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Behavioral Neuroendocrinology of Female Aggression

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Summary and Keywords

Aggressive behavior plays an essential role in survival and reproduction across animal species—it has been observed in insects, fish, reptiles, and mammals including humans. Even though specific aggressive behaviors are quite heterogeneous across species, many of the underlying mechanisms modulating aggression are highly conserved. For example, in a variety of species arginine vasopressin (AVP) and its homologue vasotocin in the hypothalamus, play an important role in regulating aggressive behaviorssuch as territorial and inter male aggression. Similarly in the medial amygdala, activation of a subpopulation of GABAergic neurons promotes aggression, while the prefrontal cortex exerts inhibitory control over aggressive behaviors. An important caveat in the aggression literature is that it is focused primarily on males, probably because in most species males are more aggressive than females. However, female aggression is also highly prevalent in many contexts, as it can affect access to resources such as mates, food, and offspring survival. Although it is likely that many underlying mechanisms are shared between sexes, there is sex specific variation in aggression, type, magnitude, and contexts, which suggests that there are important sex differences in how aggression is regulated. For example, while AVP acts to modulate aggression in both male and female hamsters, it increases male aggression but decreases female aggression. These differences can occur at the extent of neurotransmitter or hormones release, sensitivity (i.e., receptor expression), and/or molecular responses.

Keywords: aggression, sex differences, testosterone, progesterone, oxytocin, vasopressin

Introduction

Aggressive behaviors are a critical component of the competition for resources such as food, shelter, and mating opportunities. The expression of aggression may differ within and between individuals, populations, and species (King, 1973; Miczek, Faccidomo, Fish, & DeBold, 2007), and different forms of aggression can have different underlying neural and genetic networks (de Boer, Olivier, Veening, & Koolhaas, 2015; Takahashi & Miczek, 2014). However, for most species males engage in more intense and/or more frequent bouts of aggression than females, which may explain why research examining the mechanisms of aggression has historically been focused on males. For example, Darwin's writings on aggressive competition focused on the "Law of Battle" and highlighted the evolution of specialized weapons used by males in many species (Darwin, 1859).

Much of what we know about neural and hormonal mechanisms underlying aggression comes from studies of different species of rodents. An important discovery from these studies is that different neuroendocrine mechanisms are engaged to regulate different forms of aggression (Adams, 2006; Blanchard & Blanchard, 1977; Miczek, Fish, & De Bold, 2003; Takahashi & Miczek, 2014). Known forms of aggression include *offensive, defensive and escalated aggression. Offensive aggression* is associated with competition for resources, and attacks are usually targeted at non-vulnerable body areas of the opponent (Crawley, Schleidt, & Contrera, 1975; Miczek & O'Donnell, 1978). *Defensive aggression* is a response to fear-inducing stimuli, and as such is characterized by escape and threat behaviors. Here, attacks are usually directed towards vulnerable body areas such as the face of the threatening individual (Blanchard & Blanchard, 2003). More recently, the term *escalated aggression* has been introduced to describe what appears to be maladaptive behavior (de Almeida, Ferrari, Parmigiani, & Miczek, 2005; Haller & Kruk, 2006; Miczek, Faccidomo, de Almeida, Bannai, Fish, & Debold, 2004). Like offensive aggression, escalated aggression can be intense, and like defensive aggression attacks are directed towards more vulnerable body parts. However, in escalated aggression social signals are disregarded and attacks may continue even after an opponent has signaled defeat with submissive postures or signals. For these types of aggression, almost all mechanistic studies have been conducted on males. Most of what is known about the mechanisms of female aggression is from the context of *maternal aggression*, which is expressed by pregnant and early post partum females with the aim of offspring defense (Erskine, Barfield, & Goldman, 1978; Haney, Debold, & Miczek, 1989). This type of aggression is typically directed towards unfamiliar males. However, female aggression can be expressed in a variety of other contexts across taxa, which reflects its adaptive value (Rosvall, 2013A; Stockley & Bro-Jørgensen, 2011). In humans, physical aggression among women is relatively rare (Card, Stucky, Sawalani, & Little, 2008; Crick, Ostrov, &

Kawabata, 2007), but verbal aggression or manipulation of interpersonal relationships (Crick et al., 2007) is common (Benenson, 2013; Thornton, Graham-Kevan, & Archer, 2012; Vaillancourt, 2013) and frequently associated with physical and psychiatric problems (Kaltiala-Heino & Fröjd, 2011; Odgers et al., 2008; Pajer, 1998). This has led to a growing appreciation of the significance of aggressive behaviors in females, and a corresponding increase in the number of studies examining the underlying neuroendocrine mechanisms.

This introductory section will briefly summarize the forms of female aggression that have been best described in both non-human and human animals; the following sections will focus on the underlying neuroendocrine mechanisms in males and females. Importantly, while it has been proposed that offensive and defensive aggression subtypes are likely regulated by different mechanisms (Takahashi & Miczek, 2014), different forms of female aggression can include both defensive and offensive components (Lucion & de Almeida, 1996; Parmigiani, Rodgers, Palanza, Mainardi, & Brain, 1989). For males, most of the studies cited here are focused on offensive aggression, unless stated otherwise. Therefore, when talking about mechanisms of male and female aggression, we will focus on the context in which aggression is expressed.

Forms of Female Aggression

Maternal aggression is a defensive behavior in which offspring are protected, usually against conspecific individuals. This evolutionarily ancient form of behavior is present across the animal kingdom (DeVries, Winters, & Jawor, 2015; Figler, Twum, Finkelstein, & Peeke, 1995; Rosvall, 2013B; Sinn, While, & Wapstra, 2008). In mammals, maternal aggression usually takes the form of aggressive confrontation of male intruders by pregnant or lactating females (de Almeida, Ferreira, & Agrati, 2014; Palombit, 2012) and appears to have evolved as a strategy to prevent infanticide (Palombit, 2012). Interestingly, increased aggression in this context has been associated to down-regulation of physiological stress response and reduced anxiety (Gammie, D'Anna, Lee, & Stevenson, 2008; Hahn-Holbrook, Holt-Lunstad, Holbrook, Coyne, & Lawson, 2011). Remarkably, in some species in which both sexes make a considerable effort in caring for offspring, males also show increased aggressive behaviors to protect their young (Trainor, Finy, & Nelson, 2008A).

Territorial aggression, in which a resource of physical location is defended from competitors, is typically considered to be a male-typical behavior. A territory usually consists of resources such as food, shelter, and/or breeding sites, and maintaining exclusive access to these resources can increase fitness (Grant, 1993; Maher & Lott, 2000). Territorial aggression by females has been observed in fish (Ziadi-Kuenzli & Tachihara, 2016), reptiles (Jaeger, Kalvarsky, & Shimizu, 1982; Woodley & Moore, 1999), birds (Gowaty & Wagner, 1988), rodents (Ribble & Salvioni 1990), and non-human primates (Pusey & Schroepfer-Walker, 2013). Female territorial behavior can consist of defense of an individual territory or the territory of a social group. In most cases both males and females use signals or other types of indirect aggression to settle competitions without resorting to physical conflict (Cant & Young, 2013; Parker & Rubenstein, 1981; Vaillancourt, 2013). If a conflict is not settled through these indirect measures, aggression between females can be intense and lead to serious injury or even death, as seen in chimpanzees (*Pan troglodytes*) (Townsend, Slocombe, Emery Thompson, & Zuberbühler, 2007).

Competition for mates is usually considered a male typical behavior. Nonetheless, female competition for mates has been described in a variety of taxa, ranging from fish to mammals (Fernandez-Duque & Huck, 2013; Gavish, Sue Carter, & Getz, 1983; Matsumoto & Yanagisawa, 2001; Yasukawa & Searcy, 1982). This type of female competition is common in monogamous species, in which males provide resources other than sperm such as paternal care or access to a territory (Rosvall, 2011). Interestingly, female-female competition for mates can be substantial even in polygynous species. In these, competition can arise when there is a limited number of high quality mates and/or sperm (Preston, Stevenson, Pemberton, & Wilson, 2001; Wedell, Gage, & Parker, 2002), when there is a reduced males to females ratio (Charlat et al., 2007; Rusu & Krackow, 2004), when the breeding season is very short (Forsgren, Amundsen, Borg, & Bjelvenmark, 2004), or to ensure protection from infanticide (Palombit, Cheney, & Seyfarth, 2001; Stockley & Bro-Jørgensen, 2011).

Neuroendocrine Mechanisms of Female Aggression

The neuroendocrine basis of aggression has been mostly described in male rodents, and has been reviewed elsewhere (de Boer, Olivier, Veening, & Koolhaas, 2015; Falkner & Lin, 2014; Nelson & Trainor, 2007; Takahashi & Miczek, 2014). Interestingly, many of the same neural circuits that control male-male aggression are also important for maternal aggression (Gammie, 2005). However, studies of aggression outside the context of maternal defense have revealed important sex differences in the neuroendocrine mechanisms of aggression (Greenberg & Trainor, 2015; Pagani et al., 2015; Scott, Prigge, Yizhar, & Kimchi, 2015; Veenema, Bredewold, & DeVries 2013). Here we will highlight these discoveries and compare and contrast how neural circuits, steroid hormones, and neuropeptides modulate aggressive behaviors in females and males.

