

谷氨酸一钠损毁下丘脑弓状核对大鼠血浆皮质酮的影响

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提要 新生期大鼠 ip 谷氨酸一钠破坏下丘脑弓状核神经元后, 大鼠垂体重量明显减轻, 细胞数略少于对照组, 肾上腺无变化, 血浆皮质酮的基础值及应激后的升高水平均与各相应的对照组相比无明显差别, 结果表明下丘脑弓状核对垂体-肾上腺皮质系统的功能影响不大。

关键词 谷氨酸一钠; 新生期大鼠; 弓状核; 垂体-肾上腺皮质系统; 血浆皮质酮; 应激

下丘脑弓状核对垂体内分泌功能有调节作用。用谷氨酸一钠(monosodium glutamate, MSG)选择性破坏下丘脑弓状核神经元, 可产生许多内分泌功能障碍^(1,2), 大鼠性腺、甲状腺萎缩, 血浆促黄体生成素(LH)、促甲状腺素(TSH)、♂动物生长激素(GH)降低, ♀动物生乳素(PL)增加等。但极少报道弓状核对垂体-肾上腺皮质系统的影响。本实验观察新生期大鼠 ip MSG 对垂体-肾上腺皮质系统的影响。

方 法

出生后 d 5 的♂大鼠随机分成 MSG 组和对照组。MSG 组大鼠 ip 28.75% MSG(江苏无锡县制药厂生产) 2 g/kg, qd × 5 d。对照组大鼠用 10% NaCl(与 MSG 等渗)作等容量的对照处理。两组大鼠同窝饲养至 d 100 进行实验。

将大鼠放在应激箱内, 通过箱底金属栅给予不能躲避的间歇性脚底电刺激 30 min 造成大鼠应激, 刺激参数: 50 Hz, 60 V, 每隔 0.9 s 刺激 0.1 s。

实验均在 8:00~10:00 AM 进行, 实验后大鼠在 1 min 内完成断头取血, 用竞争性蛋白结合分析法测定血浆皮质酮⁽³⁾。脑组织连同脑垂体和肾上腺经 10% 福尔马林固定后, 作连续

切片和染色, 进行组织学鉴定。并且分别计算 MSG 组和对照组大鼠的脑组织切片中每个油镜视野内弓状核神经元数。

结 果

形态学改变 MSG 组大鼠下丘脑弓状核内神经元($75 \pm SD 11$ 个/视野)比对照组(185 ± 16 个/视野)减少约 60%(图 1 见后图版 1)。

垂体重量改变 MSG 组大鼠的垂体重量为(25 ± 4 mg/kg)比对照组大鼠(32 ± 6 mg/kg)明显减轻($p < 0.01$)。MSG 组和对照组大鼠的肾上腺重量分别为 105 ± 22 和 120 ± 32 mg/kg, 差别不明显。

MSG 组大鼠的垂体细胞数略少于对照组, 形态无变化, 而肾上腺束状带细胞排列成束, 胞体大, 胞核清晰, 与对照组相比未见特殊异常。

血浆皮质酮的变化 在安静情况下, MSG 组大鼠的血浆皮质酮基础值($0.58 \pm 0.14 \mu\text{mol/l}$)与对照组($0.72 \pm 0.20 \mu\text{mol/l}$)无明显差别。在应激情况下, MSG 组血浆皮质酮($0.99 \pm 0.41 \mu\text{mol/l}$)和对照组血浆皮质酮($1.13 \pm 0.44 \mu\text{mol/l}$)间亦无明显差别, 但都比安静状态下血浆皮质酮的基础值有明显升高。

讨 论

下丘脑弓状核紧靠第三脑室, 是下丘脑内侧基底部(MBH)的一个重要核团, 已有资料表明弓状核在调节垂体-性腺轴和垂体-甲状腺轴中起重要作用^(1,2)。那么弓状核在调节垂体-肾上腺皮质系统的功能中作用如何呢? 本实验的结果表明, 用 MSG 选择性破坏大鼠弓状核神经元后, 垂体重量明显减轻, 但血浆皮质酮的基础水平并不降低, 垂体 ACTH-皮质酮应激

反应仍然存在。由此可见,弓状核破坏后对垂体-肾上腺皮质系统的功能影响不大,这与近来的资料⁽⁴⁾相类似。

肾上腺皮质激素释放激素(CRH)对垂体-肾上腺轴起着主要的调节作用,由于CRH广泛存在于下丘脑内侧基底部⁽⁵⁾,并且MSG能选择性地破坏下丘脑弓状核的神经元胞体,而对路过的神经纤维无明显影响,因而对一些经过弓状核到达正中隆起释放CRH⁽⁶⁾或CRH样作用物质⁽⁷⁾的神经纤维也就影响不大。这些都支持了弓状核被破坏后,垂体-肾上腺皮质系统的功能不受重要影响的可能性。

致谢 方几希、韩志新、殷伟平协助组织学切片工作。

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Acta Pharmacologica Sinica 1986 Mar; 7 (2) : 104-105

Influence of hypothalamic arcuate nucleus lesion with monosodium glutamate on plasma corticosterone in neonatal rats

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ABSTRACT The purpose of present experiment was to assess the role of hypothalamic arcuate nucleus (ARC) in regulation of pituitary-adrenocortical system. The plasma corticosterone was determined by competitive protein-binding radioassay.

