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Ethological Causes and Consequences of
the Stress Response

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SYNOPSIS. Stress involves real or perceived changes within an organism or in the environment that activate an organism’s attempts to cope by means of evolutionarily ancient neural and endocrine mechanisms. Responses to acute stressors involve catecholamines released in varying proportion at different sites in the sympathetic and central nervous systems. These responses may interact with and be complemented by intrinsic rhythms and responses to chronic or intermittent stressors involving the hypothalamic-pituitary-adrenal axis. Varying patterns of responses to stressors are also affected by an animal’s assessment of their prospects for successful coping. Subsequent central and systemic consequences of the stress response include apparent changes in affect, motivation, and cognition that can result in an altered relationship to environmental and social stimuli. This review will summarize recent developments in our understanding of the causes and consequences of stress. Special problems that need to be explored involve the manner in which ensembles of adaptive responses are assembled, how autonomic and neurohormonal reflexes of the stress response come under the influence of environmental stimuli, and how some specific aspects of the stress response may be integrated into the life history of a species.

INTRODUCTION

Stressors are real or perceived challenges to an organism's ability to meet its real or perceived needs. In most vertebrates the responses that have evolved to cope with such challenges are constrained by a threshold for detection of challenge, for attention based on real or perceived relevance, and for capacity to respond at any particular
level once the challenge is detected. Depending on the intensity and timing of the stressor, each of these can vary independently.

Stressors that challenge homeostasis, often regarded as the most urgent of needs, are the best known. When an organism’s competence to maintain homeostasis within a specific range is exceeded, responses are evoked that enable the organism to cope by either removing the stressor or facilitating coexistence with it (Antelman and Caggiula, 1990). While many stressors can evoke dramatic neural and endocrine responses, a more modest or subclinical response may be exhibited in response to milder stimuli. These responses may build on or extend homeostatic mechanisms or they may be more or less tightly linked to homeostatic responses in a hierarchical manner creating a functional continuum. For example, such a hierarchical system was described for thermoregulation in mammals by Satinoff (1978) in which more recently evolved regulatory mechanisms are invoked when more conservative ones are unable to restore balance. This continuum is expressed in numerous physiological responses, often measured as an inverted U (Sapolsky, 1997). Although the inverted-U physiology of stress hormones such as corticosteroids presents the conundrum of opposing actions at different dosages, progress over the last twenty years has elucidated some of the physiology involved. For example, both membrane-bound (Orchinik et al., 1991) and two types of intracellular receptors (Ruel et al., 1987) help explain how acute glucocorticoid responses may differ from long-term responses. Membrane receptors acting by means of G proteins stimulate more rapid cellular response than classic receptors that act as transcription factors: type I (mineralocorticoid or MR) and type II (glucocorticoid or GR) receptors. Based on the differences in affinity and capacity, type II receptors only become bound during circadian or stress-evoked peaks in plasma glucocorticoids. We can now envision how a gradual succession from low levels of corticosteroids binding to type I receptors turns the corner on the U as progressively higher titers bind more effectively to type II receptors (Ruel et al., 1987). Basal levels of glucocorticoid (involving type I receptors) are proactively permissive for defense mechanisms at tonic and circadian levels, while reactively suppressive actions are invoked at higher, stress-induced levels of glucocorticoid (involving type II receptors) and help protect the organism from a damaging overcompensation that elevated levels of hormone might induce (Meijer et al 2000). Many if not all of the hormones involved in stress responses possess, in addition to their direct effects, pleiotropic or collateral consequences that may or may not reinforce the direct or primary effect. It is likely that many of these other effects can provide the basis of mechanisms that might serve other, unrelated adaptive needs.

