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SOCIAL STATUS AND HEALTH IN HUMANS AND OTHER ANIMALS

Robert M. Sapolsky
Departments of Biological Sciences, Neurology, and Neurological Sciences, Stanford University, Stanford, California 94305-5020; Institute of Primate Research, National Museums of Kenya, Karen, Nairobi, Kenya; email: sapolsky@stanford.edu

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■ **Abstract** Dominance hierarchies exist in numerous social species, and rank in such hierarchies can dramatically influence the quality of an individual's life. Rank can dramatically influence also the health of an individual, particularly with respect to stress-related disease. This chapter reviews first the nature of stress, the stress-response and stress-related disease, as well as the varieties of hierarchical systems in animals. I then review the literature derived from nonhuman species concerning the connections between rank and functioning of the adrenocortical, cardiovascular, reproductive, and immune systems. As shown here, the relationship is anything but monolithic. Finally, I consider whether rank is a relevant concept in humans and argue that socioeconomic status (SES) is the nearest human approximation to social rank and that SES dramatically influences health.

INTRODUCTION

Most of us were subjected in ninth-grade biology to a barely remembered concept that forms a cornerstone of physiology. This is the idea of homeostasis, in which various physiological endpoints—blood pressure, heart rate, body temperature, and so on—are at their optimal levels. [The term allostasis has been introduced to update the homeostatic concept. The older term implies a single optimal set point for any measure, maintained by local regulatory mechanisms, whereas allostasis encompasses the fact that regulation is organism wide and that optimal set points are constantly in flux (Schulkin 2003).]

Regardless of the term used, maintaining physiologic balance is essential to health. A stressor is any physical or psychological factor that perturbs or threatens to perturb homeostasis, and stress is the state of homeostatic imbalance. The body reestablishes homeostasis by marshalling neural and endocrine adaptations that collectively constitute the stress-response.

The environment produces endless means of perturbing homeostasis. Evading a predator, pursuing a prey, a sustained drought, or the harassments of social subordination all challenge homeostasis and mobilize the stress-response. Such mobilization can be life-saving; yet, a central point of this chapter is that excessive activation of the stress-response increases the risk of various stress-related diseases.

Naturally, there is considerable interindividual variation in how readily stress-related disease occurs. This arises from variation in (*a*) the amount of stressors an organism is exposed to (frequency, duration, and severity); (*b*) the adaptiveness of the stress-response mobilized (whether it is activated only in response to legitimate stressors, as well as the speed and magnitude of its activation and recovery); and (*c*) the sources of coping available.

Here, I review one source of variability among social animals: How does the rank of an individual in a social hierarchy influence the pattern of stressors to which it is exposed, the nature of the physiological stress-response it mobilizes, and its sources of coping? And thus, how does social rank influence patterns of stress-related disease?

Taking into consideration nonhuman species, with an emphasis on primates, I first outline the natures of the stress-response, stress-related disease, coping, and dominance hierarchies in social organisms. I review the relationship between social rank and stress-related physiology (recognizing that few readers are or wish to be physiologists). I then consider some critical factors that modulate the rank/physiology relationship. Finally, I consider whether rank/stress physiology relations are relevant to humans.

THE STRESS-RESPONSE AND ITS PATHOGENIC POTENTIAL

The Adaptive Nature of the Stress-Response

Why is it adaptive for the body to mobilize the same stress-response in the face of markedly differing stressors (e.g., evading a predator while injured as opposed to pursuing a prey while starving) (reviewed in McEwen 2002, Sapolsky 2004)? Despite obvious variability, most physical stressors place some similar demands on the body. Critically, coping with any acute physical stressor demands transfer of energy from storage sites to exercising muscle. Such transfer should be as rapid as possible, accomplished by increased blood pressure and heart rate. Furthermore, long-term building projects that are not essential to immediate survival are inhibited until more auspicious times. Such triaging includes inhibiting digestion, growth, tissue repair, and reproduction. Immune defenses are enhanced, pain perception is blunted, cognition is enhanced, and sensory thresholds are sharpened.

Activating some neural and endocrine systems, and inhibiting others, accomplishes these adaptations. The initial wave of activation occurs within seconds; the sympathetic nervous system is stimulated to release epinephrine (adrenaline)

from the adrenal glands and norepinephrine (noradrenaline) from nerve endings throughout the body. Within minutes, glucocorticoids (such as hydrocortisone or cortisol) are secreted from the adrenals as the final step of a cascade beginning in the hypothalamus. This triggers the release of a pituitary hormone that, in turn, stimulates the adrenals. Although there is stress-induced secretion of other hormones (e.g., glucagons, beta-endorphin, prolactin), the bulk of the stress-response is mediated by glucocorticoids and the sympathetic catecholamines (i.e., epinephrine and norepinephrine).

The primary neuroendocrine axis inhibited during stress is the parasympathetic nervous system, which works in opposition to the sympathetic system. The secretion of insulin, various digestive hormones, growth hormone, and reproductive hormones are also inhibited.

Collectively, these adaptations are essential for surviving an acute physical stressor. The precise pattern of stress-hormone secretion varies somewhat with each stressor (e.g., some stressors are dominated by catecholamine secretion, others by glucocorticoid secretion). Nonetheless, all stressors mobilize a broadly similar stress-response.

The importance of the stress-response is best demonstrated by diseases in which it fails (e.g., Addison's or Shy Drager's diseases, in which there is impaired secretion of glucocorticoids and sympathetic catecholamines, respectively). Such maladies, if untreated, can prove fatal when an individual attempts something physically taxing.

Chronic Stress and the Emergence of Stress-Related Disease

Despite the adaptiveness of the stress-response, chronic stress can be pathogenic. At one time, it was erroneously believed that chronic stress "exhausts" the stress-response (i.e., the adrenals become depleted of epinephrine and glucocorticoids, the pancreas is depleted of glucagon, and so on). However, the pathologies of chronic stress emerge because the stress-response, if chronically activated, can become as damaging as the stressor itself.

Thus, if energy is constantly mobilized, it is never stored, which produces muscle atrophy, fatigue, and an increased risk of insulin-resistant (adult-onset) diabetes. And, although hypertension is vital to sustaining a sprint from a predator, chronic hypertension damages blood vessels and, when combined with the metabolic stress-response, predisposes these vessels toward atherosclerosis.

