

Aggressive Behavior

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Introduction

Aggression is one of the most important social behaviors. It is displayed by virtually all animals and serves a wide range of adaptive functions. Aggressive behaviors enable the acquisition and defense of limited valuable resources, such as mates, territories, shelters, and food. However, aggressive encounters are also a costly investment in terms of time, energy, predation risk, and physical injury. Animals evaluate the costs and benefits of competing for resources in search of maximal fitness payoffs to make the decision to fight or give up. In this way, aggressive behaviors mediate the establishment of dominance and subordination relationships among competitors that enable access to a given limited resource. In the context of choosing a mate, for example, the contenders evaluate strength and establish dominance in front of a potential mate. In solitary or territorial species, aggression implies exclusive use of a resource, but in gregarious species, aggression sets relations of dominance and establishes hierarchies. Consequently, aggressive interactions have a direct impact on the survival and reproductive success of individuals.

Historically, studies on aggression have focused on male–male competition. However, aggression is not exclusive to males and occurs not only during competition for a mate. Female–female contests, although less studied, are also seen in nature. Just like in males, preferential access to a resource is advantageous for females, which drives this behavior. Aggression has been defined in a variety of ways. Aggression has traditionally been defined as an overt behavior with the intention of inflicting physical damage upon another individual. Different classifications of aggressive interactions have been proposed, although they have only more recently been defined around the context in which they occur: 1- Territorial aggression; 2- Disputes over food; 3- Aggression to establish relationships of dominance; 4- Parental aggression; 5- Aggression for sexual competition; 6- Antipredator aggression; 7- Irritable aggression.

The neuroendocrine mechanisms of aggressive behavior are conserved in vertebrates and have been principally studied during the breeding season. These studies have focused on male–male interactions and on the role of gonadal steroid hormones, predominantly testosterone (T), as the primary factor regulating aggression. The objective of this article is to provide a brief overview of the endocrine mechanisms modulating aggressive behavior, describing the role of both gonadal and nongonadal steroids. In addition, we discuss the neural circuits that underlie aggression and the role of androgens in promoting aggressive behaviors in primates.

Endocrine Mechanisms of Aggression

Gonadal Steroids

Traditionally, blood-borne gonadal steroids have been the primary focus of neuroendocrine studies on aggression (Fig. 1). The potential role of androgens in aggressive behavior was first explored in roosters in the mid-19th century by Arnold Berthold. In his classic “ablate and replace” study, Berthold removed the testes of sexually immature male chickens and found that castrated male chickens, called capons, exhibited a decrease in the development of certain secondary sex characteristics, such as wattles and a prominent comb; and a decrease in male-typical behaviors, including aggression and mating behaviors. However, when testes were transplanted into capons, the capons exhibited normal behavioral and morphological development. Berthold also observed via dissection that the testes had developed additional vasculature following transplantation, suggesting that a blood-borne factor secreted by the testes is essential for proper behavioral, physiological, and morphological development in roosters. This compound, which Berthold termed “productive verhältniss der hoden,” is now known as the androgen T. Berthold’s experiment was the first to provide evidence that gonadal T regulates aggression in males.

Since the publication of Berthold’s work, numerous studies have provided evidence that circulating gonadal steroids, such as T and estradiol (E₂), promote aggression by binding directly to androgen receptors (AR) and estrogen receptors (ER) in the brain, modulating neural pathways relevant to aggressive behavior. Sex steroid secretion is particularly important in facilitating aggression during the breeding season, when aggressive behavior may be crucial to acquiring a mate and maintaining territorial boundaries. In seasonally-breeding vertebrates, the gonads grow before the breeding season and regress following the termination of breeding. Circulating sex steroid concentrations fluctuate alongside gonadal growth and regression, and high levels generally occur during the breeding season and basal or nondetectable levels occur during the nonbreeding season.

While gonadal steroid secretion is important in modulating aggressive behaviors, recent work indicates that these behaviors are not exclusively regulated by gonadal steroids. There is now strong evidence that the brain is capable of metabolizing circulating gonadal steroids and precursors (such as dehydroepiandrosterone, DHEA) and even synthesizing these steroids *de novo* from cholesterol (Fig. 1). Additional steroidal and nonsteroidal hormones play a role in modulating aggression, including glucocorticoids, a class of steroids known for their antiinflammatory and immunosuppressive effects.