At one level, coping with challenge is what life is all about. Stress is inevitable, and as Hans Selye emphasized, a necessary part of life (1976). There is, however, a problem
defining stress as coping with a challenge. The implication that stress is something to be avoided is a legacy from the clinical perspective dominated by the mandate to remediate dysfunction, including the stress-evoked ‘diseases of adaptation.' H. Selye (1976) himself tried to correct this one-sided over-generalization by distinguishing ‘eustress’ from ‘distress,’ but the legacy lives on. The perception that stress responses, by reallocating resources from growth to coping with a challenge, are ‘not good,’ suggests that stress facilitated coping is forced upon organisms by occasional unfortunate circumstances. However, organisms exist in continually changing environments and their very existence can be construed as an expression of that coping. The mechanisms that have evolved to cope with change are organized within a nested hierarchy. The most conservative functions deal with homeostasis, the most ancient and urgent of needs. But homeostasis is not an organism’s only need, and Bruce McEwen’s succinct definition is an excellent beginning to a fuller appreciation of that fact: ‘Stress may be defined as a threat, real or implied, to the psychological or physiological integrity of an individual’ (McEwen, 1999a:1).

Survival in the changing external environment depends on the flexible stability of an organism’s internal environment, which is itself always in flux. The flexibility is attributable to the fact that physiological functions can proceed effectively within a range of more or less tolerance for deviation from their respective setpoints. The necessity for organisms to attain some measure of independence from the vagaries of the external environment was described by Claude Bernard in the 19th century. Bernard recognized that the stability of the milieu interieur depended on ensembles of compensating mechanisms (Bernard, 1878). Fifty years later, Walter Cannon (1929) introduced the term ‘homeostasis’ to describe the dynamic, interactive nature of these mechanisms in maintaining the stability of the internal environment. He further identified the autonomic nervous system (ANS) as an orchestrator of responses when an organism is suddenly challenged. The famous ‘fight or flight’ response is one such ensemble of responses. Within a decade, Hans Selye shifted attention from the ANS to the adrenal glands by identifying a General Adaptation Syndrome (GAS) in which the initial sympato-adrenomedullary system (SAMS) response to an emergency was augmented by an hypothalamic-pituitary-adrenal (HPA) response as the body mobilized resources to cope with a sustained stressor (Selye, 1936; 1937). This was of particular interest to medical science as the coping mechanisms of the stress response became seen, over time, as potentially deadly ‘diseases of adaptation.’ Here, organs fail as their resources become reallocated to deal with a continuing stressor, possibly leading to exhaustion and death (Selye 1946). By the early 1970's, stress was no longer viewed mainly as a threat to survival, and Seymour Levine (1971) was able to state that the normal expression of adaptive behavior depends upon some optimal level of stress.
Stressors may be acute, sequential, episodic, chronically intermittent, sustained, or anticipated (Sapolsky et al. 2000). Alternative strategies may be evoked by the organism’s perception or experience of their effectiveness in coping. The clinical view of the stress response was that it was largely nonspecific, but it has become clear that many stressors evoke specific combinations of physiological and behavioral responses depending in part on their respective potentials for effective coping in a given context. Stressors perceived as uncontrollable will evoke different ensembles of responses than those believed to be controllable (see for example, Cabib and Puglisi-Allegra 1996, Huether 1996). Stressors also can be additive, creating the appearance of a trivial stressor having a disproportionate effect. The endocrine environment may also be a significant variable for the action of stress hormones.

The present understanding of stress and behavior has been nicely summarized in several reviews. In the early 1970's, Mason (1971) called attention to the potency of psychological stimuli in the stress response. A large literature has emerged since Christian's original insights in the 1950's and 1960's that at high densities, mammals manifest enlarged adrenals indicative of increased stress and also showed increased mortality and reduced reproduction. These *psychoendocrine* effects reinforced perceptions of stress in terms of Selye's (1946, 1976) *General Adaptation Syndrome* (see Christian & Davis 1964, Christian 1980). Lee and McDonald (1985) reviewed this and related literature and appealed for additional research and more direct evidence for the effects of stress in natural populations. Axelrod and Reisine (1984) summarized the multiple regulatory mechanisms and interactions of stress hormones, identifying corticotropin (ACTH) as a principal nexus; Goldstein (1987) provided a helpful collation of stress-induced actions of the sympathetic nervous system; Johnson and colleagues (1992) reviewed mechanisms with an emphasis on the dynamic nature of endocrine and behavioral mechanisms. Neural pathways were recently reviewed by Van de Kar and Blair (1999) who pointed that prolactin, oxytocin, and renin have been neglected as stress-sensitive endocrine systems because they are coordinated by slightly different neural pathways. Saavedra (1999) recently reviewed evidence of a widespread role for angiotensin in modulating stress and corticotrophic releasing hormone (CRH), which has significant stress response coordinating functions aside from its triggering a corticosterone response (e.g. Leshner, 1978; Koob, 1991)