Neural Substrates

Aggressive behaviors rely on activity from neurobiological circuits controlling social behaviors (social behavior neural network) as well as motivation (mesocorticolimbic dopamine pathway). The components of these networks have been identified in part through studies assessing expression of immediate early genes, like c-fos and EGR-1, which can be considered as indirect markers of neuronal activity. Studies using techniques such as region specific lesions, pharmacological manipulations, and/or optical stimulation have been used to directly test how specific microcircuits regulate aggressive behaviors.

The Social Behavior Neural Network

Sarah Newman (1999) proposed that a social behavior network (SBN) consisting of the medial amygdala (MeA), bed nucleus of the stria terminalis (BNST), lateral septum (LS), periaqueductal gray (PAG), and the medial preoptic area (MPOA)/anterior hypothalamus (AH), work together to modulate social behaviors such as aggression in mammals (Figure 1). These nodes are reciprocally connected, and all express steroid hormones receptors (de Boer et al., 2015). Importantly, this network is evolutionarily conserved across diverse vertebrate taxa (Goodson & Kingsbury, 2013; Greenberg & Trainor, 2015).

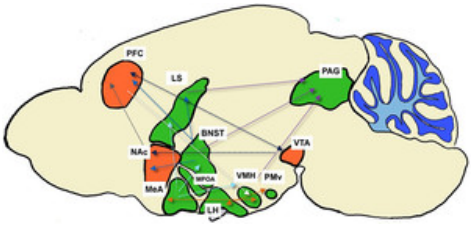

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Figure 1. Simplified neural circuits and connections associated with aggressive behavior. Green: Social behavior neural network. Orange: M esocorticolimbic dopamine system. PFC = prefrontal cortex, LS = lateral septum; NAc = nucleus accumbens; MeA = medial amygdala; BNST = bed nucleus of the stria terminalis; MPOA = hypothalamic medial preoptic area; LH = lateral hypothalamus; VMH = ventromedial hypothalamus; PMv = ventral premammilary nucleus; VTA = ventral tegmental area; PAG = periaqueductal gray.

Drawing by Natalia Duque-Wilckens.

Medial amygdala: For rodents, olfaction is the main sensory input regulating social behaviors, and the MeA plays a crucial role in processing sensory information coming from the olfactory bulb. The MEA sends efferent connections to the LS, BNST, and hypothalamus (Canteras, Simerly, & Swanson 1995; Dong, Petrovich, & Swanson, 2001b). The more posterior subregions of the medial amygdala are sexually dimorphic whereas more anterior subregions are not (Cooke, Tabibnia, & Breedlove, 1999). Both subregions appear to play a role in maternal aggression. Highly aggressive lactating female mice show increased c-fos immunoreactivity in anterior MeA compared to lactating females displaying low levels of aggression after being exposed to a male intruder (Gammie & Nelson, 2001). Another study examined the effect of engaging in maternal aggression on EGR-1 expression in the dorsal posterior MeA (MeApd). Females that engage in aggression have increased expression of EGR-1 in MEApd compared to females that do not engage in aggression (Hasen & Gammie, 2006). Interestingly, in males the MeApd is activated following either aggressive interactions (territorial aggression) as well as sexual behavior (Kollack-Walker & Newman, 1995, 1997; Veening et al., 2005). This suggests a strong connection between reproduction and aggression, at least in males.

Bed nucleus of the stria terminalis: Together with LS, the BNST constitutes an overlapping node between SBN and the mesolimbic dopamine (DA) system. The role of BNST on aggression has been better studied in males, where multiple circuits within BNST have been implicated (Masugi-Tokita, Flor, & Kawata, 2016; Shaikh, Brutus, Siegel, & Siegel, 1986; Veenema, Beiderbeck, Lukas, & Neumann, 2010). Following maternal aggression, the anterolateral BNST (BNSTl) has increased c-fos immunoreactivity (Gammie & Nelson, 2001). Similarly, female California mice that engage in aggression with a female intruder have increased phosphorylated extracellular regulated kinase (ERK) in the BNSTl compared to females that engage in a sham aggression test (Silva, Fry, Sweeney, & Trainor, 2010). This finding is consistent with studies showing increased c-fos in the BNSTl following maternal aggression, as phosphorylation of ERK facilitates transcription of c-fos (Monje, Hernández-Losa, Lyons, Castellone, & Gutkind, 2005). The BNSTl sends projections to LS and lateral hypothalamus, as well as to nuclei of the mesocorticolimic pathway (Dong & Swanson, 2004), so it is likely that the actions of this area on aggression involve more than one pathway. The anteromedial part of BNST (BNSTm) is also highly connected to nuclei involved in aggression, including LS, anterior MeA, lateral VMH, paraventricular nucleus (PVN), Nucleus accumbens (NAc), and ventral tegmental area (VTA). (Dong & Swanson, 2006). The connection between the BNSTv and VTA is especially intriguing as GABAergic projections from BNSTv promote appetitive responses while excitatory glutamatergic projections promote aversive responses. Although the role of this pathway has not been examined in the context of aggressive behavior, both aversion (Resendez, Kuhnmuench, Krzywosinski, & Aragona, 2012) and reward (Fish, De Bold, & Miczek, 2002, 2005) have been described as important properties mediating aggression.

Hypothalamus: The hypothalamus has long been recognized as an important locus mediating aggression (Lammers, Kruk, Meelis, & Poel, 1988; Lipp & Hunsperger, 1978; Siegel, Roeling, Gregg, & Kruk, 1999; Siegel & Pott, 1988). Initial experiments on male rodents (Koolhaas, 1978; Kruk, 1991) and cats (Siegel & Pott, 1988) showed that electrical stimulation of a hypothalamic attack area would trigger intense expression of aggressive behaviors. This attack area was later determined to consist of the lateral hypothalamus (LH) and ventromedial hypothalamus (VMH). After engaging in maternal aggression, female mice have increased EGR-1 immunoreactivity in both VMH and LH (Hasen & Gammie, 2006). Additionally, lesions of the VMH strongly reduce maternal aggression towards male intruders (Hansen, 1989). Curiously, the anterior part of VMH is an important node in a circuit that inhibits other types of maternal behavior such as nursing and pup retrieval (Mann & Babb, 2004; Sheehan et al., 2001; Sheehan & Numan, 1997). The onset of maternal aggression closely tracks the onset of other maternal behaviors in rodents (Mayer & Rosenblatt, 1984), so an important question is how VMH signaling changes so that it can go from inhibiting the combination of maternal behavior and aggression in virgin animals to promoting aggression in lactating females. It was recently

shown that a specific population of progesterone receptor (PR) expressing neurons in the ventrolateral subregion of VMH is key for territorial aggression and sexual behavior in males (Yang et al., 2013A). Interestingly, in females these PR neurons in ventrolateral VMH regulate sexual behavior but have no effect on maternal behavior. It is possible that non-PR expressing neurons in ventrolateral VMH may be more important for modulating maternal aggression or that more lateral subregions of VMH play a more significant role.

The MPOA is another nucleus that is very important for maternal behaviors. Immediate early gene studies have shown that MPOA activity is increased when postpartum females engage in both maternal care (Fleming, Suh, Korsmit, & Rusak, 1994; Numan & Numan, 1995) and maternal aggression (Gammie & Nelson, 2001; Hasen & Gammie, 2006; Motta et al., 2013). Lesion studies have confirmed that MPOA is essential for the display of maternal care (Numan, Corodimas, Numan, Factor, & Piers, 1988), but no study has specifically assessed the effects of MPOA lesion on maternal aggression. The role of the MPOA may generalize to paternal aggression in monogamous species in which males provide parental care. California mouse fathers are more aggressive than virgin males and have significantly more c-fos immunoreactive cells in the MPOA than virgins following a resident-intruder aggression (Trainor, Finy, & Nelson, 2008B).

Finally, the hypothalamic ventral premammilary nucleus (PMv), which has important functions for reproductive behaviors (Cavalcante, Bittencourt, & Elias, 2006; Kollack-Walker & Newman, 1995; Leshan, Louis, Jo, Rhodes, Münzberg, & Myers, 2009), was recently identified as a critical node modulating maternal aggression but not other aspects of maternal care (Motta et al., 2013). The PMv is reciprocally connected with regions important for social behaviors like MeA, VMH and LH (Canteras, Simerly, & Swanson, 1992). Engaging in maternal aggression significantly increases c-fos expression in PMv, and excitotoxic lesions of PMv inhibit aggressive behavior towards a male intruder in female lactating rats without affecting other behaviors including social investigation and pup nursing, licking and grooming (Motta et al., 2013). Interestingly, lesioned rats also show significantly less c-fos expression in anterior BNSTv, MPOA, VMH, and LH compared to unlesioned rats that display strong aggressive behavior, suggesting that the PMv has a key activating role in the maternal aggression neural network.

Lateral Septum: Immediate early gene studies suggest that increased activity in LS is negatively associated with aggression in both males and females (Goodson, Evans, & Soma, 2005; Lee & Gammie, 2007). Consistent with these data, inactivation or lesions of LS increases intraspecific male aggression in a variety of species (Albert & Chew, 1980; Goodson, Eibach, Sakata, & Adkins-Regan, 1999; Potegal, Blau, & Glusman, 1981; Ramirez, Salas, & Portavella, 1988; Slotnick, McMullen, & Fleischer, 1973). Similarly, activation of $GABA_A$ receptors in LS inhibits both maternal aggression (Lee & Gammie, 2009) and male-male (offensive) aggression (McDonald, Markham, Norvelle, Albers, & Huhman, 2012; Wong et al., 2016). A recent study showed that optogenetic activation of the pathway of LS projecting to VMHvl is sufficient to inhibit male territorial aggression without affecting other social and sexual behaviors (Wong et al., 2016). It is unclear whether this circuit is also important for maternal aggression. Further investigation of how the LS-VMH circuit changes with the onset of maternal behavior and aggression is needed.