Deferring digestion, growth, repair, and reproduction during an acute stressor is adaptive but, if chronic, increases the risk of peptic ulcers, irritable bowel syndrome, impaired growth and tissue repair, irregular ovulatory cycles, and erectile dysfunction. Furthermore, the stimulation of immunity in response to stress soon gives way to immune suppression and impaired defenses against infectious disease. Finally, although short-term stress enhances cognition, chronic stress disrupts it and impairs synaptic plasticity as well as the birth of new neurons, atrophies dendritic processes in neurons, and increases the incidence of neuron death.

These findings constitute a double-edged sword: In the face of a typical mammalian stressor (i.e., a brief physical challenge), the stress-response is essential. However, during chronic stress, the stress-response is pathogenic. This fact is particularly relevant to socially complex species such as primates that generate chronic stress for purely psychological reasons.

Psychological Stress

As noted, the stress-response can be mobilized in anticipation of a homeostatic perturbation (reviewed in Levine et al. 1989). If that perturbation does occur, the anticipatory stress-response is adaptive. But if the threat is imagined, the costly and disruptive stress-response will have been mobilized for no reason. Critically, humans and other cognitively sophisticated species may mobilize the stress-response for purely psychological or social reasons, in the absence of a threat to homeostasis, which explains our proclivity toward stress-related disease. Such psychological stressors may be local and ongoing events with no physiological reality (e.g., the stressfulness of public speaking), or they may be displaced in space and time (e.g., war on another continent or contemplation of one's eventual death). Obviously, social subordination often may involve physical stressors, but there can be a surfeit of psychological stressors as well. Thus, I review the components of psychological stressors.

LACK OF PREDICTABILITY A stress-response is more likely when an organism lacks predictive information about a stressor. This has been demonstrated in studies in which two rats received identical patterns of intermittent shocks. However, one rat was given a warning (e.g., a bell) five seconds before each shock and, as a result, was less likely to mobilize a stress-response or develop a stress-related disease than was the yoked control rat.

LACK OF CONTROL The importance of a sense of control was shown by a similar pairing of rats. In this case, one rat was trained previously in an active avoidance task (e.g., lever pressing) that decreased the likelihood of shock. In the experimental situation, the lever was disconnected (i.e., was a placebo). Nevertheless, lever pressing gave the rat a sense of control, thereby decreasing its risk of stress-related disease.

LACK OF OUTLETS FOR FRUSTRATION In this situation, two rats received identical shocks, but one had access to a source of distraction or displacement (food, a running wheel, a bar of wood to gnaw on), which prevented a stress-response. Importantly (and unfortunately), the ability to aggressively attack another individual is a highly effective coping outlet. Such "displacement aggression" is common among primates, including humans.

THE INTERPRETATION OF THE STRESSOR A severe abdominal pain can be a source of anxiety, which will likely elicit a stress-response. However, if the pain indicates

that a drug is effectively killing cells of a liver tumor, this same pain might elicit euphoria. Thus, the interpretation of an external stressor can influence the physiological response to it. An experimental demonstration of this effect might involve a rat receiving 10 shocks in the first hour and 20 in the second, while a second rat receives 50 shocks in the first hour, followed by 20 in the second. Thus, the first rat receives a total of 30 shocks and the second receives 70. Nevertheless, at the end of the second hour, the second rat will have the smaller stress-response, because the second rat's circumstances improved over time, whereas the first rat's worsened.

LACK OF SOCIAL SUPPORT Numerous studies demonstrate the capacity of social support to blunt the stress-response in the face of numerous homeostatic challenges.

SOCIAL RANK AND ITS RELATIONSHIP TO STRESS

Over the years, notions about the nature of dominance hierarchies among animals have varied. At one extreme, some investigators have considered rank as defining an animal's life, equating it with reproductive success and Darwinian fitness. In contrast, some have questioned whether hierarchy is meaningful to an animal or whether it is a construct imposed by humans (i.e., an animal merely categorizes conspecifics as dominate or subordinate to it, rather than assigning an ordinal rank) (Rowell 1974). Most investigators would agree that a hierarchy is meaningful to animals within it and that it is an emergent property of the group rather than the mere aggregation of individual dyadic relations (Chase et al. 2002); however, rank is not necessarily synonymous with Darwinian fitness (e.g., Bercovitch 1993).

Various criteria have been used to determine rank. Some investigators have based rankings on outcomes of agonistic interactions. At the other extreme, some have derived hierarchies from subtle attentional fields (patterns of eye contact and avoidance of eye contact among group members). Rankings based on the unequal distribution of contested resources have been most common.

Hierarchical structure varies among species. For example, hierarchies can be (*a*) gender-specific or involve both sexes; (*b*) hereditary or labile; (*c*) linear or contain circularities (i.e., A > B > C > A); or (*d*) situational, with rank fluctuating as a function of the resource contested or the presence of allies. There also is variability according to what sorts of stressors accompany a particular rank. I now consider the stressors associated with subordination in hierarchies in which subordination is a state imposed forcefully from above. As discussed in Why Are There Inconsistencies in These Data?, below, there are dramatic exceptions to this picture.

It is easy to imagine that subordination can produce an excess of physical stressors. Subordinate animals may have to work harder for calories, or be calorically deprived. If a member of a prey species, subordinate animals are more likely to be exposed to predators [by being forced into the periphery in variations on the Geometry of the Selfish Herd (Hamilton 1971)]. Subordinate animals also are more likely to be the subjects of unprovoked displacement aggression.

Subordination also carries numerous psychological stressors. Each physical stressor just noted also involves a lack of control and predictability. Moreover, subordinate animals have relatively few coping outlets (e.g., being able to displace aggressive frustration onto someone with lower ranking) and fewer means of social support (e.g., social grooming). Thus, within the classical picture of a dominance hierarchy, subordinate animals are likely to suffer excessive physical and psychological stressors (but see section below for exceptions to this pattern).

