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Review article

Aggression: Perspectives from social and systems neuroscience

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ABSTRACT

Exhibiting behavioral plasticity in order to mount appropriate responses to dynamic and novel social environments is crucial to the survival of all animals. Thus, how animals regulate flexibility in the timing, duration, and intensity of specific behaviors is of great interest to biologists. In this review, we discuss how animals rapidly respond to social challenges, with a particular focus on aggression. We utilize a conceptual framework to understand the neural mechanisms of aggression that is grounded in Wingfield and colleagues' Challenge Hypothesis, which has profoundly influenced how scientists think about aggression and the mechanisms that allow animals to exhibit flexible responses to social instability. Because aggressive behavior is rooted in social interactions, we propose that mechanisms modulating prosocial behavior may be intricately tied to mechanisms of aggression. Therefore, in order to better understand how aggressive behavior is mediated, we draw on perspectives from social neuroscience and discuss how social context, species-typical behavioral phenotype, and neural systems commonly studied in relation to prosocial behavior (i.e., neuropeptides) contribute to organizing rapid responses to social challenges. Because complex behaviors are not the result of one mechanism or a single neural system, we consider how multiple neural systems important for prosocial and aggressive behavior (i.e., neuropeptides and neurosteroids) interact in the brain to produce behavior in a rapid, context-appropriate manner. Applying a systems neuroscience perspective and seeking to understand how multiple systems functionally integrate to rapidly modulate behavior holds great promise for expanding our knowledge of the mechanisms underlying social behavioral plasticity.

1. Introduction

To successfully navigate variable environments, animals dynamically regulate a variety of biological responses. These responses are often rapid and involve multiple levels of biological organization, including gene expression, protein synthesis, and behavior. Animal behavior is remarkably sensitive to variation in biotic and abiotic environmental conditions. Neural and endocrine systems mediate behavioral flexibility by transducing environmental information and by coordinating integrated, and often adaptive, behavioral responses (Snell-Rood, 2013; Hau and Goymann, 2015). For example, the activity of many endocrine axes (e.g. the hypothalamic-pituitary-gonadal, HPG, axis) are regulated by the social context, and the hormones regulated by these axes (e.g. gonadal steroids) in turn modulate context-appropriate social and sexual behaviors (Oliveira, 2004).

A fundamental question in behavioral neuroscience concerns the causes and consequences of variation in behavior among individuals, species, and contexts. While the proximate mechanisms that promote context-specific behavior are not entirely clear, flexibility in the timing, duration, and intensity of particular behaviors allows animals to succeed in dynamic and novel environments (Dingemanse and Wolf, 2013; Hau and Goymann, 2015).

In this review, we focus on aggression, discussing sources of behavioral variation and a few of the neural systems that contribute to its modulation. While certain aspects of aggression differ between species, there are broad similarities in how taxonomically diverse animals respond to competition and social instability (Nelson and Trainor, 2007). In many species, testosterone (T) is a potent hormonal mediator in responding to social challenges, such as territorial intrusions from conspecifics. Wingfield and colleagues originally proposed the Challenge Hypothesis as a way to explain temporal variation in T and aggression (Wingfield et al., 1990). The Challenge Hypothesis posits that during periods of social instability, T surges in males are correlated with the expression of territorial aggression. T levels are predicted to return to baseline because of trade-offs imposed by the multitude of potential costs of maintaining high T, including interference with paternal care, increased predation, and impaired immune function (Wingfield et al., 2001). Originally explored in birds, there has been support for the

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Challenge Hypothesis across taxa (Oliveira et al., 2002; Wingfield, 2017), including invertebrates (Tibbetts and Huang, 2010).

The Challenge Hypothesis has profoundly influenced how we think about aggression and the mechanisms that allow for animals to exhibit flexible responses to social instability. In the decades since its introduction, the Challenge Hypothesis has provided a framework for investigations into the social modulation of androgen production (e.g., Oliveira, 2004), behavioral and life-history trade-offs (e.g., Hau, 2007), and neurosteroidogenesis (e.g., Pradhan et al., 2010). Importantly, it appears that there is considerable variation among species in the endocrine control of territorial aggression. For example, simulated territorial intrusions do not consistently induce an elevation in T (Goymann et al., 2007) and T surges do not always increase aggression (Hau, 2007; Goymann et al., 2015).

In order to understand how animals mount rapid, context-appropriate responses to social challenges, it may be necessary to look beyond a single hormone. Offensive and defensive responses to social instability are a consequence of reciprocal interactions between an individual's genotype, phenotype, and environment. Multiple peripheral and central systems are involved in the detection and processing of environmental stimuli, and in coordinating appropriate behavioral output. Drawing on perspectives from social and systems neuroscience, we explore how social context, behavioral phenotype, and the neural systems that modulate (and are regulated by) behavior contribute to organizing rapid responses to social challenges.

2. Characterizing social interactions

2.1. Defining aggression

Broadly defined, aggression is an overt behavior or signal of imminent behavior with the capacity to inflict physical harm on another individual (Nelson, 2006). Early ethologists classified aggression into three distinct categories: prey-directed, predator-directed, and conspecific-directed (Lorenz, 1966). In deference to the very different functions of heterospecific- and conspecific-directed aggression, others have offered more restrictive definitions that limit aggression to social behaviors directed at conspecifics (Moyer, 1968; Nelson, 2006). Although aggressive behavior may yield competitive advantages in terms of obtaining or defending resources, there are steep costs in terms of time, energy, and risk of injury (Nelson, 2006). Given these costs and benefits, there are profound fitness consequences to expressing the appropriate level of aggression at the appropriate time.

While we will limit our discussion of aggression to agonistic interactions with conspecifics, it is important to consider that in behaving animals, aggression is intertwined with other social behaviors. Because animals rapidly transition between different/opposing social behaviors, a full understanding of how and why animals respond aggressively requires considering the broader social context.

2.2. Aggression – social context and species-typical behavior

Across taxa, animals experience similar kinds of challenges and opportunities (e.g. resource defense, reproduction) (O'Connell and Hofmann, 2011). Identical stimuli induce a variety of responses depending on the social/environmental context, or an animal's internal physiological state. The Challenge Hypothesis focuses on aggression during periods of social instability, such as during the establishment of territories. During periods of social instability, the potential payoff of engaging in conflict is higher – animals have the potential to acquire resources (i.e., access to mates, territory, sustenance, dominance rank). In addition to inducing overt aggression, social challenges can lead to a suite of physiological changes that appear to be adaptive under conditions of continued social instability (Rosvall and Peterson, 2014). Indeed because of the time delay between socially-induced HPG axis activation and increases in plasma T, a primary function of socially-induced androgen production may be to prepare animals for future events. In the cichlid *A. burtoni*, social opportunity in the form of a newly vacant territory leads to an increase in aggressive and reproductive behaviors within minutes. Within 20 min, there is an induction of immediate early gene expression in the gonadotropin-releasing hormone 1 (GnRH1) neurons that sit at the apex of the HPG axis, and within 30 min there is an increase in levels of circulating androgens (reviewed in Maruska and Fernald, 2013). GnRH1 neurons show similar plasticity and social regulation across vertebrates (Stevenson et al., 2012).

Although aggression is situationally flexible, levels of aggression are often consistent across different contexts within individuals, e.g. aggression directed at same- vs. opposite-sex conspecifics (Cain et al., 2011) and nest defense and male-male competition (Duckworth, 2006). Furthermore, research examining behavioral syndromes has also demonstrated that individuals often exhibit consistent levels of boldness, aggression, sociability, and exploratory behavior across contexts (Sih et al., 2004; Reale et al., 2007; Biro and Stamps, 2008), and that behavioral tendencies associated with different ecological tasks are often correlated (Sih et al., 2004; Sih and Bell, 2008). This phenotypic integration limits behavioral plasticity and can impose fitness costs (Duckworth, 2006). Due to the trade-off between the expression of aggression and paternal care, one of the original predictions of the Challenge Hypothesis was that both baseline androgen levels and socially-stimulated androgen levels should vary with mating strategy and paternal care: during the breeding season, monogamous males that exhibit a high degree of paternal care are predicted to maintain low baseline androgen and elevate androgen levels in response to social challenges, while polygynous males with little paternal care are predicted to maintain relatively high androgen levels regardless of social challenges (Wingfield et al., 1990). Among avian species, metanalyses suggest that mating system indeed mediates challenge-induced androgen levels, with monogamous species exhibiting higher levels of androgen responsiveness (Hirschenhauser et al., 2003). However, even among species of birds with similar life histories, there is variation both in challenge-induced androgen elevation and in the degree to which androgens mediate territorial aggression (Apfelbeck et al., 2017). Across vertebrates more broadly, neither mating system nor paternal care consistently predict baseline or challenge-induced androgen levels (Hirschenhauser and Oliveira, 2006).

Both within and among taxa, there is considerable variation in the hormonal control of aggression. While there are likely many environmental and social factors that contribute to variation in the mechanisms of territorial aggression, species-specific differences in how territoriality evolved may have influenced how a primarily reproductive hormone such as T was (or was not) co-opted to modulate aggression (Apfelbeck et al., 2017). While the Challenge Hypothesis has largely been explored in territorial species, a few studies have focused on gregarious species. For example, in the nonterritorial, gregarious zebrafish, both winners and losers in staged fights exhibit an increase in circulating androgens (Teles and Oliveira, 2016). Understanding the mechanisms of aggression in nonterritorial species, and the mechanistic connections between aggressive and prosocial/reproductive behaviors could help elucidate the mechanisms that promote rapid responses to social challenges across vertebrates.

