Aggression and Territoriality☆

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Abstract

Aggressive behaviors are used to defend and maintain territories. Although there are benefits in maintaining a territory, there are also important costs, including energy expenditure and risk of injury. In most species, aggressive behaviors are highly regulated, and a variety of hormonal and neurobiological mechanisms have been identified. Recent studies have begun to outline how these mechanisms are affected by variation in the environment. In birds and rodents, different hormonal mechanisms regulate aggression in different seasons and photoperiod regimes. Modulation through social experience adds to this plasticity in aggression. In particular, previous experience winning aggressive encounters increases the probability of winning future encounters, regardless of intrinsic fighting ability. In males, this effect appears to be facilitated by testosterone, and there are hints that progesterone may be important in females. Since aggressive behavior does not occur in a vacuum, it is critical to consider how variations in the environment affect the mechanisms regulating aggression.

Keywords

Estrogens; Photoperiod; Progesterone; Resident–intruder test; Seasonal; Simulated territorial intrusion; Testosterone; Vasopressin; Winner effect

Introduction

Aggression is a complex set of behaviors intended to communicate potential harm and/or directly inflict physical damage to another individual. Although aggression is present throughout the animal kingdom, the behavioral display can be highly variable across species and even within individuals. Throughout the lifespan, aggressive behaviors can be expressed in different contexts including sexual, parental, predator-prey, and territorial conflicts. The motivation to engage in aggression can be different in each context. In the case of territorial aggression, the main motivation is defense of limited resources that are crucial for survival and/or reproduction such as food, shelter, and mating opportunities. When the distribution of resources is concentrated in a defined physical location, establishing and defending a territory can secure access to these resources and thus increase fitness of the individual (Fig. 1). Nonetheless, territorial defense can be very costly. It is energetically expensive and time consuming, and can increase the likelihood of being detected by predators (Fig. 1). When physical aggression is involved, it can increase risk of injury and even death. Therefore, animals will typically use a variety of displays to advertise their current ownership of a territory before engaging in physical aggression. For example, some species use brightly colored visual displays or advertise acoustically, while others deposit scent markings on the territory, particularly around the boundaries. When these signals are not sufficient to deter intruders, a territory holder may engage in physical aggression. Examples include wrestling between horned beetles, biting in rodents, and darting flights by birds.

Given its high cost, aggressive behavior is tightly regulated at multiple levels: information about the internal (physiological), and external (physical and social) environment is integrated by the nervous and endocrine systems to modulate behavioral responses across variable contexts. Interestingly, it appears that the same set of aggressive behaviors can be stimulated by different neuroendocrine mechanisms depending on environmental conditions, physiological state, or sex of the individual. Steroid hormones like androgens, estrogens, progesterone, and glucocorticoids as well as neuropeptide hormones such as vasopressin and oxytocin all have important roles in modulating aggressive behaviors in different contexts. We will focus on studies using rodents and birds to discuss how these hormones modulate territorial behavior in different contexts in males and females.
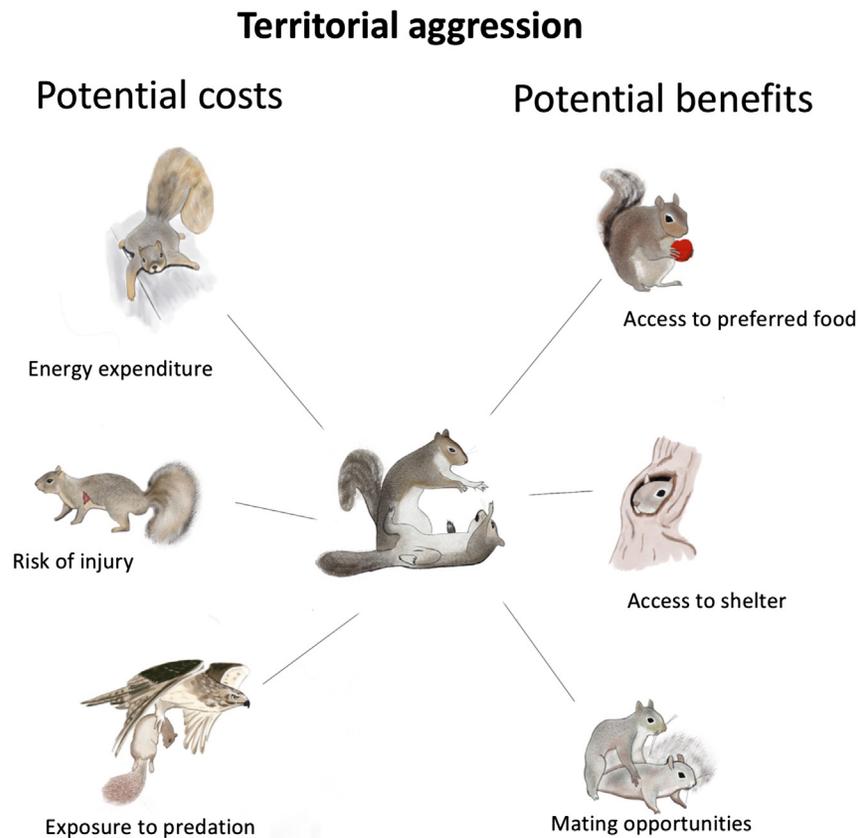
Hormonal Mechanisms of Territorial Aggression