Periaqueductal gray: The PAG is involved in the motor output of a variety of aggressive behaviors in males (Siegel & Pott, 1988; Siegel & Victoroff, 2009). It receives afferent connections from hypothalamus, BNST, and LS, and is thought to promote species-specific aggressive behaviors (Nelson & Trainor, 2007). In females, the caudal PAG (caPAG) has been associated with modulation of maternal aggression. Increased egr-1 as well as c-fos expression in the caPAG is observed following maternal aggression (Gammie & Nelson, 2001; Hasen & Gammie, 2006), and lesions to caPAG increase maternal aggression in rats (Lonstein & Stern, 1998). Further, injections of $GABA_A$ receptor antagonist into this region dose-dependently decrease maternal aggression while promoting maternal care (Lee & Gammie, 2010). Thus, the output from caPAG seems to be crucial for inhibiting maternal aggression in favor of the expression of other parental behaviors.

Mesocorticolimbic Dopamine System

Aggressive behaviors have a strong motivational component (de Almeida & Miczek, 2002; Fish et al., 2002; May, 2011), and as such are modulated by brain regions that define the salience and valence of a stimuli (Kalivas & Volkow, 2005; Love, 2014). The mesocorticolimbic dopamine pathway consists of the ventral tegmental area (VTA) and its efferent projections to the nucleus accumbens (NAc), amygdala, hippocampus, and prefrontal cortex (PFC). This circuit and the SBN interact to regulate social behaviors such as aggression (O'Connell & Hofmann, 2011). In the context of maternal aggression, it has been proposed that the presence of an intruder induces a negative affective state in the lactating dam, which results in the motivation to attack the intruder (de Almeida et al., 2014).

Ventral tegmental area: The VTA is a heterogeneous nucleus with important topographical organization (Barker, Root, Zhang, & Morales, 2016; Love, 2014). In the VTA, GABAergic neurons are important inhibitory regulators of dopamine (DA) neurons (Mathon, Kamal, Smidt, & Ramakers, 2003). Early studies showed that infusions of the GABA agonist muscimol into caudal VTA increase aggression in male rats (Arnt & Scheel-Krüger, 1979). More recently, it was shown that optogenetic stimulation of DA neurons in VTA increases isolation-induced aggression in male mice (Yu et al., 2014). These apparently conflicting results might be related to the fact that VTA is a very complex structure. The VTA has different subpopulations of neurons, and both DA and non-DA cells express GABA receptors (Mathon et al., 2003). Further, VTA neurons are capable of signaling using one or more neurotransmitters; for example, some neurons in the VTA can co-release both DA and glutamate (Zhang et al., 2015), or glutamate and GABA (Root et al., 2014). Thus, even what would appear to be a highly specific manipulation (optical stimulation of DA neurons) could result in very complex changes in neurotransmitter release. In general, little is known about the role of VTA on female aggression. Overall, most evidence points to a limited role. Immediate early gene studies observed no changes in c-fos or egr-1 in the

VTA following maternal aggression (Gammie & Nelson, 2001; Hasen & Gammie, 2006). Similarly, in lactating rats, inactivation of VTA with microinfusions of 6-hydroxydopamine (6-OHDA) does not have any effect on aggression, although it does affect pup retrieval behavior (Hansen, Harthon, Wallin, Löfberg, & Svensson, 1991). It is unclear whether the VTA plays a more important role in modulating aggression in other contexts.

Nucleus accumbens: For male aggression, there is strong evidence for an important role of the NAc. Indeed, haloperidol, an antagonist of the D2 receptors (highly expressed in NAc) was long used to reduce aggressive behaviors in mentally ill patients (de Deyn et al., 1999). However, systemic D2 inhibition has many additional adverse effects (e.g., decreased arousal and motor problems). Rodent studies have provided more targeted evidence that DA receptors within the NAc have important effects on aggression. Infusion of DA receptor antagonists into NAc significantly reduces territorial aggression in male mice (Couppis & Kennedy, 2008). In females, engaging in one episode of maternal aggression does not affect c-fos immunoreactivity in NAc (Gammie & Nelson, 2001). However, a recent study showed that female Syrian hamsters that engage in repeated displays of territorial aggression show increased spine density in NAc (Staffend & Meisel, 2012). These changes, which are mediated by decreased phosphorylation of fragile X mental retardation protein (FMRP), enhance aggressive behavior in future encounters (Been, Moore, Kennedy, & Meisel, 2016). This observation suggests that neuroplasticity within the NAc may play a role in reinforcing aggressive behavior. This is supported by findings in male rodents. Male rats that engage in regularly scheduled bouts of aggression show increased DA release in the NAc in anticipation of aggressive encounters (Ferrari, Erp, Tornatzky, & Miczek 2003). Also, male California mice that win aggressive encounters have increased androgen receptor (AR) immunoreactivity in the NAc (Fuxjager, Forbes-Lorman, Coss, Auger, Auger, & Marler, 2010). It is not clear whether these changes in DA release and AR expression result in neuroplastic changes that affect behavior.

Prefrontal cortex: In general, PFC has important inihibitory effects on aggressive behaviors across species, including humans (Nelson & Trainor, 2007; Raine & Yang, 2006). The PFC receives and sends projections to the hypothalamus, NAc, VTA, and amygdala (Gabbott, Warner, Jays, Salway, & Busby, 2005; Hoover & Vertes, 2011; Peyron, Petit, Rampon, Jouvet, & Luppi, 1998; Rosenkranz & Grace, 2002; Vertes, 2004), and is tightly associated with the serotoninergic system, a main modulator of aggressive behavior. Studies in males have shown that PFC acts primarily to inhibit aggression (Nelson & Trainor, 2007; Takahashi, Nagayasu, Nishitani, Kaneko, & Koide, 2014), although studies examining the role of specific circuitries within subregions of the PFC have shown that this relationship is more complex. For example, activation of medial PFC (mPFC), but not orbitofrontal cortex, inhibits intrasexual (territorial) aggression in males (Takahashi & Miczek, 2014). In the context of maternal aggression, the ventro orbital subregion of PFC (voPFC) has been shown to exert anti-aggressive effects (Veiga, Miczek, Lucion, & de Almeida, 2007; Veiga, Miczek, Lucion, & de Almeida, 2011).

Hormonal Mechanisms of Female Aggression

Steroid and neuropeptide hormones play an important role in coordinating aggressive behavior with other bodily functions, such as reproduction. In some cases these hormones have similar effects on aggression in both males females. However, in other cases, hormonal mechanisms important for male aggression have no or even opposite effects in females.

Steroid Hormones

Steroid hormones can be produced in the adrenal gland, gonads, and brain. Although the gonads are the most obvious source for sex differences in steroid synthesis, steroid synthesis in adrenal and brain can also differ in males and females. Neuronal effects of steroid hormones are involved in both the development of aggressive behavior and in its expression during adulthood. Prenatally, steroid hormones contribute to the organization of neural circuits (French, Mustoe, Cavanaugh, & Birnie, 2013), and during adulthood steroids participate in the modulation of aggressive behaviors and the associated physiological responses (French et al., 2013; Soma, Scotti, Newman, Charlier, & Demas, 2008).

Androgens

Gonadal Sources of Androgens

The role of androgens on aggressive behavior in males is well established (Soma, 2006). Early studies found that castration reduces aggression in males (Payne & Swanson, 1971; Vandenbergh, 1971), although now we know these effects may be mediated by estrogen in addition to testosterone (T) (discussed below). In many seasonally breeding species, increased T levels serve to coordinate increased territorial aggression with mating behavior (Wingfield, Hegner, Dufty, & Ball, 1990; Wingfield, Lynn, & Soma 2001). In females, there is increasing evidence that T is increased in the breeding season (Ketterson, Nolan, & Sandell, 2005), and high levels of T have been related to sexual behavior in women (Anders, Hamilton, Schmidt, & Watson, 2007). In addition, female T levels are higher in colonial bird species with elevated competition for nesting sites compared to solitary species that have less competition for nesting sites (Møller, Garamszegi, Gil, Hurtrez-Boussès, & Eens, 2005).

