the slower and smaller was the response to dexamethasone (Figure 6) (Sapolsky 1983b). Thus, the hypersecretion of subordinate males could be due to impaired feedback sensitivity. Their damped pituitary sensitivity to CRF can be viewed as a compensation for the neural hypersecretion.

Thus, there is a psychosocial explanation for the hypercortisolism of subordinates—they are more stressed—and a neuroendocrine explanation—they have damped sensitivity to glucocorticoid feedback. Yet, these differing explanations are interrelated. A glucocorticoid negative feedback signal is mediated by binding of the hormone to its receptor, and loss of such receptors blunts feedback efficacy. Critically, stress and its associated hypersecretion of glucocorticoids causes down-regulation of such receptors, producing feedback resistance.

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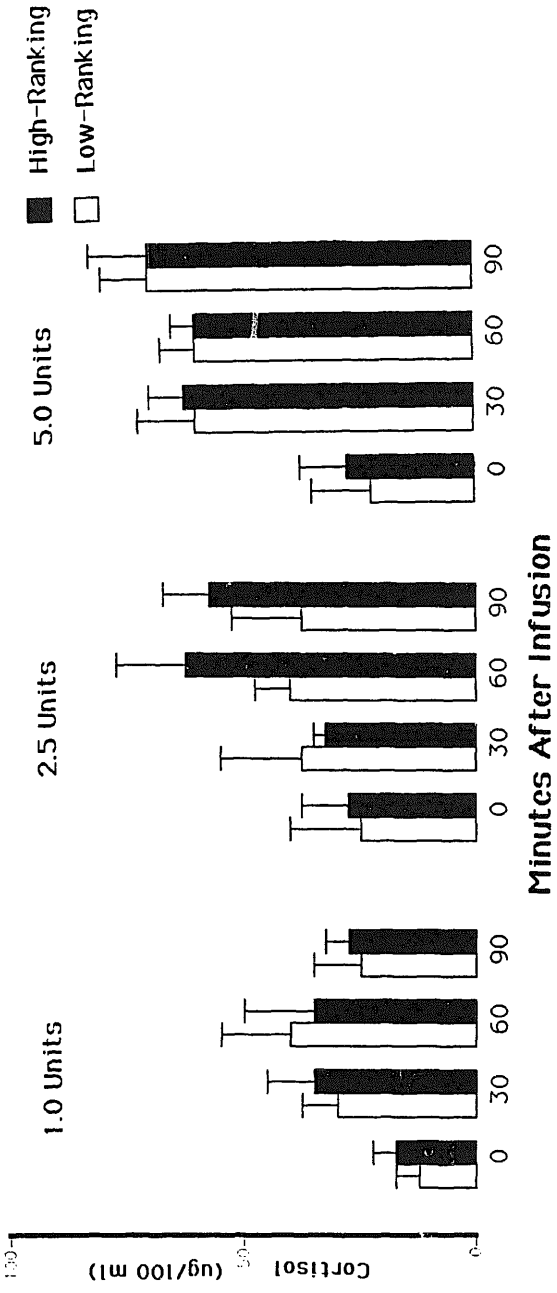


Figure 4. Adrenocortical responses to ACTH challenges, according to rank. High-ranking and low-ranking adult males (n = 6/group) were infused IV with indicated quantities of ACTH (ACTHAR, porcine ACTH), and cortisol concentrations were determined at indicated times after the challenge. The study was initiated 1 hr after anesthetization; thus, the initial sample does not represent basal values, but rather those in response to the stressor of anesthetization. The two groups did not differ, as assessed by ANOVA. Mean ± SEM. (Sapolsky, unpublished data from a 1989 study.)