2.3. Aggression and prosociality – a continuum?

Maximizing fitness requires that any aggressive behavior be fine-tuned and sensitive to context-appropriate stimuli. Generally, exhibiting overt aggression toward offspring or mates would clearly have grave fitness consequences (however, some aggression may be adaptive such as parental aggression toward offspring, which serves to reduce feeding demands on parents, minimize sibling competition, and to encourage offspring independence around weaning (Leonard et al., 1988; Vergara et al., 2010; Negayama, 2011)). Theoretically, prosocial

behavior, defined as affiliative interactions with conspecifics, and aggression cannot be exhibited at exactly the same time by a single individual. Thus, we have previously proposed that aggression and prosocial behavior, seemingly discrete behaviors, may be two sides of the same coin, operating along a sliding scale of a mechanistic continuum (Kelly and Vitousek, 2017).

Since the Challenge Hypothesis was proposed, many discoveries from the fields of neuroscience and behavioral neuroendocrinology have elucidated how the brain contributes to behaviors like aggression (Albers, 2012; Rosell and Siever, 2015). Because prosocial behavior mechanisms may be intricately tied to mechanisms of aggression, below we discuss how commonly studied social neural systems also modulate aggression and consider how these neural systems may interact to produce rapid responses to social challenges.

3. Neural mechanisms of prosociality and aggression

3.1. Neuropeptides

Neuropeptides are arguably the most commonly studied neurochemicals involved in the modulation of prosocial behavior – particularly bonding. Social behavioral functions have been described in detail for the neurohypophyseal nonapeptides (mammalian homologues: vasopressin, VP; oxytocin, OT), and to a lesser degree, for vasoactive intestinal polypeptide (VIP). The nonapeptides are strongly evolutionarily conserved, and all jawed vertebrates possess a single form of VP and a single form of OT (Acher and Chauvet, 1995; Acher et al., 1995; Hoyle, 1998). The most common vertebrate forms of VP include VP in mammals and vasotocin in non-mammalian species (Acher and Chauvet, 1995; Hoyle, 1998). The OT lineage consists of OT in mammals, isotocin in bony fish, and mesotocin in lobe-finned fish, most amphibians, birds, reptiles, and some marsupials (Hoyle, 1998; Lee et al., 2011). Given the structural and functional similarities across species, following precedence, and for ease of communication, here we will refer to all nonapeptide forms using the mammalian nomenclature (Kelly and Goodson, 2014b). VP and OT are important for regulating autonomic functions such as energy metabolism, smooth muscle functions, hydromineral balance, and cardiovascular tone (Engelmann et al., 2004; Goodson et al., 2005). They also modulate numerous behaviors including aggression, affiliation, parental care, social communication, and anxiety-like behavior (Donaldson and Young, 2008; Goodson and Thompson, 2010; Albers, 2012). VIP is best known as the major regulator of prolactin, which is a crucial hormone for various reproductive behaviors, however, recent studies have demonstrated a primary role for VIP in modulating affiliation, aggression, and parental care (Goodson et al., 2012a; Kingsbury et al., 2015; Kingsbury and Wilson, 2016; Wilson et al., 2016).

3.2. Neuropeptide-mediated social behavior and aggression

The nonapeptides promote both reproductive (i.e., bonding between mates and/or offspring) and non-reproductive affiliation (sociality; i.e., same-sex prosocial behavior). The majority of studies examining nonapeptide-mediated social behavior and aggression are conducted in a reproductive context and examine the nonapeptide receptors – VP 1a receptors (V1aRs) and OT receptors (OTRs). Perhaps most famously, anatomical comparisons between species exhibiting different mating systems have revealed that monogamous species (e.g., prairie voles, California mice, and marmosets) have high densities of V1aRs in the ventral-striato pallidum, whereas their promiscuous relatives (e.g., meadow voles, white-footed mice, and rhesus macaques) have fewer V1aRs in this region (Young, 1999; Young et al., 2001). In addition, viral overexpression of V1aR in the ventral-striato pallidum facilitates pairbonds (Pitkow et al., 2001), whereas viral down-regulation of V1aR in this region impairs pairbonds in male prairie voles (Barrett et al., 2013), demonstrating that VP receptors in the ventral-striato pallidum promote prosocial behavior in a reproductive context. Unfortunately, to our knowledge it remains unknown whether or not V1aRs in the ventral-striato pallidum also modulate aggression (i.e., most studies examining V1aRs in this area do not test aggression). The ventral-striato pallidum is part of a neural 'pairbonding circuit,' which has been studied extensively in prairie voles (Young and Wang, 2004; Young et al., 2008; Young et al., 2011). All nodes within this circuit promote pairbonding, many contain nonapeptide receptors, and depending on the species, at least one region (the paraventricular nucleus of the hypothalamus, PVN) contains nonapeptide-producing neurons. Similar to findings from assessment of V1aRs in the ventral-striato pallidum, blockade of OTRs in a key node of the pairbonding circuit, the nucleus accumbens (NAcc), prevents pairbonding, and overexpression of OTRs in this region facilitates pairbonding in female prairie voles (Liu and Wang, 2003; Ross et al., 2009). Again, OT in the NAcc has not been implicated in aggression. Interestingly, though, upregulation of dopamine receptors in the NAcc is critical for pairbond maintenance by mediating aggression toward opposite-sex strangers (Resendez and Aragona, 2013). In the quest to understand the neural circuitry underlying pairbonding, studies have utilized experimental designs that, as expected, assess pairbond formation; however, most of these studies do not directly test aggression. Yet, it is feasible that nonapeptides within the pairbonding circuit may also modulate aggression in certain contexts, either via titration of up or down regulation of protein synthesis or neural activity or via differential axonal targeting (for peptide-producing neurons). Previous studies have demonstrated behaviorally complex, multi-functional properties of nonapeptide neurons. For example, PVN VP neurons in female finches promote affiliation with same-sex conspecifics, while also promoting aggression with opposite-sex conspecifics (Kelly and Goodson, 2014a). Such studies highlight nonapeptide neural sensitivity to social context, and support the hypothesis that the mechanisms underlying prosocial behavior and aggression may be heavily intertwined.

In addition to examining nonapeptide-mediated bonding, some studies have directly investigated the role of nonapeptides in aggression. An elegant series of studies examining VP in the anterior hypothalamus (AH; not part of the pairbonding circuit) teases apart the mechanisms underlying the classic selective aggression toward novel females that is associated with pairbonding in male prairie voles. Microdialysis revealed high levels of AH VP when pairbonded males exhibit aggressive behavior toward a novel female and low levels of AH VP when pairbonded males exhibit affiliation with their partner. Interestingly, this natural pattern of selective aggression can be reversed, such that blockade of V1aRs in the AH abolishes aggression and facilitates affiliation toward a novel female (Gobrogge et al., 2017). Furthermore, viral overexpression of V1aR in the AH induces selective aggression in sexually naïve males and increases selective aggression in pairbonded males toward novel females, further highlighting a critical role of AH VP in aggression in a reproductive context (Gobrogge et al., 2009). Interestingly, though, a recent study found no correlation between AH VP neural activity and aggression (or prosocial behavior) in sexually naïve, male prairie voles that were assessed in a non-reproductive social context, stressing the importance of social context in the modulation of aggression (Kelly et al., 2018). Furthermore, studies in Syrian hamsters illustrate the importance of photoperiodic context on nonapeptide-mediated aggression, such that activation of AH V1aRs in male hamsters facilitates overt aggression, whereas blockade suppresses aggression, but only in animals housed on long 'summer-like' photoperiods. AH VP has no effects on aggression in males housed on short 'winter-like' photoperiods that exhibit sexual quiescence and have regressed gonads, suggesting that AH VP may be functionally sensitive to sex steroids (Caldwell and Albers, 2004; Albers, 2012).

The AH has also been identified as a neural hotspot for neuropeptide-mediated territorial aggression in birds (Kingsbury and Wilson, 2016). Territorial aggression positively correlates with AH VIP-ir cell number in emberizid sparrows (Goodson et al., 2012b), and knockdown

of AH VIP via antisense oligonucleotides reduces resident-intruder aggression in violet eared waxbills and nest defense aggression in zebra finches. Interestingly, knockdown of AH VIP has no effect on social preferences, courtship, pairbonding, maintenance behaviors, or anxiety-like behavior, suggesting a unique identification of an aggression-specific cell type in the brain (Goodson et al., 2012a).