STRESS/RANK CORRELATES IN INDIVIDUAL PHYSIOLOGICAL SYSTEMS

I now review the relationship between rank and functioning of some physiological systems relevant to the stress-response. The many correlations cited raise the question of whether rank drives physiology, vice versa, or neither. Many studies have employed one of two designs that allow insight into this question. In the first version, animals are initially singly housed, and physiological measures are taken before and after group formation. Thus, one can determine whether a specific physiological profile preceded or emerged only after the animal's rank was established (e.g., Morgan et al. 2000). In the second, social groups are reorganized intermittently, producing circumstances in which an animal is dominant in one group but subordinate in another. Here we try to answer whether physiological measures precede or follow the rank changes. Overwhelmingly, the distinctive physiological correlates of a particular rank emerge after the rank is achieved, suggesting that behavior drives physiology more than physiology drives behavior.

Rank, the Adrenocortical Axis, and Circulating Levels of Glucocorticoids

As noted, the two workhorses of the stress-response are the sympathetic nervous system and the release of glucocorticoids by the adrenocortical axis. These two branches typify the double-edged nature of the stress-response, in that glucocorticoids and catecholamines are essential for surviving an acute physical stressor, but are pathogenic when secreted in excess (reviewed in Sapolsky et al. 2000).

Thus, one can outline readily the ideal secretory profile of their hormones. First, under basal, nonstressed conditions, there should be minimal secretion; such basal secretion should be unchanged by a purely psychological stressor. Next, a true homeostatic challenge should provoke rapid and massive secretion. Finally, recovery should occur rapidly following stressor termination.

It is difficult to obtain accurate catecholamine measures, because these levels change within seconds. However, glucocorticoid levels change over 1–2 min, allowing extensive study of the relationship between social rank and glucocorticoid profile; these findings generally suggest that the optimal glucocorticoid profile, as outlined, is a characteristic of dominant individuals. In contrast, subordinate

animals tend toward a pathogenic profile of elevated basal glucocorticoid levels (or the enlarged adrenals that support such basal hypersecretion), a sluggish stress-response, and a delayed recovery poststress. Facets of this profile have been observed in mice (Davis & Christian 1957, Southwick & Bland 1959, Vessey 1964, Bronson & Eleftheriou 1964, Louch & Higginbotham 1967, Archer 1970, Popova & Naumenko 1972, Leshner & Polish 1979, Raab et al. 1986, Schuhr 1987, Barnard et al. 1993, Avitsur et al. 2001), rats (Barnett 1955, Korte et al. 1990, Dijkstra et al. 1992, de Goeij et al. 1992, McKittrick 1995, Blanchard et al. 1995), hamsters (Huhman et al. 1992), guinea pigs (Sachser & Prove 1986), wolves (Fox & Andrews 1973), rabbits (Farabollini 1987), pigs (Fernandez et al. 1994, McGlone et al. 1993), sugar gliders (Mallick et al. 1994), fascicularis macaques (Adams et al. 1985, Adams et al. 1987), talapoins (Keverne et al. 1982), olive baboons (Sapolsky 1990), squirrel monkeys (Manogue et al. 1975), tree shrews (Fuchs et al. 1993, 2001; Fuchs & Flugge 1995; Magarinos et al. 1996) and lemurs (Schilling & Perret 1987).

It seems reasonable that subordination is linked with elevated basal levels of glucocorticoids, given the stressors of low rank. But why should subordination be associated with a sluggish turning on and off of the stress-response? An explanation for the former is not readily available; however, there appears to be a link between basal hypersecretion and sluggish termination of glucocorticoid secretion poststress. The adrenocortical axis operates by negative feedback regulation, in which glucocorticoids inhibit the brain from triggering subsequent glucocorticoid secretion (akin to how heat, detected by a sensor in a thermostat, decreases the likelihood of the subsequent generation of heat). If glucocorticoid levels are chronically elevated, the brain's sensitivity to the hormone becomes blunted (via a down-regulatory decrease in the number of glucocorticoid receptors). As a result, the brain becomes less responsive to a glucocorticoid negative feedback signal, and sluggish in terminating glucocorticoid secretion poststress (reviewed in Sapolsky & Plotsky 1990).

Among wild baboons, subordinate animals have been found to have both excessive basal secretion of glucocorticoids and a blunted response to glucocorticoid negative feedback signals. Studies have uncovered regulatory changes at the levels of the brain, pituitary, and adrenals which explain this subordinate profile. Although the details of these findings are not relevant to the present review, the pattern of changes in a subordinate baboon was identical to what occurs in humans with major depression (in which there is often basal glucocorticoid hypersecretion and negative feedback resistance) (Sapolsky 1990). Cognitive psychologists traditionally have described depression as a state of learned helplessness; this formulation might describe as well the psychological state of a subordinate baboon.

Despite the finding of basal hypersecretion of glucocorticoids among subordinates, exceptions have been reported among rats (Blanchard et al. 1995), macaques (Chamove & Bowman 1976; van Schaik 1991; Gust et al. 1991, 1993; Bercovitch & Clark 1995), squirrel monkeys (Coe et al. 1979, Mendoza et al. 1979, Steklis 1986), marmosets (Saltzman 1994), talapoin monkeys (Keverne et al. 1982),

lemurs (Cavigelli 1999), wild dogs and dwarf mongooses (Creel et al. 1996), and wolves (McLeod et al. 1996). Below, possible resolutions of these contradictions are considered.

Rank, Cardiovascular Function, and Cardiovascular Disease

The link between stress and physiology is rarely as clear as in cardiovascular function, simply because heart rate changes noticeably in response to stressors. It has been known for decades that chronic stress increases the risk of cardiovascular disease through well-understood underlying mechanisms. These include the following: (*a*) The increase of blood pressure and heart rate via activation of the sympathetic nervous system in turn increases the risk of mechanical damage to blood vessels. (*b*) Once such damage occurs, circulating lipids and cholesterol can infiltrate into the injury site, promoting atherosclerotic plaque formation. Stress exacerbates this process by increasing circulating lipids and by decreasing levels of "good" (HDL) cholesterol, which opposes plaque formation. (*c*) Stress causes circulating platelets to aggregate at injury sites, worsening plaque formation. (*d*) Estrogen can protect blood vessels from damage, and as reviewed below, stress suppresses estrogen levels (this holds true despite the complex and controversial recent findings regarding the effects of postmenopausal estrogen replacement on cardiovascular disease). (*e*) Once coronary arteries are damaged, stress causes them to constrict (as opposed to stress-induced dilation in healthy arteries), producing myocardial ischemia during stress.