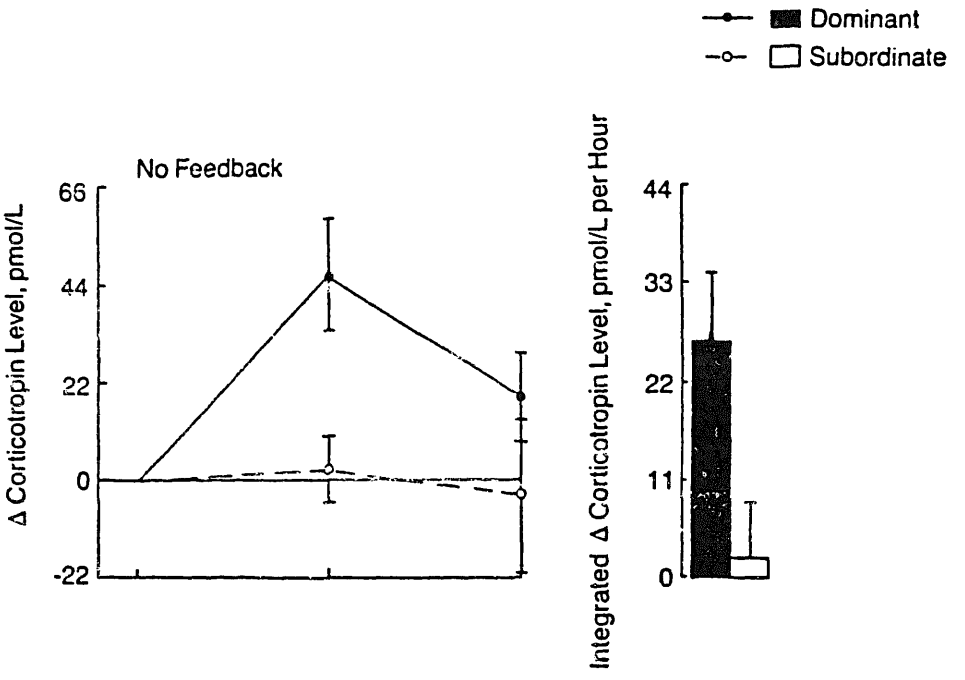


Figure 5. Absolute change in the circulating corticotropin concentrations in response to corticotropin-releasing factor (CRF) challenge among high-ranking and low-ranking males (n = 6/group), in the absence of cortisol feedback (following metyrapone administration). Dominant males were significantly more responsive to CRF. Data points were taken at 0, 15, and 30 min. Right: This rank difference was also apparent when the areas under the curve of the data from the left were generated. Dominant animals had significantly greater integrated corticotropin values than did subordinates. Mean ± SEM. (From Sapolsky 1989, from studies in the 1986 and 1987 seasons.)

This occurs in the hippocampus, one of the principal neural sites that mediate glucocorticoid feedback. The structure has high concentrations of both types of corticosteroid receptors (McEwen et al 1986). Hippocampal damage causes elevations of secretagogue mRNA levels and of circulating concentrations of secretagogues, ACTH, and glucocorticoids. Furthermore, hippocampal stimulation inhibits the axis, as does microimplantation of glucocorticoids in the structure (reviewed in Sapolsky et al 1989a, 1989b). Thus, the hippocampus is a feedback "brake" upon the axis. With chronic stress, the excessive glucocorticoids cause corticosteroid receptor down-regulation; this is a regulatory feature of all endocrine systems. The hippocampus, with its abundant corticosteroid receptors, is the most sensitive brain region to stress-induced receptor down-regulation (Sapolsky et al 1984b), and afterward, feedback is impaired; rats are feedback resistant and hypersecrete glucocorticoids basally and after the end of stress (Sapolsky et al 1984a). In humans, chronic stress also causes feedback resistance (although the intervening steps concerning receptor number have not been studied) (Baumgartner et al 1985; Ceulemans et al 1985).

Thus, I speculate that subordinate males are hypercortisolemic initially because they are stressed frequently. But with time, the stressors cause receptor down-regulation, and feedback resistance and hypercortisolism should also occur basally. Sapolsky and

