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Physiology & Behavior, Vol. 60, No. 1, 25–29, 1996

0031-9384/96 $15.00 + .00

SSDI 0031-9384(95)02246-5

Testosterone Facilitates Aggression by Modulating Vasopressin Receptors in the Hypothalamus

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Received 21 December 1994

DELVILLE, Y., K. M. MANSOUR AND C. F. FERRIS. *Testosterone facilitates aggression by modulating vasopressin receptors in the hypothalamus.* PHYSIOL BEHAV **60**(1) 25–29, 1996.—In many species, testosterone treatment facilitates offensive aggression tested in resident–intruder models. As the mechanisms of action of testosterone remain unclear, we hypothesized that testosterone interacts with neurotransmitter systems involved in the regulation of offensive aggression. We tested this hypothesis with the vasopressinergic system in golden hamsters in three separate experiments. First, we compared the density of V_1 vasopressin (AVP) receptor binding between castrated animals treated with testosterone and their untreated controls. The most noticeable difference was found within the ventrolateral hypothalamus (VLH), a site involved in the control of aggression in several species of mammals. Within this area, V_1 AVP receptor binding disappeared after castration, while being maintained by testosterone-treatment. Second, we tested behavioral effects of AVP within the VLH. Microinjections of AVP (100 nl, 1 or 100 μM) within the VLH accelerated the onset of offensive aggression in testosterone-treated animals. However, AVP-injected animals did not bite more than their vehicle-injected controls. Third, microinjections of AVP failed to activate offensive aggression in animals deprived of testosterone. As AVP receptors appeared to overlay previously described distributions of androgen and estrogen receptors in golden hamsters, we propose that testosterone facilitates the onset of offensive aggression, at least partly, through an activation of AVP receptors within the VLH.

Offensive aggression Arginine vasopressin Ventrolateral hypothalamus

IN A VARIETY of vertebrates, including primates, correlations have been established between levels of aggressiveness and testosterone or neural testosterone metabolism (4,6,29). In particular, the period of territorial aggressiveness and male sexual behavior correlates with increased testicular activity and plasma levels of testosterone during the breeding cycle of many seasonal species (5,12,33). Over 100 years ago, Berthold reported that castration decreases aggressiveness and male-typical behaviors in roosters (20). Later, Beeman reported similar effects of castration in mice, but she was also capable of restoring aggressiveness after testosterone treatment (8). In mice, strong correlations have been established between testosterone and the latency to attack an intruder in resident–intruder models of offensive aggression (32). Similarly, in golden hamsters, castration results in a reduction of offensive aggression (27). Presumably, testosterone would affect neural networks controlling offensive aggression, resulting in an enhanced arousal and predisposition towards agonistic displays. Indeed, testosterone is suspected to modulate offensive aggression by affecting several areas within the limbic system (3,7,26). For example, whereas electrical stimulation of the ventrolateral hypothalamus (VLH) induces aggression in rats, the amount of current needed to stimulate the behavior increases after castration and decreases after testosterone treatment (9). However, the exact nature of the mode of action of testosterone remains unknown. As certain neurotransmitters have been involved in the regulation of offensive aggression (16,18,23), it is conceivable that testosterone modulates specific neurotransmitter systems within the brain, particularly within areas like the VLH.

The following experiments were carried out to test the hypothesis that testosterone interacts with arginine-vasopressin (AVP), as this neurotransmitter facilitates offensive aggression when microinjected within various parts of the limbic system in rats and golden hamsters (17,21,22,28). First, we tested whether the lack of testosterone affects AVP receptor binding within any neural site. Second, we tested whether AVP microinjections within such site can affect offensive aggression in gonadally

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intact animals and testosterone-treated animals. Finally, we also tested whether castrated animals are responsive to AVP microinjections.

