

Ecological Bases of Hormone-Behavior Interactions: The “Emergency Life History Stage”¹

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SYNOPSIS. Superimposed upon seasonal changes in morphology, physiology and behavior, are facultative responses to unpredictable events known as labile (*i.e.*, short-lived) perturbation factors (LPFs). These responses include behavioral and physiological changes that enhance survival and collectively make up the “emergency” life history stage. There is considerable evidence that glucocorticosteroids, and other hormones in the hypothalamo-pituitary-adrenal (HPA) cascade, initiate and orchestrate the emergency life history stage within minutes to hours. This stage has a number of sub-stages that promote survival and avoid potential deleterious effects of stress that may result from chronically elevated levels of circulating glucocorticosteroids over days and weeks. These sub-stages may include: redirection of behavior from a normal life history stage to increased foraging, irruptive-type migration during the day, enhanced restfulness at night, and elevated gluconeogenesis. Once the perturbation passes, glucocorticosteroids may also promote recovery. Additional evidence from birds indicates that glucocorticosteroid responses to a standardized capture, handling and restraint protocol are modulated both on seasonal and individual levels. Field work reveals that these changes in responsiveness to LPFs have ecological bases, such as reproductive state, body condition etc., that in turn indicate different hormonal control mechanisms in the HPA cascade.

INTRODUCTION

Most of us interpret “emergency” responses of animals as the “fight-or-flight” response—the massive release of catecholamines by adrenal medullary cells (chromaffin) that increase heart rate, mobilize glucose, etc., within seconds (*e.g.*, Axelrod and Reisine, 1984; Sapolsky, 1987; Johnson *et al.*, 1992). This response is triggered by sudden threatening environmental events such as attack by a predator or dominant conspecific, and it serves to facilitate immediate and extreme physical exertion to escape. The fight-or-flight response is usually over within seconds (assuming successful escape) and the individual returns to normal activity within minutes. Over the past twenty years accumulating evidence

suggests another “emergency” response may exist that involves interruption of the life history cycle and re-direction of behavior and physiology towards survival. It is distinct from the “fight-or-flight” response in that it takes several minutes or even hours to develop and results in a more long-lived (hours or days, even weeks) interruption of normal activities such as breeding. This “new” phenomenon also raises questions about proximate and ultimate causations. Why has the emergency response evolved and how is it orchestrated?

Organisms have a characteristic series of life history stages that makes up their life cycle (Jacobs, 1996). A highly simplified series of life history stages in birds is presented in Figure 1. The winter (non-breeding) stage and breeding stage each have unique sets of sub-stages. Transition from stage to stage is regulated by hormone secretions, as is the activation of sub-stages within a stage. Progression of stages and timing are determined by predictable

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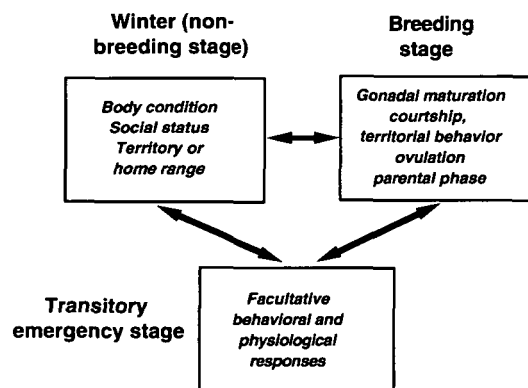


FIG. 1. A highly simplified series of life history stages in birds. The winter (non-breeding) stage and breeding stage have unique sets of sub-stages. Transition from stage to the next is regulated by hormone secretions as is the activation of sub stages within a stage. Progression from stage to stage and timing of a specific stage are determined by predictable changes in the environment (*e.g.*, photoperiod). However, the emergency life history stage may be triggered at any time by unpredictable events in the environment (see labile perturbation factors in Table 1). This transitory emergency stage has its own unique set of sub stages. After the perturbation passes, the individual can return to the original life history stage. If the perturbation was long lived then the next, or an appropriate life history stage for that time of year will be assumed. Modified from Jacobs (1996) and Wingfield *et al.* (1997).

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The unpredictable environmental factors that trigger an emergency life history stage have been termed "labile perturbation factors" (LPFs, Jacobs, 1996). It is important to understand that these factors are unpredictable (can occur at any time of year), and they are usually transitory (*i.e.*, labile), although in recent years human disturbance and pollution may result in "permanent perturbations." There are two major types of LPFs—direct and indirect (Table 1, see

TABLE 1. *Labile perturbation factors.*

Indirect	Direct
Loss of eggs or young to predator	Prolonged severe weather
Loss of eggs or young to short severe storm	Interspecific competition
Brief disturbance (<i>e.g.</i> , human)	Loss of mate
	Pollution
	Habitat change or loss
	Prolonged disturbance (<i>e.g.</i> , human)

Expanded from, Wingfield (1988, 1994).

Wingfield, 1988, 1994; Jacobs, 1996). Indirect LPFs result in loss of a nest and young, or temporary deterioration of the habitat. The individuals involved may not trigger an emergency life history stage, but may initiate a fight or flight response. Such unpredictable disturbances are over quickly and the individual continues in its life history stage appropriate for that time of year. Increased glucocorticosteroids may be, but usually are not, involved (Wingfield, 1988). Direct LPFs, on the other hand, affect the individual directly by decreasing available food resources, increasing energetic demands (*e.g.*, especially bad weather), or restricting access to resources by disturbing optimal habitat (see Wingfield, 1988). Increased interspecific competition may also result in restricted access to resources, followed by adjustment of home range and habitat partitioning (Repasky and Schluter, 1994), or at least an increase in energy required to compete for those resources. In these cases, the emergency life history stage is triggered. Although in this paper we will focus primarily on birds, the emergency life history stage concept may be widely applicable to all vertebrates—at least at the level of behavioral responses to unpredictable events (*e.g.*, Clutton Brock, 1991).

THE EMERGENCY LIFE HISTORY STAGE

There are several clearly definable events that make up the emergency life history stage in response to LPFs. These have been summarized by Wingfield and Ramenofsky (1997) and are expanded here under four major headings:

1. Deactivation of territorial behavior/disintegration of social hierarchies:

a) Reproduction and associated behavior, seasonal migration or wintering strategies are suppressed.

b) Social relationships may be suspended temporarily.

2. Activation of emergency behavior:

a) Seek or remain in a refuge. If food supply is not compromised, then the best strategy may be to find shelter and "ride-out" the LPF.

b) Move away from the source of perturbation. If food resources are compromised in any way such that negative energy balance is likely, then the best strategy would be to leave and seek alternate habitat.

c) Seek a refuge and try to ride out the LPF at first, but then leave if conditions do not improve. The time spent in a refuge before leaving may be a direct function of stored energy reserves. Note that the individual should leave while energy stores are still sufficient to fuel a flight.

3. Mobilization of stored energy reserves:

Since in 2b and c, negative energy balance is likely, then stores of fat should be tapped. In many cases gluconeogenesis may include mobilization of proteins as well.

4. Settlement in alternate habitat or return to the original site—termination of the emergency life history stage:

a) If the individual remains in its original habitat, then the normal life history stage can be assumed immediately after the LPF has abated.

b) If the individual leaves, then suitable habitat should be identified and the individual can then settle and resume the normal series of life history stages.

c) In many cases, the individual may return to its original habitat once the LPF has passed.

d) Recovery following an emergency life history stage may be a critical component of the whole process.

Evidence to date suggests that the behavioral and physiological components of the emergency life history stage are similar, if not identical, at all times of year, and regardless of the life history stage from which it may have been triggered (Jacobs, 1996).