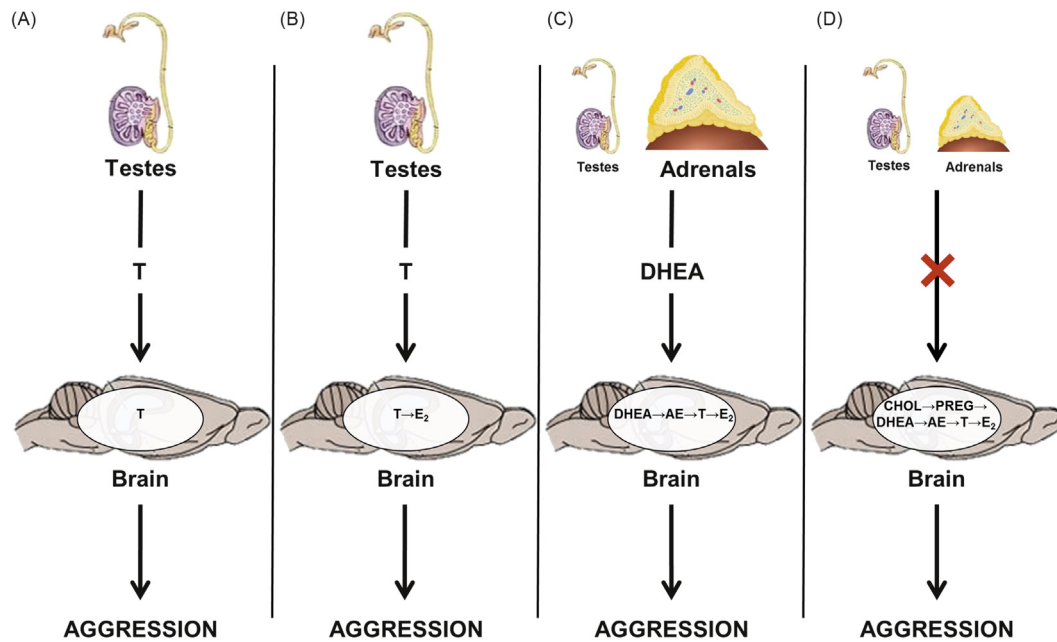


Fig. 1 Pathways by which steroids could affect aggression. (A) Gonadal testosterone (T) acts directly on the brain; (B) gonadal T is converted locally to estradiol (E₂); (C) adrenal dehydroepiandrosterone (DHEA) is converted locally to T and/or E₂; (D) neurosteroids are produced locally de novo from cholesterol (CHOL) via conversion to pregnenolone (PREG), DHEA, androstenedione (AE), T, and E₂, in the absence of gonadal and adrenal steroid production.

Nongonadal Steroids

DHEA is an inert androgen precursor that can be metabolized in tissues that express the appropriate steroidogenic enzymes. DHEA is secreted by the adrenal glands and can travel in the circulation as DHEA-S, the sulfated form of DHEA. Once DHEA has reached a target organ or tissue, it can bind with very low affinity to several different types of hormone receptors, including AR, ER, and glucocorticoid receptors. Alternatively, DHEA can be locally converted to active steroid hormones, such as T and E₂, which can then bind with high affinity to AR and ER, respectively.

The role of DHEA in modulating aggressive behavior has mostly been studied in vertebrates that exhibit year-round aggression, particularly some birds and rodents. Both males and females of these species exhibit territorial aggression outside of the breeding season, despite the fact that the gonads are regressed and circulating androgen levels are low. In fact, the aggressive behaviors observed in nonbreeding some birds are qualitatively and quantitatively similar to those displayed during the breeding season, while Syrian and Siberian hamsters actually show increased aggression toward conspecifics when they are not in breeding condition. Interestingly, these animals also consistently display elevated plasma, gonadal, and adrenal DHEA levels, suggesting that the adrenals and gonads are secreting DHEA into the circulation during the nonbreeding season. Moreover, there is emerging evidence that nonbreeding birds and rodents can display locally elevated DHEA and androgen levels and increased AR and ER in brain regions associated with aggression. Thus, seasonally-breeding animals use two different neuroendocrine mechanisms to sustain year-round territorial aggression: breeding animals rely on circulating gonadal steroids to promote aggressive behaviors, whereas reproductively quiescent animals use circulating DHEA and DHEA synthesized de novo in the brain as an alternative source of androgens during the nonbreeding season.