Steroid Hormones

In the context of aggression, the most studied steroid hormones are the sex steroids androgens and estrogens. They are called sex steroids because they were first found to be synthesized in the male and female gonads, respectively. Now we know they can also be synthesized in other peripheral as well as nervous tissues, and that both androgens and estrogens play an important role in modulating behavior and physiology in both sexes. The effects of steroid hormones on aggressive behavior can be seen during early development (organizational mechanisms) as well as during adulthood (activational mechanisms) (French *et al.*, 2013;

Territorial aggression

Potential costs   Potential benefits

Energy expenditure

Access to preferred food

Risk of injury

Access to shelter

Exposure to predation

Mating opportunities

Fig. 1 Potential costs and benefits to engaging in aggressive interactions. Distribution of resources and mating systems will influence the relative magnitude of the listed costs and benefits. Drawing by Natalia Duque-Wilckens.

Soma et al., 2008). Some studies have also found that progesterone may have an important role in territorial aggression, although the relationship is less clear.

Androgens : Testosterone

Testosterone (T) is often a focus of studies examining hormonal mechanisms regulating aggression. Although it is usually assumed that T increases aggression in males and females, this relationship is much more complex and often depends on seasonal or social cues (Gleason et al., 2009). Under some conditions, long-term baseline serum T levels do not correlate well with behavior. This may be because when serum T reaches the brain, it can be locally converted into dihydrotestosterone (a more potent androgen) or estradiol (a potent estrogen). In addition, T can be synthesized directly in the brain from other precursors such as dehydroepiandrosterone (DHEA), a steroid that is synthesized by the adrenal glands. Therefore, how T is metabolized and or synthesized within the brain can have more significant consequences on behavior than circulating levels of serum T.

Testosterone is also a dynamic hormone and can change rapidly during a single aggressive encounter. Testosterone can exert slow as well as rapid effects on behavior through two mechanisms. To exert long-term effects, T binds to receptors that migrate to the nucleus to alter transcription. To exert acute effects, T can bind to receptors in the cell membrane or cytoplasm that activate second messenger pathways (Foradori et al., 2008). Interestingly, short-term increases in T are sometimes more closely associated with aggression and territory defense than long term changes.

Estrogens

Estrogens have an important role on aggression during development and in the adult brain. During development, T secreted by the male gonads is converted to estradiol in the brain, which results in long lasting increases in male-typical aggressive behavior. Acting in the adult brain, estrogens are mainly associated with promoting aggressive behaviors in both males and females. Like T, estrogens exert longer term effects by binding to estrogen receptors that translocate to the nucleus (Heldring et al., 2007) as well as more rapid effect binding to receptors outside of the nucleus (Heimovics et al., 2015b).

Progesterone

Progesterone is another steroid hormone that can be synthesized in male and female gonads, adrenal glands, and nervous system. Like T and estrogens, progesterone can exert its effects by binding to progesterone receptors through genomic and non-genomic mechanisms (Wendler et al., 2012). Interestingly, progesterone can also be metabolized in the brain to allopregnanolone Pinna et al., 2005). Allopregnanolone can modulate GABA receptors; therefore, progesterone can indirectly affect aggression through that pathway.

Peptide Hormones

Two peptides synthesized in the brain, arginine vasopressin (AVP) and oxytocin (OT), are known to modulate a variety of social behaviors, including aggression. Within the brain, the main region where AVP and OT are produced is the hypothalamus, from where they can be released to the peripheral circulation or to other brain areas. The action of AVP and OT can be modulated by both vasopressin receptors and oxytocin receptors (OTR), as both neuropeptides are highly homologous and can thus activate each other's receptors.

Aggression in Birds and Rodents

The majority of studies on birds are conducted in field settings. An advantage of field studies is that aggressive behaviors can be observed in a complex environment, along with the fitness consequences of aggressive behaviors. A disadvantage is that because a field setting is less controlled, it is more challenging to conduct manipulations and physiological measurements. One of the most common methods for testing aggression in birds is the simulated territorial intrusion (STI), in which a caged male is placed near a resident male and a speaker is used to play songs (Wingfield and Wada, 1989). Typically, territory holders respond to STIs with a variety of aggressive behaviors, including producing song and darting at the intruder.