It is then logical to propose that the mechanisms by which this stage is initiated, maintained and terminated may be the same at all times of year and throughout the life cycle of the individual. We propose that neuropeptides associated with the hypothalamo-pituitary-adrenal cortex (HPA) axis, adrenocorticotropin (ACTH) and glucocorticosteroids regulate the emergency life history stage (*e.g.*, Wingfield, 1994), although it is certain that other endocrine secretions may also be involved. Many hormones have been identified in classical responses to stress in vertebrates, and since many aspects of the emergency life history stage are superficially similar to stress, it is tempting to draw parallels. However, evidence is accumulating that the emergency life history stage is a mechanism by which individuals avoid stress thus enhancing survival and potentially lifetime reproductive success (Wingfield *et al.*, 1997).

The hypothalamo-pituitary-adrenal axis

It has been known for decades that a host of obnoxious agents (stressors) activate the hypothalamo-pituitary-adrenal axis resulting in marked elevation of glucocorticosteroid secretion. Although they orchestrate many of the physiological, morphological and behavioral responses to stress, other hormones are also involved (*e.g.*, Axelrod and Reisine, 1984; Munck *et al.*, 1984; Johnson *et al.*, 1992). The actions of glucocorticosteroids during this so-called "stress-response" attracted our attention at first because of the apparent parallels of the emergency life history stage and a classical stress response. Owing to constraints of space we will focus primarily on the actions of opioids and glucocorticosteroids (Table 2). Because the measurement of plasma β -endorphin levels has proved technically difficult, there are few studies addressing its action in free-living individuals. However, it is known to influence reproductive behavior, analgesia, and feeding behavior, making this peptide an ideal candidate for involvement in the emergency life history stage. We also include the peptide ACTH because it is released during the initiation of the emergency life history stage, is co-released with β -endorphin (Guillemin *et al.*,

TABLE 2. *Effect of Corticosterone in an Emergency Life History Stage.*

Rapid (i.e., short term, minutes to hours)	Chronic (i.e., long term, days to weeks)
Suppress reproductive behavior	Inhibit reproductive system
Regulate immune system	Suppress immune system
Increase gluconeogenesis	Promote severe protein loss
Increase foraging behavior	Disrupt second messenger systems
Promote escape (irruptive) behavior during day	Neuronal cell death
Promote night restfulness by lowering standard metabolic rate	Suppress growth and metamorphosis
Promote recovery on return to normal life history stage	

Modified and expanded from Wingfield (1994).

1977), and binds to opioid receptors (Terenius, 1977).

Wingfield (1994) suggested that there may be two distinct types of response to glucocorticosteroids during a stress response. By far the most well studied are chronic effects induced by many days or even weeks of exposure to continual high circulating levels of glucocorticosteroids resulting from prolonged exposure to stress. These effects (Table 2) include total failure of reproductive function, increased susceptibility to disease owing to suppression of the immune system, neuronal cell death (particularly in the hippocampus), severe protein loss (for gluconeogenesis), disruption of the arachidonic acid cascade, and inhibition of growth and metamorphosis (e.g., Axelrod and Reisine, 1984; Munck *et al.*, 1984; Johnson *et al.*, 1992; Sapolsky 1987, 1996). Although these effects have immense importance for medicine and agriculture, it is difficult to imagine how any one of these states would be adaptive for an organism in the field. Indeed death would be imminent in any of these states. Thus it is unlikely that chronic effects of high circulating levels of glucocorticosteroids have much biological significance since survival by this time would be virtually zero (Wingfield *et al.*, 1997). It is well documented that severe environmental perturbations occasionally result in massive mortality in natural populations (see Wing-

field *et al.*, 1997), but presumably there would be strong selection for mechanisms by which such deleterious states are avoided in survivors. Therefore, the short term effects of elevated glucocorticosteroids (over minutes to hours) may be highly adaptive in avoiding the severe stressed state. These short term effects are also summarized in Table 2. It is these effects that may orchestrate the emergency life history stage and avoid the clearly severe, and very likely fatal, consequences of chronic high levels of glucocorticosteroids and other hormones of the HPA axis. The evidence for short term effects of HPA hormones consistent with the emergency life history stage are as follows.

Suppression of reproductive behavior

One of the hallmarks of an emergency life history stage is that individuals redirect their activities from those typical of the normal life history stage, to others more conducive to survival. There are many accounts of abandonment of breeding territories and offspring in response to LPF-like environmental events (e.g., Gessamen and Worthen, 1982; Clutton Brock, 1991), suggesting that redirection of behavior may be widespread. At first this may appear maladaptive because reproductive success becomes zero. However, temporary suspension of breeding activity may actually enhance lifetime reproductive success by allowing an individual to survive the perturbation in good condition so that it can then breed again at the earliest opportunity.

Glucocorticosteroids.—In free-living pied flycatchers, *Ficedula hypoleuca*, implants of corticosterone reduced parental behavior in both sexes (Silverin, 1986). Nestlings were fed less, fewer fledglings resulted, and young that did fledge weighed less than fledglings from control implanted birds. Another group that received implants designed to give even higher circulating levels of corticosterone resulted in complete abandonment of nests with zero reproductive success (Silverin, 1986). In breeding male song sparrows, *Melospiza melodia*, similar implants of corticosterone resulted in marked reduction of territorial aggression. Furthermore, plasma levels of testos-

terone were still in the range typical of this period in the reproductive cycle, suggesting that corticosterone may override the effects of testosterone in activation of territorial aggression (Wingfield and Silverin, 1986). Similarly in side-blotched lizards, *Uta stansburiana*, implants of corticosterone significantly reduced home range size and activity if control implanted lizards were also present (DeNardo and Sinervo, 1994a). However, if all individuals at a site were implanted with corticosterone, there was no decrease in home range size or activity, suggesting that corticosterone may reduce the effectiveness of males in retaining their home ranges when in competition with normal males. In another experiment, it was shown that if lizards were also implanted with testosterone, then corticosterone treatment still resulted in reduced home ranges if control males were present (DeNardo and Sinervo, 1994b). These data suggest further that the effects of corticosterone override any effect of testosterone on spatial behavior. The mechanisms underlying these behavioral responses remain unknown. Glucocorticosteroids also may directly suppress reproductive behavior. Subcutaneous injection of corticosterone profoundly inhibits courtship behavior in male rough-skinned newts, *Taricha granulosa* (Moore and Miller, 1984).

β -endorphin.—The effects of opioids on reproductive behavior are well known and too extensive to cover in detail here. Experiments with antagonists and agonists have demonstrated an inhibitory role for both central and circulating opioids. In the rough-skinned newt, stress-induced inhibition of courtship can be reversed by treatment with naloxone, an opioid antagonist (Miller and Moore, 1982). In rats, central infusion of β -endorphin causes a decrease in mounting by males and an inhibition of lordosis in females (Meyerson and Berg, 1977; Sirinathsinghji, 1984). Intraventricular infusion of corticotropin-releasing factor (CRF) results in suppression of lordosis that is reversible by β -endorphin antagonists (Sirinathsinghji *et al.*, 1983a, b). In female white-crowned sparrows, *Zonotrichia leucophrys gambelii*, central infusion of β -endorphin strongly inhibits copulation solici-

tation whereas naloxone enhances it (Maney and Wingfield 1998). The mechanism of opioid-induced suppression of reproductive behavior is unknown, but there is evidence that β -endorphin acts within the brain to suppress gonadotropin-releasing hormone (GnRH) neuronal systems (see Sirinathsinghji, 1984; Fan and Ottinger, 1996).