Dynamic changes in circulating T have been shown to have an important effect on aggression in males. For example, changes in T during intrasexual aggressive encounters can have important long-term effects on aggression in future competitions (Gleason, Fuxjager, Oyegbile, & Marler, 2009). This has been called the challenge effect, which predicts that concentrations of T should be elevated during social competition to promote aggressive behaviors (Wingfield et al., 1990). Interestingly, dynamic changes in T can also affect aggression in females. For example, in

the absence of dominant males, females of the African cichlid fish (*Astatotilapia burtoni*) increase their T levels and show higher levels of territorial aggression (Renn, Fraser, Aubin-Horth, Trainor, & Hofmann, 2012). Similarly, females of the daffodil cichlid (*Neolamprologus pulcher*) and female marmosets (*Callithrix kuhlii*) show elevated levels of testosterone after aggressively defending their territories from intruders (Desjardins, Hazelden, Van der Kraak, & Balshine, 2006; Ross & French, 2011). Nonetheless, T elevation in competitive social environment is not always observed in females (Davis & Marler, 2003; Goymann, Wittenzellner, Schwabl, & Makomba, 2008; Rubenstein & Wikelski, 2005). In some cases, circulating T levels in females actually decreases following an aggressive encounter (Elekonich & Wingfield, 2000; Rubenstein & Wikelski, 2005). Currently, the functional basis for the variability in T responses to aggressive interactions in females is unclear. Although androgens are known to have important metabolic costs (Buchanan, Evans, Goldsmith, Bryant, & Rowe, 2001; Marler & Moore, 1989) and immunosuppressive effects (Hillgarth & Wingfield, 1997), they can have additional side effects in females. These can include breeding delay, altered mate choice behavior, and inhibition of maternal care (Gerlach & Ketterson, 2013; McGlothlin, Neudorf, Casto, Nolan, & Ketterson, 2004; Rosvall, 2013B). Thus, it has been hypothesized that the elevation of T in response to competition should be more common in species with low maternal care, or species in which offspring are relatively less susceptible to parental neglect (Rosvall, 2013A). In turn, species with higher maternal investment are expected to have evolved mechanisms other than elevation of T to mediate aggression. An alternative solution to limit the costs of T would be to employ local synthesis of T.

T Synthetized in the Brain

Although there is little direct evidence for T synthesis within the brain, recent data suggest that the enzymes necessary to produce T de novo from cholesterol are present in many brain areas (Do Rego et al., 2009). In addition, it is possible that T could be synthesized from dehydroepiandrosterone (DHEA), a steroid precursor that can be synthesized in nervous tissue (Corpéchot, Robel, Axelson, Sjövall, & Baulieu, 1981; Do Rego et al., 2007; Hojo et al., 2004) and adrenal glands (Labrie et al., 2005). DHEA can be metabolized into androgens and/or estrogens in peripheral tissues (Labrie et al., 2005). Data from rodents and songbirds suggest that this conversion of DHEA in to active steroids can occur in the brain where it could affect behavior (Dupont, Simard, Luu-The, Labrie, & Pelletier, 1994; Soma, Alday, Hau, & Schlinger, 2004). For example, plasma levels of DHEA are elevated in male sparrows during the non-breeding season (Maddison, Anderson, Prior, Taves, & Soma, 2012; Newman & Soma, 2009), when territorial aggression levels are still elevated but plasma T levels are very low (Soma, Schlinger, Wingfield, & Saldanha, 2003). Interestingly, levels of the enzyme necessary for conversion of DHEA to androgens, 3β-hydroxysteroid dehydrogenase/δ5-δ4 isomerase (3β-HSD), are highest during this time in centromedial and caudal telencephalon (Pradhan, Newman, Wacker, Wingfield, Schlinger, & Soma, 2010). In addition, the activity of these enzymes is further increased during territorial challenges and engaging in aggression. These data suggest that male sparrows have the potential for T synthesis within the brain, and that it can be rapidly modulated. However the hypothesis that the conversion of DHEA to T increases aggression still needs to be tested directly. In females, DHEA has been linked to territorial aggression in Siberian hamsters. During winter-like days, female Siberian hamsters show increased levels of territorial aggression and have elevated levels of serum DHEA and adrenal DHEA responsiveness (see FIGURE 2) (Rendon, Rudolph, Sengelaub, & Demas, 2015). Further, aggression reduces serum DHEA levels during short days (Rendon & Demas, 2016). One possible explanation for this result is that DHEA metabolism to androgens during aggressive encounters might reduce circulating DHEA levels, but again, this is not direct evidence that DHEA is being converted to androgens to modulate aggression, as alternative mechanisms of action are possible. For example, DHEA can bind to estrogen and androgen receptors in the brain (Webb, Geoghegan, Prough, & Miller, 2006) and has been shown to modulate action of various receptor systems known to modulate aggression including $GABA_A$ and NMDA (Bergeron, Montigny, & Debonnel, 1996; Compagnone & Mellon, 2000; Majewska, 1992). Further studies will be needed to directly test which are the underlying mechanisms mediating the effects of DHEA on aggressive behavior.

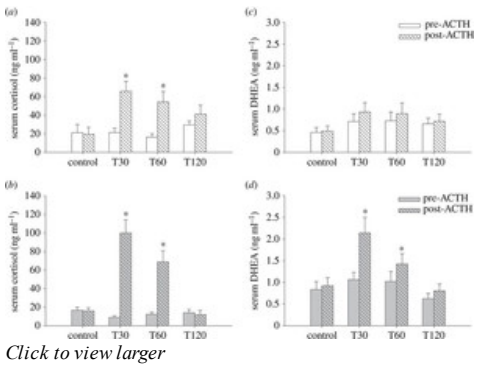

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Figure 2. ACTH challenge time course for cortisol and DHEA across photoperiods. Cortisol levels for (a) long-day females, (b) short-day females; DHEA levels for (c) long-day females and (d) short-day females receiving either an ACTH or control treatment. White bars, long days; grey bars, short days. Bar heights represent means ± *SEM* *$p < 0.05$.

Androgens: Mechanisms of Action

The effects of androgens on behavior can be mediated through direct binding to intracellular androgen receptors (AR), which then migrate to the nucleus to regulate gene expression (Heemers & Tindall, 2007). This so-called genomic effect is relatively slow, with the effects on cell function observed over a period of hours to days. Alternatively, T can rapidly influence aggressive behavior by non-genomic mechanisms in which AR are deposited in the cell membrane (Lösel & Wehling, 2003) or through intracellular AR that remain outside the nucleus. These non-genomic actions are mediated by second messenger pathways that can lead to rapid changes in cell function within seconds or minutes (Foradori, Weiser, & Handa, 2008). Membrane associated AR have been described in dendrites (Tabori et al., 2005) and axons (DonCarlos et al., 2006).

In the context of aggression, both slow and rapid effects of T have been reported. For example, a study in the coral reef fish *Stegastes nigricans* used implants of flutamide, an AR antagonist, to show that AR are necessary for the expression of territorial aggression in males but not females (Vullioud, Bshary, & Ros, 2013). Here flutamide

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was administered over the course of 5-6 days, which is consistent with slow acting genomic effects of steroid hormones. However, T can also have more rapid effects. In white-footed mice (*Peromyscus leucopus*), one pulse of T can modify territorial marking within 20 minutes, suggesting that non-genomic regulation can modify this agonistic behavior (Fuxjager, Knaebe, & Marler, 2015). Although these studies did not directly test whether these effects were mediated by AR, other evidence suggests that estrogens are not necessary. For example, male California mice (*Peromyscus californicus*) injected with T immediately following aggressive encounters are more aggressive in future encounters than males receiving saline, and this effect is not blocked by aromatase (the enzyme responsible for converting T to estrogens) inhibitor treatment (Trainor, Bird, & Marler, 2004). Rapid effects of T on aggressive behavior have also been observed in females. *Lythrypnus dalli* is a fish species that can show bidirectional sex changes. Dominant females show increased territorial aggression within 2 hours of exogenous administration of 11-ketotestosterone, a teleost analog to dihydrotestosterone, compared to controls (Pradhan, Connor, Pritchett, & Grober, 2014) (Figure 3).

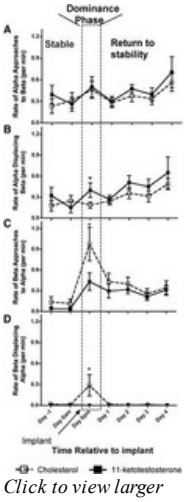

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Figure 3. Rates of agonistic interactions between alpha and beta *Lythrypnus dalli* relative to the time before or after alphas were treated with either 11-ketotestosterone or cholesterol. (A) Alpha approaches beta; (B) alpha displaces beta; (C) beta approaches alpha; and (D) beta displaces alpha. $*p < 0.05$ (n = 8 cholesterol and n = 10 ketotestosterone). Differences in aggressive behavior of both alphas and betas are seen within 2 h of implanting the alpha. Dotted lines represent the transient window of social instability that follows male removal.

Reprinted from "Contextual Modulation of Androgen Effects on Agonistic Interactions" by D. S. Pradhan, K. R. Connor, E. M. Pritchett, and M. S. Grober, 2014, *Hormones and Behavior*, *65*(1), 47–56. Copyright with permission from Elsevier.

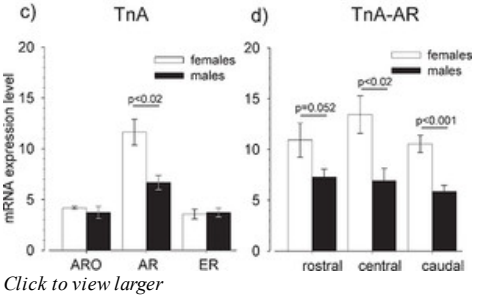

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Figure 4. Mean (± *SEM*) expression level of AR, ERα (ER) and aromatase (ARO) mRNA in nucleus taeniae (TnA) of male and female black coucals. The expression of AR mRNA in TnA was significantly higher in females than males (c), particularly in central and caudal part of the nucleus (d).