Almost all studies on rodents are conducted in the laboratory, and the most commonly used behavioral paradigm used is the resident–intruder test. The focal male (the resident) is housed in a cage for 2–5 days, and then an unfamiliar intruder is introduced into the resident's cage. In most species, male residents attack the intruder by biting the flanks or boxing with the forepaws. The frequencies of these behaviors can be a measure of the intensity of aggression. The motivation to fight can also be reflected by the latency to first attack. The resident–intruder test is designed to model a resident defending a territory, although it is only a rough approximation of natural interactions. The main advantage of laboratory studies such as the resident–intruder test is the ability to conduct a wide variety of manipulations and measurements. For example, telemetry can be used to measure physiological variables in real time, such as the heart rate or microdialysis to measures neurotransmitter release. It is also possible to conduct precise hormone manipulations that would be difficult or impossible in a field setting.

Territoriality and Aggression in Seasonally Breeding Birds

In many passerine birds, breeding occurs in the summer and males defend breeding territories. This territorial aggression is usually associated with increased baseline plasma T (Wingfield et al., 1990). In many species, males provide parental care by feeding their chicks, and both T and territorial aggression decrease while males are provisioning their chicks. Hormone manipulation experiments in several species show that artificially increasing T with an implant during the parental phase can restore territorial aggression, but at the expense of parental behavior. In some cases, however, the negative relationship between increased T and paternal behavior has been dissociated. In species such as the rufous-collared sparrow, *Zonotrichia capensis*, increasing T does not inhibit paternal behavior (Moore et al., 2004).

Additional studies suggest that the relationship between baseline T levels and aggression in birds is stronger during the breeding season. If a male song sparrow is removed from its territory, neighboring males compete to take over the recently vacated territory. If the experiment is conducted during the start of the breeding season, then the competing males have increased T. However, if the experiment is conducted outside the breeding season (autumn), then T is not increased, even though competition over the vacated territory is intense (Soma and Wingfield, 2002). Similarly, an STI conducted during the breeding season provokes an aggressive response by the resident as well as an increase in T. However, an STI conducted in the fall does not increase T, even though male residents respond aggressively. These studies suggest that T produced by the gonads is not essential for aggression outside of the breeding season. This hypothesis is supported by observations that castration of male song sparrows does not reduce aggression during non-breeding season STIs. Intriguingly, it appears that nonbreeding aggression is regulated by estrogens that are synthesized in the brain not the gonads (Soma et al., 2000).

When male sparrows are treated with an aromatase inhibitor (to block conversion of androgens to estrogens), aggression during the nonbreeding season is reduced. Interestingly, this effect is observed within 24 h, which is relatively fast for a steroid hormone manipulation. Similarly, oral administration of estradiol, a type of estrogen, increases aggression in non-breeding song sparrows (Heimovics et al., 2015a). The source of androgens for estrogen production may be the adrenal gland. The adrenal gland produces

DHEA, which is converted to the androgen androstenedione in the songbird brain. This is significant because plasma DHEA levels are elevated in nonbreeding males (Heimovics et al., 2015b). Nonetheless, nonbreeding birds treated with DHEA implants increase singing behavior but do not increase aggressive behaviors, suggesting the possibility that a minimal threshold level of DHEA is necessary to support estrogen-dependent aggression. This hypothesis is supported by observations that DHEA levels decrease when males are molting feathers, a period when males are not aggressive.

In females, some evidence suggests that T can also be associated with aggression during the breeding season. For example, females of some bird species that encounter high levels of competition for nest sites show higher levels of T than females of solitary species (Moller et al., 2005). Another example is the song sparrow, a species in which circulating T in females actually decreases following an aggressive encounter (Elekonich and Wingfield, 2000). In other cases, T is not as closely linked with aggression. Testosterone levels are not changed after an aggressive encounter in the African black coucal, a species in which females compete over males (who are the ones taking care of offspring). Instead, females of this species show a rapid decrease in circulating progesterone, which is hypothesized to down regulate aggression. The fact that there are alternative mechanisms to modulate aggression in females may be associated to the sex specific costs associated to high T levels. In females, T can delay breeding, alter mate choice behavior, and inhibit maternal care. It has been hypothesized that the role of T mediating aggressive behaviors may be more common in species which do not depend heavily on maternal care. For example in some species offspring are not highly dependent on parental care for survival or males have a more active role in offspring care (Rosvall, 2013).