Social stress can adversely affect cardiovascular function in rodents (Henry 1977), nonhuman primates (Strawn et al. 1991, Manuck et al. 1995), and humans (Williams 1989). This leads us to ask, does cardiovascular function vary with social rank? Social subordination has been associated with elevated resting blood pressure in laboratory rats, rabbits (Eisermann 1992), baboons (Cherkovich & Tatoyan 1973, Sapolsky & Share 1994), and macaques (Kaplan & Manuck 1989). This hypertension is typically secondary to elevated sympathetic activity or, less often, to elevated glucocorticoid levels. Moreover, social subordination in Old World primates of both sexes (Sapolsky & Mott 1987, Kaplan et al. 1995) has been associated with lower levels of HDL cholesterol and/or higher levels of "bad" (LDL and VLDL) cholesterol. Additionally, when macaques were fed an atherogenic diet, it was subordinate animals of both sexes who were more prone to atherosclerosis in coronary blood vessels (Kaplan et al. 1982, Herd et al. 1987, Shively & Clarkson 1994, Manuck et al. 1995), an effect mediated by the sympathetic nervous system (Kaplan & Manuck 1989). Moreover, stress-induced suppression of estrogen levels in subordinate females also contributed to vascular damage (Kaplan et al. 1995).

Thus, social subordination can increase cardiovascular disease risk. Although most of these studies involved captive animals in highly manipulated social groups (as well as, in the case cited, requiring a high-fat diet), some studies have utilized wild primates (Sapolsky & Mott 1987, Sapolsky & Share 1994), suggesting an ethological validity to this stress/disease link.

Rank and the Reproductive System

FEMALES Stress can disrupt the reproductive axis of females in numerous species through well-delineated mechanisms involving excesses of glucocorticoids, prolactin, and beta-endorphin working at the levels of the hypothalamus, pituitary, ovaries, and uterus. Such suppression can involve (*a*) lower levels of reproductive hormones, (*b*) later onset of puberty, (*c*) lower fertility rates and longer interbirth intervals, (*d*) higher miscarriage rates, and (*e*) earlier onset of a senescent decline in fertility.

Is reproductive physiology suppressed in subordinate females of various species, relative to dominant animals? Numerous studies support this picture (but see Packer et al. 1995 for one partial exception and Altmann et al. 1995 for criticisms of it). For example, among macaques living in groups with linear dominance hierarchies, subordinate females were subject to the highest rates of aggression, had the lowest rates of contact and grooming, and had elevated basal glucocorticoid levels. Critically, subordinate females had a higher rate of anovulatory cycles, with suppressed estradiol and progesterone levels. In the most severe instances, hormone levels were as low as in ovariectomized animals (Shively & Clarkson 1994).

Such reproductive suppression among subordinate animals could be due to at least four mechanisms: (*a*) "social contraception" (i.e., direct stressful harassment by dominant animals), (*b*) fewer calories, (*c*) more work required for calories, and (*d*) constitutional biology giving rise to both lower rank and impaired gonadal function. Although support for all four mechanisms has been found, the macaque studies just discussed eliminate all but social contraception. This is because these captive animals had equal access to food (eliminating *b* and *c*), and because group membership was occasionally reorganized, often producing very different rankings in different hierarchies (eliminating *d*).

When a rank/physiology relationship occurs (for example, between subordination and low estrogen levels), it is critical to ask whether the estrogen levels are low enough to impair fertility. This question has rarely been answered in these studies, because reports have tended to contain either physiological data (such as hormone levels) or demographic data (such as interbirth intervals or age at first conception), but not both.

An additional caveat: When subordination in a species is associated with reproductive suppression, it need not be the result of stress. For example, New World monkeys such as tarmarins and marmosets live in groups of a pair-bonded dominant pair and four to six subordinates. Only the dominant pair breeds, and subordinate females do not ovulate. However, this is not caused by stress; subordinate females do not have elevated levels of any key stress hormones, and have glucocorticoid levels even lower than dominant females. Moreover, subordinate females are not subject to high rates of aggression, and typically are younger sisters of the breeding female, waiting their turn to breed and helping to raise their nieces and nephews (Abbott et al. 1998). Thus, subordination is not always synonymous with stress.

MALES Stress suppresses gonadal function in males just as it does in females. This suppression takes the form primarily of low levels of circulating testosterone, due to the actions of stress hormones at the levels of the brain, pituitary, and testes. Thus, it is not surprising that social subordination has been associated with suppressed testosterone levels in laboratory rodents. However, no such consistent relationship has been found in nonhuman primates (the sources of this confusion are considered below).

Amid these inconsistencies regarding basal testosterone levels, studies of wild baboons have suggested that the response of testosterone levels to stress varies with rank. Specifically, with the onset of a stressor, testosterone levels promptly declined in subordinate males, but transiently rose in dominant ones (reviewed in Sapolsky 1991) (due most likely to rank-related differences in sympathetic nervous system function and testicular blood flow).

When rank-related differences in testosterone levels do occur, are levels sufficiently suppressed in subordinates to impair reproductive physiology and fertility? Although often assumed, this has rarely been found to be the case. With the exception that rapid changes in testosterone levels may have altered muscle physiology (Tsai & Sapolsky 1996), fluctuations of testosterone levels within the normal range have been found to have remarkably few effects on reproductive physiology or on reproductive or aggressive behavior (in contrast, levels below or above the normal range, due to castration or pharmacological androgen abuse, respectively, do alter these endpoints). When a behavior/testosterone correlation was observed, it was typically the behavior that drove the hormonal change, rather than the other way around.

A variant of this revisionism has emerged from research on orangutans. Adult male orangutans occur in two morphs: A robust, mature form involves well-developed secondary sexual characteristics, whereas a gracile, juvenile form suggests an adult animal in an arrested periadolescent state. Typically, there is only one mature male in a region of orangutans in the wild or in a captive group, and when the mature male is removed, the largest juvenile develops over the coming months into a mature male.