Perhaps the nonapeptide cell group that has been most thoroughly studied for *both* reproductive and non-reproductive affiliation is that of the VP cell population in the medial bed nucleus of the stria terminalis (BSTm). The BSTm is part of the medial extended amygdala and contains VP producing neurons in birds, reptiles, amphibians, and mammals, and also OT producing neurons in some mammals (e.g., prairie voles, rats, mice, tree shrews, common marmosets) (Wang et al., 1997; Moore and Lowry, 1998; Rood and De Vries, 2011; Knobloch and Grinevich, 2014; Ni et al., 2014; Kelly et al., 2017). The BSTm VP cell group sends axonal projections throughout the brain, but primarily to the lateral septum (LS), which contains VP and OT receptors (Kelly et al., 2011). Antisense oligonucleotide knockdown of BSTm VP production decreases sociality and increases aggression in male finches (Kelly et al., 2011; Kelly and Goodson, 2013). In addition, BSTm VP neural activity positively correlates with prosocial same-sex interactions and negatively correlates with aggressive interactions in male chickens, mice, and prairie voles (Ho et al., 2010; Xie et al., 2011; Kelly et al., 2018). Furthermore, microdialysis studies revealed that VP release in the BST of rats negatively correlates with intermale aggression (Veenema et al., 2010). Together, these studies demonstrate conserved prosocial and anti-aggressive functions of the BSTm VP cell group for males. This may extend to BSTm VP circuitry projecting to the LS given that more social species of estrildid finches have more nonapeptide receptors in the LS compared to territorial species (Goodson et al., 2009), and blockade of VP receptors in the LS reduces sociality and facilitates aggression in finches and sparrows (Goodson, 1998a; Goodson et al., 2004; Kelly et al., 2011). Interestingly, these studies suggest a relatively straightforward neural titration of prosocial and aggressive behavior by BSTm VP neurons. Activity of these neurons relates to prosocial behavior and aggression in an opposite-manner, and in all of these studies, manipulation of this cell group influences both behaviors. Such functional properties may make the BSTm VP cell group an excellent candidate for probing rapid responses to social challenges, allowing us to identify a mechanism that may underlie the rapid changes in behavior often associated with a social challenge, such as an animal positively affiliating with an individual one moment and then rapidly, aggressively chasing an intruder the next moment. This will be discussed further below.

Remarkably few data are available on OT and OTRs in aggression, and data that do exist provide mixed results, suggesting a complex relationship between OT and aggression (Goodson et al., 2015; de Jong and Neumann, 2018). OT is overwhelmingly viewed as being a prosocial hormone (but see Beery, 2015). Central OT promotes social approach in fish (Thompson and Walton, 2004), flocking in finches and sparrows (Goodson et al., 2009; Goodson et al., 2012b; Kelly and Goodson, 2014a), pairbonding in finches and voles (Young et al., 2011; Klatt and Goodson, 2013; Kelly and Goodson, 2014a), and parent-offspring bonds (Nagasawa et al., 2012; Yoshihara et al., 2018). However, OT also facilitates maternal aggression (Bosch, 2013), although OT effects on maternal aggression are dependent on the selected anxiety line of rats (Bosch and Neumann, 2012). Extremely few studies have examined the role of OT in adult, same-sex offensive aggression. A study in female Syrian hamsters demonstrated an anti-aggressive role of OT, such that OT infusion into the preoptic area-AH decreases aggression in a resident-intruder paradigm, whereas OTR antagonism facilitates aggression (Harmon et al., 2002). Consistent with this finding in hamsters, a study in territorial waxbills found that OT neural activity in several brain regions (including the AH) negatively correlates with aggression. However, in this same study, peripheral antagonism of OTRs reduced aggression, suggesting that activation of OTRs can facilitate aggression. Further analyses and discussion revealed a complex relationship between stress responsivity and OT effects on aggression (Goodson et al., 2015). Together with the findings on selected anxiety line influences on OT-mediated maternal aggression in rats, these studies suggest that aggressive functions of OT are strongly influenced by systems that modulate anxiety-like behavior and stress. Indeed, studies in humans support this and find opposing effects of synthetic OT on social and antisocial behaviors depending on sex, social context, perceived stress, and the mental health of participants (Bartz et al., 2011; Guastella and Hickie, 2016).

In summary, these studies demonstrate roles for OT and VP systems in both prosocial behavior and aggression, and reveal site- and context-specificity, species differences, and complex relationships with steroids and systems modulating stress.

3.3. Neurosteroids

Although the Challenge Hypothesis explored peripherally circulating T in relation to behavior, steroid hormones are synthesized not only by peripheral glands (i.e., gonads, adrenal, placenta), but are also produced de novo in the brain (i.e., neurosteroids) (Baulieu, 1980, 1991). De novo synthesis of neurosteroids from cholesterol in the brain appears to be conserved across vertebrate species and has been demonstrated in mammals, birds, amphibians, and fish (Baulieu, 1997; Tsutsui et al., 2000; Mellon and Vaudry, 2001; Tsutsui et al., 2006). The brain possesses several steroidogenic enzymes that produce pregnenolone, pregnenolone sulfate, 7α-hydroxypregnenolone, dehydroepiandrosterone (DHEA), progesterone, corticosterone, allopregnanolone (Allo) or epipregnanolone (only birds), androstenedione (AE), T, and estradiol-17β (E_2) from cholesterol (Tsutsui and Haraguchi, 2016). Neurosteroids have numerous biological functions and are modulators of neurogenesis and neuronal survival (Charalampopoulos et al., 2008). They are also involved in the control of neuroendocrine, behavioral, and metabolic processes, such as ingestion, sleep, blood pressure, stress, cognition, anxiety, sexual behavior, and aggression (Engel and Grant, 2001; Do Rego et al., 2009; Frye, 2009; Tsutsui et al., 2013). Social behavioral functions of neurosteroids are, somewhat surprisingly, still relatively understudied (Soma et al., 2008; Balthazart, 2010), particularly compared to effects of gonadal steroids on behavior (for reviews, see Soma et al., 2008; Wingfield et al., 2018). The roles of centrally produced steroids in social and aggressive behavior are best known for E_2, DHEA, and Allo.

3.4. Neurosteroid-mediated social behavior and aggression

The majority of research examining the role of neurosteroids in aggression comes from studies in mammals that use drugs that specifically manipulate neurosteroid concentrations (Frye, 2009; Pinna and Rasmusson, 2012). An early study in male mice showed that social isolation decreases Allo in the brain (primarily in the olfactory bulb) and is associated with an increase in aggression (Matsumoto et al., 1996). Subsequent studies found that administration of fluoxetine (a selective serotonin reuptake inhibitor) normalizes both pharmacologically-induced and social isolation-induced down-regulated Allo and reduces aggression in male and female mice (Pinna et al., 2003; Pinna et al., 2005). Together, these studies suggest an important role for Allo in the brain in the suppression of aggressive behavior. Research on neurosteroid-mediated behavior in mammals has heavily focused on providing translational insight into disorders such as PTSD, steroid-abuse, depression, and anxiety (Frye, 2009; Pinna and Rasmusson, 2012; Locci and Pinna, 2018), and as a consequence, social behavior has not been a focus of these studies. However, of particular interest from the early studies in mice is that social context regulates neurosteroid biosynthesis (i.e., social isolation decreases Allo (Matsumoto et al., 1996)). Studies in zebra finches have also demonstrated social regulation of neurosteroids. For example, microdialysis in male zebra

finches revealed an increase in forebrain E_2 levels during social interactions with a female, and an increase in E_2 and a decrease in T levels in auditory regions when exposed to another male's song (Remage-Healey et al., 2008). Further studies in finches have resulted in an extensive body of work that detail how neuroestrogens rapidly shape auditory neural circuits and mediate sensory plasticity, communication, and vocal learning (Vahaba and Remage-Healey, 2018).

Of particular relevance to the Challenge Hypothesis, early studies examining effects of seasonality on neurosteroid actions questioned a role for neurosteroids in modulating aggression outside of the breeding season. After all, despite systemic T levels being very low when seasonally breeding animals are in non-reproductive condition (Wingfield and Hahn, 1994), aggression is still capable of being displayed, and thus, must be mediated by mechanisms other than gonadal steroids. In the non-breeding season, E_2 administration increases aggression in resident-intruder tests in male song sparrows and male California mice (Trainor et al., 2007; Heimovics et al., 2015a). In the study on sparrows, administration of E_2 was delivered via ingestion of moth larvae, and in the study on mice, E_2 was injected subcutaneously, so, in the strictest sense, although the rapid effects on aggression (observed within 20 min or less of administration) were likely the result of indirect rapid actions within the brain, technically, these actions are not the result of steroids synthesized in the brain, and thus cannot be attributed to neurosteroid effects on behavior (Soma et al., 2008). These examples highlight that it is difficult to tease apart steroid vs. neurosteroid effects on behavior. However, a subsequent study in songbirds revealed that aggression in response to a stimulated territorial intrusion rapidly increases androgen synthesis in the brain during the non-breeding season. In this study, Pradhan et al. found that the enzyme 3β-hydroxysteroid dehydrogenase/Δ5-Δ4 isomerase (3β-HSD), which converts DHEA to AE, exhibits higher activity in the non-breeding season and also increases activity in response to a social challenge in several brain regions, including the ventromedial telencephalon (i.e., contains the avian medial amygdala) (Pradhan et al., 2010). This was a crucial study that elucidated a novel mechanism for the regulation of aggression based on androgens synthesized in the brain, and also demonstrated that neurosteroid activity can be acutely regulated by the social environment (Balthazart, 2010).

Although the study of neurosteroid-mediated social and aggressive behavior is still in relatively early stages (at least for the non-pharmacological study of natural behavior), this body of work demonstrates the involvement of neurosteroids in aggression (particularly DHEA and Allo) and suggests possible roles of estrogen-mediated social behavior. There remains much to understand about how neurosteroids may modulate social behaviors such as social preferences, grouping, and general prosocial contact.

