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Dynamic modulation of sociality and aggression: an examination of plasticity within endocrine and neuroendocrine systems

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Endocrine and neuroendocrine systems are key mediators of behavioural plasticity and allow for the ability to shift social behaviour across dynamic contexts. These systems operate across timescales, modulating both rapid responses to environmental changes and developmental plasticity in behavioural phenotypes. Thus, not only do endocrine systems mediate behavioural plasticity, but also the systems themselves exhibit plasticity in functional capabilities. This flexibility at both the mechanistic and behavioural levels can be crucial for reproduction and survival. Here, we discuss how plasticity in nonapeptide and steroid actions may influence the expression of, and allow rapid shifts between, sociality and aggression—behavioural shifts that can be particularly important for social interactions. Recent findings of overlap in the mechanisms that modulate social and aggressive behaviour suggest the potential for a mechanistic continuum between these behaviours. We briefly discuss the potential for a sociality–aggression continuum and novel techniques that will enable probing of the functional connectivity of social behaviours. From an evolutionary perspective, we suggest that plasticity in endocrine and neuroendocrine mechanisms of behaviour may be important targets of selection, and discuss the conditions under which we would predict selection to have resulted in differences in endocrine plasticity across species that differ in social organization.

This article is part of the themed issue 'Physiological determinants of social behaviour in animals'.

1. Introduction

To successfully navigate dynamic environments, animals must exhibit behavioural plasticity [1–3]. For example, for individuals that occupy low social ranks, exhibiting aggression towards conspecifics can be costly. But when faced with an opportunity to rise in social rank, a rapid increase in conspecific aggression could be highly adaptive, enabling individuals to defeat social rivals. The ability to rapidly shift social behaviours across dynamic contexts may be particularly important for organisms that live in social groups, because of the heightened frequency, and sometimes fitness consequences, of social interactions. Rapid flexibility (also referred to as activational plasticity [2]) in behaviour and its physiological mediators occurs when individuals adjust their phenotype to match the current context—for example, by responding more aggressively to intruders that present a greater threat. Activational plasticity can also influence the behavioural and mechanistic changes that occur across seasons or between life-history stages. Long-term changes in behaviour can result from developmental plasticity, in which early life experience has permanent effects on the mechanisms of behaviour. Developmental behavioural plasticity may enable organisms to match their behavioural phenotype to the

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environments they are likely to encounter in their lifetimes, in order to capitalize on dynamic environmental and social niches [2].

Plasticity in behaviour and its mediators arises from complex, reciprocal interactions of genotype, phenotype and the environment [4,5]. Key players in these interactions are endocrine and neuroendocrine systems, which can mediate rapid changes in behaviour, as well as trigger long-term developmental plasticity. Here, we address the role of endocrine (including neuroendocrine) systems in mediating plasticity in two classes of behaviour: sociality (defined as affiliative interactions with conspecifics) and aggression (agonistic interactions with conspecifics). Integrative molecular and evolutionary approaches that consider functioning within the whole organism (i.e. endocrine actions in the brain and body) hold high potential for elucidating the mechanisms of plasticity in social behaviour. While there are numerous mediators of behavioural plasticity, we focus on the nonapeptides and steroid hormones, both of which are widely known for their crucial involvement in the modulation of social and aggressive behaviours [6–9]. We explore the potential for a direct mechanistic link between sociality and aggressive behaviours, and highlight recent advancements in technology that show enormous promise for elucidating the mechanisms of rapid behavioural plasticity, and determining the presence of a sociality–aggression continuum. Lastly, we address whether selection operating on the plasticity of (neuro)endocrine mechanisms influences the ability to form and maintain social bonds, shaping social organization across species.

2. Nonapeptides: neuroendocrine mechanisms of social plasticity

Encountering a variety of stimuli in the social environment differentially activates neural circuits that produce complex behavioural outputs, such as social investigation, affiliation, sexual behaviour and aggression. Although there are several neural systems that modulate social behaviour, one of the most prominent neuroendocrine modulators of sociality and aggression is the nonapeptide system. This dynamic system works in concert with other limbic systems, and systems outside of the brain and spinal cord (i.e. the periphery) to produce flexible, complex suites of behaviour [1]. In this section, we explore the neuromodulatory capabilities of the nonapeptides and their diverse role in mediating behaviour across timescales.