One may speculate that the juvenile form is stress-related, with a dominant male stressing subordinates (via direct interactions or, as is more likely in the rain forest with these solitary animals, via long calls or pheromonal markings) into reproductive suppression and arrested development. However, this is not the case; juveniles do not have elevated levels of glucocorticoids or other stress hormones, nor do they have suppressed levels of reproductive hormones (instead, from an endocrine standpoint, the most stressed males are ones transitioning to mature status). Furthermore, "arrested" males are fertile and occasionally reproduce, typically through forced copulation. This evidence of reproductive success, when coupled with the low injury rate, low metabolic demands, and foraging ease of juveniles, suggests that this is not stress-induced pathology, but an alternative male strategy (Maggioncalda et al. 1999, 2000, 2002).

Finally, stress does not always suppress the testicular axis. Consider a male with an opportunity to mate following challenging and stressful male-male competition.

It would be maladaptive if the stress of that competition impaired fertility, or if impaired fertility occurred in males that mate only once a lifetime (e.g., in semelparous species in which mating is followed by programmed cell death), in species with a narrow breeding season, or in species in which, because of the transience of dominance, individuals may have only a short window of opportunity for mating. In all these circumstances, the testicular axis becomes resistant to the suppressive effects of stress (reviewed in Wingfield & Sapolsky 2003).

Thus, there has been little evidence to support the widely held assumptions that among males, social dominance is synonymous with aggression and high testosterone levels, and that subordination is synonymous with testicular suppression.

Rank and the Immune System

The first evidence to link stress and immunity, discovered in the 1930s by the pioneering stress physiologist, Hans Selye, was that chronic stress suppressed immunity. Since then, psychoneuroimmune studies have uncovered the physiological, cellular, and molecular mechanisms mediating this process, with much of the attention revolving around the immunosuppressive actions of glucocorticoids (which underlie the clinical use of glucocorticoid steroids to suppress organ rejection after transplantion, or to inhibit an overactive immune system during an autoimmune disease).

One might assume then that chronic stress-induced suppression of immunity would lead to increased risk or severity of infectious diseases. However, it has been difficult to demonstrate this under physiological circumstances. One reason for this is that such studies have relied on severe stressors, or on artificially induced diseases (such as the transplantation of a tumor into the rat), limiting the relevance of these findings to more naturalistic stressors and pathogens. A second reason is that profound immunosuppression–far greater than that achieved with the most severe stressors–is needed to compromise defenses against more serious infectious diseases.

A third reason emerged with the recent development of more sensitive immune assays. These have prompted the recognition (as noted above) that during the first hour or two of the stress-response, immunity is stimulated, rather than suppressed, and that the slower immunosuppressive effects of glucocorticoids constitute recovery from the stress-response (Dhabhar & McEwen 2001). Why is such a recovery necessary (as opposed to maintaining the immune system at a heightened level of surveillance)? Such hyperactivity is costly; and, furthermore, immune hyperactivity increases the risk of inadvertently developing an immune response to something benign (i.e., something in the environment producing an allergy, or a part of the body producing an autoimmune disease) (Munck et al. 1984).

Despite these qualifiers, stressors, including social stressors, have been found to increase the risk of the common cold and mononucleosis, and to reactivate latent viruses such as the herpes simplex virus (e.g., Cohen et al. 1991, Ader et al. 2001). However, the relationship between stress and more serious infectious diseases is less clear. For example, although social stress (in the form of frequent changes

of group composition) increased the mortality rate among monkeys infected with simian immunosuppressive virus (Capitano et al. 1998), stress seemed to accelerate the decline in immune function in only a subset of AIDS patients with certain psychosocial characteristics (Cole & Kemeny 2001). In addition, there is ample evidence of a lack of connection between stress and risk of nearly all types of cancers.

Given this picture of immune function during stress, one can make a number of tentative predictions about rank-related differences. Assuming that subordinate organisms are subject to more stressors, subordination might involve (*a*) suppression of the immune system basally, (*b*) greater risk of infectious diseases, (*c*) impairment of the transient activation of the immune response immediately following a stressor, and (*d*) an accelerated reversal of that activation.

No studies, to my knowledge, have examined *c* or *d*. There is, however, ample evidence for *a*. For example, subordinate laboratory rodents have impaired immune responses to various immune challenges (Vessey 1964, Ito et al. 1983, Raab et al. 1986, Fleshner et al. 1989, Devoino et al. 1993), and their immune cells, when cultured in petri dishes, are impaired in their responses to in vitro challenges (Raab et al. 1986, Ito et al. 1983, Fleshner et al. 1989). Similarly, subordination is associated with fewer circulating lymphocytes (or fewer key subsets of them) in pigs (McGlone et al. 1993), rhesus monkeys (Gust et al. 1993), and wild baboons (Sapolsky 1993).

There are exceptions to this rank/immunity link, however. The previous section notes that it would make little sense for the stressor of reproductive competition to suppress reproduction. Similarly, if the stressors of subordination included high rates of challenges to immunity, it would be particularly maladaptive to be immunosuppressed. Similarly, although subordination has been associated generally with immune suppression in mice, when such subordination involved frequent wounds, the immune system developed a resistance to the immunosuppressive effects of glucocorticoids (Avitsur et al. 2001).

When subordination is accompanied by immunosuppression, does this translate into a greater risk of infectious disease? Not surprisingly, given the complications discussed, there has been no consensus on this matter. On the one hand, subordination increases the risk of succumbing to a leukemia-causing virus in mice (Ebbesen et al. 1991), or of developing a respiratory infection when exposed to a cold-causing virus in macaques (Cohen et al. 1997). On the other hand, dominant rodents and primates have the most severe parasitic infections (Hausfater & Watson 1976; Barnard et al. 1993, 1994), and dominant chimps have the highest rates of respiratory infections (Masataka 1990).

The link between stress and increased risk of infectious disease is one of the weaker links in stress pathophysiology. It is not surprising, then, that the link between social subordination and increased risk of infectious disease is also quite weak.