Because seasonally-breeding vertebrates rely on an alternative mechanism to maintain aggression year-round, physiological indicators of season are critical in signaling animals to shift from one mechanism to another throughout the year. In particular, the hormone melatonin plays an essential role in initiating the physiological and endocrine changes necessary for this "seasonal switch." Melatonin is secreted by the pineal gland, a structure located between the cerebellum and cerebral cortical hemispheres of the brain. Melatonin secretion is high at night and low during the day; therefore, changes in day length, or photoperiod, result in changes in the pattern and duration of melatonin secretion. For example, some species of seasonally-breeding rodents, such as Siberian hamsters, breed during the summer, when there is a longer photoperiod than the winter. During the summer, the duration of melatonin secretion is short compared to the winter. Circulating melatonin binds to melatonin receptors on the gonads and adrenals, resulting in a decrease in gonadal steroid synthesis and an increase in adrenal DHEA synthesis (Fig. 2).

While the adaptive value of this alternative neuroendocrine mechanism of aggression is still being investigated, there are several potential benefits of utilizing DHEA as a source of androgens during the nonbreeding season. For example, maintaining high levels of aggression year-round may benefit individuals by allowing them to gain access to limited food resources and to defend their territories. Furthermore, the regulation of aggressive behaviors via this mechanism is far less energetically costly than sustaining high levels of circulating sex steroids. During the breeding season, a significant amount of energy is devoted to reproductive

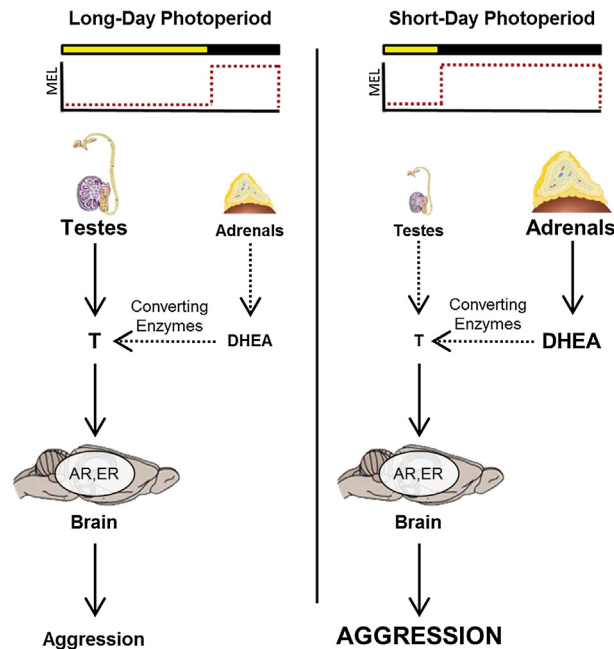


Fig. 2 Theoretical model describing the relationships among melatonin (MEL) secretion, seasonal changes in serum testosterone (T) and dehydroepiandrosterone (DHEA) levels, and aggression in male Siberian hamsters. During the summer, long-day photoperiods (LD) result in a short duration of MEL secretion compared to the short-day photoperiods (SD) of the winter. During LD, T is secreted from the testes, whereas during SD, DHEA is secreted by the adrenal glands. Dotted lines and smaller font sizes symbolize less abundant levels of hormones than solid lines and larger font sizes. T can either directly bind to neural androgen receptors (AR) or can be locally converted to estradiol and bind to neural estrogen receptors (ER). Differences in AR and ER binding and/or abundance in the brain result in changes in aggression, and in hamsters, there is higher aggression during SD than LD.

physiology, such as maintaining the gonads, often at the expense of other physiological processes. Thus, by using an inert precursor (e.g., DHEA), which can be locally and quickly converted to androgens, an individual can exhibit year-round aggression without incurring the energetic costs of maintaining high gonadal steroid levels.

