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Brief communication

Vasopressin facilitates play fighting in juvenile golden hamsters

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ABSTRACT

Vasopressin facilitates aggression in adult hamsters. Whether this neuropeptide has a similar role in play fighting remains unknown. The goal of the present study was to identify whether vasopressin controls play fighting in juvenile golden hamsters as well. Juvenile male golden hamsters were tested for play fighting after microinjections of a vasopressin V1A-receptor antagonist, Manning compound, either 0, 9, or 90 μM, into the anterior hypothalamus. The treatment selectively inhibited offensive aspects of play fighting in experimental animals. Attack frequencies were significantly decreased by both doses of Manning compound. In addition, the high dose of the receptor antagonist increased attack latencies, decreased bite frequencies, and decreased the averaged number of attacks per contact bout. Together, these results show that vasopressin controls offensive behaviors throughout development from play fighting in juveniles to aggression in adults.

1. Introduction

Play fighting is a juvenile form of agonistic behavior preceding adult aggression [1,2]. In hamsters, this behavior is initiated before puberty, peaks in early puberty around postnatal day 35 (P-35) and gradually matures into adult aggression in late puberty [3,4]. Differing from adult aggression, play fighting is characterized with more repetitive attacks and contact bouts during agonistic interactions than adults [4–6]. In addition, juvenile hamsters target the face of the protagonist in play fighting, while adult hamsters focus on the lower belly and rump [4,7,8]. However, the neural mechanisms regulating play fighting remain poorly understood. Nevertheless, we recently noted an enhanced activation of vasopressin (AVP) neurons in the hypothalamus after the consummation of play fighting in juvenile hamsters, suggesting a participation of this peptide in the regulation of this behavior [9].

The possibility that AVP is associated with play fighting is interesting as this peptide plays a key role in the control of offensive aggression in adult male hamsters [10]. In adult hamsters, microinjections of AVP into the anterior hypothalamus (AH) facilitate offensive aggression [11]. This treatment reduces the latency of the resident to bite the intruder and increases the total number of bites. In contrast, microinjections of an AVP V1A-receptor antagonist into the AH inhibit offensive aggression [12]. The source of AVP neurons associated with aggression in adults has been addressed in this species. In hamsters, AVP neurons are primarily located within the supraoptic nucleus (SON), the paraventricular hypothalamic nucleus (PVN), the nucleus circularis (NC), and the suprachiasmatic nucleus (SCN) [13,14]. Neurons within the NC and the medial division of the supraoptic nucleus (mSON) are the likely sources of AVP involved in the control of aggression. Indeed, agonistic behaviors are inhibited after lesions of the NC and the mSON, but not other areas [13,14]. In addition, AVP neurons in the same two areas also show enhanced *c*-Fos expression in adult hamsters during the consummation of offensive aggression [15]. As AVP neurons within the same areas are also activated after the performance of play fighting behavior [9], it is possible that AVP also plays a role in the offensive component of play fighting behavior. This possibility was tested in the present study through microinjections of an AVP V1A-receptor antagonist into the AH.

2. Methods

2.1. Animals and treatment

The present study was carried out with male golden hamsters (*Mesocricetus auratus*) bred in the laboratory from a colony originating from Harlan Sprague Dawley (Indianapolis, IN, USA). All litters were culled to six pups (4 males, 2 females) by P-7. All males were weaned on P-25 and housed individually in Plexiglas cages (20×33×13 cm). The hamsters were kept under a reversed light cycle (14:10 light/dark cycle and lights off at 10:00 h) and received food and water *ad libitum*. Their body weights were measured weekly to monitor their development. The studies were conducted in early puberty on P-35 around the time of peak play fighting activity in this species [3,5]. All procedures were performed according to the National Institutes of Health guidelines approved by the Institutional Animal Care and Use Committee of the University of Texas at Austin and conducted in an AALAC-accredited facility. All procedures were optimized for minimizing the number of animals used and the suffering of animals.

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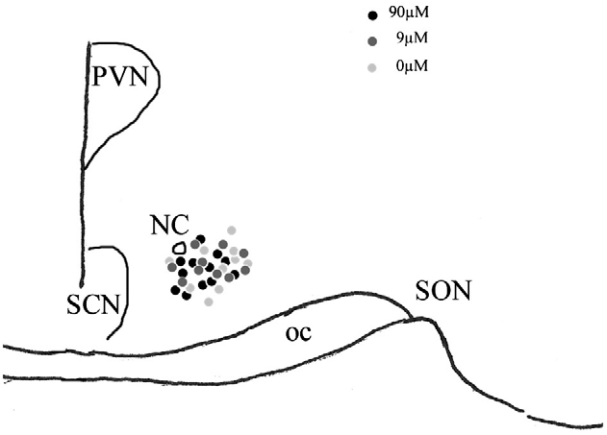

Fig. 1. A summary figure showing correct microinjection sites within the AH (light grey circle: 0 μM, grey circle: 9 μM, and black circle: 90 μM). NC: nucleus circularis; OC: optic chiasm; PVN: paraventricular hypothalamic nucleus; SCN: suprachiasmatic nucleus; SON: supraoptic nucleus.

2.2. Experimental design

On P-30, male hamsters were pre-tested for agonistic behavior for 10 min individually in the presence of a smaller (10–20% lighter) and younger unfamiliar male intruder. This resident/intruder procedure favors offensive responses by residents [16]. Animals performing no attack on intruders were excluded from the current study, which usually occurs one in every fifteen animals. Then, animals were divided into three homogeneous groups based on their body weight and the agonistic behaviors observed in this test.