To summarize, there have been numerous reports that socially subordinate animals have elevated basal glucocorticoid levels, hypertension, increased risk of

cardiovascular disease, and, in females, reproductive dysfunction. The evidence of links between social subordination and reproductive dysfunction among males, and of sufficient immune suppression which seriously compromises disease resistance has been far weaker. However, despite the solid evidence that social subordination is more predisposing toward pathophysiology in the realm of adrenocortical, ovarian, and cardiovascular function, there have been many exceptions to that pattern. I now consider some factors that give rise to these exceptions.

WHY ARE THERE INCONSISTENCIES IN THESE DATA? MODIFIERS OF RANK/PHYSIOLOGY RELATIONSHIPS

Throughout this review, a picture has been developing in which socially subordinate animals are subject to a disproportionate share of physical and psychological stressors and show the physiological indices of chronic stress. However, social subordination may not always be disproportionately stressful (as shown with marmosets and tamarins). Moreover, as is clear from the literature reviewed, subordination is not always associated with the physiological indices of stress. I now review some of the modifiers underlying these exceptions.

What Does Rank Mean in a Particular Species?

In the stereotypical picture of a hierarchy, dominant individuals impose resource inequities on subordinates through force or threats of force. This scenario is typical of most Old World primates. Among such species, enforcement may arise from psychological rather than physical means, since dominant individuals rarely had the highest rates of aggression (Altmann et al. 1995, Blanchard et al. 1995).

Yet, in cooperative breeding species, subordination is not enforced from above and does not involve a disproportionate share of stressors. Thus, subordination was not associated with elevated glucocorticoid levels among cooperatively breeding species such as marmosets and tamarins (Abbott et al. 1998), white-browed sparrows, Florida scrub jays, naked mole-rats, dwarf mongooses, and wild dogs (Faulkes & Abbott 1996, Creel et al. 1996, Creel 2001). Therefore, there is a social context in which rank occurs, and this may differ by species.

This insight extends beyond comparing cooperative and noncooperative breeders. Other qualitative features of dominance also vary among species, such as whether dominant animals constantly need to aggressively reassert their dominance, whether alternative strategies to dominance are available (such as the arrested juvenile stage of orangutans), and so on.

This variation was studied systematically in a meta-analysis of the dozen or so primate species x gender cases for which data have been published concerning the relationship between rank and basal glucocorticoid levels. Across the examples, levels in subordinate individuals ranged from approximately one-half those of dominant animals (marmosets) to 50% higher than dominants (male baboons, female talapoins). Experts on the behavior of each species were recruited to

answer a scaled questionnaire regarding quality of life for dominant and subordinate individuals (i.e., In this species, does aggression play a big role in maintaining dominance? How unequally divided are resources? Do subordinate animals have kin present in the group? How often do ranks change?). The relationship between rank and basal glucocorticoid levels was more heavily affected by the quality of life of subordinate animals than that of dominant ones. Specifically, the best predictors of elevated basal glucocorticoid levels among subordinate individuals (or low basal levels among dominant individuals) were (*a*) high rates of harassment by dominant animals and (*b*) few coping outlets for subordinate animals (Abbott et al. 2003).

What Does Rank Mean in a Particular Population?

In addition to interspecific variation in qualitative features of dominance, there is also intraspecific variation, and the physiological correlates of rank vary systematically with this. For example, the elevated basal glucocorticoid levels typically seen among subordinate female macaques or subordinate male baboons were not observed in troops that happened to have low rates of aggression and high rates of affiliative support (Gust et al. 1993, Wallner 1996, Sapolsky & Share 2004). Moreover, in the baboon study, subordination in a typical troop also involved physiological indices of anxiety (specifically, elevated signaling by endogenous benzodiazepines, anxiety-reducing compounds whose synthetic versions include Valium and Librium), but these did not occur in the less aggressive, more affiliative troop (Sapolsky & Share 2004).

Stable Versus Unstable Dominance Hierarchies

Within any given dyad, dominance interactions can strongly reinforce the status quo. Thus, individual number 5 in a hierarchy may win 90% of interactions with individual number 6, but lose 90% of them to number 4. In a stable hierarchy, interactions up and down the rankings typically reinforce the status quo this strongly.

In contrast, if number 5 wins only 51% of interactions with number 6, their relationship is unstable, and their relative positions of dominance may soon switch. On rare occasions, dominance relations throughout an entire hierarchy become destabilized, albeit not necessarily to this extent. The instabilities tend to be somewhat local (i.e., the animal destined to be number 3 when the hierarchy stabilizes wins nearly all interactions with the animal destined to be number 20; it is the interactions with the future numbers 1, 2, 4, and 5 that are unstable). Among wild populations, instability can arise because of the death, immigration, or emigration of a key individual, or the formation or dissolution of a key coalition. In captive populations, instability is characteristic of the first months after a social group is formed.

As mentioned above, in species in which subordination is enforced from above, it is the dominant individuals who have the fewest stressors. However, this is true

only in stable hierarchies. In unstable ones, competition and instability center on the higher ranks, with dominant individuals experiencing the most physical stressors and the greatest sense of loss of control and predictability.

It is not surprising, therefore, that the advantages of dominance, as measured by stress-related physiology, disappear during periods of hierarchical instability. At such times, high rank was no longer associated with lower basal glucocorticoid levels (Keverne et al. 1982 for talapoin monkeys, Coe et al. 1979, Mendoza et al. 1979 for squirrel monkeys, Chamove & Bowman 1976, Gust et al. 1991 for rhesus monkeys, Sapolsky 1993 for wild baboons). Furthermore, dominant males were most likely to develop coronary artery atherosclerosis (Manuck et al. 1995 for macaques), to be immunosuppressed, and to have the highest incidence of respiratory infections (Masataka 1990 for chimpanzees). Moreover, in unstable hierarchies, dominant males have the highest testosterone levels (Sackser & Prove 1986 for guinea pigs, Rose et al. 1971 for macaques, Eberhart & Keverne 1979 for talapoins, Coe et al. 1979, Mendoza et al. 1979 for squirrel monkeys, Sapolsky 1993 for baboons).

What Is the Personal Experience of Rank in One's Species and Population?

The qualitative features of rank not only vary on the inter- and intraspecies level, but also translate on a basic level into very different individual experiences, which influence physiology accordingly. For example, among wild female baboons, the more often an individual was harassed by a particularly aggressive male, the more immunosuppressed she became (Sapolsky 1993). Similarly, among macaques, the less social contact an animal had, the more immunosuppressed it became (Boccia et al. 1997).