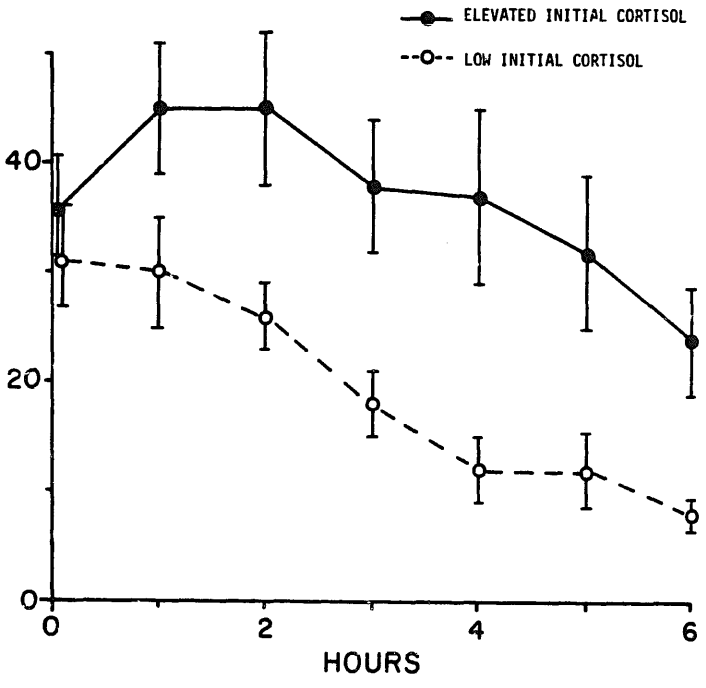


Figure 6. Cortisol concentrations after 5 mg dexamethasone administration. Subjects were divided into the 50% with below-average basal cortisol concentrations (solid line, $n = 5$, consisting of 4 high-ranking males and 1 low-ranking male, with mean basal cortisol concentrations of 13 ± 2 μg/100 ml) and the 50% with above-average concentrations (broken line, $n = 6$, consisting of 3 low-ranking and 3 middle-ranking males, with a mean basal cortisol concentration of 36 ± 4 μg/100 ml). The two groups did not differ in body weight. (From Sapolsky 1983b, based on studies from the 1982 season.) We recently replicated the observation that dominant male baboons are more responsive to dexamethasone than are subordinates (in a study of 60 male yellow baboons [*Papio cynocephalus*]) (Sapolsky and J. Altmann, in preparation).

Plotsky (1990) explain details about the experimental underpinnings of these speculations.

The Hypercortisolism of Subordinate Baboons and Depressed Humans

Many features of this story resemble the HPA abnormalities in depression. A vast literature demonstrates the hypercortisolism and dexamethasone resistance in depression (cf. APA Taskforce 1987). The role of stress as a predisposing factor in depression has been emphasized (Anisman and Zacharko 1982; Gold et al 1988). In that vein, we have speculated about corticosteroid receptors and feedback regulation to explain why only some depressives are dexamethasone resistant (Sapolsky and Plotsky 1990). Given those themes, how similar is the hypercortisolism of the subordinate baboon and the depressed human?

Initially, quite similar, basal hypercortisolism occurs in both cases. In subordinate baboons, this has only been studied in the morning. In depressives, hypercortisolism

can occur throughout the circadian cycle, but is most pronounced in the evening (Sachar et al 1980). In both instances, there is feedback resistance, as manifested by dexamethasone resistance, and the hypersecretion is at least partially neurally driven, as evidenced by blunted responsiveness to CRF in depressives (Holsboer et al 1984; Gold et al 1986; Risch et al 1988) and subordinate baboons. Finally, in both cases, the hypercortisolism might be pathogenic. Subordinate baboons have suppressed HDL-cholesterol concentrations (Sapolsky and Mott 1987) and lymphocyte counts (Sapolsky and Mott, unpublished data), and both appear to arise from the hypercortisolism. In depressives, the hypercortisolism can be somewhat immunosuppressive (Calabrese et al 1987).

Uncertainties and divergences between the two models occur involving the causes of the hypersecretion. First, in neither case is it clear *which* hypothalamic secretagogue is hypersecreted. Even if pituitary responsiveness to CRF is decreased, it need not be CRF that is hypersecreted; the blunting could be a compensation for hypersecretion of another secretagogue. CRF may be the culprit; cerebrospinal fluid (CSF) concentrations of CRF are elevated in depressives (Nemeroff et al 1984; Banki et al 1987). However, CSF CRF reflects both hypothalamic and nonhypothalamic CRF, and circadian fluctuations in CSF CRF concentrations and HPA activity are not parallel (Garrick et al 1987; Kalin et al 1987); thus, aberrant CSF CRF concentrations need not imply aberrant concentrations of hypothalamic CRF. Pituitary responsiveness to vasopressin is also diminished in depressives (Carroll 1972; Krahn et al 1985), and hypersecretion of both CRF and other secretagogues [vasopressin (von Bardeleben et al 1985) or catecholamines (Lamberts et al 1986)], may occur; others have questioned the physiological relevance of these studies (Hermus et al 1987). At present, no data are available with the baboon model as to which secretagogue is involved.

Second, it is not certain in either model why the pituitary is less responsive to CRF. As noted, it could be due to enhanced glucocorticoid feedback inhibition and/or intrinsic loss of responsiveness to CRF. In the baboons, the blunted ACTH response in the presence of metyrapone suggests intrinsic loss of responsiveness. However, it is not known whether the enhanced feedback signal adds to the blunting. In the human, it is not clear which mechanism applies. One recent study suggests that the problem is enhanced feedback, because following metyrapone treatment, depressives had a large ACTH response to CRF (von Bardeleben et al 1988). Unfortunately, however, metyrapone-treated depressives were compared with healthy controls rather than with metyrapone-treated healthy controls, making it impossible to tell if there is any intrinsic loss of sensitivity.