3.5. Rapid effects

Neurosteroids may act via genomic ('nuclear initiated') and non-genomic ('membrane initiated') mechanisms on neural circuits to regulate behavior. Operating via non-genomic mechanisms, neurosteroids can have more immediate, rapid-signaling effects than steroids secreted by peripheral glands that act through classic nuclear steroid receptors (Simon, 2002; Frye, 2009). Rapid effects on physiology and behavior have been demonstrated for all major classes of steroids, but are best described for E_2 (Wendler et al., 2010; Heimovics et al., 2015b). Because of rapid, large fluctuations in local neurosteroid concentration and the presence of steroidogenic enzymes at presynaptic boutons, several researchers propose that neurally-synthesized steroids, particularly E_2, may act more like neuromodulators and neurotransmitters than classic hormones (Balthazart et al., 2006; Saldanha et al., 2011; Remage-Healey, 2014). Several studies have shown that brain estrogens can have rapid actions on neuronal excitability within seconds to minutes of treatment (Saldanha et al., 2011). Given that neurosteroids can be synthesized quickly, can reach high local concentrations, and are in close proximity for receptor binding, neurosteroids are more likely than systemic steroids to operate via rapid, non-genomic mechanisms (Schmidt et al., 2008; Heimovics et al., 2015b). This suggests that neurosteroids may be particularly important in the modulation of rapid behavioral responses to social challenges.

It has become quite common to read about 'rapid effects of steroids.' But can neuropeptides exhibit rapid effects on behavior as neurosteroids can? The nonapeptides exhibit complex signaling properties, including volumetric release from dendrites, axons, and soma (Landgraf and Neumann, 2004; Ludwig and Leng, 2006). Volumetric release from dendrites and soma allow for large quantities of peptides to travel to distal sites in the brain, representing nonapeptide actions that occur in a diffusive, slow, and global manner (Ludwig et al., 1994; Ludwig, 1998). Although most studies typically examine how nonapeptides maintain stable characteristics of behavioral phenotype, nonapeptides also exhibit targeted axonal, fast actions, allowing for the ability to execute distinct, rapid responses to external stimuli (Stoop, 2012; Kelly and Vitousek, 2017). Numerous studies have shown that nonapeptides can alter behavior on relatively fast timescales. For example, peripheral injection of VP alters calling behavior in frogs within 30 min (Kime et al., 2007), central infusion of VP into the LS induces decreases in aggression in waxbills within 5 min (Goodson, 1998b), and central infusion of VP into the AH increases flank marking in hamsters within 5 min (Ferris et al., 1984). Furthermore, optogenetic studies have demonstrated rapid actions of nonapeptide stimulation on unprecedented timescales. For example, optogenetically stimulating OT release from hypothalamic neurons projecting to the central amygdala robustly decreases freezing responses in fear conditioned rats, and the onset of the freezing behavior is observed as quickly as 2 s after 20 s of blue light (BL) stimulation, with an average onset time of 21.5 ± 9.7 s after BL stimulation. Furthermore, freezing behavior returns after 70 ± 21 s upon termination of the 20 s BL stimulation (Knobloch et al., 2012). This study provides a clear demonstration of the ability for OT to have rapid effects on behavior, and similar rapid actions are expected of VP (Stoop, 2012, 2014).

Taken together, both neurosteroids and nonapeptides exhibit physiological properties that allow for rapid effects on behavior. Thus, they are not only capable of, but are most likely involved in modulating rapid behavioral responses to social challenges.

4. Integrating systems in the brain

4.1. Neuropeptide–neurosteroid interactions

Given that neuropeptides and neurosteroids both modulate social and aggressive behaviors, and are both capable of rapidly modulating behavior, the question arises as to whether these systems interact in the brain to produce behavior in a context-appropriate manner. Recent studies have shown that neurosteroid biosynthesis is finely regulated by neurotransmitters such as glutamate and GABA, and of particular interest here, also by VP and OT (in frogs and birds; Do Rego et al., 2009). In birds, hypothalamic and extra-hypothalamic AROM-containing cell bodies that are responsible for the production of neuroestrogens are innervated by VPergic fibers (Viglietti-Panzica et al., 1994; Balthazart, 1997). In frogs, neurons expressing 3β-HSD, which converts DHEA to AE, appear to be contacted by VP- and OT-positive fibers (Do-Rego et al., 2006). Importantly, stimulating VP and OT increases neurosteroid production, and this production is suppressed by a 3β-HSD inhibitor, providing evidence that VP activates neurons that express steroidogenic enzymes (Do-Rego et al., 2006; Do Rego et al., 2009).

Just as neuropeptides regulate neurosteroids, the opposite is also true. In vitro studies in rat hypothalamic VP and OT neurons and dissociated axon terminals demonstrated that OT release is modulated by Allo, progesterone, and 17β-estradiol, whereas VP release is modulated by Allo (Widmer et al., 2003). Together these studies provide evidence of nonapeptide-neurosteroid interactions, however, the consequences

of such interactions on aggression and social behavior remain to explored.

4.2. A candidate region for examining neuropeptide–neurosteroid interactions on responses to social challenges

Can neurosteroids rapidly modulate nonapeptide-mediated aggression and/or social behavior? As discussed above, the nonapeptides can act as neuromodulators (Stoop, 2012), and optogenetic stimulation of nonapeptide neurons can result in behavioral changes in less than 30 s (Knobloch et al., 2012). Given the potential for rapid effects of nonapeptides on behavior, it is worth considering whether neurosteroids can regulate nonapeptide neuromodulation of behavior.

Earlier we discussed behavioral functions of nonapeptide cell groups. Although OT has earned a reputation for being primarily prosocial (Neumann, 2008; Carter and Porges, 2013; Bosch and Young, 2018) and VP is largely thought of as being pro-aggression (Ferris, 2005; Albers, 2012), the manner in which nonapeptides affect social and aggressive behavior is complex. Numerous studies have indeed associated VP with aggression, such as selective aggression in pair-bonded prairie voles (Gobrogge et al., 2007), flank marking in hamsters (Ferris et al., 1984), and agonistic calling in frogs (Ten Eyck and ul Haq, 2012). However, studies have also demonstrated pro-social functions of VP, such that VP injections reduce aggressive interactions in pupfish (Lema and Nevitt, 2004) and in territorial lizards (Dunham and Wilczynski, 2014), and blockade of VP receptors reduces gregariousness in finches (Kelly et al., 2011). Studies also show that VP can exert prosocial or pro-aggressive behavior depending on social context, as well as the sex and social status of the animal (Semsar et al., 2001; Bredewold et al., 2014). Importantly, behavioral effects of nonapeptides also strongly depend on the site of action and the source of peptide (Kelly and Goodson, 2014b).

Of particular interest here is the VP cell group of the BSTm. Several studies have suggested prosocial and anti-aggressive functions of the BSTm VP neuronal population. For example, mice selected for low aggression have more BSTm VP neurons than mice selected for high aggression (Compaan et al., 1993), and VP-ir fibers in brain regions highly innervated by the BSTm (e.g., the LS) negatively correlate with aggression in rats (Everts et al., 1997). In addition, we previously demonstrated that the BSTm VP cell group directly promotes sociality and inhibits aggression in opportunistic finches (Kelly et al., 2011; Kelly and Goodson, 2013). Furthermore, neural activity in this cell group positively correlates with prosocial behavior and negatively correlates with aggression in several species, including mice, chickens, and prairie voles (Ho et al., 2010; Xie et al., 2011; Kelly et al., 2018). Interestingly, the BSTm VP cell group represents one of the most phylogenetically widespread sex differences in the vertebrate brain, with males having significantly more neurons than females (De Vries and Panzica, 2006; Kelly and Goodson, 2013). Studies have shown that in seasonally breeding species, the BSTm VP cell group is strongly sensitive to sex steroids, such that this cell group decreases in size outside of the breeding season (De Vries and Buijs, 1983; De Vries and Panzica, 2006; Goodson et al., 2012b). However, even taking seasonal plasticity of BSTm VP into consideration (i.e., when this population is reduced in size), greater numbers of BSTm VP cells still negatively correlate with aggression and positively correlate with prosociality outside of the breeding season, implying functional consistency regardless of high or low levels of circulating steroids (Goodson et al., 2012b). Importantly, together these studies set a precedent that the BSTm VP neuronal population is sensitive to steroids. But is the BSTm VP cell group sensitive to neurosteroids? Interestingly, AROM colocalizes within the cell body of BSTm VP neurons in zebra finches (Kabelik et al., 2009), suggesting that neurosteroids may indeed be capable of regulating BSTm VP release on rapid timescales. Thus, we think that the BSTm VP cell group represents an excellent candidate for early studies to probe the potential for neurosteroid-nonapeptide interactions in the modulation of rapid behavioral responses to social challenges. We hypothesize that brain androgens are converted via AROM within BSTm VP neurons, which then have rapid estrogenic effects on BSTm VP release, such that estrogens may suppress VP release to cause a decrease in prosociality in order to allow for a rapid increase in aggression. For seasonal breeders, this may serve as a mechanism to rapidly modulate aggression outside the breeding season when it is better to avoid the cost of high levels of systemic T.

4.3. An emerging frontier

A few studies have examined the effects of gonadal steroids on VP-mediated aggression and social recognition (Delville et al., 1996; Thompson and Moore, 2003; Bolborea et al., 2010; Bychowski et al., 2013), however, to our knowledge, how neurosteroids influence nonapeptide-mediated behavior remains an open question. In order to elucidate neurosteroid-neuropeptide interactive effects on behavior, we must first identify colocalization sites within the brain. Nonapeptide distributions (as well as other neuropeptides, such as VIP and galanin (Joo et al., 2004; Kuteeva et al., 2004)) have been well-characterized in numerous species (Goodson et al., 2003; Goodson and Thompson, 2010; Rood and De Vries, 2011; Ni et al., 2014). Similarly, steroidogenic enzymes and androgen and estrogen receptors have been characterized for several species (Tsutsui et al., 2006; Do Rego et al., 2009; Pang et al., 2013). However, few studies have sought to characterize colocalization of neuropeptides (e.g., peptide-producing neurons and receptors) with steroidogenic enzymes and steroid receptors – a necessary step in determining how neurosteroid-neuropeptide interactions may affect behavior.