Photoperiod and Aggression in Rodents

In many mammalian species, like bird species, seasonal changes in aggression can be induced by light cycles, or photoperiod. In many species of rodents, reproduction is inhibited in winter months, which is largely triggered by exposure to short days. In males, reproductive inhibition usually involves regression of the testes and a sharp decrease in T levels. Conventional thinking would then suggest that aggression levels should be reduced in winter-like short days. However, male aggression across a wide variety of hamsters and mice is increased in short days despite reduced T (Laredo et al., 2014; Munley et al., 2018). Evidence from several species suggests that the increased aggression observed in short days may be independent of changes in T. For example, in Siberian hamsters, Phodopus sungorus, there is natural variation in the reproductive responses to photoperiod, and some individuals maintain large testes size and increased T in short days. In a resident–intruder aggression test, these 'nonresponsive' individuals attack an intruder more often than individuals housed in long days with equivalent testes sizes and T levels. Complementary evidence is seen in the California mouse (Peromyscus californicus), a species in which short days do not reduce testes size or T levels. Despite the absence of reproductive responses, male California mice are more aggressive in resident–intruder tests when housed on short days (Trainor et al., 2008). These studies suggest that changes in T secreted by the testes cannot explain the effect of short days on aggression in these species.

An alternative mechanism of regulation may be adrenal steroids: In hamsters, adrenal steroids play a role in plasticity in aggression (Soma et al., 2015). For example, removing the adrenal cortex of Siberian hamsters blocks the effects of short days on aggression. It is likely that these effects are mediated by DHEA, as Siberian hamsters have increased DHEA levels in short days, and treatment with cortisol, another hormone produced in the adrenals, did not affect aggression in males housed in long or short days. Nonetheless, DHEA implants did not increase aggression in hamsters housed in long days. Together, this suggests that a minimal threshold of DHEA is required to promote aggressive behavior, or that short days induce changes in systems that are affected by DHEA or its metabolites, like steroid receptors. DHEA could also affect aggressive behavior in females: for example, female Siberian hamsters show increased levels of territorial aggression and have elevated levels of serum DHEA and adrenal DHEA responsiveness during short vs long days (Rendon et al., 2015a).

Another mechanism regulating aggression in rodents may be estrogens. As discussed above, in birds it appears that downstream estrogenic metabolites play a critical role in regulating aggression outside of the breeding system. A series of studies in mice of the genus Peromyscus demonstrate that estrogens have important effects on male aggression, and that photoperiod plays an important modulating role. In Peromyscus, estrogens increase male aggression in short days (Trainor et al., 2007), as would be predicted based on results from sparrows. In old field mice (P. polionotus), estrogens decrease aggression in long days, but in short days, estrogens increase aggression. These different effects of estrogen on behavior may be explained by the receptors involved. Lines of Mus musculus in which estrogen receptor α (ERα) or estrogen receptor β (ERβ) have been deleted suggested that ERα increases aggression and ERβ decreases aggression. Intriguingly, ERα expression across several hypothalamic and limbic brain nuclei increases in short days, whereas ERβ expression in the brain increases in long days. These results appeared very relevant to aggressive behavior, as they supported the hypothesis that different estrogen receptors may have opposite effects. However, in old field mice, agonists that selectively activated either ERα or ERβ decreased aggression when mice were housed in long days but increased aggression when mice are housed in short days. This suggests that in this species acute activation of ERα and ERβ can have similar effects on aggressive behavior but that these effects are modulated by the environment. In particular, photoperiod appears to alter whether these receptors act via genomic or nongenomic mechanisms.

To assess whether photoperiod influences how estrogens regulate gene expression, microarrays were used to measure the expression of genes that are estrogen dependent. In the bed nucleus of the stria terminalis (a brain region that regulates aggression), estrogen-dependent gene expression was up-regulated in mice housed in long days compared to short days. These data suggest that estrogens may decrease aggression in long days by promoting gene expression. In contrast, gene expression does not appear

to be a central component of estrogen action in short days. This is further supported by findings in California mice and old-field mice, where a single injection of estradiol increases aggression within 15 min if the mouse is housed in short days, but has no effect if the mouse is housed in long days. This rapid response after estradiol administration during the short days suggests that the effect is mediated by nongenomic mechanisms. Recent work has highlighted that steroids such as estradiol can phosphorylate kinases, regulate ion channels, or alter neurotransmitter release. All of these effects could contribute to rapid changes in aggressive behavior.

Studies in females have also found that activation of estrogen receptors can modulate aggression. Gonadectomized female mice treated with selective ERα or ERβ agonists have increased agonistic behaviors, although the type of aggression seems to differ. Female mice treated with selective ERα agonists have increased aggressive attacks towards intruders (Clipperton-Allen et al., 2011) while females treated with selective ERβ agonists show increased non-attack agonistic behaviors such as social investigation and dominance (Clipperton-Allen et al., 2010). Intriguingly, a study found that knockout of ERα increases levels of offensive aggression in females, which suggests an inhibitory effect of ERα during early development (organizational mechanism) (Ogawa et al., 1998). The extent to which environmental factors such as photoperiod affects estrogen receptor modulation of aggression in females has not been directly tested yet. However, the strong effects of photoperiod on adrenal hormone modulation of aggression in hamsters (Rendon et al., 2015b) suggests that estrogen-sensitive neural circuits of aggression are affected by environmental cues.