(a) Neuromodulatory capabilities of the nonapeptides

Central nonapeptide circuits play phylogenetically widespread roles in the modulation of sociality, aggression and stress responses [7,10,11]. These evolutionarily conserved circuits arise primarily from magnocellular and parvocellular neurons in the preoptic area and hypothalamus that produce: (i) arginine vasotocin (Ile^3-VP) in non-mammalian vertebrates or arginine vasopressin (Arg^8-VP) in mammals, and (ii) isotocin (Ser^4, Ile^8-OT) in bony fishes; mesotocin (Ile^8-OT) in lobe-finned fishes (e.g. lungfish), most amphibians, reptiles, birds and some marsupials; or oxytocin (Leu^8-OT) in most eutherian mammals [12–14]. In addition to these cell groups, virtually all tetrapods exhibit accessory cell groups in the medial extended amygdala and extrahypothalamic regions [7,15]. While the nomenclature is taxon-specific, because all jawed vertebrates express a single form of VP and a single form of OT, for ease of communication, we will here refer to all forms of the nonapeptides as VP and OT [16].

The nonapeptides exhibit complex signalling properties, including peptide release from axon terminals, dendrites and soma [17,18]. Volumetric release from dendrites and soma allows for the release of vast quantities of peptides into peripheral circulation via axon terminals in the posterior pituitary, and independently, for the release of large quantities of peptide into the brain, which can modulate very distal sites [19–21]. Thus, nonapeptides can modulate different brain structures in a multimodal manner—through a targeted axonal, fast and focal manner as well as in a diffusive, slow and global manner [22]. This signalling diversity has allowed the nonapeptide system to be adapted for numerous physiological and behavioural functions. Furthermore, the distinct targeted and diffusive modes of action result in capabilities for very different types of dynamic functional control, such as executing rapid responses to external stimuli as well as for maintaining stable characteristics of behavioural phenotype. It is this dynamic nature of the non-apeptide system that solidifies its role as a key player in the modulation of various types of complex social behaviours, and has allowed it to both exhibit highly conserved functions and remarkable plasticity over evolutionary time.

(b) Neuromodulatory patterning: the potential for a sociality–aggression continuum

Sociality and aggression are two distinctly different, yet complimentary behaviours that may in large part be modulated by the same neural systems [6,23]. Extensive anatomical studies in estrildid finches that vary in social structure highlight a mechanistic link between these two behaviours, such that nonapeptide immunoreactive neuron distribution and receptor density reflect differences in territoriality and sociality. For example, territorial species have fewer VP cells in the bed nucleus of the stria terminalis (BSTm; part of the extended medial amygdala) and less OT and VP receptor binding sites in the lateral septum (LS) compared to gregarious species [24,25]. In addition, experiments that directly manipulate peptide production demonstrate a functional link between sociality and aggression. For example, when VP production in the BSTm is knocked down via antisense oligonucleotides, both sociality and aggression are affected in male zebra finches, with antisense males exhibiting an increase in aggression and decrease in gregariousness [26]. When the VP neuronal population of the paraventricular nucleus of the hypothalamus (PVN) is knocked down via antisense, again, both sociality and aggression are impacted in males and females, although in a sex-specific manner [27]. Furthermore, in emberizid sparrows that flock (i.e. are social) in the winter and are territorial in the spring/summer (the breeding season), there is a shift in neurochemical profiles for VP, OT, corticotropin releasing hormone (CRH) and vasoactive intestinal polypeptide. Together, these findings raise an interesting question—is sociality an active suppression of the mechanisms that allow for aggression and territoriality (or vice versa)?

The expression of these two behaviours may be inextricably linked, although not necessarily in a simplistic or straightforward manner. It is also important to consider the

interplay between OT and VP, particularly given the binding promiscuity observed within the non-apeptide system [28,29]. Although OT has not been extensively studied specifically in relation to aggression, it is well known for having anxiolytic properties and strongly promoting different types of affiliative behaviours [30]. While it is crucial to understand distinct properties and functions of VP and OT separately, insight into the mechanisms underlying complex social behaviour may best be gained from studying the nonapeptide system as one whole functioning unit that interacts with other neural systems and peripheral tissues and feedback mechanisms to produce dynamic and flexible behaviour. Neuromodulators such as VP and OT probably exert effects via coordinated release in multiple brain regions, effectively modifying functional connectivity across the numerous nodes of behavioural regulatory networks [1,16].

(c) Timing: stable characteristics versus rapid responses

In an ever-changing environment, there are often appropriate times and places to behave either socially or aggressively. In response to this, the brain has evolved neural systems that are capable of maintaining stable traits and/or being plastic. The nonapeptide system is unique in that its multimodal manner of functioning has been adapted to both maintain stable behavioural characteristics within an organism's life and also allow an organism to rapidly exhibit appropriate, context-specific behavioural responses to social changes in the environment.