On P-35, hamsters ($n=52$) were anesthetized with isoflurane (3% for onset and 2% for maintenance) and placed in a stereotaxic apparatus. A small incision was made in the skin above the skull. A small hole was drilled into the skull. Microinjections were made through a 33-gauge needle attached to a 1 μl Hamilton syringe by PE 20 tubing, which was lowered to the AH through the hole. The animals were injected with either 0 μM ($n=19$), 9 μM ($n=13$), or 90 μM ($n=20$) Manning compound [d$(CH_2)_5$Tyr(Me)AVP, Sigma, St. Louis. MO]), dissolved in saline (100 nl). Manning compound is a long-lasting vasopressin V1A-receptor antagonist, which inhibited agonistic behaviors in hamsters for at least 12 h in a previous study [17]. The dosage used in this experiment has been optimized in previous studies on agonistic behavior in hamsters [12,18]. The stereotaxic coordinates of the AH for hamsters at P-35 were 1.1 mm anterior to bregma, 1.7 mm lateral to midline, and 7.6 mm down from the dura at an 8° angle from the midline. The incisor bar was leveled at +1.5 mm. After the stereotaxic injection, a local anesthesia (2% Xylocaine, Astra USA Inc., Westborough, MA) was applied to the wound before closure. The entire procedure (including anesthesia) took less than 15 min. The animals woke up less than 2 min after the gas anesthesia was removed and were brought back to the animal room. Three hours later, animals were fully active and were observed for agonistic behaviors during a 10-minute encounter with another unfamiliar intruder. Agonistic encounters of animals were videotaped with a digital video camera (Sony Digital 8 Handycam, Sony Corporation of America, New York, NY, USA) for later review. After testing, all animals were sacrificed and their brains were fixed by immersion in 10% Acrolein and then sliced into 40 μm-thick coronal sections with a freezing rotatory microtome. A Nissl stain was performed on these tissues to localize the position of the tip of the 33-gauge microinjection needle. Thirty-two animals with correct injection sites in the AH

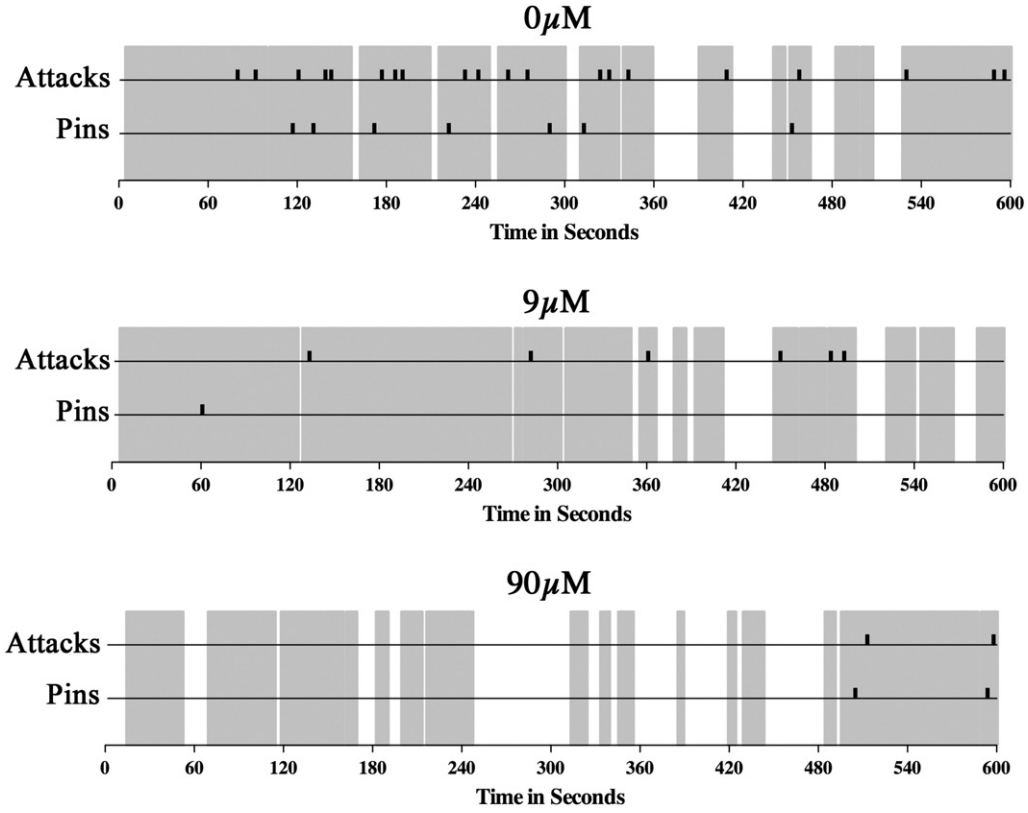

Fig. 2. Examples of the temporal distribution of agonistic behaviors (attacks and pins) and the contact time for three representative animals receiving different doses of Manning compounds: 0 μM, 9 μM, and 90 μM. The tests lasted for 10 min and the agonistic behaviors were recorded on a second-by-second basis. Each bar represents a single attack or pin. Grey shaded areas indicate the contact time.