Promotion of gluconeogenesis

Glucocorticosteroids play a key role in promoting gluconeogenesis, especially from protein, in many vertebrate taxa (Chester-Jones *et al.*, 1972). In mammals, glucocorticosteroids play a central role in metabolic responses to stress by sustaining gluconeogenesis by increasing the supply of hepatic gluconeogenic precursors and by maintaining glycogen availability in the liver (*e.g.*, Fujiwara *et al.*, 1996). Acute increases in glucocorticosteroids increase the gluconeogenic conversion of alanine to glucose by elevating uptake of alanine by the liver, and may also be accompanied by a transient decrease in insulin to further enhance gluconeogenesis (Goldstein *et al.*, 1992). Similar mechanisms may operate in birds, although increased glucose (or glycogen) may not be the only result. In song sparrows and pied flycatchers, corticosterone treatment results in apparent loss of protein from flight muscles, but no change in body weight because fat depots increased markedly (Wingfield and Silverin, 1986; Silverin, 1986). Similar effects were found in captive dark-eyed juncos, *Junco hyemalis*, (Gray *et al.*, 1990). Furthermore, although adipose lipoprotein-lipase (LPL) activity was unchanged, the concentration of LPL in muscle increased significantly even though muscle mass declined. These data are consistent with the hypothesis that flying birds utilize fatty acids as a major fuel for flight rather than glycogen.

Ward (1969) and others have suggested that the pectoralis flight muscles of birds may be important reservoirs of readily-mobilizable protein for reproduction and possibly flight. A morphological study by Kendall *et al.* (1973) of flight muscles of *Quelea quelea*, suggested that soluble proteins may be stored in mitochondria and between myofibrillar bundles in sarcoplasm.

However, such stores, if they exist, are difficult to quantify morphologically. Honey (1990) devised a biochemical method to separate soluble and structural (contractile) proteins in avian muscle by extraction in low or high salt phosphate buffers. When corticosterone was implanted into captive house sparrows, *Passer domesticus*, there was a significant decline in body weight and particularly in weight of pectoralis muscles compared with controls. Further, this loss of mass in muscle was due to a significant decline in soluble protein fractions. Structural (myofibrillar) fractions did not differ between treatments. The influences of corticosterone, and other hormones, on gluconeogenesis and utilization of protein require further study on wild birds in different life history stages.

Regulation of the immune system

It is now well known that unpredictable events in the environment can stimulate release of cytokines and monokines. These hormones of the immune system can interact extensively with other components of the endocrine system and in turn can modify behavior (e.g., Munck *et al.*, 1984; Cunningham and De Souza, 1993). Although our knowledge of these effects in non-mammalian vertebrates is sparse, it has been demonstrated that when male Western fence lizards, *Sceloporus occidentalis*, are injected with human interleukin-1 β (IL-1), they show decreased activity (especially in the morning hours) compared to saline injected controls and untreated animals. This suppression of activity is similar to that seen in lizards infected with malaria (Dunlap and Church, 1996). The authors suggest that IL-1 may mediate pathogen-induced changes in activity. Whether these hormones may also mediate other aspects of activity in an emergency life history stage in general awaits further study.

Increase in foraging behavior

Glucocorticosteroids.—As in mammals, there is evidence that glucocorticosteroids, along with other metabolic hormones, are important in the regulation of food intake (e.g., Richardson *et al.*, 1995). Implants of metyrapone (a blocker of 11 β -hydroxylase,

an enzyme essential for the synthesis of glucocorticosteroids) decreased foraging behavior (a combination of searching, scratching, pecking, and actual food intake) in male white-crowned sparrows and replacement therapy with implants of corticosterone increased foraging (Wingfield *et al.*, 1990). However, implants of corticosterone into otherwise untreated white-crowned sparrows and song sparrows tended to increase foraging (Astheimer *et al.*, 1992), but this was not significant, and had no effect in dark-eyed juncos (Gray *et al.*, 1990). It is possible that corticosterone may play a "permissive" role in the regulation of food intake. Other factors acting centrally may also be important, as has been shown in mammals (Leibowitz *et al.*, 1984).

β -endorphin.—Endogenous opioids are well-known to affect feeding behavior, and may initiate an increase in foraging during the emergency life history stage. Intracerebroventricular beta-endorphin has been shown to increase food intake or feeding behavior in a variety of vertebrates, including rats (McKay *et al.*, 1981), pigeons (Deviche and Schepers, 1984), and white-crowned sparrows (Maney and Wingfield, 1998). Food deprivation (see Morley *et al.*, 1983) causes beta-endorphin levels to decrease in the rat hypothalamus, suggesting release of this peptide. Stress-induced feeding can be reversed by naloxone, an opioid antagonist (reviewed by Morley *et al.*, 1983). Intramuscular injection of naloxone methobromide, an antagonist that does not cross the blood-brain barrier, decreases feeding in domestic fowl (Denbow and McCormack, 1990), indicating that endogenous opioids may also modulate feeding behavior at sites outside the CNS.

Promotion of diurnal escape (irruptive/shelter) behavior

Corticosterone treatment of captive male white-crowned sparrows resulted in a decline of perch hopping activity over the day (Astheimer *et al.*, 1992), which is consistent with "shelter" behavior related to "riding out" the perturbation factor. This result is particularly compelling since food was available *ad libitum* and leaving may not

confer any advantage. However, if food was removed for 24 hours (to simulate severe storms that often reduce food resources), then corticosterone-treated birds showed a considerable increase in perch hopping activity exceeding that of controls throughout the day. These data suggest that under conditions of reduced food availability, corticosterone may actually enhance activity, possibly associated with leaving the source of perturbation. Note that this activity is during the day and not at night. In the white-crowned sparrow normal spring and autumn migratory behavior occurs at night (e.g., Wingfield *et al.*, 1990), suggesting that corticosterone-induced activity is a different phenomenon consistent with the emergency life history stage (Jacobs, 1996) and not a normal life history stage (e.g., vernal or autumn migrations). Again, central effects of hormones may be important in distinguishing whether corticosterone has an effect to decrease or increase activity. This is currently under investigation. Note also that in Western fence lizards, IL-1 decreased activity (Dunlap and Church, 1996). It is possible that such a mechanism may also operate in white-crowned sparrows.

Promotion of nocturnal restfulness

It was originally suggested that since corticosterone may have marked effects on activity of birds during an emergency life history stage, then we might predict that this glucocorticosteroid may also increase metabolic rate. In contrast, implants of corticosterone actually reduced extended metabolic rate in captive white-crowned sparrows (as measured by oxygen consumption) over night compared with controls (Buttner *et al.*, 1991). Control treated birds, as well as birds sampled before treatment, showed episodes of oxygen consumption over a 60 min sampling period at night. Corticosterone treatment did not reduce standard metabolic rate, but eliminated episodes of increased oxygen consumption with a net savings of energy over night. Similar effects were obtained in American goldfinches, *Carduelis tristis*, pine siskins, *C. pinus*, and red crossbills, *Loxia curvirostra* (Buttner *et al.*, 1991). The authors

interpreted these results as enhanced "night restfulness" in an emergency life history stage. Note also that this effect is not consistent with nocturnal migratory activity in normal life history stages of vernal and autumnal migrations, and further supports the concept of a distinct emergency life history stage with its own suite of hormonal control mechanisms.

Promotion of recovery on return to normal life history stage

Implants of corticosterone into captive song sparrows had little effect on foraging-like behavior when food was removed for 24 hours, but did greatly enhance food intake when food was returned. Similar, but less marked, effects were seen in male white-crowned sparrows that were treated with corticosterone and had food withheld for 24 hours and then refed (Astheimer *et al.*, 1992). These data suggest an additional role for corticosterone in the recovery phase after a perturbation ceases. Because of its well known role in analgesia, β -endorphin must also be considered here. This aspect of the emergency life history stage deserves further study.