Interestingly, in several cases AR expression in the brain is promoted by T, which in turn can promote sex differences in brain AR expression. In *Mus musculus*, AR expression is higher in intact males than intact females in BNST, LS, and MPOA; a difference that is abolished by gonadectomy (Lu, McKenna, Cologer-Clifford, Nau, & Simon, 1998). In addition, females treated with T implants show AR expression levels equivalent to males in these nuclei. However, this is not true for every species. For example, although T increases AR gene expression in male rats, no effects of T implants were observed on AR expression in females (Roselli, 1991). Further, in females central AR expression can be higher than in males even with lower circulating T. An intriguing example is the African black coucal (*Centropus grillii*), a cuckoo bird in which females are more aggressive than males. Females have lower levels of circulating T, but show higher expression of AR mRNA in the taeniae of the amygdala (a brain region analogous to the mammalian MeA) than males (Voigt & Goymann, 2007) (Figure 4). In addition to sex specific expression of steroid receptors, activation of the same receptors in males and females may result in sex-specific gene expression profiles. For example, in the sexually dimorphic dark-eyed junco (*Junco hyemalis*), treatment with T has different effects on gene expression in the brain for males versus females. Testosterone increased expression of aromatase in hypothalamus in both sexes, but decreased expression of monoamine oxidase A in MeA in females but not males (Peterson et al., 2013). Together, this suggests that different region specific expression pattern of AR, as well as downstream effects of AR in males vs. females, may allow females to harness the beneficial effects of T on certain behaviors while avoiding some of the adverse effects that T may cause on other behaviors, metabolism and reproduction (Wingfield et al., 2001).

Estrogens

Peripheral Sources of Estrogens

Estrogens have very important effects on aggression in both males and females. Early studies in males showed that E administration (Payne & Swanson, 1972A; Vandenbergh, 1971), as well as ovarian implantation (Payne & Swanson, 1971), could increase inter-male aggression. During development, T secreted by the male gonads is converted to estradiol (E2) in the brain, which has long lasting effects on male typical behaviors, including aggression, and inhibition of female like behaviors (Lenz, Nugent, & McCarthy, 2012; Wu et al., 2009).

Acting in the adult brain, estrogens are mainly associated with promoting aggressive behaviors. In females, estrogens promote aggression in reptiles (Rubenstein & Wikelski, 2005), birds (Rosvall et al., 2012), and mammals (Albert, Petrovic, & Walsh, 1989). The role of estrogens on aggression has been particularly well studied in the context of maternal behavior. For example, ovariectomy on gestation day 16 in rats significantly reduces maternal aggression in comparison to controls (sham operated gestating rats), and this effect can be reversed by injections of E2 (Mayer & Rosenblatt, 1987). In males, evidence from several bird and rodent species shows that E2 can

Reprinted with permission from "Sex-Role Reversal Is Reflected in the Brain of African Black Coucals (*Centropus grillii*)," by C. Voigt and W. Goymann, 2007, *Developmental Neurobiology*, *67*, 1560–1573.

increase territorial aggressive behavior (Laredo, Villalon Landeros, & Trainor, 2014). In California mice, estrogens increase territorial aggression if males are housed in cages containing corncob bedding (Trainor et al., 2008A), which increases blood levels of E2-like tetrahydrofuran-diols (Villalon Landeros, Morisseau, Yoo, Fu, Hammock, & Trainor, 2012). However, if California mice are housed on cardboard-based bedding then estrogens decrease aggression (Laredo et al., 2013). These results show that factors such as social experience and diet can have important effects how estrogens modulate aggression. These factors may alter how estrogens interact with estrogen receptors (Byrnes, Babb, & Bridges, 2009; Byrnes, Casey, & Bridges, 2012). Finally, estrogens are involved at multiple levels of processing of social information (Ervin, Lymer, Matta, Clipperton-Allen, Kavaliersv, & Choleris, 2015), and can interact with other steroid hormones and neurotransmitter systems to influence aggression (Murakami, 2016; Soma, Rendon, Boonstra, Albers, & Demas, 2015)

Brain Sources of Estrogens

Aromatase, is expressed in brain areas that regulate aggressive behaviors across species (Balthazart, Baillien, Charlier, Cornil, & Ball, 2003; Cohen & Wade, 2011; Naftolin, Horvath, & Balthazart, 2001; Shen, Schlinger, Campagnoni, & Arnold, 1995), and its activity has been associated with aggression in males and females. For example, studies in male Japanese quail (*Coturnix japonica*) showed that individual differences in territorial aggressive behavior during the breeding season were positively correlated with aromatase in hypothalamus, but no correlation was seen between circulating T and this behavior (Schlinger & Callard, 1989). Further, administration of an aromatase inhibitor, but not an AR antagonist, significantly reduces aggression in this species (Schlinger & Callard, 1990). Similarly, genetic deletion of aromatase completely eliminates territorial aggression in mice (Toda, Saibara, Okada, Onishi, & Shizuta, 2001). In general, aromatase activity in the brain is higher in males than females (Roselli, Horton, & Resko, 1985), a difference mediated by at least two mechanisms. First, aromatase enzyme velocity is higher in males than females in the BNST and VMH (Roselli, Klosterman, & Fasasi, 1996B). Second, androgen receptor is a more effective enhancer of aromatase mRNA expression in male hypothalamus than females (Roselli, Abdelgadir, Jorgensen, & Resko, 1996A). Interestingly, recent data showed that aromatase producing neurons in the MeA had important effects on aggression in both males and females (Unger, Burke, Yang, Bender, Fuller, & Shah, 2015). Aromatase expressing neurons in the MeApd were selectively ablated in adult mice, such that the MeApd was allowed to develop normally in the presence of aromatase. When these aromatase expressing neurons were lesioned, both intermale aggression and maternal aggression were reduced but reproductive behaviors were unaffected (Figure 5, A & B).

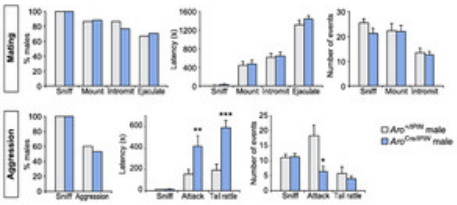

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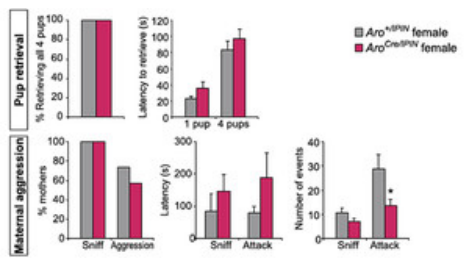
Figure 5.(a.) Ablation of aromatase + meA neurons reduces specific components of male and maternal aggression. No difference between $aro^{Cre/IPIN}$ and $aro^{+/IPIN}$ males in mating with an estrous female. Comparable percent of $aro^{Cre/IPIN}$ and $aro^{+/IPIN}$ males sniff and attack an intruder male. Aro Cre/IPIN males take significantly longer to attack and tail rattle, and they attack intruder less. (Unger et al., 2015).

Reprinted from "Medial Amygdalar Aromatase Neurons Regulate Aggression in Both Sexes," by E. K. Unger, K. J. Burke, C. F. Yang, K. J. Bender, P. M. Fuller, and N. M. Shah, 2015, *Cell Reports*, *10*(4), 453–462. Copyright (2015), with permission from Elsevier.



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Estrogen: Mechanisms of Action

Estrogens exert their action by binding to estrogen receptors (ER) ERα and ERβ, which are expressed throughout the neural circuit of aggression (Cushing, 2016; Kaiser, Kruijver, Swaab, & Sachser, 2003; Kramer, Simmons, & Freeman 2008). Similar to androgen receptors, these receptors can be expressed in the nucleus (Heldring et al., 2007; Micevych & Dominguez, 2009), as well as outside of the nucleus including dendrites, axons, and neuronal terminals (Blaustein, Lehman, Turcotte, & Greene, 1992; Milner, McEwen, Hayashi, Li, Reagan, & Alves, 2001; Milner et al., 2005; Towart, Alves, Znamensky, Hayashi, McEwen, & Milner, 2003). Activation of these receptors results in activation second messenger systems that can rapidly modulate cell function (Heimovics, Trainor, & Soma, 2015B)

Initial knock out studies assessing global effects of ER in males found that functional ERα facilitate normal expression of inter-male aggression in mice (Ogawa, Lubahn, Korach, & Pfaff, 1997; Scordalakes & Rissman, 2003). Furthermore male aggression in CD-1 mice is positively correlated with the expression of ERα in areas important for aggression including BNST, LS, and LH (Trainor, Greiwe, & Nelson, 2006). ERβ, on the other side, has been associated with the inhibition of aggression, although this effect seems to depend on age and sexual experience (Nomura et al., 2002; Ogawa et al., 1999). However, studies comparing the effects of selective ERα and ERβ agonists on male aggression have found to both increase or decrease territorial aggression depending on the light cycles used for testing (Trainor et al., 2007). Similar results have been reported in females. Gonadectomized female mice treated with selective ERα agonists have increased aggressive attacks towards intruders (Clipperton-Allen, Almey, Melichercik, Allen, & Choleris, 2011) while females treated with selective ERβ agonists show increased non-attack agonistic behaviors such as social investigation and dominance (Clipperton-Allen, Cragg, Wood, Pfaff, & Choleris, 2010). Interestingly knockout of ERα increases levels of offensive aggression in females (Ogawa, Eng, Taylor, Lubahn, Korach, & Pfaff, 1998). Together these results indicate that in males

Figure 5.(b.) Ablation of aromatase + meA neurons reduces specific components of male and maternal aggression. The vast majority of $aro^{cre/IPIN}$ and $aro^{+/IPIN}$ females retrieved all pups to the nest, and they did so with similar latencies. Comparable percentages of $aro^{Cre/IPIN}$ and $aro^{+/IPIN}$ females sniffed and attacked an intruder male. There was a significant decrease in number of attacks directed to the intruder male by $aro^{Cre/IPIN}$ females.

