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Review

The regulation of social recognition, social communication and aggression: Vasopressin in the social behavior neural network

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A R T I C L E I N F O

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A B S T R A C T

Neuropeptides in the arginine vasotocin/arginine vasopressin (AVT/AVP) family play a major role in the regulation of social behavior by their actions in the brain. In mammals, AVP is found within a circuit of reciprocally connected limbic structures that form the social behavior neural network. This review examines the role played by AVP within this network in controlling social processes that are critical for the formation and maintenance of social relationships: social recognition, social communication and aggression. Studies in a number of mammalian species indicate that AVP and AVP V1a receptors are ideally suited to regulate the expression of social processes because of their plasticity in response to factors that influence social behavior. The pattern of AVP innervation and V1a receptors across the social behavior neural network may determine the potential range and intensity of social responses that individuals display in different social situations. Although fundamental information on how social behavior is wired in the brain is still lacking, it is clear that different social behaviors can be influenced by the actions of AVP in the same region of the network and that AVP can act within multiple regions of this network to regulate the expression of individual social behaviors. The existing data suggest that AVP can influence social behavior by modulating the interpretation of sensory information, by influencing decision making and by triggering complex motor outputs.

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Contents

Introduction

A significant role for the arginine-vasotocin (AVT)/arginine-vasopressin (AVP) family of peptides in the regulation in many forms of social behavior has been well established (Caldwell et al., 2008a). This review will not attempt to comprehensively evaluate the role of AVT/AVP in controlling social behavior, but rather will provide a brief history of the early studies of AVP and social behavior and then focus primarily on evaluating the role of AVP in regulating social recognition, social communication, and aggression in mammalian species. This focus was chosen because social recognition, social communication, and aggression are essential elements in the formation and maintenance of social relationships, and the evidence indicates that AVP neural networks play a complex role in controlling each of these processes.

While the AVT/AVP family of peptides has remained structurally stable over a period of 450 million years in non-mammalian and mammalian tetrapods (Acher et al., 1999; Tessmar-Raible et al.,

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2007) the evolutionary origins of their role in the regulation of social behavior is poorly understood. Their remarkable sequence stability is thought to be the result of a common essential function, i.e., water homeostasis. Is it possible that AVT/AVP's role in social behavior evolved from its functions in water homeostasis? There is evidence that the effects of AVT on mating-related communication in female frogs are mediated by the accumulation of water and the resultant internal pressure (Diakow, 1978). Another mechanism by which AVT/AVP's regulation of water homeostasis could serve to influence social behavior is through their ability to increase urine osmolality thus concentrating the urine and the metabolic products contained within. As such, an animal's urine, particularly when concentrated, provides a great deal of information about the physiological state of that animal. It is therefore not surprising that many chemosensory cues carried in urine are essential for social communication in most tetrapods (Beynon and Hurst, 2004). A primordial role of AVT/AVP in olfactory communication may have eventually led to its involvement in the regulation of other forms of social behaviors as well.

Most of what we currently know about AVP and social behavior has come from studies that have been conducted since the early 1990s. However the origins of research on AVP's role in regulating social behavior began much earlier. The first suggestion for a possible role of AVP in social behavior came from studies in frogs from the 1940s in which injection of whole pituitary was found to induce female reproductive behavior (Noble and Aronson, 1942). The spawning reflex in fish was subsequently found to be induced by the peripheral injection of purified natural AVP (Wilhelmi et al., 1955) and synthetic AVT or AVP (Pickford and Strecker, 1977). Peripheral injection of AVT was also found to increase sexual behavior in birds (Kihlstrom and Danninge, 1972). In 1978, Diakow demonstrated that peripheral injections of AVT inhibit the release call emitted by female frogs during mating and in 1979 Moore and Zoeller reported that peripheral injections of AVT or AVP enhanced sexual behavior in newts (Diakow, 1978; Moore and Zoeller, 1979). As such, research on the roles of AVT/AVP in controlling social behavior began with studies in non-mammalian species with demonstrations that the peripheral administration of these peptides could modulate behaviors related to reproduction.