Neural Circuits of Aggression

The regulation of social behavior, such as aggression, depends on neural circuits, including the social behavior network (SBN). These well-studied neural circuits consist of reciprocally connected brain regions, or nodes, located in the forebrain, midbrain, and hindbrain. In mammals, the SBN consists of six nodes: the extended medial amygdala [the medial amygdala and the medial bed nucleus of stria terminalis (BnST)], the lateral septum (LS), the preoptic area (POA), the anterior hypothalamus, the ventromedial hypothalamus (VMH), and the periaqueductal gray. Each of these nodes regulates multiple forms of social behaviors, including sexual behavior, parental behavior, and aggressive behavior. Thus, this network works as a whole, where each region of the network responds to several social stimuli, rather than working independently in the regulation of distinct types of social behavior. In this way, the SBN receives and evaluates external stimuli, integrates them with internal physiological information, and produces an appropriate response with a distinctive circuit activity pattern, biasing behavior that is adaptive for a specific context. For example, across brain nodes, there is a particular pattern of neural activation for sexual behavior, and the same brain nodes show a different pattern of neural activation for aggressive behavior.

Social behavior emerged early in animal evolution, playing a key role in determining the survival and fitness of individuals. Therefore, its neural control is under strong evolutionary pressures. Neuroanatomical and functional studies that assessed the distribution, connectivity, and neurochemistry of the brain nodes of the SBN have found that the neural circuits that regulate social behavior are highly conserved across vertebrates and play similar roles in the regulation of these behaviors. Similarly, despite the diversity of social behaviors regulated by this network (i.e., aggression or paternal care), the neural circuits and hormones involved in modulating these behaviors have been highly conserved in all vertebrate lineages. Birds, reptiles, amphibians, and teleost fish all contain nodes of the SBN that are homologous with the mammalian counterparts described above and have similar activation patterns in similar social contexts. The conservation of these nodes enables comparative studies in different species to establish general principles among vertebrates.

More recent work suggests that a broader social decision-making (SDM) network regulates adaptive social behaviors in response to different context or stimuli. The SDM consists of both the classic SBN nodes and also the mesocorticolimbic reward system. The mesocorticolimbic reward system is composed of several interconnected nodes, including the ventral tegmental area (VTA) and

nucleus accumbens. The VTA contains dopamine-producing neurons that project to the nucleus accumbens and other forebrain regions. Signaling from these dopaminergic neurons regulates several different behaviors, including motivational aspects of motivation, preparatory behavior, and appetitive behavior. Additionally, this system is involved in positive reinforcement associated with stimuli, a fundamental aspect for the expression of appetitive-approach behaviors. The SDM network appeared early in evolution and is also highly conserved among vertebrates.

Every node of the SBN and SDM expresses sex steroid receptors, indicating that these brain regions are sex steroid-sensitive and that hormones play key roles in modulating the activity of these networks and regulating social behaviors. Sex steroids, such as androgens and estrogens, act on the central nervous system by binding to their intracellular receptors (e.g., AR and ER). Bound receptors act as transcription factors to regulate gene expression in brain cells. This genomic mechanism of action allows steroid hormones to modulate the activity of the different brain regions and act indirectly, but not deterministically, on the execution of a behavior. For instance, sex steroids can change a stimulus response threshold, such as the response threshold toward a competitor, but do not typically trigger aggression itself. The genomic effects of steroid hormones on the central nervous system require several hours or days to develop and produce persistent changes in physiology and behavior.

In addition, sex steroids can exert short-term (within 30 min) nongenomic effects, which are often mediated by plasma membrane receptors or by the allosteric modulation of neurotransmitter receptors. For example, ER, such as ER α and ER β , can be associated with the plasma membrane and rapidly regulate intracellular signaling pathways. Another membrane-associated ER is the G-protein-coupled estrogen receptor-1 (GPER-1), which is also present in the brain. Estrogens rapidly influence aggressive behaviors in birds and mammals via intracellular signaling in the SBN and SDM. These rapid effects are more prominent during short photoperiods in both mice and song sparrows. Interestingly, E₂ administration has been shown to rapidly (within 30 min) increase aggression exclusively during the nonbreeding season in both deer mice and song sparrows. These data suggest that steroids act via nongenomic mechanisms to maintain nonbreeding aggression in mammals and birds.