Reprinted from "Medial Amygdalar Aromatase Neurons Regulate Aggression in Both Sexes," by E. K. Unger, K. J. Burke, C. F. Yang, K. J. Bender, P. M. Fuller, and N. M. Shah, 2015, *Cell Reports*, *10*(4), 453–462. Copyright (2015), with permission from Elsevier.

and females ERa and ERb have distinct organizational and activational effects on aggressive behaviors.

E can act rapidly to modulate aggression. These effects may originate from the ability of aromatase activity to be rapidly modulated by environmental factors such as stress or social conflict (Balthazart et al., 2003; Dickens, Balthazart, & Cornil, 2012; Dickens, Bournonville, Balthazar, & Cornil 2014). Although the specific directional effects of E on aggression (either increasing or decreasing) depend on factors such as diet (Villalon Landeros et al., 2012), a robust observation is that rapid E action is more likely to be observed in the non-breeding season. In both *Peromyscus polionotus* (Trainor et al., 2007) and *Peromyscus californicus* (Trainor et al., 2008A), E rapidly modulates aggression in winter-like short day photoperiods but not in summer-like long day photoperiods. In *P. californicus*, the specificity of rapid E action under short days has been replicated (Laredo et al., 2014). Similarly, a study in male song sparrows showed that administration of E2 rapidly (within 20 min) increases territorial aggression in non-breeding males only (Heimovics, Ferris, & Soma, 2015A). Currently it is unclear whether rapid activation of ER modulates aggression in females, but rapid effects of E on brain and other behaviors, including memory formation (Gabor, Lymer, Phan, & Choleris, 2015), modulation of pain (An, Li, Yan, & Li, 2014), and anxiety-like behaviors (Holm, Liang, Thorsell, & Hilke, 2014), have been reported in females. This suggests that it is likely that this type of E signaling could be involved in modulation of female aggression.

Progesterone

Although progesterone (P4) production has been classically associated with female gonads, P4 can be also synthetized in male and female adrenal glands and nervous system, and has been shown to affect physiology and behavior in both sexes.

Gonadal and Adrenal P4

The effects of circulating progesterone (P4) on aggression have been mainly studied in the context of parental behaviors. In mammals, plasma levels of P4 decrease towards the end of gestation and then rise progressively from post-partum day 3 to 10 (Finley, Zhang, & Fewell, 2015; Taya & Greenwald, 1982). The administration of P4 receptor (PR) antagonist in day 6 post-partum increases maternal aggression (de Sousa et al., 2010) (Figure 6). Since maternal aggression sharply increases during peripartum and remains high for around 2 weeks post-partum, the authors proposed that increasing levels of P4 would have a role in the progressive reduction of maternal aggression after that period. The sharp decline in P4 around parturition has also been shown to be a key component of the onset of pup-directed behavior in females (Bridges, Rosenblatt, & Feder, 1978; Sheehan & Numan, 2002). Interestingly, studies using the biparental species *Peromyscus californicus* have found that in males, paternal behavior is also associated with a decrease in plasma P4 (Trainor et al., 2003) and PR expression in the BNST (Perea-Rodriguez, Takahashi, Amador, Hao, Saltzman, & Trainor, 2015; Trainor, Bird, Alday, Schlinger, & Marler, 2003). Transgenic mice lacking PR receptors show reduced aggression towards pups (Schneider et al., 2003; Schneider, Burgess, Horton, & Levine, 2009), suggesting that P4 and PR activation may facilitate infanticide.

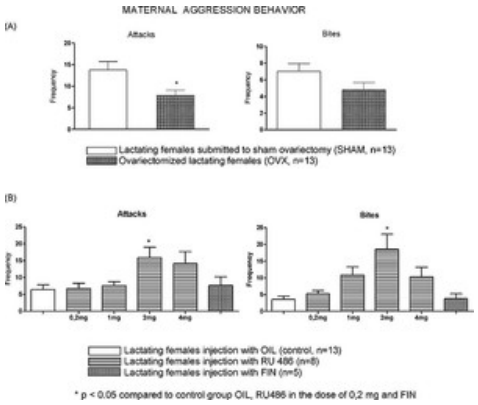

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Figure 6. (A) Maternal aggressive behavior of lactating females [6th day postpartum] who underwent SHAM surgery or ovariectomy [OVX] on the first day postpartum. A Student *t* test was used between the two experimental groups [with a $p < 0.005$ level of significance]. Indicates a significant difference between the groups [SHAM and OVX]. (B) Maternal aggressive behavior of lactating females [sixth day postpartum] that received OIL, RU 486 (0.2, 1.0, 2.0, and 4.0 mg/kg); and FIN injected SC, 2 h

Some studies have also assessed the role of P4 in the context of territorial aggression, although data here is less clear. Pharmacological manipulations of P4 in rodents have shown that P4 can both reduce and increase territorial aggression. For example, one study showed that administration of P4 reduces aggression in both male and female gonadectomized Syrian hamsters (Fraile, McEwen, & Pfaff, 1987), but another study in the same species showed that in ovariectomized females, daily injections of P4 for ten days increase intrasexual aggression (Payne & Swanson, 1972B). In female bank voles (*Clethrionomys glareolus*), administration of P4 also increases aggression (Kapusta, 1998). Similarly, studies assessing physiological levels of P4 have yielded conflicting results. One study in California mice (*Peromyscus californicus*) observed decreases in plasma P4 in females after engaging in aggression (Davis & Marler, 2003B). A similar result was observed in free-living black coucals (*Centropus grillii*), in which females also have decreased serum P4 after engaging in aggression (Goymann et al., 2008). Furthermore, females treated with P4 implants show reduced territorial aggression compared to females given empty implants. In contrast, P4 is higher in aggressive compared to non-aggressive female Iguanas (*Amblyrhynchus cristatus*) (Rubenstein & Wikelski, 2005), and studies in sparrows (*Melospiza melodia*) have found no association between P4 and female aggression (Elekonich & Wingfield, 2000B). Together, the data available suggests that while P4 seems to be necessary for the expression of maternal aggression, P4 in other contexts can have different effects,

before test one-way ANOVA was used between the three experimental groups [with a $p < 0.05$ level of significance]. Indicates a significant difference between the groups [OIL, RU 0.2 mg and FIN]. The data are expressed as mean [± *SEM*] of the frequencies of behaviors studied. The number of animals [*n*] is given between parentheses.

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and this may be related to the relationship between levels of P and other steroid hormones. During lactation, T and E2 levels are low compared to P4 (Taya & Greenwald, 1982). Interestingly, female California mice showing territorial aggression show the opposite; P4/T ratio is decreased compared to controls (Davis & Marler, 2003A). Further studies are needed to test this hypothesis.

Progesterone: Mechanisms of Action

Similar to estrogens, P4 can exert its effect through genomic and non-genomic mechanisms (Taraborrelli, 2015; Wendler, Albrecht, & Wehling, 2012). Nuclear and membrane PR are widely expressed throughout the brain (Schumacher et al., 2014), and have been related to the regulation of a variety of behaviors, including sexual behavior, social recognition and motivation (Bychowski & Auger, 2012; Frye, Koonce, & Walf, 2013; Frye, Walf, Kohtz, & Zhu, 2014; Yang et al., 2013A). To our knowledge, little work has assessed the relationship between female aggression and PR. Deletion of the PR gene has no effect on intermale aggression, but female aggression was not examined (Schneider et al., 2003). On the other hand, ablation of PR-expressing neurons in VMH reduces territorial aggressive and sexual behavior in male mice but has no effect on territorial aggressive behavior in females (Yang et al., 2013A). This line of mice may not be optimal for assessing the role of PR on aggression in females, as C57Bl6 normally do not engage in significant levels of aggression outside of maternal defense.

Intriguingly, it is possible for P4 to affect behavior independently of PR. P4 can be metabolized in the brain to allopregnanolone (Dong et al., 2001A; Pinna et al., 2008), which is an efficient positive allosteric modulator of $GABA_A$ receptors (Belelli & Lambert, 2005; Herd, Belelli, & Lambert, 2007; Pinna et al., 2000; Puia et al., 2003). The activation of $GABA_A$ by allopregananolone has been mainly associated with an inhibition of aggression. For example, aggression in socially isolation male mice is associated with reduced levels of brain allopregnanolone (Dong et al., 2001A; Pinna, Agis-Balboa, Pibiri, Nelson, Guidotti, & Costa, 2008). Further, administration of fluoxetine, which prevents declines in allopregnanolone, blocks the effects of social isolation on aggression (Pinna et al., 2003). In females, social isolation does not reduce levels of allopregnanolone, and it also does not increase aggression (Pinna, Dong, Matsumoto, Costa, & Guidotti, 2003). Interestingly, long-term administration of T in female mice results in both increased levels of territorial aggression and reduction of allopregnanolone levels (Pinna, Costa, & Guidotti, 2005), which suggests that T actions on aggression could be mediated in part by down regulation of brain allopreganolone biosynthesis.