Third, the two models differ in adrenal function. In depression, adrenals can be hypertrophic and hypersensitive to ACTH. This is shown with ACTH challenges, with imaging studies of adrenal size in situ, and by postmortem study (Nasr et al 1982; Gerkin and Holsboer 1986; Risch et al 1988; Gold et al 1986; Holsboer et al 1984; Amsterdam et al 1987, 1989; Dorovini-Zis and Zis 1987). It has thus been proposed that long-term depressive hypercortisolism causes adrenal hypertrophy, overriding the protective effects of the compensatory pituitary blunting (Gold et al 1988). The ACTH challenges of the baboons (Figure 4) indicate no adrenal hypersensitivity in subordinate males. Though the sample size is small, there was also no trend for adrenal sensitivity to increase with longer tenures as a subordinate (which would parallel the idea that the adrenal hypertrophy emerges only with long-term depression).

Finally, some of the dexamethasone resistance of depressives might be due to a shorter dexamethasone half-life; that is, less of an inhibitory signal reaches feedback sites (Lowry and Meltzer 1987). No data concerning this are available from the baboons.

Thus, the two models share the features of hypercortisolism arising from feedback resistance, and an involvement of the brain in this dysfunction. However, they are clearly not identical in terms of all the neuroendocrine details.

Is the Hypercortisolism of Subordinate Baboons Actually Related to Social Rank?

These studies suggest that social rank is an important and straightforward factor explaining individual differences in HPA function. Among complex social primates, however, it is far from straightforward. First, social ranks change over time. Second, the physiological correlates of dominance depend on the sort of society in which the dominance occurs. Finally, the physiological correlates of dominance may be more related to personality than to the dominance itself. These complexities are described below.

Social Ranks Change Over Time

Among baboons, a young growing male may eventually reverse the direction of dominance with a rival, a top-ranking male may sustain an injury, or a new male may join the troop and change the pattern of coalitions. Any of these conditions could cause waves of change in the rankings. This plasticity has an important consequence: predictions about the eventual pathogenic consequences of HPA differences must incorporate the changing status of these animals. Any season's physiology is just a still photograph of a very dynamic system.

The Physiological Correlates of Dominance Depend on the Sort of Society in Which the Dominance Occurs

There may be a consistent correlation between one's dominance rank and HPA function, but is this relationship causal? For example, dominant males tend to be prime-aged. Perhaps low basal cortisol merely results from that factor. My subsequent work suggests that the physiology does arise from rank, but is sensitive not merely to rank but to the social setting in which the rank occurs. Typically, there is a number 2 ranking male who is an heir apparent to number 1. In 1981, however, there was no male in that role. Instead, a coalition of ranks 2–7 formed and crippled the highest-ranking male in a fight, removing him from social competition. The coalition then disintegrated; any of the 6 males dominated the other males in the troop, but among themselves, sustained social instability ensued. Ranks changed daily, rates of fights and injuries increased, feeding and sexual consortships declined, coalitions formed and disintegrated rapidly. (In contrast, in other stable seasons, ranks did not shift significantly during the 3 annual months of study, and within individual dyads, the dominant individual of the pair won an average of 97% of the interactions (Sapolsky 1983a).

Critically, during this social instability, the psychological advantages of dominance in a stable setting disappeared—now, being in this top-ranking cohort involved highly

charged unpredictability and lack of control. Sitting precariously atop a shifting hierarchy is, psychologically, very different from sitting atop a stable one; just consider the likely psychological state of the Romanovs during the winter of 1917.

During the unstable 1981 season, a very different endocrine picture emerged from that in stable seasons. Dominant males were now as hypercortisolemic as were subordinates and their HPA stress responses were as sluggish. Features of testicular function unique to dominant males in stable seasons had also disappeared (Sapolsky 1983a).