Some studies have shown that GPR30, a g-protein coupled estrogen receptor, colocalizes with OT within the same neurons in the preoptic area of fish (Mangiamele et al., 2017), and the hypothalamus of rats and mice (Sakamoto et al., 2007; Hazell et al., 2009). AROM colocalizes with VP within the same neurons in hypothalamic populations as well as the BSTm in birds (Balthazart, 1997; Balthazart et al., 2006; Kabelik et al., 2009), and progestin receptors colocalize with VP within the same neurons in the BSTm and centromedial amygdala of rats (Auger and De Vries, 2002). Given this colocalization within nonapeptide neuronal populations that are known to modulate social and aggressive behavior, these studies provide an excellent starting point for future research seeking to understand the consequences of nonapeptide-neurosteroid interactions on behavior.

Other considerations for future studies may include examination of differences in neuroplasticity of neurosteroid and neuropeptide systems. For example, will neuropeptide populations that exhibit seasonal plasticity be more or less sensitive to modulation via neurosteroids or systemic steroids? Do neurosteroids exhibit functional and/or anatomical seasonal plasticity? What are the timescales in which neuropeptides can regulate neurosteroids, vice versa, and are there site-specific differences? Do neurosteroid-neuropeptide interactions differentially titrate social context in both reproductive and non-reproductive conditions? Lastly, and not considered lightly (or easily), moving further toward an integrated systems neuroscience understanding of behavior, how do neurotransmitters interact with these neural systems to modulate behavior? Seeking answers to such questions will likely prove to open several new avenues of research that aim to contribute to our understanding of the mechanisms of complex behaviors, and how animals are able to rapidly respond to changes in an ever-changing social environment.

5. Conclusions

Exhibiting behavioral flexibility in order to mount appropriate responses to an ever-changing environment is crucial to the survival of all animals. In order for the brain and body to produce a context-appropriate and species-typical behavioral response, numerous peripheral

and neural systems must be involved. With advancements in our understanding of neuroendocrine-mediated behavior and continuing advancements in technology, we can add to the foundation laid by numerous studies inspired by the Challenge Hypothesis to further understand how animals are able to rapidly respond to environmental changes, including social challenges. Given the importance of social context in modulating steroid-mediated aggression, a fruitful next step in elucidating the mechanisms of aggression is to examine how social neural systems, such as the nonapeptide system, interact with steroids in the brain to produce rapid responses to social challenges. Complex behaviors are not the result of one mechanism or a single neural system, but rather the result of neuromodulatory patterning across several integrated systems, and thus studies seeking to integrate multiple systems hold great promise for expanding our knowledge of the mechanisms underlying various behaviors, such as sociality and aggression.

References

Acher, R., Chauvet, J., 1995. The neurohypophysial endocrine regulatory cascade: precursors, mediators, receptors, and effectors. Front. Neuroendocrinol. 16, 237–289.

Acher, R., Chauvet, J., Chauvet, M.T., 1995. Man and the chimaera. Selective versus neutral oxytocin evolution. Adv. Exp. Med. Biol. 395, 615–627.

Albers, H.E., 2012. The regulation of social recognition, social communication and aggression: vasopressin in the social behavior neural network. Horm. Behav. 61, 283–292.

Apfelbeck, B., Flinks, H., Goymann, W., 2017. Territorial aggression does not feed back on testosterone in a multiple-brooded songbird species with breeding and non-breeding season territoriality, the European stonechat. Horm. Behav. 87, 89–95.

Auger, C.J., De Vries, G.J., 2002. Progestin receptor immunoreactivity within steroid-responsive vasopressin-immunoreactive cells in the male and female rat brain. J. Neuroendocrinol. 14, 561–567.

Balthazart, J., 1997. Steroid control and sexual differentiation of brain aromatase. J. Steroid Biochem. Mol. Biol. 61, 323–339.

Balthazart, J., 2010. Behavioral implications of rapid changes in steroid production action in the brain [Commentary on Pradhan D.S., Newman A.E.M., Wacker D.W., Wingfield J.C., Schlinger B.A. and Soma K.K.: Aggressive interactions rapidly increase androgen synthesis in the brain during the non-breeding season. Hormones and Behavior, 2010]. Horm. Behav. 57, 375–378.

Balthazart, J., Cornil, C.A., Taziaux, M., Charlier, T.D., Baillien, M., Ball, G.F., 2006. Rapid changes in production and behavioral action of estrogens. Neuroscience 138, 783–791.

Barrett, C.E., Keebaugh, A.C., Ahern, T.H., Bass, C.E., Terwilliger, E.F., Young, L.J., 2013. Variation in vasopressin receptor (Avpr1a) expression creates diversity in behaviors related to monogamy in prairie voles. Horm. Behav. 63, 518–526.

Bartz, J.A., Zaki, J., Bolger, N., Ochsner, K.N., 2011. Social effects of oxytocin in humans: context and person matter. Trends Cogn. Sci. 15, 301–309.

Baulieu, E.E., 1980. Steroid hormone receptors. Expos Annu Biochim Med 34, 1–25.

Baulieu, E.E., 1991. Neurosteroids: a new function in the brain. Biol. Cell. 71, 3–10.

Baulieu, E.E., 1997. Neurosteroids: of the nervous system, by the nervous system, for the nervous system. Recent Prog. Horm. Res. 52, 1–32.

Beery, A.K., 2015. Antisocial oxytocin: complex effects on social behavior. Curr. Opin. Behav. Sci. 6, 174–182.

Biro, P.A., Stamps, J.A., 2008. Are animal personality traits linked to life-history productivity? Trends Ecol. Evol. 23, 361–368.

Bolborea, M., Ansel, L., Weinert, D., Steinlechner, S., Pevet, P., Klosen, P., 2010. The bed nucleus of the stria terminalis in the Syrian hamster (*Mesocricetus auratus*): absence of vasopressin expression in standard and wild-derived hamsters and galanin regulation by seasonal changes in circulating sex steroids. Neuroscience 165, 819–830.

Bosch, O.J., 2013. Maternal aggression in rodents: brain oxytocin and vasopressin mediate pup defence. Philos. Trans. R. Soc. Lond. Ser. B Biol. Sci. 368, 20130085.

Bosch, O.J., Neumann, I.D., 2012. Both oxytocin and vasopressin are mediators of maternal care and aggression in rodents: from central release to sites of action. Horm. Behav. 61, 293–303.

Bosch, O.J., Young, L.J., 2018. Oxytocin and social relationships: from attachment to bond disruption. Curr. Top. Behav. Neurosci. 35, 97–117.

Bredewold, R., Smith, C.J., Dumais, K.M., Veenema, A.H., 2014. Sex-specific modulation of juvenile social play behavior by vasopressin and oxytocin depends on social context. Front. Behav. Neurosci. 8, 216.

Bychowski, M.E., Mena, J.D., Auger, C.J., 2013. Vasopressin infusion into the lateral septum of adult male rats rescues progesterone-induced impairment in social recognition. Neuroscience 246, 52–58.

Cain, K.E., Rich, M.S., Ainsworth, K., Ketterson, E.D., 2011. Two sides of the same coin? Consistency in aggression to conspecifics and predators in a female songbird. Ethology 117, 786–795.

Caldwell, H.K., Albers, H.E., 2004. Effect of photoperiod on vasopressin-induced aggression in Syrian hamsters. Horm. Behav. 46, 444–449.

Carter, C.S., Porges, S.W., 2013. The biochemistry of love: an oxytocin hypothesis. EMBO Rep. 14, 12–16.

Charalampopoulos, I., Remboutsika, E., Margioris, A.N., Gravanis, A., 2008. Neurosteroids as modulators of neurogenesis and neuronal survival. Trends Endocrinol. Metab. 19, 300–307.

Compaan, J.C., Buijs, R.M., Pool, C.W., De Ruiter, A.J., Koolhaas, J.M., 1993. Differential lateral septal vasopressin innervation in aggressive and nonaggressive male mice. Brain Res. Bull. 30, 1–6.

De Vries, G.J., Buijs, R.M., 1983. The origin of the vasopressinergic and oxytocinergic innervation of the rat brain with special reference to the lateral septum. Brain Res. 273, 307–317.

De Vries, G.J., Panzica, G.C., 2006. Sexual differentiation of central vasopressin and vasotocin systems in vertebrates: different mechanisms, similar endpoints. Neuroscience 138, 947–955.

Delville, Y., Mansour, K.M., Ferris, C.F., 1996. Testosterone facilitates aggression by modulating vasopressin receptors in the hypothalamus. Physiol. Behav. 60, 25–29.

Dingemanse, N.J., Wolf, M., 2013. Between-individual differences in behavioural plasticity within populations: causes and consequences. Anim. Behav. 85, 1031–1039.

Do Rego, J.L., Seong, J.Y., Burel, D., Leprince, J., Luu-The, V., Tsutsui, K., Tonon, M.C., Pelletier, G., Vaudry, H., 2009. Neurosteroid biosynthesis: enzymatic pathways and neuroendocrine regulation by neurotransmitters and neuropeptides. Front. Neuroendocrinol. 30, 259–301.