Glucocorticoids

Engaging in aggression frequently generates a significant response from the hypothalamus-pituitary-adrenal (HPA) axis (Bronson & Eleftheriou, 1965; Earley et al., 2006; Ramenofsky, 1985; Schuurman, 1980; Woodley & Moore, 1999). Although an increased glucocorticoid (GC) response is often examined in the context of losing aggressive interactions, GC have important effects on aggressive behaviors.

Glucocorticoids and Aggression

GC production has been involved in both the promotion and inhibition of aggression in humans and animals, and many times this effect is context specific. For example, while acute systemic (Haller, Albert, & Makara, 1997; Mikics, Kruk, & Haller, 2004) and hypothalamic (Hayden-Hixson & Ferris, 1991) injections of GC promote territorial aggression in male rodents, chronic administration of high levels of GC suppresses aggression in a variety of species (Leshner, Korn, Mixon, Rosenthal, & Besser, 1980; Wingfield & Silverin, 1986). Interestingly, GC seems to have a particular important effect on abnormal levels of aggression. Animals selected for high levels of aggression show lower GC responses (Carere, Groothuis, Möstl, Daan, & Koolhaas, 2003; Veenema, Meijer, de Kloet, & Koolhaas, 2003A; Veenema, Meijer, de Kloet, Koolhaas, & Bohus, 2003B), and chronic glucocorticoid deficit induced by adrenalectomy results in escalated aggression in male rats (Haller, Schraaf, & Kruk, 2001). This has also been described in humans. Aggression in habitually violent offenders (Virkkunen, 1985) and patients with antisocial personality disorder (Dolan, Anderson, & Deakin, 2001) is associated with low GC levels. In females, HPA activity has been mainly associated with the reduction of aggression. In female rodents increased corticotrophin releasing hormone (CRH) signaling, as well as administration of dexamethasone, reduces maternal aggression (Gammie, Negron, Newman, & Rhodes, 2004; Gammie, Hasen, Stevenson, Bale, & D'Anna 2005; Vilela & Giusti-Paiva, 2011). Similarly, female crows (*Corvus macrorhynchos*) show a negative correlation between cortisol metabolites and dominance rank, which is the opposite of what happens in males (Ode, Asaba, Miyazawa, Mogi, Kikusui, & Izawa, 2015). A study in young women found a negative correlation between circulating cortisol and reactive aggression (Stoppelbein, Greening, Luebbe, Fite, & Becker, 2014). The apparent promotion or inhibition of GC on aggressive behavior may be explained by the fact that GC do not directly activate or inhibit aggression circuits, rather, they modulate neurons properties (Reul & de Kloet, 1985). Thus, the activation of specific neural circuits may be the primary variable modulating aggressive response, which in turn can be secondarily modulated by GC signaling.

Glucocprticoids: Mechanisms of Action

The modulating actions of GC are regulated through two types of receptors, the mineralocorticoid receptors (MR) and the GC receptor (GR) (Herman et al., 2016). MR primarily mediates the action of baseline levels of GC, while lower affinity GR are more important for mediating the effects of stress-induced increases of GC (Myers, McKlveen, & Herman, 2012). Both MR and GR can act as transcription factors, or as mediators of rapid non-genomic signaling (Groeneweg, Karst, de Kloet, & Joëls, 2012). MR are expressed in limbic areas including PFC, LS, and hippocampus, where they co-localize with GR. GR are more widely distributed throughout the brain, but also show lower affinity for GC than MR (Reul & de Kloet, 1985). It has been a challenge to directly assess the specific role of these receptors in regulating aggression, especially GR. This is because the field does not have access to a specific GR antagonist. For example, two groups have reported that RU486 treatment reduces territorial aggression in male rainbow trout (Schjolden, Basic, & Winberg, 2009) and electric fish (Dunlap, Jashari, & Pappas, 2011). The problem for interpreting these results is that RU486 is very strong antagonist of PR receptors also, so it is impossible to rule out a role for PR. Transgenic mice overexpressing or underexpressing GR have been produced (Ridder et al., 2005), but aggressive behavior has not be examined. An ideal strategy would be to examine the effect of inducible knockout of GR in brain regions mediating aggression such as the lateral septum. For MR, the specific antagonist spironolactone has been a useful tool. Intriguingly, a single injection of spironolactone reduced offensive behavior in male rats against a naïve intruder, whereas repeated spironolactone treatment enhanced aggression (Ruiz-Aizpurua, Buwalda, & de Boer, 2013). Thus, while it is clear that GCs can modulate aggression, the specific receptors and site of action mediating these effects have not been identified. Moreover, virtually no studies examining GC regulation of aggression have been conducted in females, despite that fact that, in many rodent species, GC levels are significantly higher in females than in males (Critchlow, Liebelt, Bar-Sela, Mountcastle, & Lipscomb, 1963; Trainor, Takahashi, Silva, Crean, & Hostetler, 2010; Weiser & Handa, 2009).

Peptide Hormones: Vasopressin and Oxytocin

Arginine vasopressin (AVP) and oxytocin (OT) are highly conserved neuropeptides. They are mainly synthetized in hypothalamus, and can be released peripherally (Sivukhina & Jirikowski, 2016; Yang, Wang, Han, & Wang, 2013B) and centrally through either local synaptic or somatodendritic release (Bosch & Neumann, 2012; Dumais & Veenema, 2016; Gobrogge, Liu, Young, & Wang, 2009; Love, 2014). These neuropeptides regulate a variety of social behaviors (Albers, 2015; Caldwell & Albers, 2015; Veenema & Neumann, 2008). Although it is usually assumed that the behavioral effects of OT are mediated by OT receptor (OTR), and AVP through AVP receptor (AVPR) subtypes, recent work has highlighted the promiscuity of these receptors (Manning et al., 2012). The effects of OT can be mediated by AVPR, while AVP can also activate OTR (Anacker, Christensen, LaFlamme, Grunberg, & Beery, 2016; Ramos et al., 2013; Song, Larkin, Malley, & Albers, 2016). OTR and AVPR are expressed throughout the structures regulating aggression (Caldwell & Albers, 2015). Importantly, the expression of the neuropeptides and their receptors are regulated by gonadal hormones (Amico, Thomas, & Hollingshead, 1997; Bale, Pedersen, & Dorsa, 1995; Delville, Mansour, & Ferris, 1996; DeVries, Wang, Bullock, & Numan, 1994; Young, Muns, Wang, & Insel, 1997A). The actions of gonadal hormones may play an important role in determining the sex-specific effects of OTR and V1aR activation on behavior (Dumais & Veenema, 2016).

Vasopressin

The effects of AVP on aggression in rodents are highly sex-specific. In male rodents, AVP typically promotes aggressive behavior when the individual is in a familiar context. This effect is best described in Syrian hamsters (*Mesocricetus auratus*), in which AVP acting in the AH is necessary for the display of territorial aggressive behaviors (Ferris, Albers, Wesolowski, Goldman, & Luman, 1984; Ferris, Meenan, Axelson, & Albers 1986; Ferris & Potegal, 1988). Microinjections of AVP into ventrolateral hypothalamus (VLH) (Delville et al., 1996), MeA, and LS (Koolhaas, Moor, Hiemstra, & Bohus, 1990) have also been found to increase male territorial aggression in rodents. The effects of AVP on aggression are thought to be mediated mainly through its actions on AVPR subtype V1a (V1aR). For example, in male hamsters, oral administration of the V1aR antagonist SRX251 (Ferris et al., 2006) significantly reduces male aggression in a resident intruder test. Similarly, infusion of Manning compound (a highly specific V1aR antagonist) into the lateral ventricle reduces aggression in male California mice (Bester-Meredith, Martin, & Marler, 2005). Microinjections of Manning compound directly into medioventral BNST (BNSTmv) also increased attack latency for males tested in a resident-intruder test (Duque-Wilckens et al., 2016). The effects of V1aR on aggression can be species specific and likely mediated by species differences in social systems as well as V1aR receptor distribution (Goodson & Bass, 2001). For example, intracerebroventricular (i.c.v) infusion of either AVP or Manning had no effect on aggression in the white-footed mouse (*Peromyscus leucopus*) (Bester-Meredith et al., 2005). Both hamsters (Ferris & Potegal, 1988) and California mice (Ribble & Salvioni, 1990) are highly territorial species, while the white-footed mouse is less territorial (Metzgar, 1971). In addition to territorial aggression, AVP has been found to be an important molecule regulating selective aggression in monogamous species. Selective aggression refers to the aggression directed towards opposite sex individuals after the formation of a pair bond (Carter & Getz, 1993). After forming a pair bond with a female, male prairie voles (*Microtus ochrogaster*) treated with an i.c.v. injection of V1aR antagonist showed reduced aggression towards unfamiliar females (Winslow, Hastings, Carter, Harbaugh, & Insel, 1993). Selective aggression is absent in the polygynous montane vole (*Microtus montanus*) (Young, Winslow, Nilsen, & Insel, 1997B).