METHOD

Animals and Treatment

Adult male golden hamsters ($n = 61$) were purchased from Sprague–Dawley Laboratories (Indianapolis, IN). The animals were kept under a reverse day–light cycle (14L:10D; lights on at 1900 h), and received food and water ad lib. Some hamsters ($n = 49$) were castrated and implanted SC with a Silastic capsule (length: 22 mm; i.d.: 1.98 mm; Silastic Medical Grade Tubing, Dow Corning Midland, MI) under sodium pentobarbital (Nembutal; 35 mg/kg; Abbott Laboratories, North Chicago, IL) anesthesia. The Silastic capsules were either empty or filled with crystalline testosterone (Sigma, St. Louis, MO). One month later, the animals were sacrificed ($n = 10$) for autoradiography or tested for behavior ($n = 39$). The flank glands were observed in all testosterone-treated animals to ascertain the efficiency of the Silastic implants, as this gland is testosterone dependent (31). Indeed, all testosterone-treated animals had well-developed glands.

Vasopressin Receptor Binding

The effect of castration and testosterone replacement was tested on AVP receptor binding performed by in vitro autoradiography, as previously described (19). The animals were sacrificed by decapitation, their brains removed, frozen on dry ice, and kept at −80°C until sectioning. Coronal sections (20 μm) were cut in a cryostat set at −10°C, thaw-mounted on gelatin-coated slides, and stored at −80°C. Later, the sections were preincubated at room temperature in 0.05 *M* Tris-HCl buffer (pH 7.3) containing 100 μM NaCl and 50 μM guanosine 5′-triphosphate (Type II-S, Sigma Chemical Co.) followed by two 5-min washes in Tris buffer. Later, the sections were incubated for 1 h at room temperature in Tris buffer containing 10 m*M* $MgCl_2$, 0.01% bovine serum albumin (Fraction V, Sigma Chemical Co.), 0.05% bacitracin, and 40 IU aprotinin and 50 p*M* [^{125}I][d(CH_2)$_5$Sar^7]AVP ([^{125}I]SAVP) (19). Then the sections were washed in ice-cold Tris buffer containing 10 m*M* $MgCl_2$. Nonspecific binding was obtained by incubations containing 1 μM unlabeled AVP. Once dried at room temperature, the sections were apposed to Hyperfilm™ ^{3}H (Amersham Co., IL), in X-ray cassettes for about 3 weeks at −80°C. Following removal from the X-ray cassettes, the slides were stained with thionin to identify the labeled sites. The specificity of the autoradiography procedure has been previously reported (19). The relative density of SAVP binding was quantified with the Image software (NIMH, Version 1.47). The density of SAVP binding was estimated within standard surfaces (diameter: 0.2 mm) placed inside the VLH. As a specificity test, measures were also taken from a neighboring nucleus, the dorsomedial hypothalamic nucleus. An average of three to five independent measures (specific minus nonspecific binding) was taken for each level in each area analyzed in each animal. The results were compared between experimental animals (castrates implanted with testosterone-filled Silastic capsules; $n = 5$) and their controls (castrates implanted with empty capsules; $n = 5$).

Stereotaxic Surgery

Three weeks after castration, the animals were stereotaxically implanted with 26-gauge guide cannulae targeted at the VLH under sodium pentobarbital anesthesia. The stereotaxic coordinates of the guide cannulae were: 0.5 mm caudal to the bregma, 2.0 mm lateral to the midline, and 4.5 mm below the dura, at a 0.8° angle from the vertical plane. The nose piece was placed at the level of the interaural line. The cannulae were closed with a 33-gauge obturator extending no more than 1 mm beyond the guides. The inner cannulae (33 gauge) used for microinjections were inserted through the guides just before testing. These inner cannulae were cut to extend 4.0 mm beyond the guides.

Behavior

Offensive aggression was observed using a resident–intruder paradigm after unilateral microinjections within the VLH. After microinjecting AVP or vehicle, a nonaggressive intruder was placed in the home cage of the subjects, and behaviors were recorded for a 10-min period. The latency to bite, numbers of bites, and total duration of contact time were recorded for a 10-min exposure to an intruder. Microinjections were administered through a 33-gauge cannula lowered into the target site and connected to a 1-μl Hamilton syringe through PE-20 tubing. The animals used in these behavioral experiments were experienced fighters, as they consistently attacked intruders placed in their home cage during tests performed over a period of several weeks before the present experiments. However, castrated animals without testosterone treatment did not consistently attack intruders during these sessions. After testing, the animals were sacrificed, and their brains were fixed in 10% formalin. Later, the brains were sliced and the sections were labeled with thionin to ascertain that the tips of the injection cannulae were localized within the VLH.