Similar findings emerge with studies of captive primates, where instability can be induced by forming new social groups. Dominant males in unstable captive groups have cortisol concentrations at least as high as subordinates (Keverne et al 1982; Mendoza et al 1979; Coe et al 1979; Chamove and Bowman 1976), and do not have distinctively rapid stress responses (Coe et al 1979). As time passes and social relations stabilize, dominant males show lower basal cortisol concentrations and greater cortisol secretion in response to stress, like dominant baboons in the wild during stable seasons (Keverne et al 1982; Manogue et al 1975). This has two important implications. First, rank is not arising from the physiology; in studies of captive primates, HPA measures prior to group formation rarely predict eventual rank. Second, there is no single HPA profile of dominance. Instead, it depends on the type of society in which it occurs, and social stability seems to be a critical variable. Low basal cortisol concentrations occur in dominant males only when dominance means high degrees of social control and predictability.

The Physiological Correlates of Dominance May be More Related to Personality Traits Than to the Dominance Itself

Social primates do not merely come in two flavors—dominant or subordinate—nor can they be reduced to a simple rank. These complex individuals differ in their behavioral *traits*. Males differ as to how often they form successful cooperative coalitions, how often they play with infants, whether they displace aggression after losing a fight, and so on. Do males of similar ranks but with different behavioral traits have the same rank-specific HPA function?

We formalized different behavioral traits among dominant males, and then analyzed their HPA correlates. We found that low basal cortisol concentrations were not a marker of dominance at all, but rather a marker of a certain subset of dominant males. Dominant males without those traits were as hypercortisolemic as subordinates.

The traits themselves are striking. Dominant males with any of the following had low basal cortisol concentrations (Figure 7) (Sapolsky and Ray 1989; Ray and Sapolsky, unpublished data).

1. Differentiating well between neutral and threatening actions of a rival (as manifested by whether the subject acts differently after each of these events).
2. When a rival is indeed threatening, controlling the situation by initiating the fight.
3. Differentiating between winning and losing a fight.
4. Displacing aggression onto a third party if a fight is lost.
5. Having high rates of interactions with infants, and nonsexual interactions with females.

All of these traits occurred independently of rank; that is, these "low-cortisol" males were not merely the most dominant of the dominant cohort.

