

MISTY ROLE OF AMYGDALA IN FEMALE REPRODUCTIVE BEHAVIOR

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Sir,

The primary reproductive behavior of female quadrupeds is known as lordosis which is the bending of vertebral column during intercourse. This behavior depends on the actions of estrogens followed by progesterin in cells within defined neuronal groups in the hypothalamus and midbrain. The main neural circuit for lordosis behavior is well established. Estrogen-dependent transcription in ventromedial hypothalamic (VMH) cells sends permissive signals to the midbrain central grey, thus enabling the rest of the circuit to operate [1]. Main neural circuit of lordosis consists of medial preoptic, medial anterior and ventromedial nucleus of hypothalamus, midbrain central gray, the reticular formation and spinal cord [2].

Along with the main neural circuit of lordosis behavior, many other areas are responsible for modulating reproductive behaviors in female rats. Septum has an inhibitory role on lordosis [3] and the ventrolateral outputs may play a critical role in sending this inhibitory signal [4]. Olfactory bulb has also an inhibitory role and it is thought that they may act through a common pathway [5]. Estrogen may release the inhibition when it acts on the lateral septum [6]. Bed nucleus of stria terminalis exerts an excitatory role on lordosis [7]. In addition, area postrema (AP) lesions block suppression of estrous behavior, but not estrous cyclicity, in food-deprived Syrian hamsters. Not only that, AP has an influence on estrogen receptor expression in the VMH and arcuate nucleus [8]. The remaining brain area which is well investigated in accordance with female reproductive behaviors is the amygdala (AMY). The enhancement of lordosis following repeated coital stimulation was significantly reduced in the medial amygdala (mAMY)-lesioned rats as compared with the sham-operated controls. Since luteinizing hormone-releasing hormone (LHRH) is reported to enhance lordosis behavior in ovariectomized estrogen-primed rats, it appears likely that the effects of mAMY on lordosis are mediated through the LHRH neuronal system [9]. It was found that AMY expresses estrogen receptor (ER) messenger RNA [10]. AMY inputs from lateral septum and preoptic hypothalamus may project to AMY via stria terminalis and it was found that, estrogens may reduce the AMY inputs from the preoptic area, without affecting those from the lateral septum [11]. Estrogens may modulate the AMY output trans-synaptically, in addition to the direct postsynaptic actions [12-14]. In another study, it was reported that, the AMY lesion reduces behavioral sensitivity to estrogens in septal lesioned rat and hence AMY may be a part of the neural system mediating the increased sensitivity to E in septal lesioned rats [5]. But it was also reported that, the ER α in the VMH, but not in the medial posterodorsal AMY, is important for receptivity as well as for sexual incentive motivation [15]. To study the role of mAMY in copulatory pacing behavior it was found that lesions in the mAMY shorten the latency to enter the male compartment rather than pacing behavior in female rats, especially when they were primed with lower doses of estrogens, suggesting that mating access is regulated by the mAMY [16].

Although the mAMY is known for its effects on reproduction along with defense mechanism, more precise understanding of its role in estrogen sensitivity as well as its correlation with other brain areas in modulating reproductive behavior is yet to be established. Despite various discrete findings the picture is still very blurred.

To understand how ovarian steroids influence the GABAergic system it was found that, GABA receptor subunit gene expression was generally higher in the AMY than in the medial basal hypothalamus [17]. So, GABA receptors of medial and/or central AMY may be an important unit of estrogen sensing neural circuitry [18].

Visual sensation can modulate reproduction through mAMY along with other brain areas like piriform cortex, olfactory tubercle, lateral hypothalamus, and the bed nucleus of the stria terminalis [19-24]. But, no precise literature was available related to the association of visual sensation, mAMY and reproductive behavior.

From the present article, it is clear that, despite of many studies the role of AMY in female sex behavior is still hazy. Mainly the misty idea about the role of GABA receptors of AMY in estrogen sensitivity and the relation of visual sensation with AMY and its further effect on the reproductive behavior circuit should be cleared precisely. Finally it can be easily concluded that, a clear picture of the AMY in the map of female reproductive behavior may help to explore many new paths to reach many new destinations of basic as well as applied neurophysiology.

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