Three separate behavioral experiments were performed. During a first experiment (Experiment 1), the animals were gonadally intact and separated into two groups: control vehicle injected (artificial CSF, 100 nl, $n = 6$) and experimental AVP injected (100 nl, 100 μM, $n = 6$). During a second experiment (Experiment 2), all animals were castrated and implanted with a testosterone-filled Silastic capsule to provide comparable baselines of plasma testosterone levels. These animals received microinjections of either vehicle (artificial CSF, 100 nl, $n = 7$), 1 μM AVP ($n = 7$), or 100 μM AVP ($n = 5$). During a third experiment (Experiment 3), all animals were castrated, implanted with an empty Silastic capsule, and microinjected with either vehicle (artificial CSF, 100 nl, $n = 6$) or 10 μM AVP ($n = 6$). These animals were tested before and after microinjections. Pretests before injections were performed in comparison with testosterone-treated animals ($n = 8$).

RESULTS AND DISCUSSION

Vasopressin Receptor Binding

The distribution of specific binding was compared between castrates and castrates with testosterone throughout the limbic system. A striking difference was noted within the ventrolateral hypothalamus (VLH), an area involved in the regulation of aggression (1,9,11,15,25). In castrates with testosterone, [^{125}I]SAVP binding was observed within the VLH, spreading laterally from the ventrolateral part of the ventromedial nucleus to the medial tuberal nucleus. This binding was observed to extend rostro-caudally from the level of the middle of the ventromedial nucleus to the ventral premammillary nucleus. It is noteworthy that this distribution of [^{125}I]SAVP binding within the VLH matches previously described distributions of immunoreactivity for estrogen and androgen receptors in golden hamsters

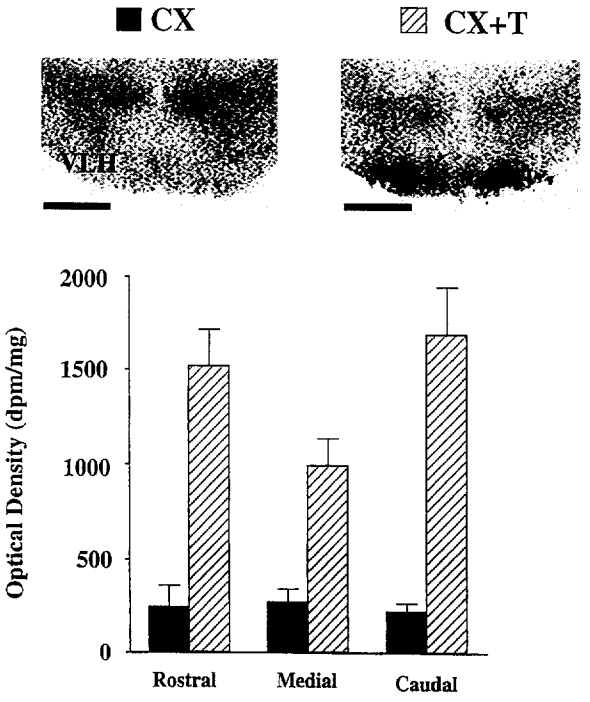

FIG. 1. Top: [^{125}I]SAVP binding in coronal sections (20 μm thick) of the medial hypothalamus in male golden hamsters. Note the absence of [^{125}I]SAVP binding within the VLH in castrated controls (Cx) compared to testosterone-treated animals (Cx + T). Scale bar: 1 mm. Bottom: Quantification of [^{125}I]SAVP binding within the VLH of castrated animals treated with testosterone (Cx + T, striped columns) and their untreated controls (Cx, filled columns). Values presented are the mean ± SEM of the optical density of the specific binding. Comparisons were made at three levels along the rostro-caudal extent of the VLH, starting at the level of the ventromedial nucleus of the hypothalamus, and finishing at the level of the ventral premammillary nucleus. The rostral level of the VLH coincided with the middle of the ventromedial nucleus, the medial level of the VLH with the caudal end of the ventromedial nucleus, and the caudal level of the VLH with the ventral premammillary nucleus. Comparisons were statistically significant for each level analyzed ($p < 0.01$, Student's t-test, two-tailed).

