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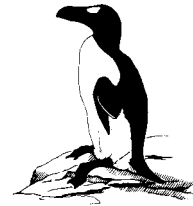
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PERSPECTIVES IN ORNITHOLOGY

ADVANCES IN AVIAN BEHAVIORAL ENDOCRINOLOGY

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BIRDS HAVE LONG been the focus of studies aimed at understanding the hormonal control of behavior. The very first “endocrine” experiment of any type is often considered the castration and testicular replacement of roosters by Berthold in 1849 (although see Wingfield and Farner 1993). In the century after Berthold, advances in the study of behavior, endocrinology, and poultry science, established the key role of the gonadal steroid hormones—testosterone (T), estradiol (E₂), and progesterone (P)—in controlling avian aggressive, copulatory, courtship, vocal, and parental behaviors. Typically, investigators examined the effects of gonadectomy in males and females, followed by gonadal hormone replacement, or replacement of the entire gonad. In some cases, hormones were implanted directly into the brain.

Avian behavioral endocrinology saw great advances between the 1960s and 1980s as investigators developed the use of radioimmunoassay (RIA) to measure concentrations of sex steroid hormones in blood, radioactive-steroid autoradiography to determine steroid-sensitive targets in the brain, sex steroid antagonists, and enzyme inhibitors to block sex steroid action or steroid production. That period also saw the development of biochemical, immunocytochemical, and molecular techniques for understanding the chemistry and cell biology of the sex steroid receptors, the metabolism of steroid hormones by the brain, and the anatomy of

complex neural circuits. Together with research on other vertebrate species, several key principles in behavioral endocrinology became established.

Steroid actions on brain and behavior.—Sex steroids permanently influence the developing avian brain. Those “organizational” effects establish the foundation for the performance of masculine or feminine behaviors by adults. In general, ovarian estrogens feminize and demasculinize the developing hypothalamus and perhaps other brain areas known to control copulatory behaviors (Adkins 1981, Balthazart et al. 1996). Treatment of developing female Zebra Finches (*Taenopygia guttata*) with estrogens can masculinize their brain regions controlling song, but there is some doubt as to whether song-system development naturally depends on estrogens in the male Zebra Finch, or in males of other oscine songbirds (Schlinger 1998). Adult behavioral expression is also influenced by sex steroids, but the effects in adults are typically transient and called *activational*. In males, androgens and estrogens activate masculine copulatory and aggressive behaviors, including song. In females, estrogens and progestins activate female receptive behaviors and parental care (Wingfield et al. 1999).

Plasma sex steroid concentrations and behavior.—Measures of sex steroid concentrations in blood of developing and adult birds generally confirm the close relationship between gonadal activity, elevated hormone levels in blood, and the organization and activation of reproductive

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behaviors (Wingfield and Farner 1993). In the most general sense, E_2 is elevated in the blood of developing females, but not males (although there may be exceptions). In seasonal breeding birds, T is elevated in the blood of males only during periods of reproduction, usually coincident with territorial, courtship, and copulatory behaviors. In adult females, E_2 is elevated when females are receptive to males and during laying, whereas P is elevated in some species during incubation and brooding. There are exceptions to this latter rule, notably in some tropical birds with year-round low levels of circulating sex steroids, including when breeding (see also below).

Mechanism of sex steroid action.—Sex steroids act on target tissues by binding to intracellular receptors. Bound receptors bind to specific sequences of DNA, or *promoters*, that then regulate the transcription of specific genes. In fact, birds have played a key role in the elucidation of fundamental molecular mechanisms derived from studies of hormone action (O'Malley 1995). In the brain, cells in specific brain regions express intracellular steroid receptors, and in those cells, sex-steroid regulated genes influence cell survival, morphology, physiology, or activity (McEwen and Alves 1999). Although some sex-steroid-dependent genes have been identified, we are a long way from knowing the full suite of genes up- or down-regulated by steroids in the avian brain (Clayton 1997).