A review of the diversity of glucocorticoid actions in the stress response by Sapolsky, Romero, and Munck (2000) provides a valuable synthesis of the seeming contradictory functions of glucocorticoids. The permissive actions of glucocorticoids that are based on tonic levels associated with homeostasis are seen to be distinct from the suppressive and stimulatory actions that result from stress-induced elevation of circulating levels. Sapolsky et al. also express an appreciation for the ethological
perspective in developing what they have termed the "preparative" functions of glucocorticoids, and caution laboratory researchers to be sensitive to the organism's perspective of what constitutes a stressor.

**TABLE 1 SEE BELOW**

Understanding the causes and consequences of stress in nonhumans has taken on an urgency of its own as a result of a growing concern for animal welfare as well as a search for more robust and relevant animal models. This interest in the role of stress in life history has proven a valuable counterbalance to well-intentioned perceptions by scientists and citizens who too frequently view stress in a stereotypical way as necessarily deleterious. Ignorance of the real needs of the animals (echoing Sapolsky's appeal for appreciation for the unique needs of the subject) is most obvious when well cared for animals fail to thrive or reproduce. Further, freedom from stress attainable in the laboratory is as serious as inadvertently introduced stress in compromising the external validity of findings. Attempts to bring perspective to this issue are proliferating (for example, Broom and Johnson 1994, SCAW 2000) some of which target nonmammalian vertebrates (e.g., Schaeffer et al., 1992; Warwick et al., 1995, Greenberg 1994). Other efforts try to deal with issues of definition and clarity. For example, Moberg (1999) has attempted to identify the boundary between stress and distress at a point where the cost of coping impairs functions critical to well-being.

**COMPLICATIONS IN DEFINING CAUSE AND CONSEQUENCE:**

**LESSONS FROM ETHOLOGY**

Stress researchers and physiological ethologists often emphasize that stress is evoked by a *perceived* challenge to the status quo as well as a physical experience. Since we now more fully understand that not all change is bad and not all stressors are deleterious there is renewed attention to the relationships between stress and emotion. The now distant dispute between proponents of the James-Lange theory of emotion (that the experience of an emotion was secondary to the physiological events) and WB Cannon's view (that physiological changes were subsequent to an emotional experiences) persists because, as Leshner (1978) points out, both positions have some validity. Leshner's review of the problem concluded that at least some hormones may have a general effect on arousal which then feeds back to evoke enhanced
catecholamine and glucocorticoid responses. For example, maternal care-giving is positively correlated with cortisol levels in humans (Fleming et al., 1987) but may also be accounted for by enhanced attention to stimuli. Therefore, endocrine and neuroendocrine events proceed in an interdependent manner to regulate multiple, variable stress responses, each unique, but influenced by previous events (Summers, 2001). Taking this a step further, arousal can evoke an emotion which will be tested against experience and cognition and then by means of a positive feedback loop can lead to progressively more focused expression.

The hormonal mechanisms responsible for behavioral changes during mild stress are rarely obvious, as many stress hormones have structurally-related and biologically active counterparts with multiple receptors and receptor subtypes. For example, corticotrophin releasing hormone (CRH) and the structurally related peptide urocortin act on multiple receptor types to rapidly inhibit feeding. It has is only recently that researchers have been able to identify the respective contribution of each peptide and receptor type to stress-induced alterations in feeding (Cone, 2000). In some respects the selective facilitation or inhibition of normal behavioral patterns evoked by mild stress is analogous to ‘subclinical’ symptoms of a disease.