Effects of Experience on Aggression

Within a season, individual variation in male territorial aggression may also occur. For example, in a contest over a territory, residents often have an advantage. The reasons can be related to traits intrinsic to the territory owner, such as fighting ability or size. Alternatively, interactions between the territory owner and the physical environment, such as familiarity with the territory can also play a role. Individual variation in territorial aggression can be induced through a variety of mechanisms. For example, the behavioral response to an intruder can vary based on familiarity. The 'dear enemy' phenomena suggests that there is a lower aggressive response to a neighbor, but this may only be accurate as long as the boundaries remain stable. This effect has been described in both birds and frogs.

Another example of social influences is related to past experience such as the ability to win a contest with an intruder (Reiger et al., 2018). The loser effect is well established. An individual that loses an encounter is more likely to lose future encounters as a result of long-lasting changes in the brain and neuroendocrine system. In contrast, in many species an individual that wins an encounter is more likely to win future encounters, regardless of intrinsic competitive ability. *Peromyscus* mice have become a model system for investigating the mechanisms underlying the winner effect. The California mouse is monogamous and defends territories year-round while the white footed mouse (*P. leucopus*) is promiscuous and significantly less territorial. In a laboratory study, inexperienced male California mice were randomly assigned to win between 1 and 3 encounters as residents in the resident–intruder test. After this training phase, the residents were tested against a larger intruder. The probability of winning an aggressive encounter against a larger opponent increased in direct relationship to the amount of winning experiences (Oyegible and Marler, 2005). The mechanism underlying the winner effect appears to be associated to T secretion after the aggressive encounter. In birds, androgens often rise briefly after a male is challenged by another male. Studies in California mice have also found that there is a transient increase in T, and that it modulates both future aggression and the probability of winning. This was demonstrated in castrated male California mice with T implants that mimicked baseline T levels, but prevented males from increasing T after an aggressive encounter. Males given a T injection after winning (to mimic the normal T challenge response) became more aggressive in future encounters while males given a placebo injection showed no change in aggression (Trainor et al., 2004). This effect was blocked by aromatase inhibitor treatment, suggesting that experience–induced aggression is mediated by androgen receptors rather than through conversion of T to estrogen. Furthermore, winning experience alone in males can induce an increased ability to win future encounters in an additive fashion with T. Interestingly, the strength of the winner effect in a familiar area can differ between species, as the white footed mouse displays a diminished winner effect.

The white-footed mouse does not experience a T surge at the same time after an encounter as the California mouse (Fuxjager et al., 2010). The species comparison between the monogamous California mouse and polygynous white-footed mouse raises the hypothesis that residency, may influence the development of the winner effect (Fuxjager and Marler, 2010). This is further supported by findings that T pulses, expressed in California mice but not white-footed mice, can induce a preference for a location (Zhoa and Marler, 2016). This preference may be important for establishing or modulating residency and territoriality. In fact, California mice do not display a full robust winner effect unless they have the 'home advantage,' regardless of intrinsic fighting ability. For territorial animals, an individual's fitness depends on its ability to win aggressive disputes. During the establishment of a territory, frequent aggressive encounters may occur as individuals compete for or expand their territories. Because the costs of aggression can be high, a residency-dependent winner effect might be adaptive because it would allow individuals to adjust their winning ability in a context-specific manner. Together, the results suggest that the separate effects of past winning experience, territoriality, and exposure to transient increases in T can induce changes in an individual's aggressive behavior. Testosterone itself may influence future winning ability, but this influence is stronger when it is coupled with the experience of winning a fight and territoriality.

Winner effects may also be present in females, although the mechanisms appear to differ from males. Specifically, accumulating evidence suggests that winner effects may depend more on progesterone than T in females. For example, the dominant hormonal change for female California mice after winning an aggressive encounter is a rapid decrease in progesterone (Davis and Marler, 2003), similar in profile to the rapid increase in T observed during encounters in males. An identical decrease in progesterone during

aggressive encounters was observed in female black coucals (*Centropus grillii*) (Goymann *et al.*, 2008). In addition, female coucals treated with progesterone implants were less aggressive than females receiving empty implants, suggesting that a transient decrease in progesterone may indeed facilitate increased aggression. Again, the transient nature of steroid hormones is a consistent response associated with winning aggressive encounters. In both Syrian hamsters (*Mesocricetus auratus*) and bank voles (*Clethrionomys glareolus*), chronic administration of progesterone increased intrasexual aggression (Payne and Swanson, 1972; Kapusta, 1998). It is possible that acute and chronic changes in progesterone could affect different pathways, with rapid changes having stronger effects on inhibitory GABA receptors (which can be activated by progesterone metabolites) and chronic changes having stronger effects through the transcriptional effects of progesterone receptors. Together, these studies suggest that the effects of progesterone on aggressive behavior depends on contexts and species, and may be mediated by different mechanisms other that progesterone receptor activation.