Furthermore, androgens and estrogens produced by the testes and ovaries are under neural influence via the hypothalamic-pituitary-gonadal axis, or HPG axis. Within the HPG axis, the hypothalamus integrates neuroendocrine input and environmental stimuli to regulate the release of gonadotropin-releasing hormone (GnRH). In turn, GnRH secretion stimulates the release of luteinizing hormone (LH) and follicle-stimulating hormone (FSH) from the pituitary gland, which then stimulate gonadal secretion of sex steroids. Circulating sex steroids reach virtually every cell in the body, including cells in the central nervous system, where they can bind to sex steroid receptors and regulate cells in the SBN and SDM.

In addition, the brain itself is capable of locally synthesizing sex steroids. The brain possesses the necessary enzymes for steroidogenesis and can synthesize active sex steroids called neurosteroids, either from inert androgen precursors or de novo from cholesterol (Fig. 1). Neurosteroids can regulate behavior via actions on the SBN and SDM. For example, in male song sparrows, aromatase, the enzyme that converts androgens into estrogens, is critical for the expression of aggression in the breeding season, but also in the nonbreeding season when circulating sex steroids are nondetectable. Several brain regions, such as the POA, VMH, and BnST, contain elevated levels of aromatase year-round. In contrast, aromatase activity decreases in the nucleus taeniae, which is similar to the mammalian medial amygdala, during the molt, when song sparrows also show a decrease in aggressive behaviors. Similarly, the activity of brain 3 β -hydroxysteroid dehydrogenase/ Δ 5- Δ 4 isomerase (3 β -HSD), an enzyme that catalyzes the conversion of DHEA to an active androgen, changes with season and social context. In song sparrows, 3 β -HSD activity is higher during the nonbreeding season in multiple brain areas, and an aggressive social stimulus rapidly increases enzymatic activity in the brain. Together, these data provide evidence of neural steroidogenesis and suggest that local neurosteroid production occurs predominantly during the nonbreeding season.

Androgens and Primate Aggression

Aggression has been a central topic of human and nonhuman primate research for many years. Considerable research indicates the importance of environmental and social influences on the development and expression of primate aggressive behavior. Dominance is the most well-known social influence on aggression, and dominance hierarchies are a major organizing principle in many primate societies. Although the hormonal mechanisms of primate aggression have been studied, much less is known compared with rodents. Most recent studies on the neuroendocrine regulation of primate aggression measure androgens in feces or urine. While collection of such samples can be performed noninvasively, excreted levels frequently represent a fraction of plasma levels. In general, high rates of aggression are positively correlated with elevated androgen concentrations in nonhuman primates. This correlation is observed in seasonal changes in androgens and aggression, sex differences in aggression, and increased aggression at puberty.

Many primates reproduce seasonally and have provided information on correlations between androgens and aggression. Seasonality in testicular activity, with peak circulating T concentrations during the mating season, has been reported in many primates. Although exceptions exist, the majority of research suggests that androgens do not necessarily cause aggression in nonhuman primates. In Japanese macaques, seasonal increases in circulating androgens precede seasonal increases in aggression by several months, suggesting that these hormones do not affect aggression in a simple causal way. Further, experimental elevations of T do not reliably increase aggression. In rhesus monkeys, injections of human chorionic gonadotropin stimulated T production, but had no consistent effect on rates of aggression. Rather, increased circulating T levels were associated with an intensification of existing behavior, which did not disrupt group stability. In contrast, injections of T propionate increased aggression in dominant male long-tailed macaques (*M. fascicularis*), but increased submission in subordinates. When male marmosets (*Callithrix jacchus*) were castrated as neonates and tested as adults, they displayed high rates of aggression with female partners and low rates with

male partners. Moreover, the effects of neonatal castration on aggressive behavior were reversed by T treatment in adulthood. Several studies have found that castration of adult males has little effect on their aggressive behavior, suggesting that gonadal T is not required to maintain aggression in adults.

Some studies in nonhuman primates also suggest that aggressive behavior can be unrelated to fecal T levels, but related to adrenal androgens. Although the adrenal gland can secrete high levels of DHEA and DHEA-S, the role of adrenal DHEA and DHEA-S in primate aggression is largely unknown. One study assessed circulating DHEA-S levels in a population of wild baboons and found high DHEA-S concentrations in both male and female baboons, showing marked age-related decreases in both sexes. DHEA-S levels were not compared with aggressive behaviors, however.