Indeed, the expression of many behavioral patterns are stress-sensitive in that their expression may be secondary to neurotransmitter or hormone-induced increases in non-specific arousal and selective attention (see Mason 1968, Nelson, 2000). The actions of stress hormones may also be constrained by the activity of other hormones and by environmental circumstances. For example, the rapid behavioral (perch-hopping) response to corticosterone in white-crowned sparrows is influenced by photoperiod. During a long-day (breeding) photoperiod, but not short-day (winter) photoperiod, corticosterone will increase activity (Breuner & Wingfield, 2000). Testosterone is subject to seasonal variation and social dynamics in many species, and its activity appears to facilitate or enhance responsiveness to stressors both directly and indirectly. Reduced androgen, such as might be seen in subordinate males, appears to ameliorate the normal stress-evoking effects of certain stimuli (e.g., Greenberg et al., 1984 in the lizard, Anolis). As a female counterpart to the ‘fight-or-flight’ responses of males, Taylor (et al. 2000) proposed a ‘tend-and-befriend’ response to stress in females where (for example) the effects of oxytocin are moderated by the presence of estrogen and endogenous opioids. In this response, female mammals under stress will manifest enhances caregiving and attachment behavior.

**Stress-sensitive Behavior**

There is a great diversity of adaptive behavioral patterns that appear to have built on specific elements of the stress axes (Table 1). No tabulation can be exhaustive but the
one we have assembled underscores the diversity of effects at different levels of specificity. It is a continuing challenge to distinguish primary from secondary behaviors: Are the effects of stress on behavior a consequence of hormones acting directly on specific neural structures mediating actions? Might they be collateral actions on secondary targets? Or might the manifest behavioral pattern be secondary to enhanced attention, arousal, cognitive activity, or even sensitivity of sensory receptors? Collateral effects are particularly rich sources of alternative behavioral patterns. For example, releasing factors such as corticotropic releasing hormone (CRH) (Koob et al., 1993) and pituitary hormones frequently have multiple target tissues. CRH has many behavioral effects mediated through CNS receptors in addition to its central role in simulating pituitary ACTH secretion and indeed, CRH may arguably be the principle coordinating regulator of central stress responsiveness, influencing central serotonergic (Price et al., 1998; Lowry et al., 2000) and catecholaminergic activities (Dunn and Berridge, 1987; Curtis et al., 1997). CRH is also believed to have direct effects on behavioral patterns such as locomotion (Lowry and Moore, 1991), startle responses (Pelton et al., 1997), and learning (Radulovic et al., 1999; Wang et al., 2000). Significant direct central (extra-adrenal) effects of ACTH are also well known (Leshner, 1971). Thus, simply administering exogenous corticosterone as a way of determining its effects on behavior is complicated by the fact that while it may act directly on a target tissue, it may also be acting indirectly by means of feedback suppression of CRF or ACTH (Brain, 1972). In addition, as mentioned above, the same hormone can have opposite effects when present at different absolute amounts or temporal regimens. Opposing actions of adrenal axis hormones and central stress peptides stem from an inverted-U dose physiology that is a part of a framework of optimal stress response mechanisms (Sapolsky, 1997). Also, different receptor types have alternative effects when stimulated by the same hormone and different stressors can evoke different patterns of endocrine response. For example, the stimuli involved in an aggressive exchange between two males competing for social dominance will elicit comparable corticosteroid release in both animals, but following such an encounter, the winner will also experience a testosterone surge (e.g., Coe et al., 1982 for squirrel monkeys; Greenberg and Crews, 1990 for lizards). Further, if they continue in a long-term dominant-subordinate relationship, the subsequent responsiveness to stress-evoking stimuli will be different in the two animals.

THE EVOLUTION OF STRESS AND BEHAVIORAL COPING MECHANISMS

Autonomic responses are among the richest sources of adaptive behavioral patterns. Tightly yoked somatic and autonomic effects involving sympathetic activation and
occasionally, parasympathetic rebound, have been identified in situations that involve frustration or conflict (Morris, 1956). It is reasonable to imagine that in sophisticated decision-making organisms such as humans, there is competence to reflect, at least in part, on processes that guide the selection of alternative ensembles of adaptive pathways such as “fight or flight” (Cannon, 1929), “flee or freeze” (Rand, 1964, for a lizard), or active versus passive coping (Bandler et al., 2000). Interestingly, in humans, there is evidence that at a critical level of acute stress, cognitive mechanisms of the prefrontal cortex are suppressed and more rapid, conservative responses are invoked (Arnsten 1997, 2000); chronic stress may also work through several other long-term mechanisms to impair cognitive function (McEwen and Sapolsky, 1995).