In most species, sex steroids bind to receptors in neurons in the hypothalamus, archistriatum, and midbrain—areas shown by other means to be involved in reproductive, aggressive, and vocal behaviors. An important exception to that pattern has been documented in many oscine songbirds, in which additional steroid-sensitive neurons are found in fore-brain structures associated with the learning and expression of song (Metzdorf et al. 1999). Those brain regions can be sexually dimorphic in size and other characteristics, correlated with differences in singing by males and females (Nottebohm and Arnold 1976).

Brain steroid metabolism.—Sex steroids in blood often undergo enzymatic conversions into active or inactive products in the brain. This brain steroid metabolism markedly influences the organizational and activational effects of circulating sex steroids, especially the actions of T

in male birds (Balthazart et al. 1996, Schlinger 1997). In the avian brain, T can be converted into E_2 by the enzyme aromatase; into 5α -dihydrotestosterone (5α -DHT) by the enzyme 5α -reductase; or into 5β -DHT, by the enzyme 5β -reductase. Whereas 5β -DHT is considered an inactive compound (Hutchison and Steimer 1981), E_2 and 5α -DHT are active metabolites of T and bind to and activate intracellular receptors for estrogen and androgen, respectively. Consequently, androgenic or estrogenic pathways in the brain (and thus behaviors) can be independently activated by T derived from blood.

Those ideas in large part constitute the current conceptual foundation for the hormonal control of avian behavior. However, concepts continue to be redefined as cellular and molecular biological tools are used increasingly on a greater diversity of bird species. In what follows, we describe some birds whose physiology differs from established paradigms. We also describe some new mechanisms that expand our views of the kinds of neural circuits on which steroids act, the ways in which hormones are made available to neural circuits, and the ways in which neurons respond to hormonal signals. Although our focus here is on the sex steroid hormones, studies on nonsteroidal hormones and the adrenal glucocorticoids are contributing to our understanding of hormone action on the brain.

HORMONES AND BRAIN DEVELOPMENT

The dogma that the gonads use steroid hormones to direct sexually dimorphic brain development may not apply to all avian neural systems. Estrogens, and to a lesser degree androgens, can masculinize song-control regions of developing female Zebra Finches, suggesting that they do so naturally in males. However, there is no evidence that the testes are involved in masculine brain development (Arnold 1997). Thus, whereas steroids may still be involved, they are likely not derived from the gonads. It is possible that steroidal or nonsteroidal signals produced directly in the brain dictate full song-system development, independent of the gonads (Arnold 1997, Schlinger 1998).

In addition to the possibility that such "brain autonomous" mechanisms help guide the growth of some sexually dimorphic neural cir-

cuits, laying females may also deposit sex steroids into the yolk that might influence the way the embryonic brain develops (e.g. Schwabl 1993). Those steroids originate in the mother, and thus represent a nontraditional source of sex steroids to the developing brain.

HORMONAL CONTROL OF COMPLEX COURTSHIP BEHAVIORS

Males of many species of birds perform complex visual displays to attract and to stimulate females. Surprisingly, little is known about the hormonal control of those behaviors. We have begun studies of Golden-collared Manakins (*Manacus vitellinus*) that live in Panamanian rainforests. Like many other manakins (Pipridae), adult males develop a brilliant and conspicuous plumage, establish an arena, and display to females in leks. Those visual displays often involve rapid jumps, short dances, brief flights, and the erection of feather tracts, particularly those under the throat. Males appear to vocalize only weakly. However, they punctuate their displays with loud sounds, or wingsnaps, apparently produced by upward flips of the wings. Females are cryptically colored and do not perform these behaviors.

Those displays resemble other kinds of hormone-dependent behaviors, such as oscine birdsong and copulatory behaviors; they are associated with reproduction and they are produced by males only. We hypothesized that sex-steroid-sensitive and sexually dimorphic neural circuits and peripheral muscles might exist in manakins to control those remarkable behaviors. We have evidence supporting both hypotheses. First, we have found extensive binding of radioactive-T (or metabolites of T) in cells of the manakin spinal cords, especially in regions of the cord containing motoneurons controlling the wing and leg muscles. We found more of such T-accumulating cells in males than in females (Schultz and Schlinger 1999). Secondly, we have found sexually dimorphic properties (overall muscle size, fiber size, and myosin content) of two wing muscles that might generate the rapid upward flips of the birds' wings used in wingsnapping (Schlinger et al. 2001, B. Schlinger pers. obs.).