VP and OT have been extensively examined in relation to stable characteristics associated with distinct behavioural phenotypes and species differences. As mentioned above, non-apeptide immunoreactive neuron distribution and receptor density reflect differences in territoriality and sociality in estrildid finches, such that more social species have significantly more VP neurons in the BSTm and greater VP and OT receptor densities in the LS (a primary projection site of BSTm VP neurons) [24,25]. Similar comparative studies demonstrate that the BST-LS VP anatomical difference observed in estrildid finches also distinguishes mating systems in voles, such that the promiscuous meadow vole exhibits more BST VP cells and greater LS VP receptor densities compared with the monogamous prairie vole [31–33]. In addition, developmental studies show that variation in the early life social environment impacts social behavioural phenotype and nonapeptide neural profiles that persist into adulthood [34,35] and even transgenerationally [36].

Nonapeptides not only maintain long-term, stable characteristics, but VP and OT also act quickly to induce rapid behavioural changes. There are numerous studies that demonstrate acute behavioural effects of nonapeptides by employing methods of peripheral pharmacological manipulations of VP–OT systems. Such studies have investigated the effects of exogenous peptide or peptide receptor antagonists on courtship, social hierarchy, aggression and cooperative behaviour in various species of frogs, fishes and mammals [37–45]. In many of these studies, differences in social behaviour are observed within minutes of peripheral administration of peptide or an antagonist, with effects lasting from 30 min to 2 h (e.g. [41,42]). Similarly, central manipulations of VP–OT systems demonstrate that antagonism of peptide receptors influences pair bonding behaviour within 1.5 h of administration [46,47].

Of particular importance for this review are studies that show rapid changes in non-apeptide levels in the brain in the absence of pharmacological manipulation. A 30 min agonistic social interaction is enough time to elicit differences in VP, and to a lesser extent OT, levels throughout the brain (assessed via high performance liquid chromatography) in zebrafish [48]. Similarly, changes in neuropeptide mRNA levels are observed with only 1 h of acute social isolation, as well as following chronic, repeated social isolation [49,50]. Furthermore, microdialysis studies enable monitoring of local extracellular concentrations of peptide within specific brain regions, and can provide information about potential signal function during behaviour [11]. For example, microdialysis revealed a significant elevation in septal VP release measured in a 30 min interval during aggressive inter-male interactions in rats [51], and a significant increase in OT release in the PVN and central amygdala during a 10 min maternal aggression test [52]. These studies not only serve as an example of rapid nonapeptide neural responses that correlate with rapid changes in behaviour, but also show that similar mechanisms within the brain (e.g. same neuropeptides and brain regions) underlie social and aggressive behaviours.

The nonapeptide system has long been recognized as being plastic given that: (i) peptide release is multimodal and can occur on different timescales, and (ii) nonapeptide anatomy and functioning can change over evolutionary time [53]. However, the mechanisms of this functional plasticity have been largely unexplored, primarily owing to limitations in technology. With recent advances in genetic engineering (e.g. optogenetics), we are beginning to examine the properties of rapid functioning within the nonapeptide system. An elegant experiment optogenetically manipulating hypothalamic axonal OT release demonstrates that behavioural changes can be seen in as little as 2 s, and on average 20 s, after stimulation of OT axons [54]. In this experiment, the authors demonstrated that local blue-light-induced endogenous release of OT robustly decreases fear response in rats [54]. This experiment is one of the first to truly demonstrate the rapid neuromodulatory capabilities of the non-apeptide system. Similar experiments evoking hypothalamic OT neuronal release demonstrate rapid suppression of nociception and the promotion of analgesia in rats [55]. The technology of opto- and chemogenetics is revolutionizing the field of neuroscience [56,57] and will enhance our understanding of functional connectivity within the nonapeptide system by allowing for the manipulation of specific neural pathways under tight temporal control in order to determine their role in context-specific behaviour. Although, to date, there are few opto- or chemo-genetic studies directly investigating non-apeptide modulation of sociality and aggression (particularly in non-model organisms [58]), several laboratories are beginning to use such technology to examine aspects of social behaviour (e.g. [59]). Unfortunately, optogenetic technology is limited to laboratory studies; however, advances in chemogenetics can allow for manipulative neural studies in common garden experiments. For example, designer receptors exclusively activated by designer drugs (DREADDs) can be site-specifically infused into the brain without having permanent hardware affixed to the animal, allowing for activation or deactivation of specific cells with an acute peripheral injection of clozapine N-oxide (selectively activates DREADDs) [57]. Such technology not only allows for manipulative neural

experiments to be conducted in freely behaving animals, but also allows researchers to probe the mechanisms underlying rapid changes in behaviour in a manner that is impermanent (i.e. DREADD activation lasts for approx. 2–6 h). Similarly, although only appropriate for laboratory studies, optogenetics allows for neural manipulations on an even tighter temporal scale such that neural activity can be returned to baseline in seconds [57,60]. The ability to stimulate and inhibit non-apeptide circuits (at will) vastly expands the types of experiments we can conduct in order to examine functional plasticity within the nonapeptide system and examine how this neural functional plasticity relates to rapid changes in behaviour. While the nonapeptide system is a key player in the neuromodulation of rapid social responses, this system works in concert with several peripheral endocrine mechanisms, which we will explore next.