“In animals, almost invariably, a change in behavior is the crucial factor initiating evolutionary innovation,” Mayr tells us (1988:408). Also, selection pressures can be altered by behavior that modifies the environment in which an animal must survive and thrive (see Deacon's 1997 review of Mark Baldwin). The role of stress in guiding the evolution of coping mechanisms cannot be overestimated. It is likely that stress responses are a first means of dealing with altered selection pressures caused by the inevitable environmental changes organisms are subjected to. A further link is likely between the stress-evoked changes in behavior when confronted with novel selection pressures and the ultimate changes identifiable as evolutionary innovations which seem more abundant in rapidly changing environments (Jablonski & Bottjer 1990, Hoffmann & Hercus 2000). As brains change in response to specific selection pressures, the larger contexts in which resolutions to act are made involve the systems that subsume motivation, affect, and cognition. More conservative coping strategies are reasonably mediated by more ancient parts of the brain (Paradiso et al., 1999). Candidate mechanisms for the developmental influence of stress on brain and behavior, including the effects of corticosteroids impairing the growth of specific neural areas (see Thomas and Devenport 1988) and the integrity of the highly plastic hippocampus, are now well established (for example, Fuchs & Flugge 1998 and McEwen 1999b for recent reviews). Indeed, a major influence of stress on the evolution of brain structures that selectively respond to stressful stimuli or are activated by stress hormones was suggested by Huether (1996) in his conceptualization of a “central adaptation syndrome.

### CONCLUSIONS AND NEED FOR FUTURE STUDY

One of the more striking effects of confronting such a diverse assortment of stress-sensitive phenomena is vivid sense of the versatility and flexibility of the system. The stress response is orchestrated by a deeply embedded, highly conservative sense of
biological priorities and an impressive economy. By assembling and reassembling a relatively small number of possible responses into a diversity of new combinations, natural selection deals with an almost infinite array of possible challenges. Clearly, physiological stress responses need not be manifest as conveniently conspicuous behavioral patterns or pathologies to have adaptive significance. As David Goldstein (1990) put it, they can be evoked whenever an organism experiences “expectations --- whether genetically programmed, established by prior learning, or deduced from circumstances [that] do not match the current or anticipated perceptions of the internal or external environment (p. 243). In addition, the modulation of stress responses by perceived control or helplessness (see Cabib and Puglisi-Allegra [1996], and see Seligman [1975], Seligman et al., [1975]) allows us to envision how an animal’s perception of the prospects for future remediation of a mismatch can influence the expression of an appropriate compensating response (Bandler et al., 2000).

Stress research is compelling not only as a fascinating puzzle that helps make sense of many previously scattered observations, but is also compelling medically and socially. Medical researchers (and then the rest of us) began with Selye's insights about diseases of adaptation, the clinical expressions of chronic stress. However, we have learned much about developmental neuroplasticity. Early nurturing experiences (Liu et al. 1997), prenatal stress syndrome, and brief but intense episodes of stress (such as childhood abuse), have all been implicated in causing enduring neurological changes. In this regard it is significant that a significant number of violent criminals have atypical autonomic responses (Raine et al., 2000), often associated with early experiences of intense stress. The principal function of stress is protective and many elements of the stress response can also be viewed as a kind of cure --- chemotherapy without drugs, in Antelman and Caggiula’s terms-- but sometimes the cure can be worse than the disease. This was Walter Cannon’s insight when he wrote that the development of pathological functions in a system is quite consistent with its usual performance of normal functions.

The adaptive value of responsiveness to stressors in animals in nature may provide invaluable information regarding the dynamics and flexibility of neuroendocrine stress responses. Absolute levels of transmitters or hormones may not matter in the production of significant and adaptive results. Relative elevation or inhibition accruing from previous experience may adjust specific neural centers to produce relevant output specifically related to the appropriate environmental context. The neural mechanisms for transduction of relevant information are of necessity very plastic, with many transmitter, neuromodulator and peripheral hormone systems interacting. These systems influence behavioral and physiological stress responses, but are also influenced by that output.
Our goal in this brief review has been more to provoke than postulate. The references selected from the vast literature of overlapping behavioral, neurological, and endocrine reports applicable to stress were exemplary, not exhaustive. Many of the findings about the reciprocity of behavioral patterns and stress physiology underscores the fact that systems usually expressed as an ensemble are often cobbled together by multiple selection pressures. A sense of this opens researchers to creative hypotheses and the value of the comparative method. By training and disposition, researchers apply Occam's Razor to available evidence no matter how fragmentary, but if the prevailing views of mechanisms cannot assimilate or accommodate new data, new views must be sought.

