

Monoaminergic modulation of pathways mediating sensory-evoked PAD in the hemisectioned spinal cord of the mouse

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This study was aimed at investigating the monoaminergic control on neural networks interposed in primary afferent depolarization (PAD) pathways in the juvenile mouse. In the hemisectioned spinal cord preparation with hindlimb attached, we examined the effects of the monoamines 5-HT, DA and NA on sensory-evoked PAD, intraspinal microstimulation ( $\mu$ Stim)-evoked PAD, and excitability of sensory intraspinal terminals. PAD was inferred from dorsal root potentials (DRPs) recorded at L4 and elicited by electrical stimulation of low threshold sensory afferents of selective hindlimb nerves, or by  $\mu$ Stim ( $<10 \mu\text{A}$ ) applied at the intermediate zone, where maximal extracellular field potentials evoked by low threshold sensory afferents were recorded (just dorsal to the central canal). Excitability changes were inferred from the compound antidromic potential (CAP) using Wall's technique. 5-HT and DA ( $10 \mu\text{M}$ ) depressed markedly (30% of control) sensory-evoked (TIB, SU and DP, 2xT) and NA ( $100 \mu\text{M}$ ) depressed TIB-evoked DRP to 12% of control, while orthodromic afferent volleys recorded from the sciatic were not affected. 5-HT, and DA ( $50 \mu\text{M}$ ) depressed  $\mu$ Stim-evoked PAD to about 30% of control but CAPs recorded at L4 were only slightly depressed. These data suggest that monoaminergic actions occur on interneuronal pathways mediating PAD, more than directly on fibers giving, or receiving PAD. This modulation may be equivalent to the reconfiguration of short-latency FRA-induced PAD after L-DOPA injection in the cat.

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