Donaldson, Z.R., Young, L.J., 2008. Oxytocin, vasopressin, and the neurogenetics of sociality. Science 322, 900–904.

Do-Rego, J.L., Acharjee, S., Seong, J.Y., Galas, L., Alexandre, D., Bizet, P., Burlet, A., Kwon, H.B., Luu-The, V., Pelletier, G., Vaudry, H., 2006. Vasotocin and mesotocin stimulate the biosynthesis of neurosteroids in the frog brain. J. Neurosci. 26, 6749–6760.

Duckworth, R.A., 2006. Behavioral correlations across breeding contexts provide a mechanism for a cost of aggression. Behav. Ecol. 17, 1011–1019.

Dunham, L.A., Wilczynski, W., 2014. Arginine vasotocin, steroid hormones and social behavior in the green anole lizard (Anolis carolinensis). J. Exp. Biol. 217, 3670–3676.

Engel, S.R., Grant, K.A., 2001. Neurosteroids and behavior. Int. Rev. Neurobiol. 46, 321–348.

Engelmann, M., Landgraf, R., Wotjak, C.T., 2004. The hypothalamic-neurohypophysial system regulates the hypothalamic-pituitary-adrenal axis under stress: an old concept revisited. Front. Neuroendocrinol. 25, 132–149.

Everts, H.G., De Ruiter, A.J., Koolhaas, J.M., 1997. Differential lateral septal vasopressin in wild-type rats: correlation with aggression. Horm. Behav. 31, 136–144.

Ferris, C.F., 2005. Neuroplasticity and aggression: an interaction between vasopressin and serotonin. In: Nelson, R.J. (Ed.), Biology of Aggression. Oxford University Press, New York, pp. 163–175.

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.

Frye, C.A., 2009. Neurosteroids' effects and mechanisms for social, cognitive, emotional, and physical functions. Psychoneuroendocrinol 34 (Suppl. 1), S143–S161.

Gobrogge, K.L., Liu, Y., Jia, X., Wang, Z., 2007. Anterior hypothalamic neural activation and neurochemical associations with aggression in pair-bonded male prairie voles. J. Comp. Neurol. 502, 1109–1122.

Gobrogge, K.L., Liu, Y., Young, L.J., Wang, Z., 2009. Anterior hypothalamic vasopressin regulates pair-bonding and drug-induced aggression in a monogamous rodent. Proc. Natl. Acad. Sci. U. S. A. 106, 19144–19149.

Gobrogge, K.L., Jia, X., Liu, Y., Wang, Z., 2017. Neurochemical mediation of affiliation and aggression associated with pair-bonding. Biol. Psychiatry 81, 231–242.

Goodson, J.L., 1998a. Territorial aggression and dawn song are modulated by septal vasotocin and vasoactive intestinal polypeptide in male field sparrows (*Spizella pusilla*). Horm. Behav. 34, 67–77.

Goodson, J.L., 1998b. Vasotocin and vasoactive intestinal polypeptide modulate aggression in a territorial songbird, the violet-eared waxbill (Estrildidae: *Uraeginthus granatina*). Gen. Comp. Endocrinol. 111, 233–244.

Goodson, J.L., Thompson, R.R., 2010. Nonapeptide mechanisms of social cognition, behavior and species-specific social systems. Curr. Opin. Neurobiol. 20, 784–794.

Goodson, J.L., Evans, A.K., Bass, A.H., 2003. Putative isotocin distributions in sonic fish: relation to vasotocin and vocal-acoustic circuitry. J. Comp. Neurol. 462, 1–14.

Goodson, J.L., Lindberg, L., Johnson, P., 2004. Effects of central vasotocin and mesotocin manipulations on social behavior in male and female zebra finches. Horm. Behav. 45, 136–143.

Goodson, J.L., Evans, A.K., Lindberg, L., Allen, C.D., 2005. Neuro-evolutionary patterning of sociality. Proc. Biol. Sci. 272, 227–235.

Goodson, J.L., Schrock, S.E., Klatt, J.D., Kabelik, D., Kingsbury, M.A., 2009. Mesotocin and nonapeptide receptors promote estrildid flocking behavior. Science 325, 862–866.

Goodson, J.L., Kelly, A.M., Kingsbury, M.A., Thompson, R.R., 2012a. An aggression-specific cell type in the anterior hypothalamus of finches. Proc. Natl. Acad. Sci. U. S. A. 109, 13847–13852.

Goodson, J.L., Wilson, L.C., Schrock, S.E., 2012b. To flock or fight: neurochemical signatures of divergent life histories in sparrows. Proc. Natl. Acad. Sci. U. S. A. 109 (Suppl. 1), 10685–10692.

Goodson, J.L., Schrock, S.E., Kingsbury, M.A., 2015. Oxytocin mechanisms of stress response and aggression in a territorial finch. Physiol. Behav. 141, 154–163.

Goymann, W., Landys, M.M., Wingfield, J.C., 2007. Distinguishing seasonal androgen responses from male-male androgen responsiveness-revisiting the Challenge Hypothesis. Horm. Behav. 51, 463–476.

Goymann, W., Villavicencio, C.P., Apfelbeck, B., 2015. Does a short-term increase in testosterone affect the intensity or persistence of territorial aggression? — an approach using an individual's hormonal reactive scope to study hormonal effects on behavior. Physiol. Behav. 149, 310–316.

Guastella, A.J., Hickie, I.B., 2016. Oxytocin treatment, circuitry, and autism: a critical review of the literature placing oxytocin into the autism context. Biol. Psychiatry 79,