(10,24,34,35). In contrast, no such [^{125}I]SAVP binding was observed in castrates (Fig. 1). The density of [^{125}I]SAVP binding within the VLH was assessed and compared between groups at three different levels along the rostro-caudal extent of the VLH. Although elevated levels of binding density were measured within the rostro-caudal extent of the VLH in castrates with testosterone, binding density was much reduced in castrates without testosterone (Fig. 1). Furthermore, the absence of testosterone had no apparent or significant effect on [^{125}I]SAVP binding within the dorsomedial hypothalamic nucleus and other neighboring sites. These data indicate the existence of a close relationship between gonadal steroid hormones and AVP receptors within the VLH.

Behavior

As the VLH is involved in the regulation of aggressive behavior (1,9,11,15,25), we then decided to test whether offensive aggression can be affected by AVP microinjections within this area. During Experiment 1, the animals were gonadally intact. The results (Fig. 2, Exp. 1) showed that animals microinjected with AVP attacked and bit readily the intruders, whereas vehicle-injected animals were slower [$t(11) = 2.57$, $p < 0.05$, Student's t-test, two-tailed]. However, other variables, such as the numbers of bites and contact times, did not differ significantly between the groups.

This experiment was replicated under Experiment 2 with castrated animals implanted with a testosterone-filled Silastic capsules. Again, AVP microinjections resulted in an activation of offensive aggression [latency to bite: $F(2, 16) = 5.3$, $p < 0.05$, ANOVA] (Fig. 2, Exp. 2). Animals injected with either 1 or 100 μM AVP were faster to attack and bite the intruders ($p < 0.05$ and $p < 0.01$, respectively, Fisher's PLSD post hoc tests). As for the previous experiment, AVP microinjections had no significant effect on the number of bites and contact time. Two hamsters injected with 1 μM AVP were implanted dorsal to the VLH, in the lateral hypothalamus. These animals were not responsive to AVP injections and were not used for data analysis. The results obtained from these animals also indicate that the AVP-facilitated offensive aggression is specific to the VLH and not sites located dorsally along the cannula track.

Our results show that AVP receptors within the VLH are testosterone dependent. Therefore, it is likely that the effects of AVP on offensive aggression should also be testosterone dependent (i.e., animals deprived of testosterone should not be responsive to AVP microinjections within the VLH). This possibility was tested under Experiment 3 using castrated animals without testosterone treatment. One vehicle-injected animal was implanted outside the VLH and was removed from data analysis. In this experiment, AVP microinjections did not affect the latency to attack and bite the intruders [latency to bite: AVP: 488.3 ± 233.9 s; CSF: 499.2 ± 225.4 s; $t(9) = 0.08$, $2p > 0.1$]. Most animals (9 out of 12) did not bite the intruders during the tests, as expected from previous observations that castration reduces aggressive behavior in golden hamsters (27). In fact, these animals spent little time in contact with the intruders, as they tended to ignore them, escape from them, or even displayed submissive postures. During pretesting, these residents spent 206 ± 107 s in contact with the intruders, whereas testosterone-treated animals ($n = 8$) spent 360 ± 135 s [$t(17) = 2.78$, $2p < 0.01$]. Nevertheless, a few hamsters (one to two per group) attacked the intruders readily after their introduction in the cages. Taken together, our results

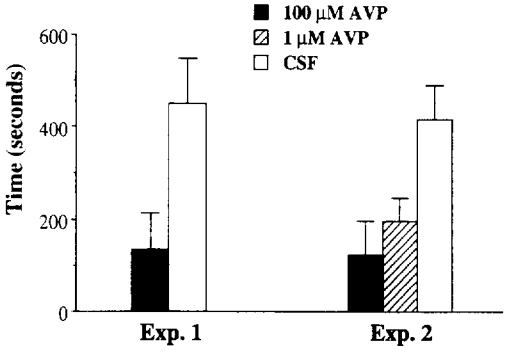

FIG. 2. Latencies of bites (mean ± SEM) in male golden hamsters exposed to an intruder placed inside their home cage. The subjects were tested after unilateral microinjections of AVP (100 nl, 1–100 μM) or vehicle (artificial CSF, 100 nl) within the VLH. During a first experiment (Exp. 1), the animals were gonadally intact experienced fighters, whereas during the second experiment (Exp. 2), the animals were experienced fighters that were castrated and implanted with testosterone-filled Silastic capsules.