Peptide Hormones: Species, Context and Sex Specific Regulation of Aggression

The expression of peptide hormones and their receptors are tightly regulated by gonadal hormones in some species (Delville *et al.*, 1996; Miller *et al.*, 1992) and gonadal hormones may play an important role in determining the sex-specific effects of the peptide hormones vasopressin and oxytocin (and associated homologues) and activation on behavior (Dumais and Veenema, 2016). Both have been closely linked with aggression. The effects of arginine vasopressin (AVP) on territorial aggression are highly dependent on social context and sex. For example, in the strongly territorial California mouse, blocking V1aR in males reduces territorial aggression. Similarly, in the highly territorial Syrian hamster AVP acting in anterior hypothalamus is necessary for the display of male territorial aggression (Ferris *et al.*, 1984). On the other hand, intracerebroventricular infusions of a V1aR antagonist has no effects on aggression in the less territorial white-footed mouse (Bester-Meredith *et al.*, 2005). These species-specific effects of AVP in males have been hypothesized to be mediated by variation in the distribution of receptors activated by AVP. The effects of AVP on aggression are also highly sex specific. In non-lactating females, inhibition of V1aR in anterior hypothalamus increase territorial aggression in female hamsters (Gutzler *et al.*, 2010). The mechanisms for sex-specific effects of the vasopressin receptor V1aR on aggression are not well understood. One possibility is V1aR expression is typically quantified using techniques such as autoradiography, which does not have single cell resolution. New approaches such as single cell RNA sequencing may provide important insights in to whether there are sex differences in which cell types expression V1aR. The structurally similar neuropeptide OT also modulates aggression in a sex- and context-specific fashion. For example, acute administration of OT either intracerebroventricularly or intranasally reduces territorial aggression in male rats (*Rattus norvegicus*) (Calcagnoli *et al.*, 2013). Similarly, blocking OTR results in increased territorial defense in this species. In females OT also seems to reduce aggression but only if they are not lactating. For example, female knock out mice that do not express OT are more aggressive than wild-type females (Ragnauth *et al.*, 2005), and injections of OT into the hypothalamus reduces aggression in female Syrian hamsters (Harmon *et al.*, 2002). In contrast, OT seems to enhance aggression when females are lactating. For example, OT infused into the central amygdala increases aggression in lactating rats (Bosch *et al.*, 2005).

Although pharmacological experiments have outlined how AVP and OT can modulate aggressive behaviors, it is unclear how these neuropeptides are influenced by territorial encounters. Is vasopressin released during territorial encounters and if so which brain circuits are affected? Do rapid changes in T (in males) or progesterone (in females) induce long term changes in AVP or OT production? If so, do these changes influence behavior in future aggressive encounters?

Future Directions

Territorial aggression is a major component of social systems that fluctuates seasonally as well as within breeding seasons. The plasticity in this behavior in response to a variety of physical and social aspects of the environment may reflect the multitude of selection pressures that can shape territorial behavior. Androgen manipulations have revealed the costs in a variety of species, including field manipulations with Mountain spiny lizards (*Sceloporus jarrovi*) and dark-eyed juncos (*Junco hyemalis*). In the appropriate contexts, territorial aggression is highly beneficial for gaining access to resources. The study of mechanisms controlling these behaviors is
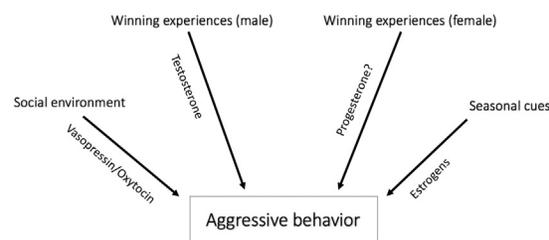
Fig. 2   Hormones and neuropeptides mediating the effects of experience on aggressive behavior. Drawings by Natalia Duque-Wilckens.

proving to be rewarding because of the striking plasticity of the behavior. New neural mechanisms are continually being discovered that reveal the complexity of control of aggression. Investigations into how different aspects of the environment influence territorial aggression through varying mechanisms and how this information is integrated at a neural level represents an opportunity for researchers interested in plasticity of behavior and neural mechanisms (Fig. 2).

References

Bester-Meredith, J.K., Martin, P.A., Marler, C.A., 2005. Manipulations of vasopressin alter aggression differently across testing conditions in monogamous and nonmonogamous Peromyscus mice. Aggressive Behavior 31, 189–199.

Bosch, O.J., Meddle, S.L., Beiderbeck, D.I., Douglas, A.J., Neumann, I.D., 2005. Brain oxytocin correlates with maternal aggression: Link to anxiety. Journal of Neuroscience 25, 6807–6815.

Calcagnoli, F., De Boer, S.F., Althaus, M., Den Boer, J.A., Koolhaas, J., 2013. Antiaggressive activity of central oxytocin in male rats. Psychopharmacology 229, 639–651.