Those results reveal a new sexually dimorphic, steroid-sensitive neuromuscular system in birds. Given that complex physical displays

are common in males of many bird species, but less so in females, and that displays require precise coordination of several peripheral neuromuscular systems, we would expect that (a) the spinal cord of birds may gain appreciation as an important sex steroid target, and that (b) additional muscles may be found that differ between males and females to allow for performance of those behaviors. Sex steroids might play a substantial role in the neuromuscular coordination underlying some of the truly spectacular displays of Birds of Paradise, many Galliformes, Anseriformes, and Bustards, to name just a few.

HORMONAL CONTROL OF WINTER AGGRESSION

The activation of male territorial aggression during the breeding season by gonadal T has been well studied in a variety of avian species (Wingfield et al. 1990). Some birds also defend territories in the nonbreeding season, when the gonads are regressed and plasma T levels are basal (Soma and Wingfield 1999). Winter territoriality has generally been thought to be independent of the activational effects of T. However, there is now evidence that sex steroids are involved, and the source of those hormones may not be the gonads.

Male Song Sparrows (*Melospiza melodia morphna*) in Washington state robustly defend territories throughout the year, except briefly during prebasic molt (Wingfield and Hahn 1994). Although aggression is expressed in the nonbreeding season, at that time the testes are completely regressed and plasma levels of T, 5 α -DHT, and E₂ are nondetectable (Soma and Wingfield 1999). Even castration does not reduce winter aggression (Wingfield 1994). Nevertheless, treatments of free-living male Song Sparrows with aromatase inhibitors, with or without androgen-receptor blockers, significantly reduces winter aggression (Soma et al. 1999b, 2000a, b). These data suggest that winter aggression is sex-steroid dependent, and in particular is dependent on estrogen.

Where are these sex steroids coming from? Nongonadal organs, such as the adrenals and brain, may produce sex steroids. The adrenal glands may secrete the inert androgen dehydroepiandrosterone (DHEA) which can be converted to active sex steroids by cells within the brain (Vanson et al. 1996). DHEA has been

identified in the plasma of wild winter Song Sparrows, at levels nearly eight times higher than plasma T and androstenedione (Soma et al. 2000c). Moreover, treatments with exogenous DHEA can activate singing in wild autumnal Song Sparrows, and stimulate growth of one song-control nucleus (Soma et al. 2000c). This is the first report of plasma DHEA in a songbird and of DHEA effects on avian brain and behavior. Given its potential importance in birds, DHEA should probably be measured more frequently when plasma steroids are measured in other bird species.

In addition, there is accumulating evidence that the brain can produce its own steroids *de novo* from cholesterol (neurosteroids) (Compagnone and Mellon 2000). Using biochemical measures of enzyme activity, direct measures of brain steroids in castrated birds, and molecular analyses of gene expression, steroidogenic enzymes necessary for the production of E_2 from cholesterol have been found in avian brains (Tsutsui and Yamazaki 1995, Vanson et al. 1996, Tsutsui and Schlinger, 2001). Possession of that steroidogenic machinery gives the brain the capacity to synthesize active sex steroids directly from cholesterol, independent of the gonads or adrenals. Not only might concentrations of sex steroids in the brain differ considerably from those detected in blood, but also steroid-dependent neural actions may persist when peripheral steroid synthesis is undesirable or unavailable, such as in wintering birds. Developmentally, estrogens might be synthesized only locally in the telencephalon of male Zebra Finches to masculinize the neural circuits controlling song (see above). Presumably, those estrogens would not reach steroid-sensitive neurons in the hypothalamus that would be inappropriately feminized by those same hormones.