show that castrated animals are not sensitive to AVP microinjections within the VLH, suggesting that the VLH is a site integrating the actions of testosterone and AVP in the regulation of offensive aggression.

The modulation of offensive aggression involves a large number of neurons located within many areas other than the VLH (2). In fact, it is likely that the behavior is controlled by neural networks involving many parts of the brain, each integrating particular stimuli or motor output relevant to the display of offensive aggression (2). In hamsters, AVP is known to affect offensive aggression from the anterior hypothalamus (17,28) and, as shown by the present data, the VLH. In rats, AVP microinjections within the medial amygdala and the lateral septum also facilitate offensive aggression (21,22). Interestingly, AVP microinjections within the VLH only affected the latency to attack the intruders. The number of bites received by the intruders was not affected by AVP. Therefore, microinjections of AVP within the area did not increase the intensity of aggressiveness (i.e., the number of bites), but rather facilitated the onset of the behavior (i.e., the latency to bite). These results contrast with recent observations made after microinjections of AVP in the anterior hypothalamus (Ferris, Melloni, and Delville, unpublished observations). Microinjections of AVP within the anterior hypothalamus not only shortened the latency to bite the intruders but also increased the number of bites inflicted to them. These data suggest different roles for the VLH and the anterior hypothalamus in the regulation of offensive aggression. Although the anterior hypothalamus regulates the intensity of the behavior, the VLH participates in the onset of the behavior.

In the present experiments, castrates were not responsive to AVP microinjections, suggesting that the VLH is a site of action for testosterone in the regulation of offensive aggression. However, castrates spent less time in contact with the intruders than testosterone-treated animals. These results suggest that testosterone affects the motivation and the onset of the behavior. The release of AVP in the VLH would only affect the onset of the behavior. Therefore, it is likely that testosterone affects offensive aggression through other sites beside the VLH. The results of the experiment with castrates (Experiment 3) should be taken under this restriction. In rats, it is unclear whether AVP microinjected within the VLH affects offensive aggression. Interestingly, castration increases the threshold of current necessary to activate offensive aggression by electrical stimulation of the VLH (9). Microinjections of AVP within other sites (i.e., lateral septum, amygdala) facilitate offensive aggression in rats (21,22). However, the behavior can be activated by AVP microinjections within these sites in castrated animals (21,22). In rats, castration does not affect the density of AVP receptor binding within these sites (30), while reducing the density of AVP neurons and terminals (13,14). These observations, although reinforcing the distinctiveness of the VLH in golden hamsters, also suggest that the area is involved in the onset of offensive aggression in both species. Furthermore, this aspect of the behavior is facilitated by testosterone treatment in golden hamsters as well as in rats.

In gonadally intact animals and testosterone-treated castrates, AVP microinjections within the VLH facilitate the onset of offensive aggression. Blockade of AVP receptors within this site might delay the behavior. This point was addressed in a pilot experiment (Delville, Mansour, and Ferris, unpublished observations). Bilateral microinjections of a selective V_1 AVP receptor antagonist did not delay the onset of aggression in testosterone-treated castrates that were experienced fighters. Unfortunately, the exact location of cannulae tips was not recorded in this test, and it is unclear whether the antagonist successfully inhibited AVP receptor binding in this pilot experiment. Nevertheless, this observation is consistent with the concept that the VLH is not involved in the appetitive elements of the behavior. The animals injected with the antagonist were still aggressive, and it is likely that blocking AVP receptors in the VLH is not enough to suppress the various stimuli that trigger agonistic responding in a resident–intruder paradigm.