Even in a stable hierarchy, there will be pockets of instability (e.g., numbers 8 and 9 may be in the process of switching ranks). One might predict that higher rates of such unstable interactions would be associated with higher glucocorticoid levels (insofar as instability generates a milieu of low predictability). In a study of wild male baboons, this was found to be the case for unstable interactions with animals directly below a subject in the hierarchy; in contrast, no correlation with glucocorticoid levels occurred with the rate of unstable interactions with the individuals above the subject in the hierarchy (Sapolsky 1992). This is logical: Unstable interactions with immediate subordinates indicate that they are gaining on an individual, clearly a cause of stress. In contrast, unstable interactions with higher-ups are not signs of stress, but of impending career advancement. In such cases, the stress-reducing effects of the perception of improving circumstances outweighs the stressful effects of unpredictability in the situation.

Personality as a Filter for the Experience of Rank

It is not anthropomorphic to discuss personality and temperament among animals (i.e., stable affective styles that bias how the individual responds to stimuli) (Clark

& Boinski 1995). Part of the variability in the rank/stress physiology relationship reflects the intervening variable of personality. Studies of both captive macaques (Suomi 1987) and wild baboons (Sapolsky & Ray 1989, Ray & Sapolsky 1992) indicated a "hot reactor" personality style. These are animals whose ongoing behaviors are disrupted easily by novelty, who have trouble distinguishing neutral from threatening stimuli, and who tend to lack effective coping outlets for stress (such as seeking social affiliation). This style bears a striking resemblance to the Type A profile in humans, in which neutral stimuli are atypically interpreted as threatening (Williams 1989). Strikingly, after controlling for rank, such individuals were found to have elevated basal glucocorticoid levels (Suomi 1987, Sapolsky & Ray 1989, Ray & Sapolsky 1992, Virgin & Sapolsky 1997) and an increased risk for atherosclerosis (Manuck et al. 1983, 1989). Moreover, these traits remained stable over time in these individuals, and arose from both genetic (Scanlon et al. 1985) and developmental (Clark & Boinski 1995) influences.

Summary

As discussed above, the social rank of an animal influences stress-related physiology. However, there is no monolithic rank/physiology relationship, either across or within species. In many circumstances, social subordination involves exposure to high rates of physical and psychological stressors, a tendency toward chronic activation of the stress-response, and an increased risk of stress-related diseases. Nonetheless, there are numerous exceptions to this profile, depending upon the social context in which rank occurs and the personality with which the individual reacts to that rank and context.

HUMANS, RANKS, AND HIERARCHY

In discussing relationships between rank and stress-related disease and physiology in humans, one must question whether humans have meaningful ranks and hierarchies. This is important for a number of reasons: (*a*) As exemplified by contemporary hunter-gatherer societies, much of human history probably has been spent in fairly egalitarian groups. (*b*) Humans belong to multiple hierarchies and tend to value most the one in which they rank highest. Consider, for example, the mailroom clerk who is also the best player on the company's softball team. The place in the former hierarchy may be dismissed as "just a job," whereas the latter may be emphasized and become a source of considerable self-esteem. (*c*) Humans readily alter the psychological meaning of a rank. Consider a novice runner who manages to complete a marathon, finishing 1000th, versus the anticipated winner who faded to number 5. Despite the differences in the formal ranking, the former is likely to be more pleased than is the latter.

Thus, although some studies have examined rank/physiology correlates in humans (e.g., the endocrine responses in winners versus losers of a wrestling match),

there are many reasons to question the relevance of such studies to understanding human health. However, a reasonable argument can be made for one realm of human experience in which hierarchical ranking is meaningful in the context of stress and health: socioeconomic status (SES).

Socioeconomic Status and Health

Data stretching back centuries demonstrate that each step down the SES ladder increases the morbidity and mortality for numerous diseases. This gradient, documented in all industrialized societies, is considerable, with mortality rates due to some diseases differing by an order of magnitude between the highest and lowest echelons of SES ladders (reviewed in Syme & Berkman 1976, Adler et al. 1993, Evans et al. 1994, Wilkinson 2000).

In theory, health can both influence and be influenced by SES. Although there is evidence for poor health causing a downward SES spiral, prospective studies have amply documented that SES can precede health status and be highly predictive of it.

How does SES influence health (and is stress relevant)? Generally speaking, there have been psychosocial and nonpsychosocial explanations offered. Although the latter most readily come to mind, they do not begin to explain the SES/health gradient.

Health Care Access, Lifestyle Risk Factors, and Protective Factors

To those oriented toward nonpsychosocial explanations of how SES influences health, the most obvious explanation is the fact that poverty limits access to health care. This is certainly the case in the United States, where poorer people have fewer preventative checkups, longer waits for medical procedures, less access to new experimental procedures, and so on. However, health care access alone does not explain the existence of an SES/health gradient: (*a*) This gradient occurs in countries with socialized medicine and has even worsened during the time when universal health care was instituted. (*b*) The SES gradient is indeed a variable slope: Rather than there being a threshold of poverty below which health care access (and health) declines precipitously, health declines with every step in the SES gradient, starting at the wealthiest and progressing through the middle class. (*c*) A gradient exists for diseases whose incidences are unchanged by access to preventative health care (e.g., juvenile diabetes or rheumatoid arthritis) (reviewed in Pincus & Callahan 1995, Wilkinson 2000).

These findings have shifted the focus from nonpsychosocial explanations to differential exposure to risk factors and protective factors. As one descends the SES slope, the incidence of smoking, drinking to excess, obesity, sedentary lifestyles, poor diets, proximity to toxic dumps, and so on all increase. Moreover, lower SES also translates into fewer protective factors (e.g., fewer safe parks, fewer health club memberships). However, careful multivariate studies indicate that the major

risk and protective factors account for only approximately one third of the SES gradient.