Overview

Experimental evidence to date thus supports the concept of an emergency life history stage that can be triggered by increased circulating levels of corticosterone. Although other hormones are undoubtedly involved, it seems clear that the transitory elevation of glucocorticosteroids above normal baseline levels and daily or seasonal changes (levels A and B of Wingfield *et al.*, 1997; Fig. 2) to high concentrations often associated with stress (level C of Wingfield *et al.*, 1997; Fig. 2), results in a suite of physiological and behavioral responses. These redirect the individual quickly from "non-essential" activities such as reproduction, territorial behavior, and social hierarchies, to behaviors associated with surviving the perturbation. In this way the individual minimizes the possibility of metabolic debilitation, thus avoiding the detrimental effects of chronic stress and prolonged high levels of glucocorticosteroids. Most of the evidence given here comes from birds, but it is highly likely that the concept

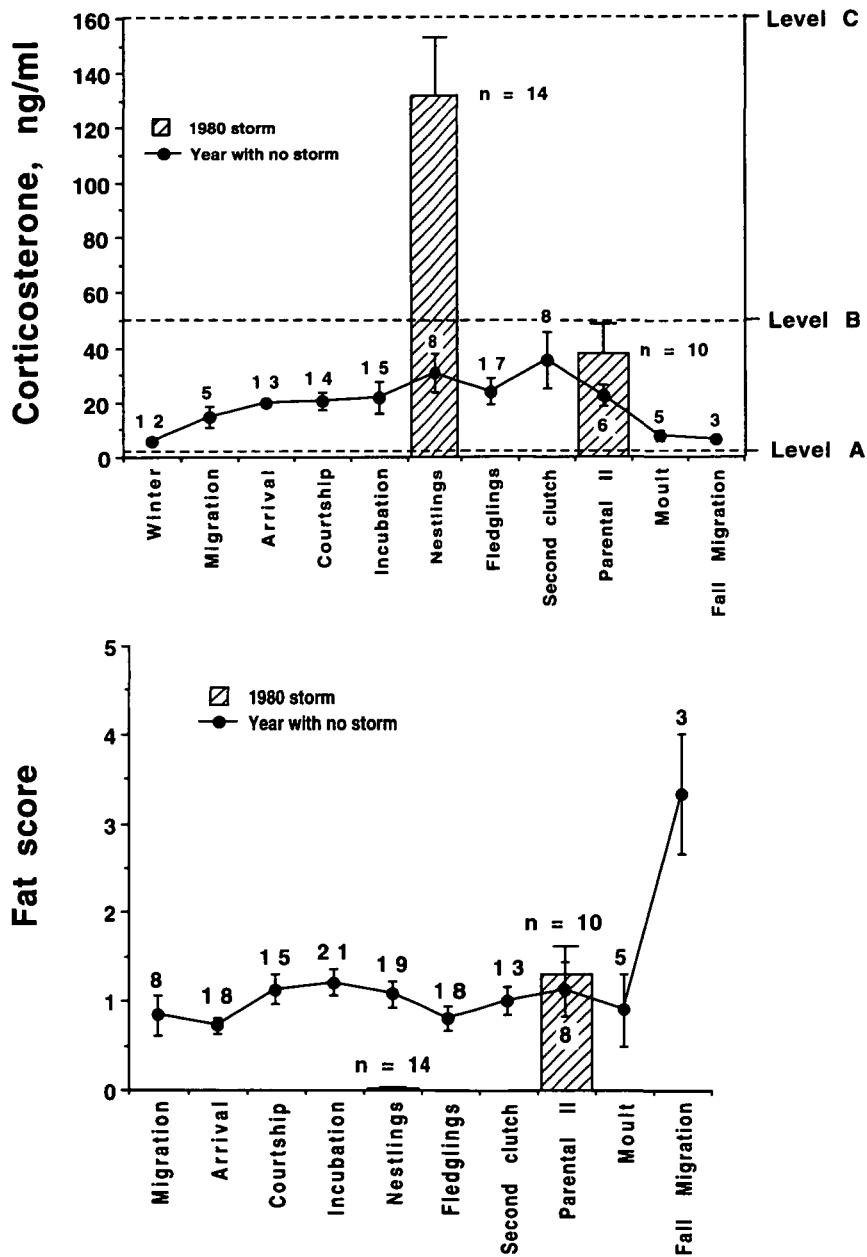


FIG. 2. Changes in circulating plasma levels of corticosterone (top panel, solid line) and fat depot (lower panel, solid line) in male Puget Sound white-crowned sparrows (*Zonotrichia leucophrys pugetensis*) during a normal breeding cycle (i.e., no labile perturbation factors). Note the depiction of stages and sub-stages during the breeding period (X axis). In May 1980 a prolonged rain and wind storm resulted in abandonment of nests and territories. Renesting occurred in June and July after weather conditions became more normal. The cross hatched bars show that corticosterone levels were greatly elevated over the year with no storm even though all birds were in the same reproductive sub-stage. Later, when renesting occurred, corticosterone levels had returned to normal. Level A is the absolute baseline of corticosterone, Level B is the limit of normal variation of corticosterone levels in the absence of perturbation factors (i.e., normal daily and seasonal cycles); and Level C is the limit of variation above Levels A and B. Perturbations factors result in transitory increases in corticosterone above Level B (see Wingfield *et al.*, 1997 for details). Fat depot (cross hatched bars, lower panel) in males during the storm were depleted but had returned to normal when renesting. Numbers by points are sample sizes, vertical bars are standard errors of the means. Top panel from Wingfield *et al.* 1997; bottom panel redrawn from Wingfield and Farner (1978) and Wingfield *et al.* (1983).

of emergency life history stage is applicable to other vertebrate taxa as well. The next question is, do individuals in the field that are challenged by LPFs actually show elevations in glucocorticosteroids, and how quickly is the emergency life history stage initiated?

TRIGGERING THE EMERGENCY LIFE HISTORY STAGE: THRESHOLDS AND TIME COURSE

Field investigations of bird populations responding to labile perturbation factors

There are now numerous studies indicating that individuals responding to unpredictable events in the environment, such as direct LPFs, show elevated levels of corticosterone in blood consistent with development of an emergency life history state. This response appears to occur both in breeding and non-breeding life history stages, and may be possible in other stages as well (see Wingfield, 1984, 1988, 1994 for reviews). For example, male white-crowned sparrows that had abandoned their nests and territories in response to a severe and prolonged storm in May 1980 had greatly elevated circulating levels of corticosterone compared with males sampled in a year with no storm (Fig. 2, top panel; Wingfield and Farner, 1978; Wingfield *et al.*, 1983). Note that in the year with fair weather, baseline corticosterone levels increased in breeding males (within level B of Wingfield *et al.*, 1997) and declined thereafter (level A of Wingfield *et al.*, 1997). Later in the season, after the severe storm had passed and birds were reneating, plasma levels of corticosterone had returned to normal for that time of year (*i.e.*, within level B, Fig. 2). During the storm, subcutaneous fat depots were virtually depleted, but returned to normal after the storm had passed when reneating was initiated (Fig. 2, bottom panel). Thus corticosterone levels were high when an emergency life history stage had been triggered. It has been suggested that the effects of corticosterone on territorial behavior do not occur via suppression of sex steroid hormones that normally activate reproductive behavior. In Figure 3 it can be seen that during the storm of 1980, male white-crowned sparrows had normal levels of luteinizing hormone and

testosterone, thus supporting the hypothesis that corticosterone may be acting directly to suppress expression of reproductive behavior rather than indirectly through decreased secretion of sex steroids that activate such behavior. Further research is needed to determine the mechanisms and locus of corticosterone action in this regard.