In summary, the present data point to the VLH as a site of action for AVP in the regulation of offensive aggression. The responsiveness of the VLH to AVP depends on the presence of testosterone. Indeed, testosterone maintains AVP receptors within the VLH, and AVP activates offensive aggression only in testosterone-treated animals. Although it is likely that the modulation of offensive aggression by testosterone involves more than AVP receptors within the VLH, the present data indicate a causal relationship between removal of testosterone and the decrease in the ability of AVP to stimulate offensive aggression. This conclusion is further supported by the apparent overlay of AVP binding sites with neurons containing gonadal steroid receptors (10,24,34,35), suggesting a colocalization of both receptor types within VLH neurons.

ACKNOWLEDGEMENTS

These studies were supported by grants BNS-9121097, NS 30199 and MH 52280 awarded to Craig Ferris. The authors are grateful to Marcus Mercier, Eran Hayden, and Eric Quan for technical assistance and to Rich Melloni for helpful comments on the manuscript.

REFERENCES

1. Adams, D. B. Defence and territorial behaviour dissociated by hypothalamic lesions in the rat. Nature 232:573–574; 1971.
2. Adams, D. B. Brain mechanism for offense, defense, and submission. Behav. Brain Sci. 2:201–241; 1979.
3. Albert, D. J.; Dyson, E. M.; Walsh, M. L. Intermale social aggression: Reinstatement in castrated rats by implants of testosterone propionate in the medial hypothalamus. Physiol. Behav. 39:555–560; 1987.
4. Albert, D. J.; Walsh, M. L.; Gorzalka, B. B.; Siemens, Y.; Louie, H. Testosterone removal in rats results in a decrease in social aggression and a loss of social dominance. Physiol. Behav. 36:401–407; 1986.
5. Balthazart, J. Hormonal correlates of behavior. In: Farner, D. S.; King, J. R.; Charles, K. C., eds. Avian biology, vol. 3. New York: Academic Press; 1983:221–365.
6. Barfield, R. J.; Busch, D. E.; Wallen, K. Gonadal influence on agonistic behavior in the male domestic rat. Horm. Behav. 3:247–259; 1972.
7. Bean, J. B.; Conner, R. Central hormonal replacement and home-cage dominance in castrated rats. Horm. Behav. 11:100–109; 1978.
8. Beeman, E. A. The effect of male hormone on aggressive behavior in mice. Physiol. Zool. 20:373–405; 1947.
9. Bermond, B.; Mos, J.; Meelis, W.; van der Poel, A. M.; Kruk, M. R. Aggression induced by stimulation of the hypothalamus: Effects of androgens. Pharmacol. Biochem. Behav. 16:41–45; 1982.
10. Clancy, A. N.; Whitman, C.; Michael, R. P.; Albers, H. E. Distribution of androgen receptor-like immunoreactivity in the brains of intact and castrated male hamsters. Brain Res. Bull. 33:325–332; 1994.