The first reports of behavioral effects of AVP in mammals came from studies of learning and memory. David de Wied and his coworkers found that destruction of the neurohypophysis resulted in the acceleration of conditioned avoidance and that AVP was responsible for these effects (de Wied, 1965; de Wied and Bohus, 1966; van Ree et al., 1978). These findings stimulated a great deal of research which examined whether AVP had direct effects on learning or whether these effects were mediated by the arousing, aversive or visceral properties of AVP (for a review see Dantzer and Bluthe, 1993). In the mid 1970s, central administration of AVP in mice was found to produce another behavioral phenomenon that was characterized by a "state of hyperactivity with excessive scratching and grooming behavior…[and at] high doses, squeaking, foraging, jumping and stereotyped head-to-tail circling" (Dunn et al., 1976; Rees et al., 1976; Meisenberg, 1981). In the late 1970s and early 1980s studies on the behavioral effects of AVP continued to focus primarily on learning and memory and this work was extended to paradigms involving learned submissive behavior (Roche and Leshner, 1979; Siegfried et al., 1984). However, during this time period there were no reports suggesting AVP involvement in the regulation of mammalian *social* behavior and, in fact, there was some evidence that AVP did not alter social behavior (Crawley et al., 1981). The first indication that AVP might play a significant role in regulating *social* behavior came in 1978 when Delanoy et al. reported that lysine vasopressin injected into the cerebroventricular system of hamsters produced "a dramatic bout of flank marking" (Delanoy et al., 1978). Flank marking is a form of scent marking used extensively by hamsters in social communication. Interestingly, this "report" was limited to a two-sentence paragraph in the discussion section of a paper that reported the behavioral effects of neurohypophyseal peptides in mice. The ability of AVP to induce high levels of flank marking in hamsters was "rediscovered" serendipitously some six years later (Ferris et al., 1984). Ferris et al., 1984, not only confirmed a role for AVP in the regulation of social behavior, they were also the first to identify a specific site within the brain where AVP acted to regulate a social behavior.

The "social behavior neural network"

Over the last 20 years it has become clear that there is a large degree of overlap in the neural circuitry controlling different social behaviors. As proposed by Newman (1999), data obtained using a wide variety of experimental approaches has led to the concept of a "social behavior neural network" composed of neural groups in the lateral septum, extended amygdala, midbrain, preoptic area, several hypothalamic nuclei as well as other limbic structures. As noted by Newman, "all of these areas fulfill several important criteria for nodes in a social behavior network. Each is reciprocally interconnected with all of the others, all are populated with neurons that contain gonadal hormone receptors, and each of these areas has been identified as an important site of regulation or activation in more than one social behavior." Further, there is a substantial body of evidence that this network is involved in controlling a wide range of social behaviors including both offensive and defensive aggression, social recognition/memory, sex behavior, parental behavior and social communication (Newman, 1999). According to this view, a specific social behavior is an emergent property of the pattern of activity across the network and not the result of turning a specific structure in the network "on" or "off". While this concept is attractive, the challenge is finding ways to critically test this overarching hypothesis. Newman suggests "A first step might be to actively look for evidence of multiple social-behavior functions in studies where identifiable cells groups (e.g., cells in a specific transmitter circuit) are being manipulated or monitored."

AVP in the "social behavior neural network"

AVP and AVP receptors are found throughout the social behavior neural network and, as would be expected if AVP circuitry is critical in the control of social behavior, the distribution of AVP and AVP receptors within specific sites of the network can vary depending on *species, hormonal state, sex* and *social experience*. There is considerable evidence that many of the effects of AVP on social behavior are mediated by the V1a AVP receptor subtype and this receptor is found extensively throughout the social behavior neural network. While this review will be limited to discussion of the effects of AVP that are mediated by V1a receptors there is recent evidence that the V1b AVP receptor subtype is also important for the regulation of social behavior (see Caldwell, this volume).

There are significant *species* differences in the distribution of V1a receptors and some of these differences appear to underlie species differences in social behavior (Insel et al., 1993). For example, non-monogomous male Montane voles have few V1a receptors in the ventral pallidum (VP) compared to monogamous Prairie voles, and induction of V1a receptors in the VP of Montane voles by viral vector gene transfer results in pair bonding similar to that seen in Prairie voles (Young and Wang, 2004). There are also species differences in the distribution of AVP in the social behavior neural network. For example, in most species so far studied there are prominent AVP projections from the bed nucleus of the stria terminalis (BNST) and the medial amygdala (MeA) to the lateral septum (LS), however in Syrian hamsters these projections appear to be completely absent (Dubois-Dauphin et al., 1990; Albers et al., 1991). The extent to which species differences in AVP circuitry contribute to species differences in social behavior is not known. It is clear however that