These responses are not restricted to the breeding stage. Severe winter weather that triggered emergency life history stages was accompanied by elevated plasma levels of corticosterone in dark-eyed juncos (Rogers *et al.*, 1993); Harris' sparrows, *Zonotrichia querula*, (Rohwer and Wingfield, 1981); and common diving petrels, *Pelecanoides urinatrix*, (Smith *et al.*, 1994). It seems likely, then, that not only does the emergency life history stage have common characteristics regardless of the normal life history stage during which it may be triggered, but also that it is dependent upon a rise of glucocorticosteroids and other hormones of the HPA axis.

Time courses

Another question that needs to be resolved is how quickly a LPF can trigger an emergency life history stage. This will, of course, depend to a great extent on the severity and intensity of the LPF. Experimental evidence suggests that in white-crowned sparrows food withdrawal results in a decrease in blood glucose and an increase in free fatty acid levels for up to 22 hours of fasting. Plasma levels of corticosterone rose within at least 2 hours of fasting (Richardson, 1996). More recent evidence suggests that even as little as one hour without food stimulates an increase in corticosterone levels and heightened perch hopping activity (S. Lynn and J. C. Wingfield, unpublished). Furthermore, non-invasive administration of corticosterone to male white-crowned sparrows (via feeding of meal worms injected with vehicle, or corticosterone) resulted in an increase in plasma levels of corticosterone within 5–10 min and heightened perch hopping activity within 15 min (Breuner *et al.*, 1998). These data clearly suggest that even short term fasting, as would be expected during onset of a severe storm (a form of direct LPF), results in an

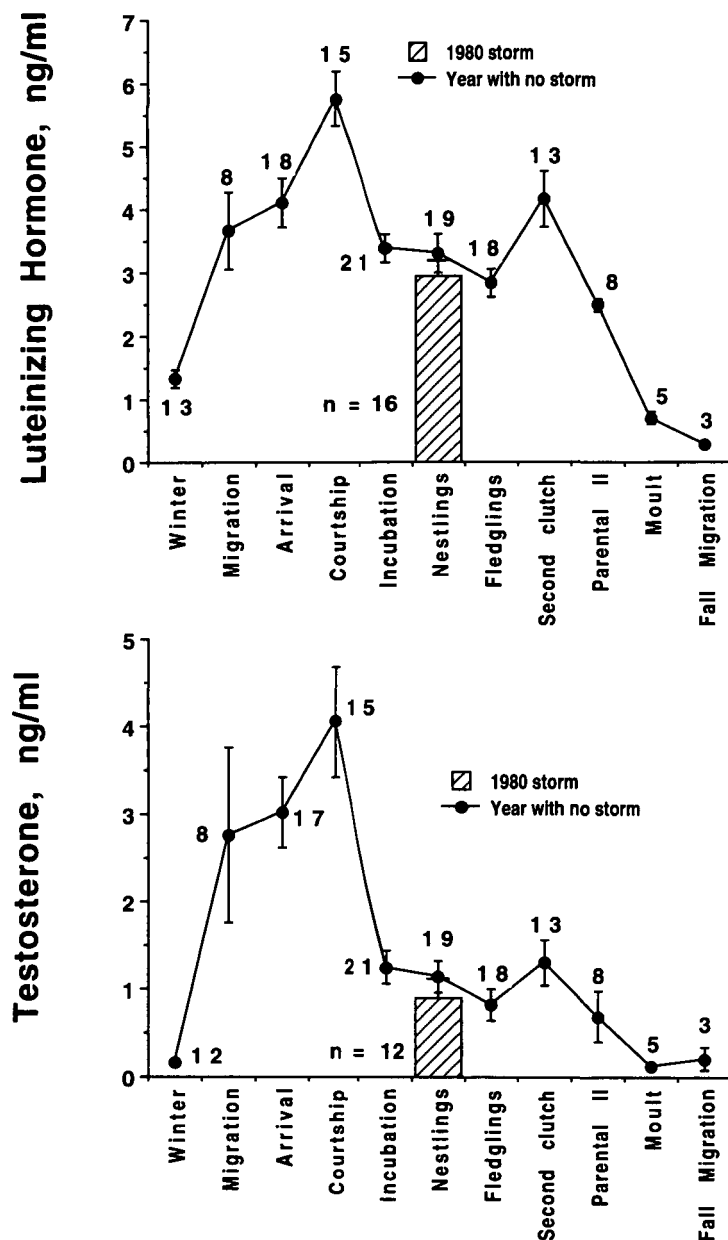


FIG. 3. Changes in circulating plasma levels of luteinizing hormone (LH, top panel, solid line) and testosterone (lower panel, solid line) in male Puget Sound white-crowned sparrows (*Zonotrichia leucophrys pugetensis*) during a normal breeding cycle (*i.e.*, no labile perturbation factors). Note the depiction of stages and sub-stages during the breeding period (X axis). In May 1980 a prolonged rain and wind storm resulted in abandonment of nests and territories. Renesting occurred in June and July after weather conditions became more normal. The cross hatched bars show that both LH and testosterone levels were not affected by the storm even though nests and territories were abandoned—behavior known to be under control of sex steroids in this species. Numbers by points indicate sample sizes, vertical bars are standard errors of the means. Redrawn from Wingfield and Farmer (1978) and Wingfield *et al.* (1983).

increase in circulating glucocorticosteroids within at least an hour, and that behavioral responses consistent with the emergency life history stage follow within minutes. These phenomena are entirely consistent with observations of bird populations in the field as they respond to natural LPFs. Mechanisms of these rapid responses are currently under investigation.

A model to explain biological context for the emergency life history stage at population and individual levels

The evidence is compelling that the emergency life history stage can be triggered within minutes to hours at any, or at least most, times of year, and by a whole spectrum of LPFs. However, the question of whether a common physiological pathway exists by which such diverse environmental information is transduced into secretions of the hypothalamo-pituitary-adrenal axis remains. Much further work is needed at the central level, but we have postulated the following scheme which may provide a unifying framework to explain how physical and social LPFs may trigger an emergency life history stage under extremely diverse conditions. We admit that the model is very simplistic, but we feel it has heuristic value as a beginning to understand the possible common themes underlying these phenomena.

The framework is based on a simple energetic theme. Here we propose E to represent the energy required by an individual to survive day to day and pursue its activities as demanded by the progression of normal life history stages. It makes no assumptions or adjustments for specialized nutrient requirements, vitamins etc., although these could easily be worked into the model if nutritionists required. We then suggest the following:

- EG = Energy to be gained from food in environment
- EE = Existence energy (*i.e.*, maintenance-level = resting metabolic rate)
- EI = Energy required to obtain food, process and assimilate it under ideal conditions

EO = Additional energy required to obtain food under non-ideal conditions

In the theoretical example given in Fig. 4, EG, EI and EE remain constant over time (such as the annual cycle of seasons). In reality of course, they will vary as a function of predictable changes in the environment. We have kept them constant here for simplicity and illustrative purposes. Normally, $EG - (EI + EO + EE) > 0$ and thus the individual should remain in an appropriate life history stage. However, if a LPF should occur, (Fig. 4), then the additional energy required to obtain food may increase such that $EG - (EI + EO + EE) < 0$. In this case the individual should trigger an emergency life history stage. Note that once the perturbation passes and $EG - (EI + EO + EE) > 0$ once again, then the individual can return to a life history stage appropriate for that time. On the other hand, if alternate habitat is discovered that allows positive energy balance, then the individual may settle there and resume its normal life history stage. The duration of the perturbation factor can vary resulting in short duration of the emergency life history state (Fig. 4, upper panel) to long duration (Fig. 4, lower panel). In the latter case, the original life history stage may be inappropriate, and the next stage, or most appropriate one may be assumed. In this way the individual is able to adjust life history stages to maximize survival and ultimately lifetime reproductive success in response to both predictable and unpredictable environmental events.