The lesson beyond the obvious one of humility in the face of nature's imagination, has been one of openness to the myriad possibilities for the organization and reorganization of the relatively small numbers of ways that hormones, brains, and behavior can interact. We have often heard that research has become a more collaborative affair. This is the only solution to the problem of the isolation that attends explorations of great disciplinary depth. A continuing challenge must be to enhance the reciprocal influences of the laboratory and the real world in which traits of interest have evolved. This will require renewed efforts at mutual understanding for researchers specializing in the unique questions and methodologies of each research approach. Efforts must be taken to place the limited validity of highly controlled laboratory studies at the service of less exact field research, and to present the insights of observers in the real world to bench scientists. In a small way this resembles the tension between ideologies of freedom and control that plague all would-be collaborative social groups, but the richness of the reward justifies all possible efforts.

ACKNOWLEDGMENTS

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REFERENCES


### TABLE 1. REPRESENTATIVE BEHAVIORAL RESPONSES TO STRESS

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<thead>
<tr>
<th>Parameter</th>
<th>Response</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>AROUSAL/ATTENTION</strong></td>
<td>Epi intensified but does not evoke affect</td>
<td>Teichner, 1968</td>
</tr>
<tr>
<td></td>
<td>Stress narrows attention onto specific stimuli</td>
<td>Teichner, 1968</td>
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<td></td>
<td>GC (acute) enhances salience of stimuli</td>
<td>Hayden-Hixson, pers comm</td>
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<tr>
<td>Sensory Thresholds</td>
<td>CRH facilitates acoustic startle</td>
<td>Koob, 1991</td>
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<tr>
<td>Social defeat diminishes nocioception in mice</td>
<td>Miczek et al., 1982</td>
<td></td>
</tr>
<tr>
<td>Handling and species-specific stress-evoking odors cause analgesia in rats</td>
<td>Fanselow and Sigmundi, 1986</td>
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</table>

**ACTIVITY**

<table>
<thead>
<tr>
<th>Locomotor</th>
<th>CRH-induced in familiar habitat</th>
<th>Sutton et al., 1982</th>
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<tr>
<td>CRH-inhibited in unfamiliar habitat</td>
<td>Britton et al. 1982</td>
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<tr>
<td>CRH-induced walking and swimming (newt)</td>
<td>Lowry et al., 1996</td>
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<tr>
<td>Feeding and grooming correlated with subsequent increase in GC</td>
<td>Shiraishi et al., 1984</td>
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<tr>
<th>Dispersal</th>
<th>GC/stress involved in seasonal population dispersal in birds</th>
<th>Lee and McDonald, 1985; Silverin, 1997; Wingfield et al., 1997</th>
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<thead>
<tr>
<th>Exploratory</th>
<th>GC restores exploratory activity eliminated by adrenalectomy</th>
<th>Veldhuis et al., 1982</th>
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<tr>
<th>Exploratory</th>
<th>CRH enhances effects of novelty</th>
<th>Koob, 1991</th>
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<tr>
<td>ACTH excitatory with novel stimulus and inhibitory with prolonged stimulus</td>
<td>Oades, 1979</td>
<td></td>
</tr>
<tr>
<td>ACTH but not GC impairs habituation to an acoustic stimulus and reduces exploration</td>
<td>File, 1978</td>
<td></td>
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<tr>
<td>Some elements enhanced, others impaired in lizards; stress affects ameliorated in castrates</td>
<td>Greenberg, 1985</td>
<td></td>
</tr>
<tr>
<td>GC evokes rapid (nongenomic) locomotor response in rats in novel but not familiar cage</td>
<td>Sandi et al., 1996</td>
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</table>

