Abstract View

MONOSYNAPTIC AND LONGER-LATENCY REFLEXES IN THE MOUSE SPINAL CORD DIFFER IN THEIR RESPONSES TO DOPAMINE AND D2-LIKE AGONISTS AND ANTAGONISTS

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The modulatory actions of dopamine (DA) on spinal cord circuits are still poorly understood, although the actions of other monoamines (e.g. serotonin and noradrenalin) have been analyzed in much detail. The only source for spinal DA comes from descending DA axons in the dorsal hypothalamus, which have a widespread projection pattern and send collaterals throughout the spinal cord. We have reported earlier that dopaminergics modulate spinal circuits in wild type and D3 receptor knockout mice, and we now show that this modulation is different for monosynaptic and longer latency reflexes.

Spinal cords of mice (C57/BL6, postnatal days 6-17) were stimulated (500 μA, 100 μs) on dorsal lumbar segments L2-L5, and reflexes were recorded from corresponding ventral roots. Monosynaptic (0-3 ms after reflex onset) and longer-latency reflex responses (5-60 ms after reflex onset) were compared with the responses during bath application of DA and D2-like agonists and antagonists.

The monosynaptic reflex response was reduced consistently by DA (1-100 μM), as well as by the D2 and D3 agonists bromocriptine, quinpirole, pergolide and PD 128907. However, the longer-latency reflex response was increased by DA concentrations up to 20 μM, and during pergolide application. The D3 antagonists GR 103691 and nafadotride both increased the monosynaptic reflex response, but had a mixed effect on the longer latency component.

Our data indicate that descending DA neurons can modulate the strength of the monosynaptic reflex amplitude using both D2- and D3-receptor pathways, while the longer latency, and presumably polysynaptic, spinal reflex circuits might be modulated by different sets of DA receptors. Unraveling these differences might have important consequences for pharmaco-therapeutical strategies that target the spinal cord.

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