MODULATION OF ADRENOCORTICAL
RESPONSES TO LABILE PERTURBATION
FACTORS

There are now several lines of evidence suggesting that in birds, the sensitivity of the hypothalamo-pituitary-adrenal (HPA) axis to LPFs changes at the population level (*i.e.*, among life history stages) and at the individual level (*e.g.*, Wingfield, 1994; Wingfield *et al.*, 1995). Most of these studies have been conducted in the field and use a standardized test to determine the sensitivity of the HPA axis. Because different species are acclimated to widely different habitats, and any one population may move

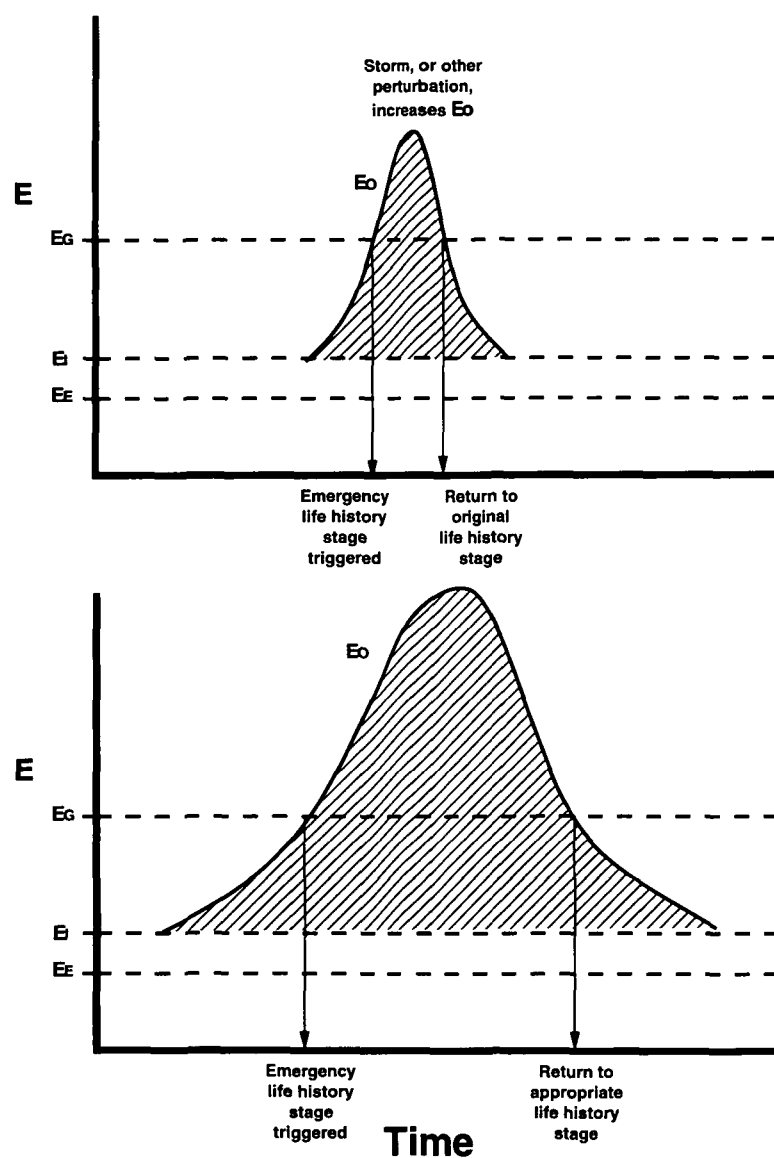


FIG. 4. Theoretical depiction of when an emergency life history stage should be triggered. Over time (such as annual cycle of seasons), we assume here that E (energy as defined by all nutrients an organism needs to survive and breed), and its components EG = energy to be gained from food in environment; EE = existence energy; and EI = energy required to obtain food, process and assimilate it under ideal conditions remain constant. EO = additional energy required to obtain food under non-ideal conditions increases because of a storm or other labile perturbation factor. Then if $EG - (EI + EO + EE) > 0$ the individual should remain in an appropriate life history stage. If $EG - (EI + EO + EE) < 0$ then the individual should trigger an emergency life history stage. Periods of the perturbation may be short (upper panel) or long (lower panel). Note also that EG , EI and EE may all change seasonally but in a predictable manner. EO increases as a result of unpredictable perturbation factors.

among several habitats during its annual cycle of life history stages, it is not possible to use "natural" LPFs such as temperature extremes, food restriction etc. across all species. Thus, we have developed the "capture stress" protocol in which birds are captured, handled and restrained in a cloth bag for periods up to one hour. During this time small blood samples are collected at intervals (within 1–2 min of capture and then at 5, 10, 30 and 60 min of handling and restraint) for measurement of corticosterone. We can then compare the baseline value at capture (indicative of the level in the free-living animal just prior to capture), the rate and degree of increase, and the maximal level of corticosterone attained (*e.g.*, Wingfield, 1994). While we admit that this procedure is not the most perfect way to assess sensitivity of the HPA axis to LPFs, all populations thus far studied react to capture with increased heart rate, respiration rate, and struggle to escape as expected (individuals try to avoid what to them is likely a predation attempt). It also is a powerful way to stimulate marked increases in circulating glucocorticosteroid levels and although this protocol bears little relation to many of the diverse LPFs known, it does allow us to assess sensitivity of the HPA axis as a measure of responsiveness to acute unpredictable stimuli. We can test many individuals and populations in exactly the same way and thus test hypotheses as to why modulation or individual differences exist. With these provisos in mind we can then test hypotheses as to possible ecological bases of these endocrine phenomena under field conditions that in turn indicate appropriate laboratory experiments to explore mechanisms further.

Modulation at the population level

Wingfield (1988) suggested that seasonal suppression of the HPA axis during the reproductive life history stage may be related to severity of habitat. Although it may be highly adaptive to trigger an emergency life history stage at most times of year, if the breeding season is very short, especially in severe habitats such as the Poles, deserts, high altitudes etc., then it may be advantageous to suppress sensitivity of the HPA

axis to LPFs—at least temporarily. Failure to do so may result in frequent interruption of breeding attempts in severe climates. Blunting sensitivity to LPFs would increase costs of breeding and may even increase mortality, but the pay off is increased reproductive success. Evidence for suppression of the sensitivity of the HPA axis to the capture and handling protocol has been obtained for arctic birds (Wingfield *et al.*, 1995) and birds of the Sonoran Desert (Wingfield *et al.*, 1992). Furthermore, this suppression is most marked when in the parental sub stage (Wingfield *et al.*, 1995). Note, however, that this is not a universal property of the HPA axis in birds in extreme habitats. Some actually increase sensitivity of the HPA axis to LPFs (Astheimer *et al.*, 1995; Wingfield *et al.*, 1995) for reasons as yet unknown.

There is also evidence that even if sensitivity of the HPA axis to LPFs is suppressed, if the LPF is prolonged, then birds have the ability to reactivate adrenocortical responses and trigger an emergency life history stage before individuals become debilitated. A population of arctic breeding Lapland longspurs, *Calcarius lapponicus*, subjected to a 3 day storm in mid June, remained on their nests for several days but then began to abandon as weather conditions failed to improve. Birds captured after abandoning their nests had a rate of increase in corticosterone following the capture and handling protocol that was almost an order of magnitude higher than before the storm (Astheimer *et al.*, 1996). These data emphasize the ability for modulation of sensitivity of the HPA axis to LPFs in both directions. Such modulations allow birds to adjust expression of the emergency life history stage very precisely to environmental conditions and to their life cycle. Mechanisms underlying these modulations are just beginning to be investigated.