Although the study of DHEA as a regulator of aggression in humans has received some limited experimental attention, much less is known about this mechanism compared to nonprimates. However, alterations in DHEA and DHEA-S have been implicated in a range of psychiatric disorders in humans. Circulating levels of DHEA-S are generally 1000-fold higher than levels of DHEA in humans. Studies from rodents and birds suggest that androgens, such as DHEA, may regulate aggression in situations where aggression seems otherwise T-independent. Adrenal DHEA appears to play a role in human aggression, as indicated by studies on "conduct disorder," typically defined as a collection of symptoms, including aggression directed toward people or animals, destruction of property, theft, and serious violations of rules. Prepubertal boys with conduct disorder were found to have higher levels of plasma DHEA-S, but not T, than normal control boys. Also, DHEA-S concentrations were correlated with the intensity of aggression as rated by parents and teachers. In another study, plasma DHEA-S concentrations were found to be higher in boys with conduct disorder than in boys with attention-deficit/hyperactivity disorder (ADHD) or normal controls. Recent research has examined circulating levels of cortisol, DHEA and DHEA-S in delinquent adolescent boys diagnosed with conduct disorder compared with healthy controls. Hormone levels were correlated with aggression as determined by the Child Behavior Checklist and the Overt Aggression Scale. Delinquent boys had higher DHEA-S levels than control boys, but did not show any differences in either DHEA or cortisol. Collectively, these data suggest a relationship between DHEA-S and aggression, at least in male adolescents diagnosed with clinically-relevant psychiatric conditions.

Adrenal androgen precursors may also contribute to the regulation of aggression in human females. Adolescent and adult females with congenital adrenal hyperplasia who were exposed to high levels of adrenal androgen precursors in the prenatal and early postnatal periods were found to have greater self-reported aggression ratings than were control females. More recently, adrenal androgen precursor levels have been determined in adolescent girls diagnosed with conduct disorder. Specifically, blood samples were drawn from adolescent girls with either conduct disorder or no psychiatric disorder, and samples were assessed for DHEA, DHEA-S, cortisol, and gonadal androgens and estrogens. Girls with conduct disorder scored higher on a clinical aggression scale and demonstrated significantly lower cortisol to DHEA ratios, but did not differ from control girls on any other hormone measurement. Furthermore, girls diagnosed with aggressive conduct disorder had lower cortisol to DHEA ratios than those with nonaggressive conduct disorder.

In addition to regulating aggression in adolescents, DHEA also modulates aggressive behaviors in adults. In a study of alcohol withdrawal, serum levels of DHEA-S and cortisol, as well as DHEA-S response to treatment with an exogenous steroid, dexamethasone, were determined in adult alcohol-dependent or healthy control males. Alcohol-dependent subjects displayed reduced basal and dexamethasone-induced levels of DHEA-S compared with control subjects in late alcohol withdrawal. When alcohol-dependent subjects were separated into high and low aggression groups, lower basal DHEA-S levels were seen only during early alcohol withdrawal in high aggression individuals, whereas DHEA was lower only during late withdrawal in low aggression individuals relative to control subjects. In contrast, dexamethasone-induced decreases in DHEA-S were observed during both early and late alcohol withdrawal, whereas lower DHEA levels were only seen during early withdrawal. While the meaning of these results is not entirely clear, these data suggest an important link between DHEA and aggressive behavior, at least under conditions of drug withdrawal. Whether a link between DHEA, DHEA-S and aggressive behavior exists in healthy adult and adolescent males and females remains to be determined. Regardless, it is clear that considerably more research on DHEA and human aggression is needed.

Conclusions

Aggression is displayed by virtually all organisms and allows males and females to compete for access to limited resources. Many studies have examined the endocrine and neural regulation of aggression, and these studies highlight the importance of steroid hormones, such as testosterone, and brain circuits, such as the social behavior network. Aggression occurs in a wide variety of physiological and environmental contexts, and the neuroendocrine regulation of aggression varies across contexts as well. For example, in the breeding season, gonadal sex steroids play important roles in the modulation of aggressive behavior, but in the nonbreeding season, neurally-synthesized sex steroids are critical. Furthermore, sex steroids affect aggressive behavior via both genomic and nongenomic mechanisms, and the balance between these two mechanisms of action depends on the physiological and environmental contexts. Despite the variability of aggressive behaviors across species, the relevant neural circuits and neuroendocrine mechanisms are generally well conserved. Therefore, comparative studies in different vertebrate taxa can shed light on common mechanisms and establish general principles in the control of aggression.