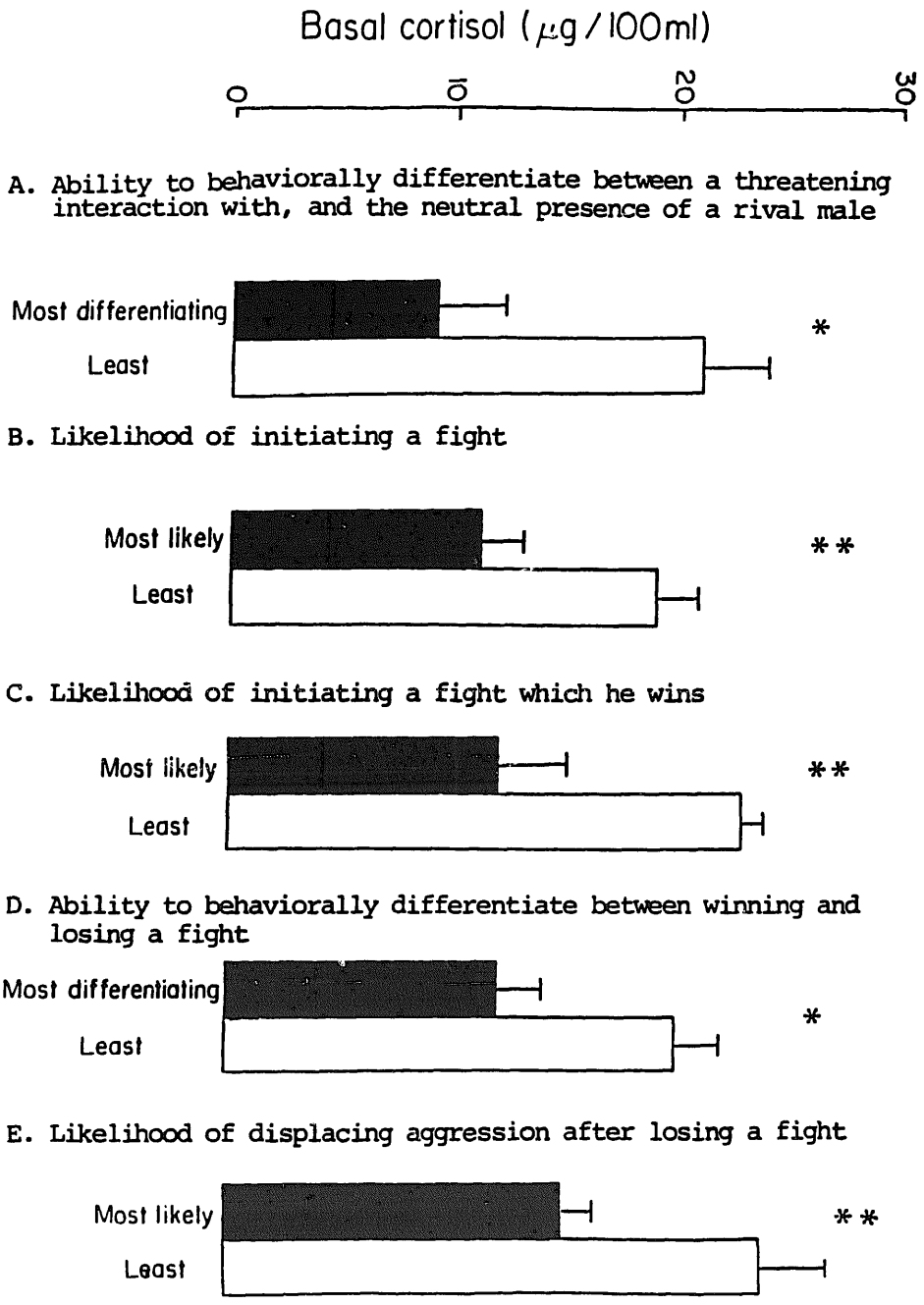


Figure 7. Behavioral extremes that were associated with differing basal cortisol concentrations. Numbers indicate circulating cortisol concentrations in μg/100 ml. (From Sapolsky and Ray 1989.) *$p < 0.05$. **$p < 0.01$.

If one were giving stress management courses to baboons, these are the behaviors he or she would encounter. They reflect high degrees of social skillfulness, predictability, control, outlets for frustration, and social affiliation. Showing this further, the low cortisol males not only tended to initiate their fights, but they won more of the fights they initiated than the ones they did not initiate (in contrast to the usual picture with baboons), that is, they pick the right fights. These traits appear to be beneficial, as low-cortisol dominant males stay in the dominant cohort significantly longer than do high-cortisol dominant males. Moreover, their behavioral traits and low cortisol secretion are evident from the beginning of their dominant tenure, and are consistent over a number of years, suggesting that these may represent stable personality traits.

Thus, what initially seemed to be an endocrine marker of dominance actually marks a style of dominance. The 1981 season suggested that low basal cortisol concentrations are seen in dominant males only when the hierarchy is stable, that is, only when there are psychological advantages to dominance. The subsequent study of behavioral traits suggests that low basal cortisol concentrations are then seen only in dominant males who can perceive those psychological advantages. The magnitude of this effect is striking: the difference in cortisol concentrations between dominant and subordinate males is smaller than the difference among dominant males with different behavioral traits. (The obvious studies of styles of subordinance are now in progress.) These findings echo the classic ones demonstrating hypercortisolism among parents of children dying of cancer (Friedman et al 1963; Wolff et al 1964): the extent of hypercortisolism was very sensitive to the coping style of the parents, with less secretion among those with religious rationalizations about the illness, those who denied the facts of the disease, or those who could lose themselves in the details of managing the disease.

Conclusions

Selye and his intellectual descendants conceived of the HPA axis purely in terms of the magnitude of the external stressor, the extent to which homeostatic balance was disrupted by an insult. The studies showing, for example, the linearity between the magnitude of a hemorrhage and the extent of HPA activation (Gann 1969) are within that intellectual tradition. It is a vastly difficult yet pleasing task to try to understand the axis in these purely bioengineering terms. Yet Weiss's rat studies (1970, 1984), the studies of the parents of children with cancer, and these baboon personality studies show that the axis is too rich and complex to be studied only on these mechanistic levels. In each case, two organisms are exposed to equivalent external insults—a schedule of electric shocks, the pain of watching a child with cancer—a rank that cannot be changed. Yet, the psychological filters with which those insults are perceived influence the HPA response at least as much as do the insults themselves. It is this psychoendocrine level of study of these baboons that is most satisfying and one that I believe will most likely generalize ourselves: the realization that these baboons are subtle enough to see life either as glasses half full or half empty. Their physiology and no doubt, our own, is profoundly sensitive to this difference.

These studies were made possible by the longstanding generosity of the Harry Frank Guggenheim Foundation. Field assistance was supplied by Richard Kones, Francis Onchiri, Hudson Oyaro, Diane Rich, Lisa Share, and Reed Sutherland.

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