# Deep Lumbar Neurons Control Ejaculation

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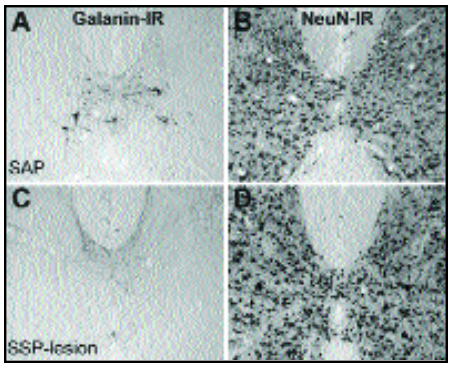


**Fig. 2** Photomicrographs of galanin-IR (A, C) and NeuN-IR (B, D) in L4 of a representative SAP(Aand B) and SSP-les (C and D) animal. Galanin is visibly reduced, while NeuN labeling shows no reduction in SSP-les males.

SSP-SAP treatment resulted in two groups, rats with complete lesions of LSt neurons (SSP-les; defined by less than 1/3 of the number of LSt cells observed in untreated rats), and rats with incomplete or misplaced lesions (SSP-il). No lesions were present in SAP-treated males (SAP). SSP-les animals had fewer galanin-immunoreactive (IR; Fig. 2) and NK-1r-IR neurons than SSP-il or SAP animals. Despite the severe reduction in LSt neurons in SSP-les rats, there was no overall reduction in numbers of NeuN-IR cells (Fig. 2), indicating the selectivity of the lesions. Furthermore, the lesions were restricted to the area surrounding the central canal and did not affect the number of NK-1r expressing neurons in the dorsal horn.

LSt lesions had dramatic effects on sexual behavior. Lesions completely disrupted display of ejaculatory behavior in SSP-les males and seminal plugs were uniformly absent upon examination of the female partner throughout the testing session. In contrast, SSP-il and SAP males continued to ejaculate regularly following surgery. Furthermore, ablation of LSt neurons selectively blocked ejaculatory behavior without affecting other components of sexual behavior. SSP-les animals did not differ from the SSP-il or SAP animals in number of mounts or intromissions (Fig. 3).

It is well established that ejaculation is a reflex and ejaculatory reflexes remain intact when control by supra-spinal sites is eliminated, suggesting the existence of a spinal ejaculation generator. However, the anatomical site of such an ejaculation generator is yet unknown. Our data suggest that LSt cells form a critical component of the ejaculation generator.

To further test this hypothesis, effects of LSt cell lesions on expression of ejaculatory reflexes were investigated. We utilized a model that reliably mimics ejaculatory responses in anesthetized, spinalized male rats, i.e. the urethro-genital reflex model.[3] In short, urethral stimulation induces characteristic

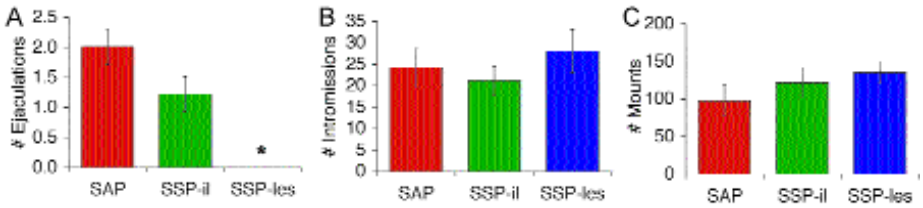


**Fig. 3** LSt neuronal lesioning abolishes ejaculatory behavior but has no effect on number of mounts or intromissions. Presented here are group means ± SEM of numbers of ejaculations (A), intromissions (B), and mounts (C).

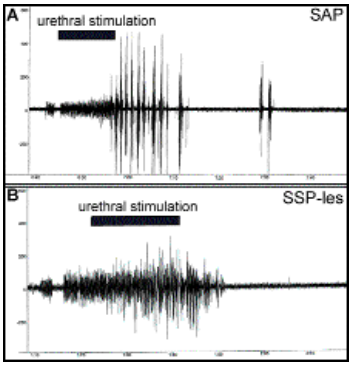


**Fig. 4** EMG of BCM bursting in urethral genital reflex model. Characteristic bursting patterns evoked by urethral distention observed in SAPcontrol rats (A) are disrupted in LSt lesioned rats (B).

organized bursting patterns of peripheral nerves, smooth muscles and striated muscles, including the bulbocavernosus muscle (BCM). In the present study, urethrogenital reflexes were investigated in males with complete and incomplete LSt lesions, and SAP controls. LSt cell lesions were performed using SSP-SAP as described above and BCM bursting patterns were monitored. LSt lesions dramatically disrupted ejaculatory reflexes. In SSP-les males, urethral stimulation resulted in asynchronous, low amplitude, short bursts of the BCM (Fig. 4B). In contrast, urethral stimulation resulted in a characteristic organized and synchronized series of 8 to 12 BCM bursts in SSP-il and SAP control males (Fig. 4A). Together these data demonstrate that LSt cells play a pivotal role in control of ejaculatory reflexes and behavior.

**REFERENCES**

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2. Truitt WA, Coolen LM (2002) *Science* 297(5586):1566-1569.
3. McKenna KE, Chung SK, McVary KT (1991) *Am J Physiol* 261(5 Pt 2) R1276-R1285.

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