Modulation at the individual level

Although there may be marked modulation of sensitivity of the HPA axis to LPFs among populations or within populations from season to season, within these cohorts there may be considerable individual variation. Again, the question "why" comes to

the fore. In white-throated sparrows (*Zonotrichia albicollis*), social status and body mass are negatively correlated with intensity of the adrenocortical response to capture, handling and restraint. This may explain individual differences at least partially (Schwabl, 1995). We have also found that body condition, particularly the size of fat depots, correlates negatively with sensitivity of the HPA axis to the capture protocol in a number of arctic species (Wingfield, 1994; Wingfield *et al.*, 1995), although this is by no means a universal phenomenon. Individual variation in sensitivity of the HPA axis to LPFs in other species may be entirely different ecological bases as yet unknown. Endocrine mechanisms underlying these modulations also remain to be determined.

CONCLUSIONS

Evidence to date is compelling that an emergency life history stage exists that can be triggered at any, or most, intervals in the life cycle of vertebrates. It is distinct from the "flight-or-flight" response because it takes minutes to hours to develop (rather than seconds), and can be triggered by perturbations of the environment that may not necessarily be immediately life threatening (*e.g.*, bad weather). The hormones involved, however, may show considerable overlap between the "flight-or-flight" response and the emergency life history stage, but the behavioral and physiological effects and mechanisms of action are likely to be different. This emergency life history stage may play a major role in maximizing overall lifetime fitness by redirecting individuals away from non-essential activities (such as reproduction) during environmental perturbations. This would allow survival in the best conditions possible so that when the LPF passes the individual can return to its normal life history stage. If this were reproduction, then reneesting would follow. Given the current information available, we can suggest that the behavioral and physiological characteristics of the emergency life history stage should be identical, regardless of the time of year when it is triggered. Also, we suggest that the interactions of hormones in the HPA axis to orchestrate

this stage should be similar at all times of year (but not necessarily among all populations). Further research will now address these issues of action and mechanisms underlying the interaction of glucocorticosteroids and peptides. Of particular interest are the receptors, locations and mechanisms of action at the central level. Most of the work cited here focuses on the class Aves, but we suggest that the emergency life history stage may be triggered in other vertebrates in essentially similar ways. These lines of research also transcend traditional boundaries of ultimate and proximate mechanisms in biological disciplines by incorporating theoretical modeling, ecology, behavior, physiology, endocrinology and cell and molecular mechanisms.

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REFERENCES

- Astheimer, L. B., W. A. Buttemer, and J. C. Wingfield. 1992. Interactions of corticosterone with feeding, activity and metabolism in passerine birds. *Ornis Scand.* 23:355–365.
- Astheimer, L. B., W. A. Buttemer, and J. C. Wingfield. 1994. Gender and seasonal differences in the adrenocortical response to ACTH challenge in an arctic passerine, *Zonotrichia leucophrys gambelii*. *Gen. Comp. Endocrinol.* 94:33–43.
- Astheimer, L. B., W. A. Buttemer, and J. C. Wingfield. 1995. Seasonal and acute changes in adrenocortical responsiveness in an arctic breeding bird. *Horm. Behav.* 29:442–457.
- Axelrod, J. and T. D. Reisine. 1984. Stress hormones: Their interaction and regulation. *Science* 224: 452–459.
- Breuner, C., A. L. Greenberg, and J. C. Wingfield. 1998. Non-invasive corticosterone treatment rapidly increases activity in Gambel's white-crowned sparrows (*Zonotrichia leucophrys gambelii*). *Gen. Comp. Endocrinol.* (In press).
- Buttemer, W. A., L. B. Astheimer, and J. C. Wingfield. 1991. The effect of corticosterone on standard metabolic rates of small passerines. *J. Comp. Physiol. B* 161:427–431.
- Chester-Jones, I., D. Bellamy, D. K. O. Chan, B. K. Follett, I. W. Henderson, J. G. Phillips, and R. S. Snart. 1972. Biological actions of steroid hormones in non-mammalian vertebrates. *In* D. R.