3. Steroid hormones and social plasticity

In addition to the rapid effects of nonapeptides on aggression and sociality, slower acting and longer-lasting hormonal responses also play an important role in mediating and priming social behaviour. While many hormones can influence social behaviours, steroid hormones are particularly integrative and far-reaching coordinators of phenotypic plasticity. These chemical messengers are both peripherally derived and produced in the brain. They operate both through classic genomic routes and through rapid non-genomic routes to effect changes in gene expression, cellular function, and neuronal activity, in ways that influence the expression of social and aggressive behaviour. Steroid hormones can also function as central regulators of nonapeptides and other neuroendocrine signals [61,62], and act as behavioural neuromodulators [9]. Because of the multiple pathways by which steroid hormone systems can produce an effect, they operate over a broad range of timescales. Early products of the activation of steroid hormone axes can produce behavioural effects within seconds [63,64]. Once released, circulating steroids have a half-life measured in minutes, but their effects on phenotype can last substantially longer. Under some conditions steroid hormones can produce permanent and even transgenerational effects on social behaviour, apparently through epigenetic mechanisms [65,66].

(a) Glucocorticoids: rapid enhancement of perception of and responses to social information

Both the rapid response to social stimuli, and the formation and maintenance of social bonds, can be strongly influenced by glucocorticoid hormones (e.g. corticosterone and cortisol). At low levels, glucocorticoids mediate metabolic function. These hormones also rise in response to social and other challenges, functioning as central coordinators of the stress response. Glucocorticoids, and the peptide hormones that affect their release—including VP—can rapidly affect affiliative behaviour and aggression. The direction of effect often depends on the duration of exposure. While acute increases in glucocorticoids can promote aggression [67], chronic glucocorticoid exposure tends to suppress aggression and increase social avoidance [68,69]. Chronically elevated glucocorticoids can also impair the formation and maintenance of social bonds by reducing the expression of affiliative behaviours central to these bonds [68,70]. Differences in the behavioural effects of glucocorticoids across timescales of exposure probably stem, at least in part, from the cumulative effects of glucocorticoid exposure on other signalling systems. For example, while acute glucocorticoid increases induce rapid changes in aggression through both non-genomic and genomic mechanisms [67,71], chronic glucocorticoid exposure increases social avoidance by facilitating glucocorticoid receptor activation in dopaminoceptive neurons [67].

Because glucocorticoids are highly sensitive to social context (e.g. [72–74]), individuals that inhabit different social environments, or occupy different positions within a social hierarchy, often show persistent differences in glucocorticoid physiology [75–77]. The immediate social environment also influences how stressors are perceived. Social animals from titi monkeys to great tits produce a weaker hormonal response to stressors in the presence of a conspecific than when experiencing the same stimulus alone, a phenomenon termed 'social buffering' [78–80]. Pre-existing social bonds can also influence the response to social isolation and to stressors experienced alone [77,81]. The pervasive influence of social context on the response to stressors—which may be mediated in part through socially mediated changes in nonapeptides [82]—suggests that variation in social integration may impact the ability of populations to withstand environmental challenges. Tightly integrated social groups—and their members with the strongest social bonds—may be more resistant to moderate stressors, or those that influence a small subset of the population at any given time, through social buffering. However, highly integrated groups may also be more vulnerable to challenges that influence group dynamics.

Glucocorticoids also influence learning and memory, and are intimately involved in matching the physiological and emotional states of social group members [83,84]. Upon reunion with partners that were subjected to a stressor out of sight, prairie voles rapidly increase circulating corticosterone to levels highly correlated with those of their stressed mate, and increase OT-dependent consolation behaviours [85]. In humans, this kind of emotional contagion is far more prevalent among friends and family members than between strangers. In a clever series of experiments, Martin *et al*. [84] demonstrated that this effect is glucocorticoid mediated. In both humans and mice, when glucocorticoid synthesis and binding are pharmacologically blocked, emotional contagion occurs even between strangers [83]. Through their effects on learning and memory, glucocorticoids can also influence how individuals perceive and respond to social information [83]. The glucocorticoid mediation of spatial and declarative memory has been widely demonstrated; recent evidence suggests that these hormones are also important facilitators of social learning, and mediate the reconsolidation and recall of social memories [86–88].