Clipperton-Allen, A.E., Almey, A., Melichercik, A., Allen, C.P., Choleris, E., 2011. Effects of an estrogen receptor alpha agonist on agonistic behaviour in intact and gonadectomized male and female mice. Psychoneuroendocrinology 36, 981–985.

Clipperton-Allen, A.E., Cragg, C.L., Wood, A.J., Pfaff, D.W., Choleris, E., 2010. Agonistic behavior in male and females: Effects of an estrogen receptor beta agonist in gonad-ectomized and gonadally intact mice. Psychoneuroendocrinology 35, 1008–1022.

Davis, E.S., Marler, C.A., 2003. The progesterone challenge: Steroid hormone changes following a simulated territorial intrusion in female Peromyscus californicus. Hormones and Behavior 44, 189–198.

Delville, Y., Mansour, K., Ferris, C., 1996. Testosterone facilitates aggression by modulating vasopressin receptors in the hypothalamus. Physiology & Behavior 60, 25–29.

Dumais, K.M., Veenema, A.H., 2016. Vasopressin and oxytocin receptor systems in the brain: Sex differences and sex-specific regulation of social behavior. Frontiers in Neuroendocrinology 40, 1–23.

Elekonich, M.M., Wingfield, J.C., 2000. Seasonality and hormonal control of territorial aggression in female song sparrows (Passeriformes: Emberizidae: Melospiza melodia). Ethology 106, 493–510.

Ferris, C.F., Albers, H.E., Wesolowski, S.M., Goldman, B.D., Luman, S.E., 1984. Vasopressin injected into the hypothalamus triggers a stereotypic behavior in golden hamsters. Science 224, 521–523.

Foradori, C.D., Weiser, M.J., Handa, R.J., 2008. Non-genomic actions of androgens. Frontiers in Neuroendocrinology 29, 169–181.

French, J.A., Mustoe, A.C., Cavanaugh, J., Birnie, A.K., 2013. The influence of androgenic steroid hormones on female aggression in 'atypical' mammals. Philosophical Transactions of the Royal Society of London B, Biological Sciences 368, 20130084.

Fuxjager, M.J., Marler, C.A., 2010. How and why the winner effect forms: Influences of contest environment and species differences. Behavioral Ecology 21, 37–45.

Fuxjager, M.J., Montgomery, J.L., Becker, E.A., Marler, C.A., 2010. Deciding to win: Interactive effects of residency, resouces and 'boldness' on contest outcome in white-footed mice. Animal Behaviour 80, 921–927.

Gleason, E.D., Fuxjager, M.J., Oyegbile, T.O., Marler, C.A., 2009. Testosterone release and social context: When it occurs and why. Frontiers in Neuroendocrinology 30, 460–469.

Goymann, W., Wittenzellner, A., Schwabl, I., Makomba, M., 2008. Progesterone modulates aggression in sex-role reversed female African black coucals. Proceedings of the Royal Society B-Biological Sciences 275, 1053–1060.

Gutzler, S.J., Karom, M., Erwin, W.D., Albers, H.E., 2010. Arginine-vasopressin and the regulation of aggression in female Syrian hamsters (Mesocricetus auratus). European Journal of Neuroscience 31, 1655–1663.

Harmon, A.C., Huhman, K.L., Moore, T.O., Albers, H.E., 2002. Oxytocin inhibits aggression in female Syrian hamsters. Journal of Neuroendocrinology 14, 963–969.

Heimovics, S.A., Ferris, J.K., Soma, K.K., 2015a. Non-invasive administration of 17b-estradiol rapidly increases aggressive behavior in non-breeding but not breeding male song sparrows. Hormones and Behavior 69, 31–38.

Heimovics, S.A., Trainor, B.C., Soma, K.K., 2015b. Rapid effects of estradiol on aggression in birds and mice: The fast and the furious. Integrative and Comparative Biology 55, 281–293.

Heldring, N., Pike, A., Andersson, S., et al., 2007. Estrogen receptors: How do they signal and what are their targets. Physiological Reviews 87, 905–931.

Kapusta, J., 1998. Gonadal hormones and intrasexual aggressive behavior in female bank voles (Clethrionomys glareoulus). Aggressive Behavior 24, 63–70.

Laredo, S.A., Villalon Landeros, R., Trainor, B.C., 2014. Rapid effects of estrogens on behavior: Environmental modulation and molecular mechanisms. Frontiers in Neuroendo-crinology 35, 447–458.

Miller, M.A., Devries, G.J., Alshamma, H.A., Dorsa, D.M., 1992. Decline of vasopressin immunoreactivity and messenger-rna levels in the bed nucleus of the stria terminalis following castration. Journal of Neuroscience 12, 2881–2887.