- Idler (ed.), *Steroid in non-mammalian vertebrates*, pp. 414–480. Academic Press, New York.
- Clutton Brock, T. H. 1991. The evolution of parental care. Princeton University Press, Princeton.
- Cunningham, E. T., Jr. and E. B. De Souza. 1993. Interleukin-1 receptors in the brain and endocrine tissues. *Immunol. Today* 14:171–176.
- DeNardo, D. F. and B. Sinervo. 1994a. Effects of corticosterone on activity and home range size of free-ranging male lizards. *Horm. Behav.* 28:53–65.
- DeNardo, D. F. and B. Sinervo. 1994b. Effects of steroid hormone interaction on activity and home range size of male lizards. *Horm. Behav.* 28:273–287.
- Denbow, D. M. and J. F. McCormack. 1990. Central versus peripheral opioid regulation of ingestive behavior in the domestic fowl. *Comp. Biochem. Physiol.* 96C:211–216.
- Deviche, P. and G. Schepers. 1984. Intracerebroventricular injection of ostrich beta-endorphin to satiated pigeons induces hyperphagia but not hyperdipsia. *Peptides* 8:691–694.
- Dunlap, K. D. and D. R. Church. 1996. Interleukin-1 β reduces daily activity level in male lizards, *Sceloporus occidentalis*. *Brain, Behav. Immun.* 10:68–73.
- Fan, Y. and M. A. Ottinger. 1996. Inhibition of hypothalamic chicken gonadotropin-releasing hormone (cGnRH) by opioid peptides in vitro. *Soc. Neurosci. Abstr.* 22:625.2.
- Fujiwara, T., A. D. Cherrington, D. N. Neal, and O. P. McGuiness. 1996. Role of cortisol in the metabolic response to stress hormone infusion in the conscious dog. *Metabolism* 45:571–578.
- Gessamen, J. A. and G. L. Worthen. 1982. *The effect of weather on avian mortality*. Utah State Printing Services, Logan.
- Goldstein, R. E., G. W. Reed, D. H. Wasserman, P. E. Williams, D. Brooks Lacey, R. Buckspan, N. N. Abumrad, and A. D. Cherrington. 1992. The effects of acute elevations in plasma cortisol levels on alanine metabolism in the conscious dog. *Metabolism* 41:1295–1303.
- Gray, J. M., D. Yarian, and M. Ramenofsky. 1990. Corticosterone, foraging behavior, and metabolism in dark-eyed juncos, *Junco hyemalis*. *Gen. Comp. Endocrinol.* 79:375–384.
- Guillemin, R., T. Vargo, J. Rossier, S. Minick, N. Ling, C. Rivier, W. Vale, and F. Bloom. 1977. Beta-endorphin and adrenocorticotropin are secreted concomitantly by the pituitary gland. *Science* 197:1367–9.
- Honey, P. K. 1990. Avian flight muscle *Pectoralis major* as a reserve of proteins and amino acids. MS Thesis, University of Washington, 85 pp.
- Jacobs, J. 1996. Regulation of life history stages within individuals in unpredictable environments. Ph.D. Thesis, University of Washington.
- Johnson, E. O., T. C. Kamilaris, G. P. Chrousos, and P. W. Gold. 1992. Mechanisms of stress: A dynamic overview of hormonal and behavioral homeostasis. *Neurosci. Behav. Rev.* 16:115–130.
- Kendall, M. D., P. Ward, and S. Bacchus. 1973. A protein reserve in the *pectoralis major* flight muscle of *Ouelea quelea*. *Ibis* 115:600–601.
- Leibowitz, S. F., C. R. Roland, L. Hor, and V. Squillari. 1984. Noradrenergic feeding via the paraventricular nucleus is dependent upon circulating corticosterone. *Physiol. Behav.* 32:857–864.
- Maney, D. L. and J. C. Wingfield. 1998. Central opioid control of feeding behavior in the white-crowned sparrow, *Zonotrichia leucophrys gambelii*. *Horm. Behav.* (In press).
- McKay, L. D., N. J. Kenney, N. K. Edens, R. H. Williams, and S. C. Woods. 1981. Intracerebroventricular beta-endorphin increases food intake of rats. *Life Sci.* 29:1429–1434.
- Meyerson, B. and M. Berg. 1977. Influence of beta-endorphin on exploratory, social, and sexual behavior in the male rat. *Acta Pharmacol. Toxicol. (Copenh)* 40:Suppl 1, 1–27.
- Miller, L. J. and F. L. Moore. 1982. Evidence that an opioid peptide inhibits sexual behavior in rough-skinned newts. *Amer. Zool.* 22:92.
- Moore, F. L. and L. J. Miller. 1984. Stress-induced inhibition of sexual behavior: Corticosterone inhibits courtship behaviors of a male amphibian (*Taricha granulosa*). *Horm. Behav.* 18:400–410.
- Morley, J. E., A. S. Levine, G. K. Yim, and M. T. Lowy. 1983. Opioid modulation of appetite. *Neurosci. Biobehav. Rev.* 7:281–305.
- Munck, A., P. M. Guyre, and N. J. Holbrook. 1984. Physiological functions of glucocorticosteroids in stress and their relation to pharmacological actions. *Endocrine Rev.* 5:25–44.
- Rogers, C. M., M. Ramenofsky, E. D. Ketterson, V. Nolan, Jr., and J. C. Wingfield. 1993. Plasma corticosterone, adrenal mass, winter weather, and season in non-breeding populations of dark-eyed juncos (*Junco hyemalis hyemalis*). *Auk* 110:279–285.
- Rohwer, S. and J. C. Wingfield. 1981. A field study of social dominance; plasma levels of luteinizing hormone and steroid hormones in wintering Harris' sparrows. *Z. Tierpsychol.* 47:173–183.
- Repasky, R. R., and D. Schluter. 1994. Habitat distributions of wintering sparrows along an elevational gradient: Tests of the food, predation and microhabitat structure hypotheses. *J. Anim. Ecol.* 63:569–582.
- Richardson, R. D. 1996. Central regulation of food intake in the white-crowned sparrow. Ph.D. Thesis, University of Washington.
- Richardson, R. D., T. Boswell, B. D. Raffety, R. Seeley, J. C. Wingfield, and S. C. Woods. 1995. NPY increases food intake in white-crowned sparrows: Effect in short and long photoperiods. *Am. J. Physiol.* 268:R1418–R1422.
- Sapolsky, R. M. 1987. Stress, social status, and reproductive physiology in free-living baboons. In D. Crews (ed.), *Psychobiology of reproductive behavior: An evolutionary perspective*, pp. 291–322. Prentice-Hall, Englewood Cliffs, New Jersey.
- Sapolsky, R. M. 1996. Why stress is bad for your brain. *Science* 273:749–750.
- Schwabl, H. 1995. Individual variation of the acute adrenocortical response to stress in the white-throated sparrow. *Zoology* 99:113–120.

- Silverin, B. 1986. Corticosterone-binding proteins and behavioral effects of high plasma levels of corticosterone during the breeding period. *Gen. Comp. Endocrinol.* 64:67–74.
- Sirinathsinghji, D. J. S. 1984. Modulation of lordosis behavior of female rats by naloxone, beta-endorphin, and its antiserum in the mesencephalic central gray: Possible modulation via GnRH. *Neuroendocrinol.* 39:222–230.
- Sirinathsinghji, D. J. S., L. J. Rees, J. Rivier, and W. Vale. 1983a. Corticotropin-releasing factor is a potent inhibitor of sexual receptivity in the female rat. *Nature* 305:232–235.
- Sirinathsinghji, D. J. S., P. E. Whittington, A. Audsley, and H. M. Fraser. 1983b. Beta-endorphin regulates lordosis in female rats by modulating LHRH release. *Nature* 301:62–64.
- Smith, G. T., J. C. Wingfield, and R. R. Veit. 1994. Adrenocortical response to stress in the common diving petrel, *Pelecanoides urinatrix*. *Physiol. Zool.* 67:526–537.
- Terenius, L. 1977. Opioid peptides and opiates differ in receptor selectivity. *Psychoneuroendocrinol.* 2: 53–58.
- Ward, P. 1969. The annual cycle of the yellow-vented bulbul, *Pycnonotus goavier*, in a humid equatorial environment. *J. Zool. Lond.* 157:25–45.
- Wingfield, J. C. 1984. The influences of weather on reproduction. *J. Exp. Zool.* 232:589–594.
- Wingfield, J. C. 1988. Changes in reproductive function of free-living birds in direct response to environmental perturbations. In M. H. Stetson (ed.), *Processing of environmental information in vertebrates*, pp. 121–148. Springer-Verlag, Berlin.
- Wingfield, J. C. 1994. Modulation of the adrenocortical response to stress in birds. In K. G. Davey, R. E. Peter, and S. S. Tobe (eds.), *Perspectives in comparative endocrinology*, pp. 520–528. National Research Council Canada, Ottawa.
- Wingfield, J. C., C. Breuner, and J. Jacobs. 1997. Corticosterone and behavioral responses to unpredictable events. In R. J. Etches and S. Harvey (eds.), *Avian endocrinology*, in press. J. Endocrinology Ltd., Bristol.
- Wingfield, J. C., and D. S. Farner. 1978. The endocrinology of a naturally breeding population of the white-crowned sparrow (*Zonotrichia leucophrys pugetensis*). *Physiol. Zool.* 51:188–205.
- Wingfield, J. C., and D. S. Farner. 1979. Some endocrine correlates of reneating after loss of clutch or brood in the white-crowned sparrow (*Zonotrichia leucophrys gambelii*). *Gen. Comp. Endocrinol.* 38:322–331.
- Wingfield, J. C., M. C. Moore, and D. S. Farner. 1983. Endocrine responses to inclement weather in naturally breeding populations of white-crowned sparrows. *Auk* 100:56–62.
- Wingfield, J. C., K. M. O'Reilly, and L. B. Astheimer. 1995. Ecological bases of the modulation of adrenocortical responses to stress in Arctic birds. *Am. Zool.* 35:285–294.
- Wingfield, J. C., and M. Ramenofsky. 1997. Corticosterone and facultative dispersal in response to unpredictable events. *Ardea*. (In press)
- Wingfield, J. C., H. Schwabl, and P. W. Mattocks, Jr. 1990. Endocrine mechanisms of migration. In E. Gwinner (ed.), *Bird migration*, pp. 232–256. Springer-Verlag, Berlin.
- Wingfield, J. C., and B. Silverin. 1986. Effects of corticosterone on territorial behavior of free-living male song sparrows, *Melospiza melodia*. *Horm. Behav.* 20:405–417.
- Wingfield, J. C., J. P. Smith, and D. S. Farner. 1982. Endocrine responses of white-crowned sparrows to environmental stress. *Condor* 84:399–409.
- Wingfield, J. C., C. M. Vleck, and M. C. Moore. 1992. Seasonal changes in the adrenocortical response to stress in birds of the Sonoran Desert. *J. Exp. Zool.* 264:419–428.

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