234–242.
Harmon, A.C., Huhman, K.L., Moore, T.O., Albers, H.E., 2002. Oxytocin inhibits aggression in female Syrian hamsters. J. Neuroendocrinol. 14, 963–969.
Hau, M., 2007. Regulation of male traits by testosterone: implications for the evolution of vertebrate life histories. Bioessays 29, 133–144.
Hau, M., Goymann, W., 2015. Endocrine mechanisms, behavioral phenotypes and plasticity: known relationships and open questions. Front. Zool. 12.
Hazell, G.G., Yao, S.T., Roper, J.A., Prossnitz, E.R., O'Carroll, A.M., Lolait, S.J., 2009. Localisation of GPR30, a novel G protein-coupled oestrogen receptor, suggests multiple functions in rodent brain and peripheral tissues. J. Endocrinol. 202, 223–236.
Heimovics, S.A., Ferris, J.K., Soma, K.K., 2015a. Non-invasive administration of 17beta-estradiol rapidly increases aggressive behavior in non-breeding, but not breeding, male song sparrows. Horm. Behav. 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. Integr. Comp. Biol. 55, 281–293.
Hirschenhauser, K., Oliveira, R.F., 2006. Social modulation of androgens in male vertebrates: meta-analyses of the challenge hypothesis. Anim. Behav. 71, 265–277.
Hirschenhauser, K., Winkler, H., Oliveira, R.F., 2003. Comparative analysis of male androgen responsiveness to social environment in birds: the effects of mating system and paternal incubation. Horm. Behav. 43, 508–519.
Ho, J.M., Murray, J.H., Demas, G.E., Goodson, J.L., 2010. Vasopressin cell groups exhibit strongly divergent responses to copulation and male-male interactions in mice. Horm. Behav. 58, 368–377.
Hoyle, C.H.V., 1998. Neuropeptide families: evolutionary perspectives. Regul. Pept. 73, 1–33.
de Jong, T.R., Neumann, I.D., 2018. Oxytocin and aggression. Curr. Top. Behav. Neurosci. 35, 175–192.
Joo, K.M., Chung, Y.H., Kim, M.K., Nam, R.H., Lee, B.L., Lee, K.H., Cha, C.I., 2004. Distribution of vasoactive intestinal peptide and pituitary adenylate cyclase-activating polypeptide receptors (VPAC1, VPAC2, and PAC1 receptor) in the rat brain. J. Comp. Neurol. 476, 388–413.
Kabelik, D., Kelly, A.M., Goodson, J.L., 2009. Dopaminergic regulation of mate competition aggression and aromatase-Fos colocalization in vasotocin neurons. Neuropharmacology 58, 117–125.
Kelly, A.M., Goodson, J.L., 2013. Functional significance of a phylogenetically widespread sexual dimorphism in vasotocin/vasopressin production. Horm. Behav. 64, 840–846.
Kelly, A.M., Goodson, J.L., 2014a. Hypothalamic oxytocin and vasopressin neurons exert sex-specific effects on pair bonding, gregariousness and aggression in finches. Proc. Natl. Acad. Sci. U. S. A. 111, 6069–6074.
Kelly, A.M., Goodson, J.L., 2014b. Social functions of individual vasopressin-oxytocin cell groups in vertebrates: what do we really know? Front. Neuroendocrinol. 35, 512–529.
Kelly, A.M., Vitousek, M.N., 2017. Dynamic modulation of sociality and aggression: an examination of plasticity within endocrine and neuroendocrine systems. Philos T R Soc B 372.
Kelly, A.M., Kingsbury, M.A., Hoffbuhr, K., Schrock, S.E., Waxman, B., Kabelik, D., Thompson, R.R., Goodson, J.L., 2011. Vasotocin neurons and septal V_{1a}-like receptors potently modulate songbird flocking and responses to novelty. Horm. Behav. 60, 12–21.
Kelly, A.M., Hiura, L.C., Saunders, A.G., Ophir, A.G., 2017. Oxytocin neurons exhibit extensive functional plasticity due to offspring age in mothers and fathers. Integr. Comp. Biol. 57, 603–618.
Kelly, A.M., Saunders, A.G., Ophir, A.G., 2018. Mechanistic substrates of a life history transition in male prairie voles: developmental plasticity in affiliation and aggression corresponds to nonapeptide neuronal function. Horm. Behav. 99, 14–24.
Kime, N.M., Whitney, T.K., Davis, E.S., Marler, C.A., 2007. Arginine vasotocin promotes calling behavior and call changes in male Tungara frogs. Brain Behav Evolut 69, 254–265.
Kingsbury, M.A., Wilson, L.C., 2016. The role of VIP in social behavior: neural hotspots for the modulation of affiliation, aggression, and parental care. Integr. Comp. Biol. 56, 1238–1249.
Kingsbury, M.A., Jan, N., Klatt, J.D., Goodson, J.L., 2015. Nesting behavior is associated with VIP expression and VIP-Fos colocalization in a network-wide manner. Horm. Behav. 69, 68–81.
Klatt, J.D., Goodson, J.L., 2013. Oxytocin-like receptors mediate pair bonding in a socially monogamous songbird. Proc. R. Soc. Lond. Ser. B Biol. Sci. 280, 20122396.
Knobloch, H.S., Grinevich, V., 2014. Evolution of oxytocin pathways in the brain of vertebrates. Front. Behav. Neurosci. 8, 31.
Knobloch, H.S., Charlet, A., Hoffmann, L.C., Eliava, M., Khrulev, S., Cetin, A.H., Osten, P., Schwarz, M.K., Seeburg, P.H., Stoop, R., Grinevich, V., 2012. Evoked axonal oxytocin release in the central amygdala attenuates fear response. Neuron 73, 553–566.
Kuteeva, E., Calza, L., Holmberg, K., Theodorsson, E., Ogren, S.O., Hokfelt, T., 2004. Distribution of galanin and galanin transcript in the brain of a galanin-overexpressing transgenic mouse. J. Chem. Neuroanat. 28, 185–216.
Landgraf, R., Neumann, I.D., 2004. Vasopressin and oxytocin release within the brain: a dynamic concept of multiple and variable modes of neuropeptide communication. Front. Neuroendocrinol. 25, 150–176.
Lee, A.G., Cool, D.R., Grunwald Jr., W.C., Neal, D.E., Buckmaster, C.L., Cheng, M.Y., Hyde, S.A., Lyons, D.M., Parker, K.J., 2011. A novel form of oxytocin in New World monkeys. Biol. Lett. 7, 584–587.
Lema, S.C., Nevitt, G.A., 2004. Exogenous vasotocin alters aggression during agonistic exchanges in male Amargosa River pupfish (Cyprinodon nevadensis amargosae). Horm. Behav. 46, 628–637.
Leonard, M.L., Horn, A.G., Eden, S.F., 1988. Parent-offspring aggression in moorhens. Behav. Ecol. Sociobiol. 23, 265–270.
Liu, Y., Wang, Z.X., 2003. Nucleus accumbens oxytocin and dopamine interact to regulate pair bond formation in female prairie voles. Neuroscience 121, 537–544.
Locci, A., Pinna, G., 2018. Social isolation as a promising animal model of PTSD comorbid suicide: neurosteroids and cannabinoids as possible treatment options. Prog. Neuro-Psychopharmacol. Biol. Psychiatry 92, 243–259.
Lorenz, K., 1966. On Aggression. Routledge, London.
Ludwig, M., 1998. Dendritic release of vasopressin and oxytocin. J. Neuroendocrinol. 10, 881–895.
Ludwig, M., Leng, G., 2006. Dendritic peptide release and peptide-dependent behaviours. Nat. Rev. Neurosci. 7, 126–136.
Ludwig, M., Callahan, M.F., Neumann, I., Landgraf, R., Morris, M., 1994. Systemic osmotic stimulation increases vasopressin and oxytocin release within the supraoptic nucleus. J. Neuroendocrinol. 6, 369–373.
Mangiamele, L.A., Gomez, J.R., Curtis, N.J., Thompson, R.R., 2017. GPER/GPR30, a membrane estrogen receptor, is expressed in the brain and retina of a social fish (Carassius auratus) and colocalizes with isotocin. J. Comp. Neurol. 525, 252–270.
Maruska, K.P., Fernald, R.D., 2013. Social regulation of male reproductive plasticity in an African cichlid fish. Integr. Comp. Biol. 53, 938–950.
Matsumoto, K., Ojima, K., Watanabe, H., 1996. Neurosteroidal modulation of social isolation-induced decrease in pentobarbital sleep in mice. Brain Res. 708, 1–6.
Mellon, S.H., Vaudry, H., 2001. Biosynthesis of neurosteroids and regulation of their synthesis. Int. Rev. Neurobiol. 46, 33–78.
Moore, F.L., Lowry, C.A., 1998. Comparative neuroanatomy of vasotocin and vasopressin in amphibians and other vertebrates. Comp. Biochem. Physiol. C Pharmacol. Toxicol. Endocrinol. 119, 251–260.
Moyer, K.E., 1968. Kinds of aggression and their physiological basis. Communications in Behavioral Biology 2 (Pt A), 65–87.
Nagasawa, M., Okabe, S., Mogi, K., Kikusui, T., 2012. Oxytocin and mutual communication in mother-infant bonding. Front. Hum. Neurosci. 6, 31.
Negayama, K., 2011. Kowakare: a new perspective on the development of early mother-offspring relationship. Integr Psychol Behav 45, 86–99.
Nelson, R.J., 2006. Biology of Aggression. Oxford University Press, New York, NY.
Nelson, R.J., Trainor, B.C., 2007. Neural mechanisms of aggression. Nat. Rev. Neurosci. 8, 536–546.
Neumann, I.D., 2008. Brain oxytocin: a key regulator of emotional and social behaviours in both females and males. J. Neuroendocrinol. 20, 858–865.
Ni, R.J., Shu, Y.M., Wang, J., Yin, J.C., Xu, L., Zhou, J.N., 2014. Distribution of vasopressin, oxytocin and vasoactive intestinal polypeptide in the hypothalamus and extrahypothalamic regions of tree shrews. Neuroscience 265, 124–136.
O'Connell, L.A., Hofmann, H.A., 2011. Genes, hormones, and circuits: an integrative approach to study the evolution of social behavior. Front. Neuroendocrinol. 32, 320–335.
Oliveira, R.F., 2004. Social modulation of androgens in vertebrates: mechanisms and function. Advances in the Study of Behavior 34, 165–239 (Academic Press).
Oliveira, R.F., Hirschenhauser, K., Carneiro, L.A., Canario, A.V.M., 2002. Social modulation of androgen levels in male teleost fish. Comparative Biochemistry and Physiology - B Biochemistry and Molecular Biology 132, 203–215.
Pang, Y., Dong, J., Thomas, P., 2013. Characterization, neurosteroid binding and brain distribution of human membrane progesterone receptors delta and {epsilon} (mPRdelta and mPR{epsilon}) and mPRdelta involvement in neurosteroid inhibition of apoptosis. Endocrinology 154, 283–295.
Pinna, G., Rasmusson, A.M., 2012. Up-regulation of neurosteroid biosynthesis as a pharmacological strategy to improve behavioural deficits in a putative mouse model of post-traumatic stress disorder. J. Neuroendocrinol. 24, 102–116.
Pinna, G., Dong, E., Matsumoto, K., Costa, E., Guidotti, A., 2003. In socially isolated mice, the reversal of brain allopregnanolone down-regulation mediates the anti-aggressive action of fluoxetine. Proc. Natl. Acad. Sci. U. S. A. 100, 2035–2040.
Pinna, G., Costa, E., Guidotti, A., 2005. Changes in brain testosterone and allopregnanolone biosynthesis elicit aggressive behavior. Proc. Natl. Acad. Sci. U. S. A. 102, 2135–2140.
Pitkow, L.J., Sharer, C.A., Ren, X., Insel, T.R., Terwilliger, E.F., Young, L.J., 2001. Facilitation of affiliation and pair-bond formation by vasopressin receptor gene transfer into the ventral forebrain of a monogamous vole. J. Neurosci. 21, 7392–7396.
Pradhan, D.S., Newman, A.E., Wacker, D.W., Wingfield, J.C., Schlinger, B.A., Soma, K.K., 2010. Aggressive interactions rapidly increase androgen synthesis in the brain during the non-breeding season. Horm. Behav. 57, 381–389.
Reale, D., Reader, S.M., Sol, D., McDougall, P.T., Dingemanse, N.J., 2007. Integrating animal temperament within ecology and evolution. Biol. Rev. Camb. Philos. Soc. 82, 291–318.
Remage-Healey, L., 2014. Frank Beach Award Winner: steroids as neuromodulators of brain circuits and behavior. Horm. Behav. 66, 552–560.
Remage-Healey, L., Maidment, N.T., Schlinger, B.A., 2008. Forebrain steroid levels fluctuate rapidly during social interactions. Nat. Neurosci. 11, 1327–1334.
Resendez, S.L., Aragona, B.J., 2013. Aversive motivation and the maintenance of monogamous pair bonding. Rev. Neurosci. 24, 51–60.
Rood, B.D., De Vries, G.J., 2011. Vasopressin innervation of the mouse (*Mus musculus*) brain and spinal cord. J. Comp. Neurol. 519, 2434–2474.
Rosell, D.R., Siever, L.J., 2015. The neurobiology of aggression and violence. CNS spectrums 20, 254–279.
Ross, H.E., Freeman, S.M., Spiegel, L.L., Ren, X., Terwilliger, E.F., Young, L.J., 2009. Variation in oxytocin receptor density in the nucleus accumbens has differential effects on affiliative behaviors in monogamous and polygamous voles. J. Neurosci. 29, 1312–1318.
Rosvall, K.A., Peterson, M.P., 2014. Behavioral effects of social challenges and genomic mechanisms of social priming: what's testosterone got to do with it? Current Zoology