(b) Glucocorticoids: developmental plasticity and social grouping

Permanent changes in social behaviour are induced by exposure to glucocorticoids during development—a mechanism that can serve to match offspring phenotype to dynamic environments. Chickens that hatch from corticosterone-treated eggs are more aggressive as juveniles, differ in their

stress physiology (circulating glucocorticoid concentrations, receptor protein expression, and methylation of hypothalamic gene promoters), and express less arginine vasotocin than controls [89]. Intriguingly, developmental glucocorticoid exposure seems to alter social grouping by influencing the degree to which individuals associate with strangers versus social group members. Prairie voles exposed to glucocorticoids during the early postnatal period prefer to associate with unrelated individuals than with siblings, and show less alloparental behaviour than controls [90]. Similarly, zebra finches treated with corticosterone during development later have weaker associations with their parents and forage with a greater number of conspecifics, occupying more central positions in social networks [91]. Corticosterone-treated offspring also change their social learning strategy: while control finches learn social foraging tasks primarily from their parents, individuals exposed to corticosterone during development learn these same tasks almost exclusively from unrelated individuals [92]. Recent research has revealed that glucocorticoid-mediated effects on social behaviour and gene expression can persist for multiple generations after stressor exposure [65,93]. While the specific mechanisms by which transient exposure to glucocorticoids produces such far-reaching effects are not well understood, they are likely to include organizational and epigenetic effects on monoamines and nonapeptides [94,95].

The strength and persistence of glucocorticoid-mediated phenotypic effects suggests that these hormones may play a key role in increasing the likelihood or degree of social grouping, and the expression of other social behaviours. Developmental glucocorticoid exposure may therefore serve as a mechanism inducing population-level changes in the degree of sociality. If so, in populations and environments where tighter social bonds between group members are favoured, we predict that mothers will show a weaker glucocorticoid stress response during gestation and offspring care, and/or offspring will be more resistant to the effects of glucocorticoid exposure during development. By contrast, in environments where social bonds are weaker, and individuals benefit from interacting with many unrelated individuals, we predict that parents will show stronger stress responses, and/or offspring will be more susceptible to developmental glucocorticoid exposure.

(c) Androgens: priming future flexibility

Androgens have long been studied as mediators of social aggression, and of behavioural and morphological signals related to mate attraction, particularly in males. In many species, social challenges increase circulating androgen levels [96,97]. Non-agonistic interactions, including those with fertile females, and even observing the social interactions of others, can also stimulate an androgen response [98,99]. Because circulating androgens do not rise for many minutes after the activation of the hypothalamic-pituitary-gonadal axis, socially mediated androgen responses often occur too late to influence the outcome of the social event that triggered them. However, androgen responses may be particularly important in preparing organisms for the social environment they are likely to face in the near future [8]. In many taxonomically diverse species, prevailing in social contests triggers an androgen response that elevates aggression during subsequent social interactions, and this heightened aggression increases the likelihood of winning future contests [100–102]. Social interactions can also prime future responses by altering androgen receptor density and gene expression [103,104], and the androgen modulation of VP receptors in the brain [105,106].

It is important to note that the sensitivity of androgens to social challenges and contest outcomes, and the androgen mediation of social aggression, vary enormously across species. Some species show no androgen response to social challenges, and no relationship between circulating androgens and aggression [107,108]. Within species, the androgen response to social challenges and the androgen mediation of social behaviours can also differ across life-history stages and between the sexes [109,110]. Furthermore, an individual's androgen response to a given stimulus can change based on social context and prior experience [107,111]. For example, while social isolation decreases androgen formation in territorial male cichlids, this same stimulus tends to increase androgens in non-territorial males [112].

The rapid effects of androgens on aggression and affiliation may occur through a variety of pathways, including direct genomic or non-genomic effects, the aromatization of androgens to oestradiol, and androgen-mediated changes in nonapeptide and monoamine activity [111,113]. Androgen exposure during development also has widespread organizational effects on social behaviour. A series of classic studies found that female mice which develop between two male siblings—and thus are exposed to more androgens *in utero*—are more aggressive in adulthood, and more likely to compete successfully for a limited food resource [114,115]. How androgens impact social behaviour appears to be strongly dependent on the timing of exposure. For example, familial affiliation in voles is increased by prenatal testosterone exposure, but decreased by postnatal exposure [90]. The widespread developmental effects of androgen exposure on aggression and sociality may be mediated, at least in part, by changes in the androgen sensitivity of nonapeptide systems [15,116].