A ROLE FOR SEX-STERIODS IN LEARNING AND MEMORY

Recent research on birds and other vertebrates has considerably expanded the understanding of behaviors influenced by sex steroids. Included among these are steroid effects on learning and memory. Many songbirds learn how and what to sing during a finite, sensitive period as juveniles; some continue to modify their song through adulthood (Bren-

owitz et al. 1997). Those critical periods for song learning may be defined by sex steroids. Estrogens can be naturally elevated in plasma when songs are being learned; androgens can be elevated when the sensitive period for song learning closes (Marler et al. 1987). If androgen levels are experimentally elevated prematurely, the song-learning period is shortened. In contrast, removal of androgens by castration or treatment of intact males with an androgen receptor blocker extends the period of song learning (Bottjer and Johnson 1997).

The effects of T on behavior are directly coupled to effects on the physiology of neural circuits associated with song learning. N-methyl-D-aspartate (NMDA)-type glutamate receptors, which mediate some forms of excitatory neurotransmission, are critical for memory function in several vertebrates, including songbird memory for song. NMDA receptor expression is high during and decreases at the termination of song learning. T treatment prematurely decreases the expression of NMDA receptors in areas involved in song learning (Singh et al. 2000). Moreover, similar T treatments also induce changes in NMDA-receptor mediated transmission. The rate of transmission via NMDA receptors naturally increases during song learning and T treatment of juvenile Zebra Finches induces the faster NMDA-receptor mediated transmission observed in the adult song circuit (White et al. 1999). Those data demonstrate a consistent influence of steroids on the mechanisms of learning in birds.

Other forms of avian learning might also be influenced by sex steroids. The hippocampus, a brain region associated with several forms of learning and memory in mammals, is rich in the estrogen synthetic enzyme aromatase in many species of songbirds (Saldanha et al. 1998). In some hippocampal neurons, aromatase is colocalized with NMDA receptors (Saldanha et al. 1999). In mammals, sex steroids significantly influence hippocampal structure and function (McEwen and Alves 1999). They may also have important effects on the hippocampus of birds.

The ways in which sex steroids can influence hippocampal neurotransmission are diverse. Steroids can regulate the abundance of neurotransmitter on neuronal membranes (Gazzaley et al. 1996). Those kinds of effects on neurons are relatively slow, because the steroids must

bind to intracellular receptors and change gene expression. Some steroids can directly bind to and regulate neurotransmitter receptors. For example, some 5 α - and 5 β -reduced metabolites of P can directly bind to GABA_A receptors (the major inhibitory neurotransmitter system) and potentiate GABA-evoked currents in the avian hippocampus (Carlisle et al. 1998). Those effects occur within seconds, so they represent a mechanism whereby steroids can influence behavior rapidly.

A ROLE FOR SEX-STEROIDS IN PLASTICITY OF THE ADULT BRAIN

Some songbirds demonstrate structural or biochemical neural plasticity, or both, in adulthood, especially across seasons as some neural circuits expand and contract in concert with singing and with spatial memory capabilities. The magnitude of the plasticity of the adult avian brain is impressive. Those changes include seasonal alterations in neurogenesis, neuron size, dendritic organization, and synaptic input (Barnea and Nottebohm 1994, Alvarez-Buylla and Kirn 1997, Smith et al. 1997) as well as steroid-metabolizing enzymes and steroid receptors (Balthazart and Ball 1998, Soma et al. 1999a, c; Fusani et al. 2000). Because both androgens and estrogens can influence neuron number, synaptic density, dendritic arborization (DeVoogd and Nottebohm 1981), and neuronal recruitment (Hidalgo et al. 1995, Burek et al. 1995), they are good candidates as the natural signals stimulating some seasonal neuroplasticity in birds.

CONCLUSIONS

In summary, over the last few decades, we have learned a great deal about the cellular and molecular mechanisms of gonadal sex steroid control of masculine and feminine reproductive behaviors in birds. We now see that steroids influence a large number of behaviors, and do so by acting on a multiplicity of neural sites. We see that as opposed to being static, some parts of the avian brain are ever-changing, relying in part on a fluctuating sex-steroid environment. We have also learned that some behaviors previously considered independent of steroid action may indeed rely on hormones, but ones that are produced in novel sites or that

act in novel ways. There is no doubt that our concepts about the hormonal control of avian brain and behavior will continue to grow as ornithologists continue to examine new species and new behaviors.

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