Psychosocial Factors and Stress

Most researchers view psychosocial factors related to stress as major mediators of the SES/health relationship. In addition to the insufficiency of the most notable nonpsychosocial explanations, indirect support for psychosocial factors includes the following: (*a*) The poor have an excess of physical and psychological stressors (Marmot & Feeny 2000); (*b*) studies report an SES gradient related to basal glucocorticoid levels (Kristnson et al. 1997, Lupien et al. 2000); and (*c*) the strongest SES gradients occur for diseases with the greatest sensitivity to stress, such as heart disease, diabetes, metabolic syndrome, and psychiatric disorders (Wilkinson 2000).

The case for stress-related psychosocial factors has become more direct. To appreciate this, one must consider a truism: Given food, shelter, and safety sufficient to sustain health, if everyone is poor, then no one is. In modern societies, it is never the case that everyone is equally (non)poor. This paves the way for a key point about the gradient, namely that poor health is not so much the outcome of being poor, but of feeling poor, that is, feeling poorer than others. Therefore, poverty, rather than being an absolute measure, is a subjective assessment that is mired in invidiousness.

This conclusion has been demonstrated in studies that assess subjective SES (subjects were shown a picture of a ladder with ten rungs and asked to indicate where they place themselves in their society in terms of "how they are doing"). Remarkably, subjective SES was as good or better a predictor than objective SES of stress-related health outcomes (cardiovascular and metabolic measures, glucocorticoid levels). Ongoing work in this area examines how local is the community within which one makes comparisons. The media's global village allows one to make SES comparisons with vastly larger numbers of people than in traditional human experience (Adler & Ostrove 1999, Adler et al. 2000, Singh-Manoux et al. 2003, Goodman et al. 2003).

The importance of subjective SES is reinforced by more top-down economic health literature. Intrinsic to the idea that the SES/health gradient reflects feeling poorer than others is that there are societal mechanisms that make some feel poorer than others. Numerous studies have shown that poverty in a community is not as strong a predictor of crime as is poverty amid plenty, i.e., income inequality. In the United States, at both the state and metropolitan level, the higher the degree of income inequality, the worse the health, the higher the mortality rates, and the steeper the SES/health gradient (independent of the absolute level of wealth) (Wilkinson 2000). This relationship seems not to hold as strongly, if at all, in more economically egalitarian European countries (Lynch et al. 2004).

This is a critical observation, but it could arise from a subtle confound. Suppose that in a society, the bad health of the poor is more sensitive to SES factors than is the good health of the wealthy. If income were made more equal through the

transfer of wealth, this would produce a small health decline for the few wealthy and a large health improvement for the numerous poor. Although this would result in an overall improvement in health, it would be irrelevant to these psychosocial considerations. However, the finding that health is better for all strata of SES in more economically equitable communities rules out this confound (Evans 2002).

How do a subjective sense of low SES and living in an environment of income inequality adversely impact health? Amid the proposed routes, the most intriguing concerns the concept of "social capital." Although the term's definition is still evolving, it refers to salutary features of a community that transcend the level of individuals or individual networks. These features reflect trust, reciprocity, lack of hostility and cynicism, group participation, and a collective sense of efficacy. Thus, for example, social capital is high in a community with lots of volunteerism, in which doors rarely are locked, and in which people belong to effective unions and tenant organizations (most studies assay social capital with two measures, the response of people to statements such as, "Would most people take advantage of you if they got a chance, or would they be fair?," and the number of organizations people belong to).

A fascinating and robust literature has revealed two key findings: (*a*) As income inequality rises in a community, not only does crime increase, but levels of social capital also decline (Kawachi et al. 1999, Kawachi & Putnam 2001). This inverse relationship can be viewed as inevitable, in that social capital is, by definition, about reciprocity and symmetry of relationships, whereas income inequality, by definition, is about hierarchy and asymmetry (Wilkinson 2000). (*b*) Path analyses indicate that the links between income inequality, poor health, and high mortality rates are mediated predominately by the decline in social capital (Kawachi & Kennedy 2002).

Collectively, this literature makes some critical points: First, once basic needs are met, poverty alone is not as predictive of poor health as is poverty amid plenty. Second, when there is considerable poverty amid plenty (i.e., high income inequality), people tend to decrease their investment in (and expectations of) the community, thereby reducing everyone's quality of life. This decline results in more psychological stressors (because of a reduced collective sense of efficacy and control, greater need for vigilance amid increased crime, and so on) and less social support. Finally, amid the adverse community-wide consequences of income inequality and low social capital, the wealthy have disproportionate opportunities (both financial and otherwise) to obtain private means of stress-reduction, further decreasing their incentive to invest in public, community-wide means. An inevitable result of such a "secession of the wealthy" is the production of "private affluence and public squalor," which steepens the SES gradients of stress and poor health (Evans 2002).

This is a far cry from the initial assumption that the SES/health gradient is primarily about poor people having too little money to afford health care. This point has been made by the economist Robert Evans, who observed that, "Most graduate students have had the experience of having very little money, but not of poverty. They are very different things" (Evans 2002).

CONCLUSIONS

The first half of this review focuses on the relationship between social rank and either stress-related physiology or stress-related disease in nonhuman species. Although there is considerable support for a picture of social subordination involving an excess of stress and stress-related pathology, this finding is not universal among social species, and the rank/health relationship can be modified dramatically by an array of factors ranging from individual temperament to nonhuman culture. Findings such as these amply confirm the subtlety and complexity of behavior and social systems among animals other than humans.

Some related issues are then discussed in relation to humans. The transition from nonhuman to human subjects typically involves an increase in the subtleties and complexities considered. In many ways, this is the opposite of what occurs when switching from the subject of nonhuman rank and health to that of human SES and health. In contrast to the modifiers and qualifiers in the nonhuman realm, the most striking quality of the human SES/health gradient is its imperviousness. Do socially subordinate animals suffer a disproportionate share of poor health? The answer can only be, "Often, but certainly not as a rule." Do poor humans suffer a disproportionate share of poor health? The answer must be a robust, Yes—regardless of gender, age, or race; with or without universal healthcare; in culturally homogeneous societies or one's rife with ethnic tensions; and under governments with socialist or capitalist credos.

The developments of class, stratification, and poverty are fairly recent in hominid history. What these findings suggest is that nothing in the world of nonhuman sociality involves such an utterly, psychologically permeating sense of subordination as does the human invention of poverty.

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