The number of ARC neurons in monosodium glutamate (Msg)-treated rats showed a decrease of 60%, and the pituitary gland in Msg-treated group was smaller than that of control group ($p < 0.01$), but there was no obvious difference in the weights of adrenal glands. No significant

difference in corticosterone levels was seen between Msg-treated group and the control one, even after electrical foot-shock.

These results indicate that the neurons in ARC do not play an essential role in the regulation of pituitary-adrenocortical system.

KEY WORDS monosodium glutamate; neo-natal rats; arcuate nucleus; pituitary-adrenocortical system; plasma corticosterone; stress

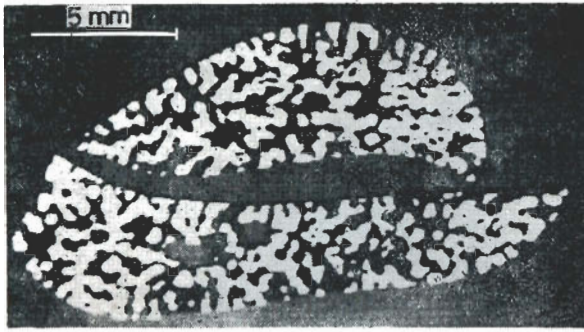


Fig 1. Section image of a mouse liver shown by image-analyzer. Figure legends total area of liver sections = 129.3 mm². Area of ChE activity = 52.8 mm² (white, 40.8%).

(See p 97)

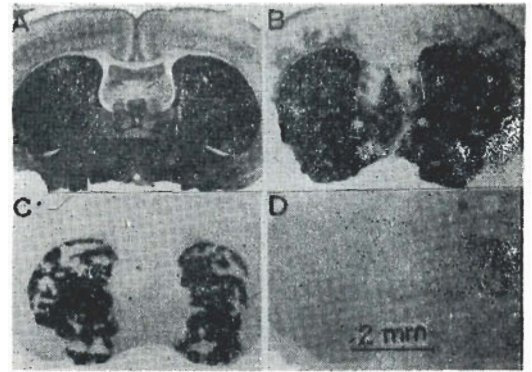


Fig 3. Brain sections (ChE histochemical stain, scale 2 mm) showing ChE inhibited areas in mice after ip 1 LD₅₀ soman. A) Normal; B) Phenobarbital + soman, showing partially inhibited areas in cerebral cortex; C) Soman, showing complete inhibition in cerebral cortex and partially inhibited areas in basal ganglia; D) TOCP + soman, showing complete inhibition in both cerebral cortex and basal ganglia.

(See p 98)

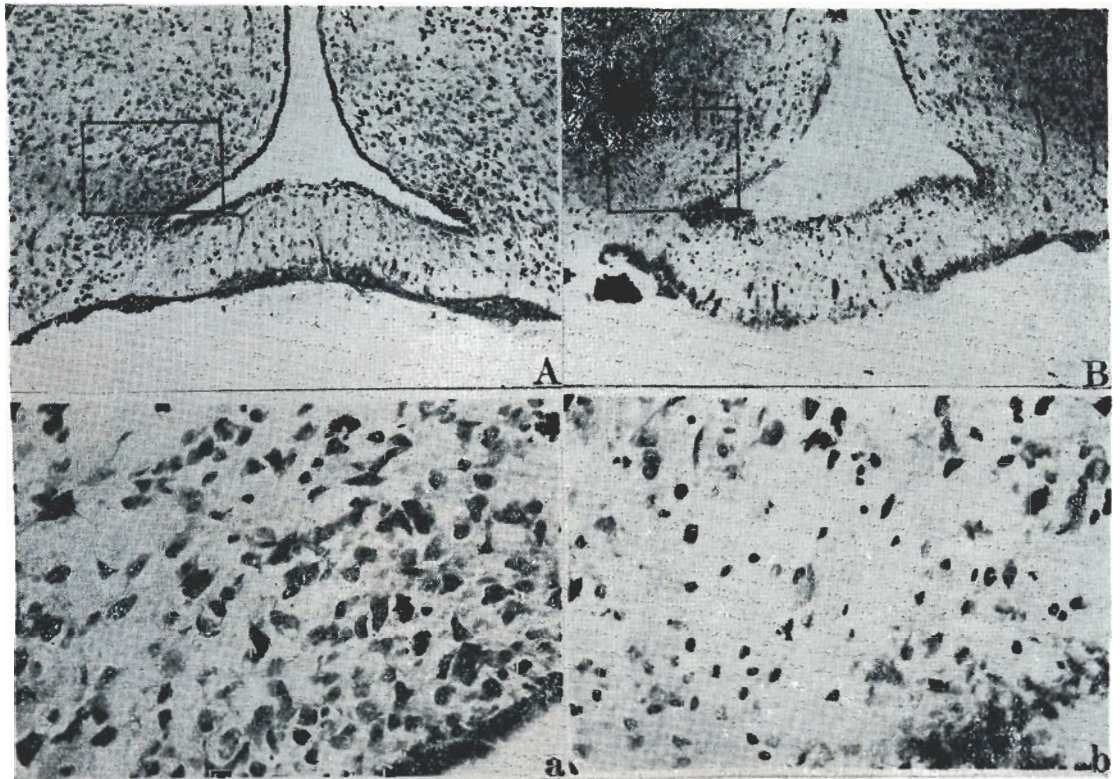


Fig 1. Neurons in hypothalamic arcuate nucleus of monosodium glutamate-treated rats (B, b) were significantly less than those of control rats (A, a). A, B × 64; a, b × 320.

(See p 104)