4. Discussion

(a) Elucidating a sociality–aggression continuum

Is there a mechanistic sociality–aggression continuum? Data that support the potential for such a continuum largely come from comparative studies of anatomy, such that differences in closely related species that vary in social behaviour are distinguished by distinct anatomical profiles within the same neuroendocrine systems [53,117,118]. For example, in estrildid finches, affiliative species have more nonapeptide cells and greater nonapeptide receptor densities in the BST-LS circuitry, which promotes affiliative behaviour [16], compared to territorial species [53]. Similarly, whole-brain nonapeptide gene expression differences distinguish social and non-social species of African cichlids [118]. Although comparisons between species that vary in grouping behaviour suggest that social and aggressive behaviour are modulated via differential activation of the same circuitry, studies within individuals are necessary to comprehensively reveal whether a mechanistic continuum exists for the modulation of social and aggressive behaviour. Perhaps the most informative way to approach this question is to determine whether it is possible to significantly manipulate sociality without affecting

aggression. As discussed earlier, direct manipulation of nonapeptide circuitry that promotes affiliative behaviour in finches also affects aggressive behaviour, such that knockdown of VP production originating from the PVN and BST cell groups inhibits grouping behaviour while concomitantly increasing aspects of aggression [26,27]. Such findings suggest a strong mechanistic link between these dichotomous behavioural outputs. However, sociality and aggression are complex behaviours, and constant manipulation/alteration of one cell group does not provide enough information to definitively conclude that a continuum exists. Further studies targeting the neural plasticity of affiliative behaviour within individuals are required to elucidate whether the absence of affiliative behaviour indicates an increased presence of aggressive behaviour, or vice versa. Recent advances in technology, such as opto- and chemo-genetics, will enable researchers to probe the plasticity of social behaviour within a single individual because of the capability to maintain an animal at its baseline state and only temporarily manipulate the brain when desired. Such technology can allow for tight control over the manipulation of social behaviour, and thus, we may be able to determine whether or not it is possible to turn on (and subsequently off) affiliative behaviour. Because control over specific circuits can be activated and deactivated numerous times, it becomes possible to examine and re-examine behaviour under different contexts, allowing for close examination of the dynamic qualities of both affiliative and aggressive behaviours.

When considering a sociality–aggression continuum, it is also important to deconstruct behaviour. Is sociality simply an absence of aggressive behaviour? Probably not, given that manipulations which influence one of these types of behaviour does not necessarily alter the other in a straightforward manner. For example, experiments knocking down BST VP production decrease affiliative behaviour in zebra finches and increase aggression towards *same-sex* conspecifics, yet aggression toward *opposite sex* conspecifics is unaffected [26]. This reveals the complexity of sociality and aggression, and their underlying mechanisms. Yes, manipulation of social circuitry affected both affiliative and aggressive behaviours, but only under some social contexts. In addition, given the extraordinary diversity observed in grouping behaviour across species, it is important to consider that there must be multiple dimensions to a sociality–aggression continuum given that the possible dynamic range of behaviours can be quite different between species. For example, territorial species may exhibit aggressive behaviour towards most conspecifics, but in territorial species that exhibit monogamy and parental care (e.g. violet eared waxbills [119]), affiliative behaviour is still exhibited, yet only in specific contexts (in this example, affiliative behaviour is directed towards pairbond partners and offspring only). On the other hand, highly affiliative species (e.g. zebra finches) exhibit robust plasticity in affiliative behaviour, and can rapidly fluctuate from affiliating with both sexes (kin or non-kin, and in sexual or non-sexual contexts) and exhibiting short bouts of aggression in mate-competition or nest defence contexts [120]. Thus, in addition to base differences in the expression of sociality/territoriality, differences in the capabilities of behavioural plasticity need to be accounted for in a potential sociality–aggression continuum.

If a sociality–aggression continuum does indeed exist, it is probably based on a multidimensional, shifting scale of concerted activity across numerous endocrine systems, including the nonapeptide system, the mesolimbic dopamine circuit and steroid hormones (e.g. figure 1). Such a dynamic, multimodal continuum strongly resembles that of the proposed social behaviour network [122,123]; such a physiological regulatory network [124] would allow for shifts in patterns of activity in response to social context and phenotypic differences.

(b) An evolutionary approach to the mechanisms of behavioural plasticity

Neuroendocrine and endocrine mechanisms are clearly central to the expression of aggression and sociality. But do individual differences in the flexibility of endocrine responses influence the outcomes of social interactions? Does selection on endocrine plasticity influence the ability to form and maintain social bonds, and shape social organization across species? It is conceivable that in dynamic social groups, individuals with greater endocrine plasticity, or those better able to initiate or terminate a response, would be better able to match their behaviour to rapidly changing contexts [3,125]. But while a multitude of research has addressed the links between endocrine expression and social behaviour, surprisingly little is known about whether variation in the scope or temporal dynamics of endocrine responses influences social outcomes. Several recent studies have used a reaction norm approach to confirm the presence of individual differences in the scope and speed of steroid hormone responses [126–129]. None have, to our knowledge, attempted to link this variation with the outcomes of social interactions, or addressed whether there are consistent individual differences in the context-dependent flexibility of other components of endocrine systems. Such flexibility could be an important way of balancing the costs and altering the outcomes of endocrine responses across dynamic contexts. For example, as described above, the social environment can influence the endocrine sensitivity of specific neural tissues by changing receptor expression [35,103]; variation in receptor expression can also predict the behavioural response to social stimuli [130]. Individual differences in socially mediated plasticity in tissue sensitivity might therefore be an important mechanism influencing behavioural flexibility across changing social contexts.