In females, there is strong evidence that AVP has important effects on maternal aggression. For example, Brattleboro rats that are congenitally deficient of AVP show markedly reduced maternal aggression (Fodor et al., 2014). Similarly, studies using rat dams selected for high (HAB) and low (LAB) anxiety behaviors show that AVP signaling is key for the expression of maternal aggression (Bosch & Neumann, 2010, 2012). The

increased anxiety levels showed by HAB rats is a result of augmented hypothalamic AVP activity, which may be due to a polymorphism in the AVP gene promoter region (Murgatroyd et al., 2004; Wigger et al., 2004). Interestingly, HAB rats also show increased levels of maternal care and aggression (Bosch, Meddle, Beiderbeck, Douglas, & Neumann, 2005A; Neumann, Krömer, & Bosch, 2005). Blockade of V1aR in the MPOA reduces maternal aggression and maternal care in HAB rats (Bosch & Neumann, 2008). Besides the MPOA, the BNST (Bosch, Pförtsch, Beiderbeck, Landgraf, & Neumann, 2010) (Figure 7) and central amygdala (Bosch & Neumann, 2010) are key sites of local signaling for V1aR regulation of maternal aggression. Finally, chronic administration of AVP to LAB can enhance maternal care and aggression to levels more similar to HAB rats (Bosch & Neumann, 2008). Activation of V1aR during lactation, but not other stages, may have an important role in modulating sensory processing in order to coordinate both motivation to engage in maternal care and aggressive defense in response to intruders. An i.c.v. injection of V1aR antagonist prior to resident intruder testing in lactating dams increases activation as measured by BOLD responses in areas associated with somatosensory processing (important for social investigation), while it reduces activation in areas associated with aggression (Caffrey, Nephew, & Febo, 2010). Importantly, the V1b receptor for AVP has been shown to also affect maternal behavior, but this receptor doesn't seem to modulate maternal aggression (Bayerl, Klampfl, & Bosch, 2014).

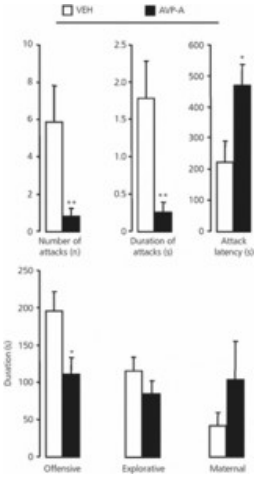

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Figure 7. Effect of blocking V1aR within BNST on maternal aggression in lactating rats. Maternal aggression was tested after bilateral local injections of vehicle (VEH) or of a V1aR antagonist (AVP-A) during the 10-min maternal defense test.

Reprinted with permission from "Maternal Behaviour Is Associated With Vasopressin Release in the Medial Preoptic Area and Bed Nucleus of the Stria Terminalis in the Rat," by O. J. Bosch, J. Pförtsch, D. I. Beiderbeck, R. Landgraf, and I. D. Neumann, 2010, *Journal of Neuroendocrinology*, *22*, 420–429.

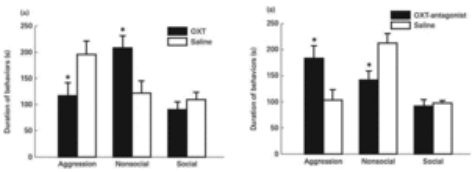

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Figure 8. (A) Duration of aggression, social, or non-social behaviors (mean ± *SEM*) if resident hamsters tested for 7 min in their home cage with a non-aggressive intruder (A) immediately after a microinjection of OT in the medial preoptic area (MPOA). (B) 30 min after a microinjection of OT antagonist in MPOA.

Reprinted with permission from "Oxytocin Inhibits Aggression in Female Syrian Hamsters," by A. C. Harmon, K. L. Huhman, T. O. Moore, and H. E. Albers, 2002, *Journal of Neuroendocrinology*, *14*, 963–969.

While V1aR activation robustly promotes maternal aggression, very different results have been observed for non-lactating females. Microinjections of Manning compound into the AH increased territorial aggression in non-lactating female hamsters (Gutzler, Karom, Erwin, & Albers, 2010). This is also surprising given that V1aR antagonist injections in AH have the exact opposite effect in male hamsters (Caldwell & Albers, 2004; Ferris & Potegal, 1988). However, similar sex-specific effects of AVP signaling within AH have been reported in hamsters. For example, injections of AVP in AH results in increased flank marking behavior in males tested in the presence or absence of another males (Ferris, Melloni, Koppel, Perry, Fuller, & Delville, 1997), while in females the same treatment increases this behavior in a non-social context (Hennessey, Huhman, & Albers, 1994). Interestingly, AVP microinjections into the anterior hypothalamus had no effect in a social context (Gutzler et al., 2010). Context-dependent effects of V1aR have also been observed in California mice. Infusion of V1aR antagonist into the BNSTmv had anxiogenic effects in both social and nonsocial contexts for males, whereas for females, V1aR antagonist had anxiogenic effects in social contexts only (Duque-Wilckens et al., 2016). Currently, the mechanisms underlying sex differences in V1aR function are unknown. One possibility is that there may be sex differences in the cell types expressing V1aR receptor. So far, most analyses of V1aR expression have been conducted with autoradiography, which does not allow for cell type analysis. The determination of which cells express V1aR in the anterior hypothalamus and BNST could provide important insights on how activation of this receptor can have such different behavioral effects in males and females.

Oxytocin

The effects of OT on aggression are highly dependent on context in males and females. For example, acute administration of OT either i.c.v (Calcagnoli, de Boer, Althaus, den Boer, & Koolhaas, 2013) or intranasally (Calcagnoli, Kreutzmann, de Boer, Althaus, & Koolhaas, 2015A) reduces intermale territorial aggression in rats. Male aggression in resident intruder tests is also enhanced after i.c.v. injection of OTR antagonist (Calcagnoli et al., 2013). These effects of OT appear to be at least partly mediated by CeA, as site-specific infusions of OT are sufficient to reduce offensive aggression (Calcagnoli, Stubbendorff, Meyer, de Boer, Althaus, & Koolhaas, 2015B). On the other side, mice with a homozygous mutation in the OT gene show significantly reduced territorial aggression (DeVries, Young, & Nelson, 1997), although in this experiment it is not possible to know if the effects of OT are organizational or activational.

In females, OT appears mostly to inhibit aggression, but only if they are not lactating. For example, female OT knockout mice are more aggressive towards other females than wild type females (Ragnauth et al., 2005). Once again it's not clear whether the effects of OT are developmental or activational. In female Syrian hamsters, site-specific injections of OT into MPOA or AH significantly reduce aggression, while the injection of OTR antagonist had the opposite effect (Figure 8) (Harmon, Huhman, Moore, & Albers, 2002). Finally, i.c.v. injections of OT inhibited aggression directed toward unrelated young in female mice, and this effect was independent of ovarian hormones or prolactin (McCarthy, 1990). In contrast, OT mostly enhances maternal aggression. For example,

correlational studies have found that OT is positively associated with maternal aggression in rats (Bosch et al., 2005), and direct infusion of OT into CeA or PVN increases maternal aggression in this species Bosch et al., 2005). Similarly, infusion of OT into CeA increases aggression in lactating hamsters (Ferris, Foote, Meltser, Plenby, Smith, & Insel, 1992). A previous study reported that infusion of antisense oligonucleotides designed to inhibit OT in PVN increases maternal aggression (Giovenardi, Padoin, Cadore, & Lucion, 1998). However, experimental knockdown of OT was not verified in these rats, so it is unclear whether the OT expression was inhibited as expected. Intriguingly, it has been suggested that effects of OT on maternal aggression are mediated by V1aR, as i.c.v. infusion of OTR antagonist has no effects on maternal aggression (Neumann, Tosch, Ohl, Torner, & Krömer, 2001) and OTR antagonist infused into CeA increased maternal aggression (Lubin, Elliott, Black, & Johns, 2003). The apparent contradictory effects of OT on female aggression might be explained partly by the recent hypothesis that a primary role of OT is to signal the salience of social stimuli regardless of the valence (Shamay-Tsoory & Abu-Akel, 2016). Thus, one possibility is that OT enhances the salience of the intruders' behaviors. In the case of non-lactating females, OT could be reducing aggression towards opponents that are not a direct threat to survival (other females, infants), which would be beneficial considering the potential costs of engaging in aggressive encounters. On the other side, since infanticide by males is a direct threat to fitness, OT would promote aggressive behavior by enhancing salience of the male intruders' behavior. Further investigation is needed to better understand the role of OT in different social contexts.

Conclusions

Although historically male and female aggression has been studied independently, much of the neural circuitry controlling aggression is shared between sexes. Furthermore, recent studies are revealing that brain circuits originally viewed as exerting sex-specific control of aggression are really context dependent. For example, the MPOA has usually been considered to be less important for male aggression. However, recent data suggest the MPOA may be important for parental aggression in both males and females. Sex differences arise more frequently in the neurochemical mechanisms used by brain circuits. This is likely a result of sex-specific adaptations to different social and reproductive roles. For example, costs associated with high levels of certain hormones like T are generally higher in females. This may have resulted in females developing alternatives to high T to facilitate the expression of aggressive behaviors. Sex differences in the neurochemical control of aggression can also occur beyond the level of the receptor. We see this with V1aR, which has pro-aggressive effects in males, but anti-aggressive effects in virgin females. Understanding the mechanisms through which these sex-specific effects are generated will be a challenging but important task for future research.

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