60, 791–803.
Sakamoto, H., Matsuda, K., Hosokawa, K., Nishi, M., Morris, J.F., Prossnitz, E.R., Kawata, M., 2007. Expression of G protein-coupled receptor-30, a G protein-coupled membrane estrogen receptor, in oxytocin neurons of the rat paraventricular and supraoptic nuclei. Endocrinology 148, 5842–5850.
Saldanha, C.J., Remage-Healey, L., Schlinger, B.A., 2011. Synaptocrine signaling: steroid synthesis and action at the synapse. Endocr. Rev. 32, 532–549.
Schmidt, K.L., Pradhan, D.S., Shah, A.H., Charlier, T.D., Chin, E.H., Soma, K.K., 2008. Neurosteroids, immunosteroids, and the Balkanization of endocrinology. Gen. Comp. Endocrinol. 157, 266–274.
Semsar, K., Kandel, F.L., Godwin, J., 2001. Manipulations of the AVT system shift social status and related courtship and aggressive behavior in the bluehead wrasse. Horm. Behav. 40, 21–31.
Sih, A., Bell, A.M., 2008. Insights for behavioral ecology from behavioral syndromes. Adv. Study Behav. 38, 227–281.
Sih, A., Bell, A., Johnson, J.C., 2004. Behavioral syndromes: an ecological and evolutionary overview. Trends Ecol. Evol. 19, 372–378.
Simon, N.G., 2002. Hormonal processes in the development and expression of aggressive behavior. In: Pfaff, D.W., Arnold, A.P., Etgen, A.M., Fahrbach, S.E., Rubin, R.T. (Eds.), Hormones, Brain, and Behavior. Academic Press, New York, pp. 339–392.
Snell-Rood, E.C., 2013. An overview of the evolutionary causes and consequences of behavioural plasticity. Anim. Behav. 85, 1004–1011.
Soma, K.K., Scotti, M.A., Newman, A.E., Charlier, T.D., Demas, G.E., 2008. Novel mechanisms for neuroendocrine regulation of aggression. Front. Neuroendocrinol. 29, 476–489.
Stevenson, T.J., Hahn, T.P., MacDougall-Shackleton, S.A., Ball, G.F., 2012. Gonadotropin-releasing hormone plasticity: a comparative perspective. Front. Neuroendocrinol. 33, 287–300.
Stoop, R., 2012. Neuromodulation by oxytocin and vasopressin. Neuron 76, 142–159.
Stoop, R., 2014. Neuromodulation by oxytocin and vasopressin in the central nervous system as a basis for their rapid behavioral effects. Curr. Opin. Neurobiol. 29, 187–193.
Teles, M.C., Oliveira, R.F., 2016. Androgen response to social competition in a shoaling fish. Horm. Behav. 78, 8–12.
Ten Eyck, G.R., ul Haq, A., 2012. Arginine vasotocin activates aggressive calls during paternal care in the Puerto Rican coqui frog, Eleutherodactylus coqui. Neurosci. Lett. 525, 152–156.
Thompson, R.R., Moore, F.L., 2003. The effects of sex steroids and vasotocin on behavioral responses to visual and olfactory sexual stimuli in ovariectomized female roughskin newts. Horm. Behav. 44, 311–318.
Thompson, R.R., Walton, J.C., 2004. Peptide effects on social behavior: effects of vasotocin and isotocin on social approach behavior in male goldfish (*Carassius auratus*). Behav. Neurosci. 118, 620–626.
Tibbetts, E.A., Huang, Z.Y., 2010. The challenge hypothesis in an insect: juvenile hormone increases during reproductive conflict following queen loss in Polistes wasps. Am. Nat. 176, 123–130.
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 nongenomic pathways. Proc. Natl. Acad. Sci. U. S. A. 104, 9840–9845.
Tsutsui, K., Haraguchi, S., 2016. Neurosteroids. In: Handbook of Hormones, pp. 537–e96-12. Available at: https://doi.org/10.1016/B978-0-12-801028-0.00096-9.
Tsutsui, K., Ukena, K., Usui, M., Sakamoto, H., Takase, M., 2000. Novel brain function: biosynthesis and actions of neurosteroids in neurons. Neurosci. Res. 36, 261–273.
Tsutsui, K., Matsunaga, M., Miyabara, H., Ukena, K., 2006. Neurosteroid biosynthesis in the quail brain: a review. J Exp Zool A Comp Exp Biol 305, 733–742.
Tsutsui, K., Haraguchi, S., Fukada, Y., Vaudry, H., 2013. Brain and pineal 7alpha-hydroxypregnenolone stimulating locomotor activity: identification, mode of action and regulation of biosynthesis. Front. Neuroendocrinol. 34, 179–189.
Vahaba, D.M., Remage-Healey, L., 2018. Neuroestrogens rapidly shape auditory circuits to support communication learning and perception: evidence from songbirds. Horm. Behav. 104, 77–87.
Veenema, A.H., Beiderbeck, D.I., Lukas, M., Neumann, I.D., 2010. Distinct correlations of vasopressin release within the lateral septum and the bed nucleus of the stria terminalis with the display of intermale aggression. Horm. Behav. 58, 273–281.
Vergara, P., Fargallo, J.A., Martinez-Padilla, J., 2010. Reaching independence: food supply, parent quality, and offspring phenotypic characters in kestrels. Behav. Ecol. 21, 507–512.
Viglietti-Panzica, C., Aste, N., Balthazart, J., Panzica, G.C., 1994. Vasotocinergic innervation of sexually dimorphic medial preoptic nucleus of the male Japanese quail: influence of testosterone. Brain Res. 657, 171–184.
Wang, Z., Moody, K., Newman, J.D., Insel, T.R., 1997. Vasopressin and oxytocin immunoreactive neurons and fibers in the forebrain of male and female common marmosets (*Callithrix jacchus*). Synapse 27, 14–25.
Wendler, A., Baldi, E., Harvey, B.J., Nadal, A., Norman, A., Wehling, M., 2010. Position paper: rapid responses to steroids: current status and future prospects. Eur. J. Endocrinol. 162, 825–830.
Widmer, H., Ludwig, M., Bancel, F., Leng, G., Dayanithi, G., 2003. Neurosteroid regulation of oxytocin and vasopressin release from the rat supraoptic nucleus. J. Physiol. 548, 233–244.
Wilson, L.C., Goodson, J.L., Kingsbury, M.A., 2016. Seasonal variation in group size is related to seasonal variation in neuropeptide receptor density. Brain Behav. Evol. 88, 111–126.
Wingfield, J.C., 2017. The challenge hypothesis: where it began and relevance to humans. Horm. Behav. 92, 9–12.
Wingfield, J.C., Hahn, T.P., 1994. Testosterone and territorial behavior in sedentary and migratory sparrows. Anim. Behav. 77–89.
Wingfield, J.C., Hegner, R.E., Dufty Jr., A.M., Ball, G.F., 1990. The "Challenge Hypothesis": theoretical implications for patterns of testosterone secretion, mating systems, and breeding strategies. Am. Nat. 136, 829–846.
Wingfield, J.C., Lynn, S.E., Soma, K.K., 2001. Avoiding the 'costs' of testosterone: ecological bases of hormone-behavior interactions. Brain Behav. Evol. 57, 239–251.
Wingfield, J.C., Wacker, D.W., Bentley, G.E., Tsutsui, K., 2018. Brain-derived steroids, behavior and endocrine conflicts across life history stages in birds: a perspective. Front Endocrinol (Lausanne) 9, 270.
Xie, J., Kuenzel, W.J., Sharp, P.J., Jurkevich, A., 2011. Appetitive and consummatory sexual and agonistic behaviour elicits FOS expression in aromatase and vasotocin neurones within the preoptic area and bed nucleus of the stria terminalis of male domestic chickens. J. Neuroendocrinol. 23, 232–243.
Yoshihara, C., Numan, M., Kuroda, K.O., 2018. Oxytocin and parental behaviors. Curr. Top. Behav. Neurosci. 35, 119–153.
Young, L.J., 1999. Frank A. Beach Award. Oxytocin and vasopressin receptors and species-typical social behaviors. Horm. Behav. 36, 212–221.
Young, L.J., Wang, Z., 2004. The neurobiology of pair bonding. Nat. Neurosci. 7, 1048–1054.
Young, L.J., Lim, M.M., Gingrich, B., Insel, T.R., 2001. Cellular mechanisms of social attachment. Horm. Behav. 40, 133–138.
Young, K.A., Liu, Y., Wang, Z., 2008. The neurobiology of social attachment: a comparative approach to behavioral, neuroanatomical, and neurochemical studies. Comparative biochemistry and physiology. Toxicology & pharmacology: CBP 148, 401–410.
Young, K.A., Gobrogge, K.L., Liu, Y., Wang, Z., 2011. The neurobiology of pair bonding: insights from a socially monogamous rodent. Front. Neuroendocrinol. 32, 53–69.