Do endocrine responses, and their plasticity, show sufficient heritable variation upon which selection could act? Phenotypic variation in circulating steroid levels has a heritable component [131,132]. The often substantial among-individual variation in nonapeptide responses could also be directly heritable [133], or could be influenced by heritable variation in steroids or other neuroendocrine regulators. Whether the plasticity of endocrine systems is itself heritable is not known [3,134], but this possibility is reinforced by findings of heritable plasticity in behavioural traits [135,136].

If selection on the mechanisms of plasticity enables more effective responses to dynamic social contexts, we would predict that, across species, context-dependent flexibility in steroid and nonapeptide responses will be associated with differences in social organization. Specifically, we would predict particularly strong selection on the endocrine mechanisms of social flexibility when: (i) social upheaval is more frequent, and more dramatic, (ii) environmental resources are highly variable, and life-history stage or optimal group size changes rapidly and unpredictably, (iii) the ability to

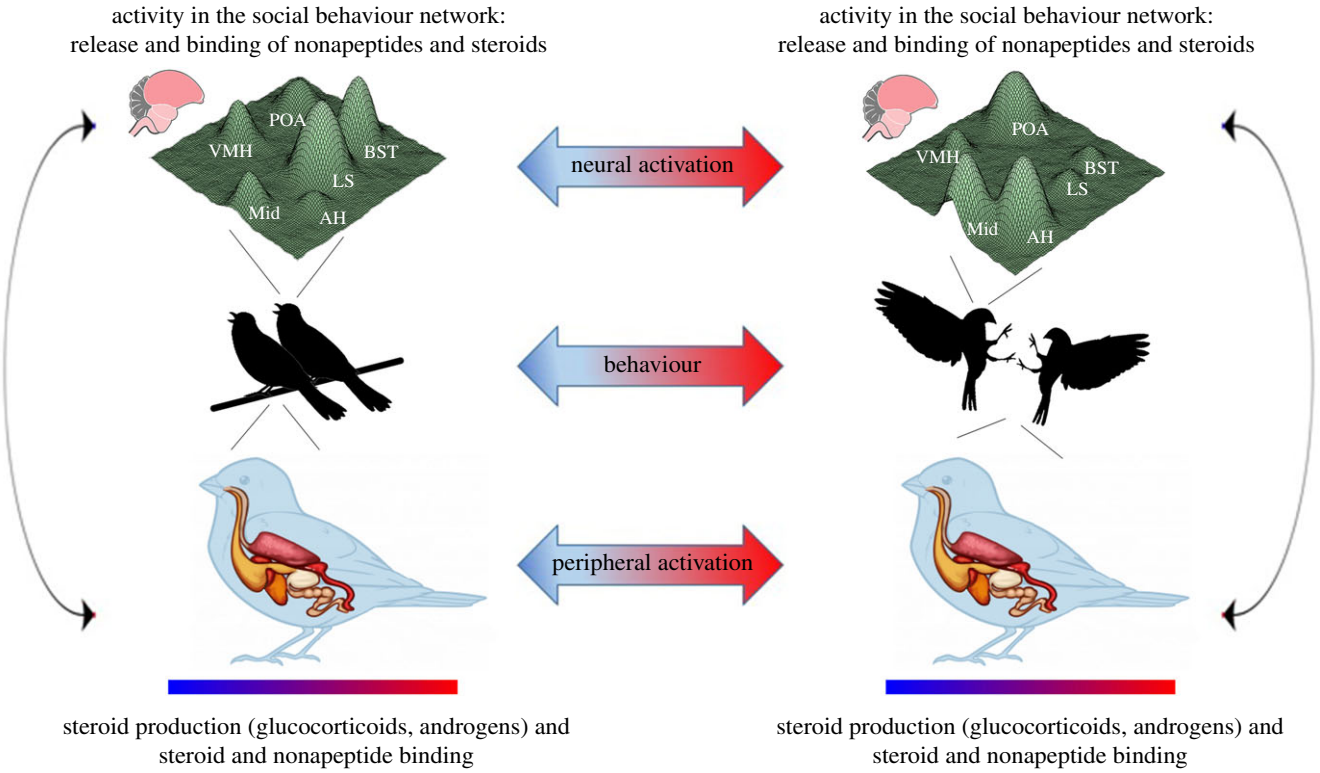

Figure 1. A simplified, hypothetical representation of a sociality–aggression continuum demonstrating reciprocal connections between peripheral and central endocrine systems. Different profiles of circulating steroid hormones and differential activation across neural nodes in the social behaviour network would reflect behavioural differences observed between an individual that is exhibiting affiliative behaviour and aggressive behaviour. Note that other endocrine systems are involved, however, for ease of visualization, here we only consider steroids and nonapeptides. We also only show primary nodes within the social behaviour network, which contains sites of steroid and nonapeptide production. In this hypothetical example, sociality is reflected by greater activity in the BST and LS (brain regions that promote sociality [16]) and lower levels of circulating testosterone, whereas aggression is reflected by lower activity in the BST and LS, greater activity in the AH (a brain region that promotes aggression [6,121]), and higher levels of circulating testosterone [111]. Thus, shifts in social behaviour are reflected by shifts in activity of the same endocrine systems. AH, anterior hypothalamus; BST, bed nucleus of the stria terminalis; LS, lateral septum; POA, preoptic area; VMH, ventromedial hypothalamus; Mid, midbrain. Bird anatomy cartoon from Dragoart.

alter social position is temporally constrained and has major fitness consequences, or (iv) it is adaptive to match the social behaviour of mates or other conspecifics. Tests of these and other hypotheses about the evolution of the mechanisms of flexible social behaviour could be a fruitful direction for future research.

5. Concluding remarks