11. Colpaert, F. C.; Wiepkema, P. R. Effects of ventromedial hypothalamic lesions on spontaneous intraspecies aggression in male rats. Behav. Biol. 16:117–125; 1976.
12. Crews, D.; Silver, R. Reproductive physiology and behavior interactions in nonmammalian vertebrates. In: Adler, N.; Pfaff, D.; Goy, R. W., eds. Handbook of behavioral neurobiology, vol. 7. New York: Plenum Publishing Co.; 1985:101–182.
13. De Vries, G. J.; Buijs, R. M.; Van Leeuwen, F. W.; Swaab, D. F. The vasopressinergic innervation of the brain in normal and castrated rats. J. Comp. Neurol. 233:236–254; 1985.
14. De Vries, G. J.; Al-Shamma, H. A. Sex differences in hormonal responses of vasopressin pathways in the rat brain. J. Neurobiol. 21:686–693; 1990.
15. Eclancher, F. S.; Karli, P. Comportement d'aggression interspécifiques et comportement alimentaire du rat: Effets de lésions des noyeaux ventro-médians de l'hypothalamus. Brain Res. 26:71–79; 1971.
16. Eichelman, B. Toward a rational pharmacotherapy for aggressive and violent behavior. Hosp. Comm. Psychol. 39:31–39; 1988.
17. Ferris, C. F.; Potegal. M. Vasopressin receptor blockade in the anterior hypothalamus suppresses aggression in hamsters. Physiol. Behav. 44:235–239; 1988.
18. Ferris, C. F.; Delville, Y. Vasopressin and serotonin interactions in the control of agonistic behavior. Psychoneuroendocrinology 19:593–601; 1994.
19. Ferris, C. F.; Delville, Y.; Grzonka, Z.; Luber-Narod, J.; Insel, T. An iodinated vasopressin (V_1) antagonist blocks flank marking and selectively labels neural binding sites in golden hamsters. Physiol. Behav. 54:737–747; 1993.
20. Forbes, T. R. A. A. Berthold and the first endocrine experiment: Some speculation as to its origin. Bull. Hist. Med. 23:263–267; 1949.
21. Koolhaas, J. M.; Van den Brink, T. H. C.; Roozendaal, B.; Boorsma, F. Medial amygdala and aggressive behavior: Interaction between testosterone and vasopressin. Aggress. Behav. 16:223–229; 1990.
22. Koolhaas, J. M.; Moor, E.; Hiemstra, Y.; Bohus, B. The testosterone-dependent vasopressinergic neurons in the medial amygdala and lateral septum: Involvement in social behaviour of male rats. In: Jard, S.; Jamison, R., eds. Vasopressin. Paris-London: INSERM/John Libbey Eurotext Ltd.; 1991:213–219.
23. Kruk, N. R. Ethology and pharmacology of hypothalamic aggression in the rat. Neurosci. Biobehav. Rev. 15:527–538; 1991.
24. Li, H.-Y.; Blaustein, J. D.; De Vries, G. J.; Wade, G. N. Estrogen-receptor immunoreactivity in hamster brain: Preoptic area, hypothalamus and amygdala. Brain Res. 631:304–312; 1993.
25. Olivier, B. The ventromedial hypothalamus and aggressive behavior in rats. Aggress. Behav. 3:47–56; 1977.
26. Owen, K.; Peters, P. J.; Bronson, F. H. Effects of intracranial implants of testosterone propionate in intermale aggression in the castrated male mouse. Horm. Behav. 5:83–92; 1974.
27. Payne, A. P. A comparison of the aggressive behaviour of isolated intact and castrated male golden hamsters towards intruders introduced into the home cage. Physiol. Behav. 10:629–631; 1973.
28. Potegal, M.; Ferris, C. F. Intraspecific aggression in male hamsters is inhibited by intrahypothalamic vasopressin-receptor antagonist. Aggress. Behav. 15:311–320; 1990.
29. Rose, R. M.; Holaday, J. W.; Bernstein, I. S. Plasma testosterone, dominance rank and aggressive behavior in male rhesus monkey. Nature 231:366–368; 1971.
30. Tribollet, E.; Audigier, S.; Dubois-Dauphin, M.; Dreifuss, J. J. Gonadal steroids regulate oxytocin receptors in the brain of male and female rats. An autoradiographical study. Brain Res. 511:129–140; 1990.
31. Vandenbergh, J. C. Effects of gonadal hormones on the flank gland of the golden hamster. Horm. Res. 4:28–33; 1973.
32. Van Oortmessen, G. A.; Dijk, D. J.; Schuurman, T. Studies in wild house mice. II. Testosterone and aggression. Horm. Behav. 21:139–152; 1987.
33. Wingfield, J. C.; Marler, P. Endocrine basis of communication in reproduction and aggression. In: Knobil, E.; Neill, J.; Ewing, L. L.; Greenwald, G. S.; Markert, C. L.; Pfaff, D. W., eds. The physiology of reproduction. New York: Raven Press, Ltd.; 1988:1647–1677.
34. Wood, R. I.; Newman, S. W. Intracellular partitioning of androgen receptor immunoreactivity in the brain of the male Syrian hamster: Effects of castration and steroid replacement. J. Neurobiol. 24:925–938; 1994.
35. Wood, R. I.; Newman, S. W. Androgen and estrogen receptors coexist within individual neurons in the brain of the Syrian hamster. Neuroendocrinology 62:487–497; 1995.