**THERMOGREGULATION**

| Hippocampal GC receptors mediate stress responsiveness to novel habitats | Hiebert et al., 2000 |
**Diurnal torpor in hummingbird**

### COGNITIVE

#### Memory and learning

<table>
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<tr>
<th>Facilitator</th>
<th>Effect</th>
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<tr>
<td>Epi</td>
<td>Facilitates acquisition</td>
<td>Smith, 1973</td>
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<td>CRH</td>
<td>Facilitates acquisition of visual discrimination</td>
<td>Koob, 1991</td>
</tr>
<tr>
<td>ACTH</td>
<td>Facilitates and corticosterone impairs imprinting</td>
<td>Martin, 1978</td>
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<td>Melanocortins</td>
<td>Facilitate habituation (toad)</td>
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<td>Enhance learned avoidance and approach behavior was contrasted with Attenuated learning</td>
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<td>Enhance learned avoidance and approach behavior was contrasted with Attenuated learning</td>
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<tr>
<td>Attenuated acquisition and performance of learned behavior</td>
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<td>McEwen et al., 1986</td>
</tr>
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<td>Stress</td>
<td>Facilitates classical conditioning in males but not females</td>
<td>Shors et al., 1992, 2000; Wood &amp; Shors, 1998</td>
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<tr>
<td>Stress-facilitated learning depends on stressor intensity, duration and context</td>
<td></td>
<td>Shors and Servatius, 1997</td>
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<tr>
<td>Stress-induced rise in natural benzodiazepine levels rise and apparently enhance the inhibitory neurotransmitter GABA, preventing retention of irrelevant information</td>
<td></td>
<td>Levine, 1971; Izquierdo and Medina, 1991</td>
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### SOCIAL DOMINANCE

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<th>Hormone</th>
<th>Effect</th>
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<tbody>
<tr>
<td>GC</td>
<td>Increases submissiveness</td>
<td>Leshner and Politch, 1979</td>
</tr>
<tr>
<td>Losers of territorial fights become subordinate in the lab (lizard)</td>
<td></td>
<td>Greenberg et al., 1984</td>
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### REPRODUCTION

<table>
<thead>
<tr>
<th>Facilitator</th>
<th>Effect</th>
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<tr>
<td>Endorphins</td>
<td>Block gonadotropin releasing factor and CS impairs gonadal responsiveness to gonadotropins</td>
<td>Sapolsky, 1994</td>
</tr>
<tr>
<td>CRF</td>
<td>Inhibits sexual behavior in female rats</td>
<td>Sirinathsinghji et al., 1983</td>
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<tr>
<td>ACTH</td>
<td>Can induce transient increase in testosterone while sustained CS suppresses testosterone</td>
<td>Moberg, 1985</td>
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<tr>
<td>Can be facilitated by presumed stress of aggressive activity</td>
<td></td>
<td>Antelman and Caggiula, 1980</td>
</tr>
<tr>
<td>Prenatal stress syndrome: stressed pregnant rats deliver feminized male pups</td>
<td></td>
<td>Ward &amp; Weisz, 1980; Greenberg and Wingfield, 1980</td>
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## DYSFUNCTIONAL COMPENSATIONS

<table>
<thead>
<tr>
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<th>1987</th>
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<tbody>
<tr>
<td>ACTH-induced stretch-yawn syndrome and grooming.</td>
<td>Gispen, 1982</td>
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<td>MSH induces grooming behavior in rats</td>
<td>O'Donohue <em>et al.</em>, 1981</td>
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<tr>
<td>Subordinate rats increase alcohol consumption</td>
<td>Blanchard <em>et al.</em>, 1993</td>
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<tr>
<td>Addictions, neuroses and psychoses precipitated by stress</td>
<td>Arnsten, 1997</td>
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<tr>
<td>Atress catalyzes hyperexcitability in fear-mediating circuits leading to anxiety disorders</td>
<td>Rosen and Shulkin, 1998</td>
</tr>
</tbody>
</table>

ACTH, adrenocorticotropic hormone; EPI, epinephrine; GC, glucocorticoid; MSH, melanocyte-stimulating hormone3)