Both the nonapeptides and steroid hormones play important roles in mediating rapid plasticity in social behaviour, as well as in maintaining stable behavioural phenotypes. VP–OT function and anatomy not only differentiate social phenotypes across species, but VP–OT can affect behaviour on varying timescales ranging from 2 s to a few hours. This functional flexibility makes the nonapeptide system temporally plastic and evolutionarily labile, and therefore, an excellent neuroendocrine system for the study of the evolution of rapid social plasticity. Similarly, steroid hormones are highly sensitive to social context and play a particularly important role in mediating and priming social behaviours, and in modulating the perception of and response to related challenges. Developmental steroid exposure can also have widespread organizational effects on social behaviour (including through epigenetic mechanisms) that may induce lifelong changes in nonapeptide responses and other endocrine systems. These systems all play a critical role in the expression of affiliative or aggressive behaviour during social encounters. As we note, the substantial overlap in the mechanisms that modulate these seemingly dichotomous behaviours suggests the potential for a mechanistic sociality–aggression continuum. Reciprocal interactions between nonapeptides and steroids mediate complex suites of behaviour, both through their dynamic effects on temporary behavioural outputs and by producing lasting changes (sometimes transgenerational) in social behavioural phenotype. Regardless of whether a sociality–aggression continuum exists, there remains individual and species variation in the plasticity of the mechanisms underlying social behaviour. We hypothesize that selection operating on social plasticity, and its mechanisms, will result in differences in endocrine plasticity across species that differ in social organization.

In order to truly understand the mechanisms underlying social and aggressive behaviour, it is crucial to consider functioning within the whole organism—peripheral and central mechanisms; the dynamic interplay between the body and the brain. Owing to feasibility issues, studies often focus on one or the other. Herein lies a major opportunity for collaborative research because studies that can examine both peripheral and central systems will lead to substantial advances in our understanding of rapid, whole-body responses to the environment. It is important to note that the mechanisms underlying plasticity in behaviour are enormously complex. In this review, we largely discuss neural and peripheral *actions* of steroids and nonapeptides.

However, when integrating 'brain and body', it will also be important to consider not only binding and actions, but also production and whether mechanistic actions are the result of peripheral- or neurally derived hormone. In addition to considering the whole organism, it is important to integrate multiple perspectives. Incorporating comparative evolutionary approaches will provide greater insight into the evolution of plasticity in social behaviour and its mechanisms [137]. Furthermore, novel tools in molecular neurogenetics that make it possible to observe activity in the brain of an individual in real time, at multiple time points, will allow us to delineate the extent to which the neural mechanisms underlying sociality are plastic, and can also elucidate whether a sociality–aggression continuum exists in the brain. We are at the beginning of an exciting new age in science, uninhibited by the obstacle of only being able to capture a snapshot of the brain from a single moment in time. The ability to probe neural circuitry, and explore neural plasticity, can be used in combination with measures and manipulations of endocrine activity to provide a comprehensive, whole-body understanding of behaviour.

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