Further Reading

- Adkins-Regan, E. (2005). *Hormones and Animal Social Behavior* (1st Edn.). Princeton: Princeton University Press.
- Demas, G. E., Polacek, K. M., Durazzo, A., & Jasnow, A. M. (2004). Adrenal hormones mediate melatonin-induced increases in aggression in male Siberian hamsters (*Phodopus sungorus*). *Hormones and Behavior*, 46, 582–591.

- Goldman, B. D. (2001). Mammalian photoperiodic system: Formal properties and neuroendocrine mechanisms of photoperiodic time measurement. *Journal of Biological Rhythms*, *16*, 283–301.
- Goodson, J. L., & Kabelik, D. (2009). Dynamic limbic networks and social diversity in vertebrates: From neural context to neuromodulatory patterning. *Frontiers in Neuroendocrinology*, *30*, 429–441.
- Heimovics, S. A., Trainor, B. C., & Soma, K. K. (2015). Rapid effects of estradiol on aggression in birds and mice: the fast and the furious. *Integrative and Comparative Biology*, *55*(2), 281–293.
- Jasnow, A. M., Huhman, K. L., Bartness, T. J., & Demas, G. E. (2000). Short-day increases in aggression are inversely related to circulating testosterone concentrations in male Siberian hamsters (*Phodopus sungorus*). *Hormones and Behavior*, *38*, 102–110.
- King, J. A. (1973). The ecology of aggressive behavior. *Annual Review of Ecology and Systematics*, *4*, 117–138.
- Laredo, S. A., Landeros, R. V., & Trainor, B. C. (2014). Rapid effects of estrogens on behavior: Environmental modulation and molecular mechanisms. *Frontiers in Neuroendocrinology*, *35*(4), 447–458.
- Maney, D. L., & Goodson, J. L. (2011). Neurogenomic mechanisms of aggression in songbirds. *Advances in Genetics*, *75*, 83–119.
- Moyer, K. E. (1968). Kinds of aggression and their physiological basis. *Communications in Behavioral Biology*, *2*, 65–87.
- Nelson, R. J. (2005). *Biology of Aggression* (1st ed.). New York: Oxford University Press.
- Newman, S. W. (1999). The medial extended amygdala in male reproductive behavior: a node in the mammalian social behavior network. *Annals of the New York Academy of Sciences*, *877*, 242–257.
- O'Connell, L. A., & Hofmann, H. A. (2011). The vertebrate mesolimbic reward system and social behavior network: a comparative synthesis. *The Journal of Comparative Neurology*, *519*, 3599–3639.
- Quiring, D. P. (1944). Transplantation of testes (by A. A. Berthold). *Bulletin of the History of Medicine*, *16*, 399–401.
- Rose, R. M., Gordon, T. P., & Bernstein, I. S. (1972). Plasma testosterone levels in the male rhesus: Influences of sexual and social stimuli. *Science*, *178*, 643–645.
- Soma, K. K., Rendon, N. M., Boonstra, R., Albers, H. E., & Demas, G. E. (2015). DHEA effects on brain and behavior: Insights from comparative studies of aggression. *The Journal of Steroid Biochemistry and Molecular Biology*, *145*, 261–272.
- Svare, B. B. (1983). *Hormones and Aggressive Behavior* (1st Edn.). New York: Plenum.
- Van Goozen, S. H. M., Matthys, W., Cohen-Kettenis, P. T., Thijssen, J. H. H., & van Engeland, H. (1998). Adrenal androgens and aggression in conduct disorder prepubertal boys and normal controls. *Biological Psychiatry*, *43*(2), 156–158.
- Wingfield, J. C., Ball, G. F., Dufty, A. M., Hegner, R. E., & Ramenofsky, M. (1987). Testosterone and aggression in birds. *American Scientist*, *75*, 602–608.
- Wingfield, J. C., Hegner, R. E., Dufty, A. M., Jr., & Ball, G. F. (1990). The "challenge hypothesis": Theoretical implications for patterns of testosterone secretion, mating systems, and breeding strategies. *The American Naturalist*, *136*(6), 829–846.