Moller, A.P., Garamszegi, L.Z., Gil, D., Hurtrez-Bousses, S., Eens, M., 2005. Correlated evolution of male and female testosterone profiles in birds and its consequences. Behavioral Ecology and Sociobiology 58, 534–544.

Moore, I.T., Wada, H., Perfito, N., et al., 2004. Territoriality and testosterone in an equatorial population of rufous-collared sparrows, Zonotrichia capensis. Animal Behaviour 67, 411–420.

Munley, K.M., Rendon, N.M., Demas, G.E., 2018. Neural androgen synthesis and aggression: Insights from a seasonablly breeding rodent. Frontiers in Endocrinology 9, 136.

Ogawa, S., Eng, V., Taylor, J., et al., 1998. Roles of estrogen receptor-alpha gene expression in reproduction-related behaviors in female mice. Endocrinology 139, 5070–5081.

Oyegible, T.O., Marler, C.A., 2005. Winning fights elevates testosterone levels in California mice and enhance future ability to win fights. Hormones and Behavior 48, 259–267.

Payne, A.P., Swanson, H.H., 1972. The effect of sex hormones on the agonistic behavior of the male golden hamster (Mesocricetus auratus Waterhouse). Physiology & Behavior 8, 687–691.

Pinna, G., Costa, E., Guidotti, A., 2005. Changes in brain testosterone and allopregnanolone biosynthesis elicit aggressive behavior. Proceedings of the National Academy of Sciences of the United States of America 102, 2135–2140.

Ragnauth, A.K., Devidze, N., Moy, V., et al., 2005. Female oxytocin gene-knockout mice, in a semi-natural environment, display exaggerated aggressive behavior. Genes, Brain and Behavior 4, 229–239.

Reiger, N.S., Fuxjager, M.J., Trainor, B.C., Zhao, X., Marler, C.A., 2018. Behavioral and neuroendocrine plasticity in the form of winner and loser effects. In: Mehta, M., Schultheiss, O.C. (Eds.), Handbook of Social Neuroendocrinology. Oxford University Press (in press).

Rendon, N.M., Rudolph, L.M., Sengelaub, D.R., Demas, G.E., 2015a. The agonistic adrenal: Melatonin elicits female aggression via regulation of adrenal androgens. Proceedings of the Royal Society B 282, 20152080.

Rendon, N.M., Rudolph, L.M., Sengelaub, D.R., Demas, G.E., 2015b. The agonistic adrenal: Melatonin elicits female aggression via regulation of adrenal androgens. Proceedings. Biological Sciences 282, 20152080.

Rosvall, K.A., 2013. Proximate perspectives on the evolution of female aggression: Good for the gander, good for the goose? Philosophical Transactions of the Royal Society B 368, 20130083.

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.

Soma, K.K., Scotti, M.-A.L., Newman, A.E.M., Charlier, T.D., Demas, G.E., 2008. Novel mechanisms for neuroendocrine regulation of aggression. Frontiers in Neuroendocrinology 29, 476–489.

Soma, K.K., Tramontin, A.D., Wingfield, J.C., 2000. Oestrogen regulates male aggression in the non-breeding season. Proceedings of the Royal Society B: Biological Sciences 267, 1089–1096.

Soma, K.K., Wingfield, J.C., 2002. Spring and autumn territoriality in song sparrows: Same behavior, different mechanisms? Integrative and Comparative Biology 42, 11–20.

Trainor, B.C., Bird, I.M., Marler, C.A., 2004. Opposing hormonal mechanisms of aggression revealed through short-lived testosterone manipulations and multiple winning experiences. Hormones and Behavior 45, 115–121.

Trainor, B.C., Finy, M.S., Nelson, R.J., 2008. Rapid effects of estradiol on male aggression depend on photoperiod in reproductively non-responsive mice. Hormones and Behavior 53, 192–199.

Trainor, B.C., Lin, S., Finy, M.S., Rowland, M.R., Nelson, R.J., 2007. Photoperiod reverses the effects of estrogens on male aggression via genomic and non-genomic pathways. Proceedings of the National Academy of Sciences of the United States of America 104, 9840–9845.

Wendler, A., Albrecht, C., Wehling, M., 2012. Nongenomic actions of aldosterone and progesterone revisited. Steroids 77, 1002–1006.

Wingfield, J.C., Hegner, R.E., Dufty, J.A.M., Ball, G.F., 1990. The 'challenge hypothesis': Theoretical implications for patterns of testosterone secretion, mating systems, and breeding strategies. American Naturalist 136, 829–846.

Wingfield, J.C., Wada, M., 1989. Changes in plasma levels of testosterone during male-male interactions in the song sparrow, Melospiza melodia: Time course and specificity of response. Journal of Comparative Physiology A 166, 189–194.

Zhoa, X., Marler, C.A., 2016. Social and physical environments as a source of individual variation in the rewarding effects of testosterone in male California mice (*Peromyscus californicus*). Hormones and Behavior 85, 30–35.