Motor nerve sensitivity changes caused by N-arachidonoyl-dopamine and capsaicin in rats

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Introduction: **In vitro data proved that capsaicin and some endogenous cannabinoid lipids including N-arachidonoyl-dopamine (NADA) can inhibit the voltage gated sodium channels (VGSC). Since these ligands can also influence TRPV1 and/or cannabinoid receptors, the peripheral motoneurons can be appropriate model for selective influence of VGSC. The goal of this study was to investigate the in vivo potency of these ligands on the facial nerve-induced vibrissal muscle activity.**

Fig. 1.

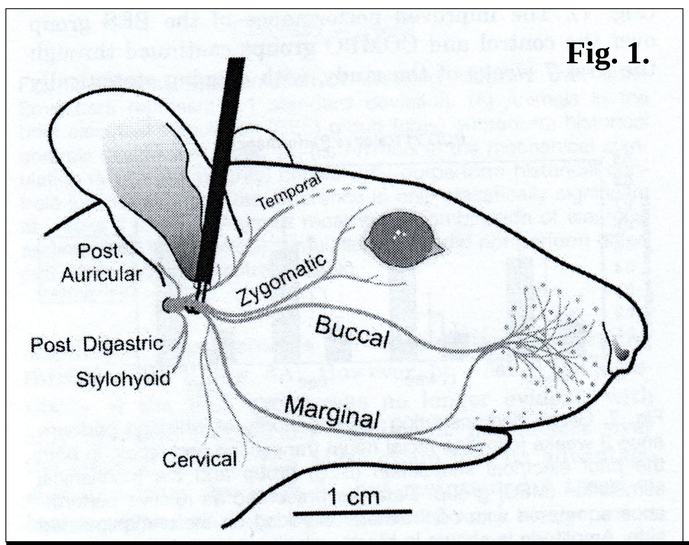


Fig. 2.

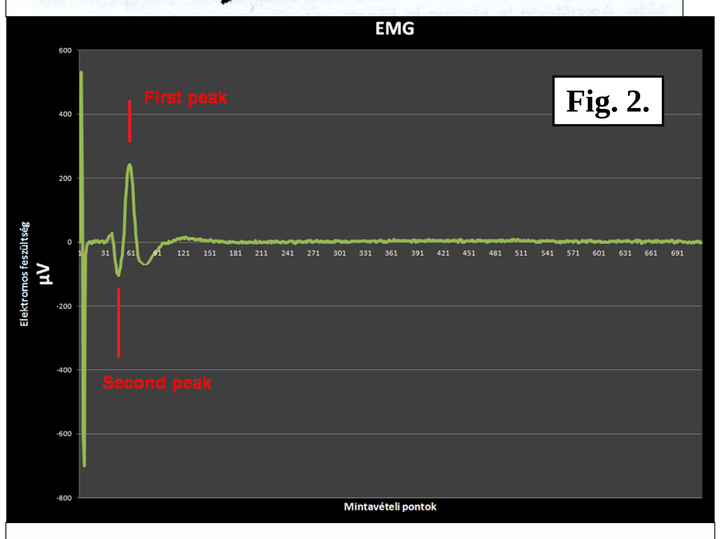
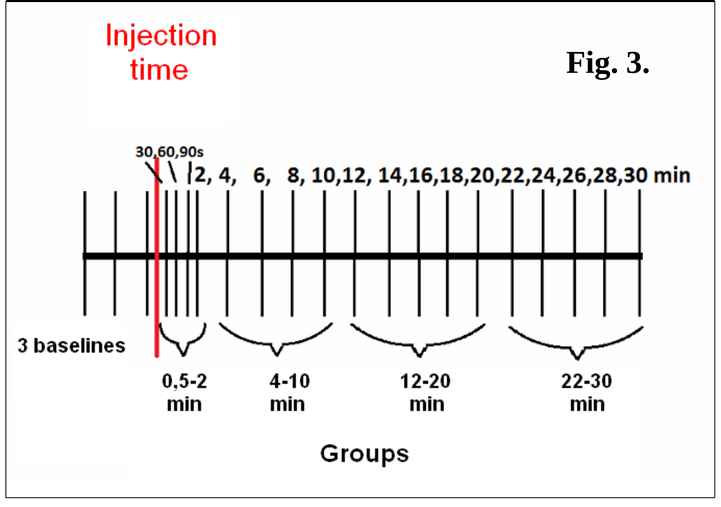


Fig. 3.



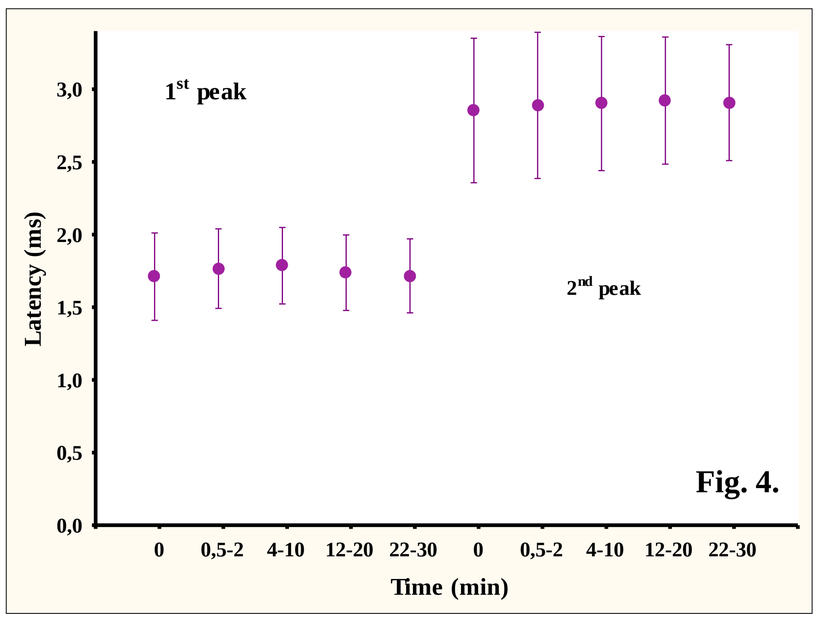


Fig. 4.

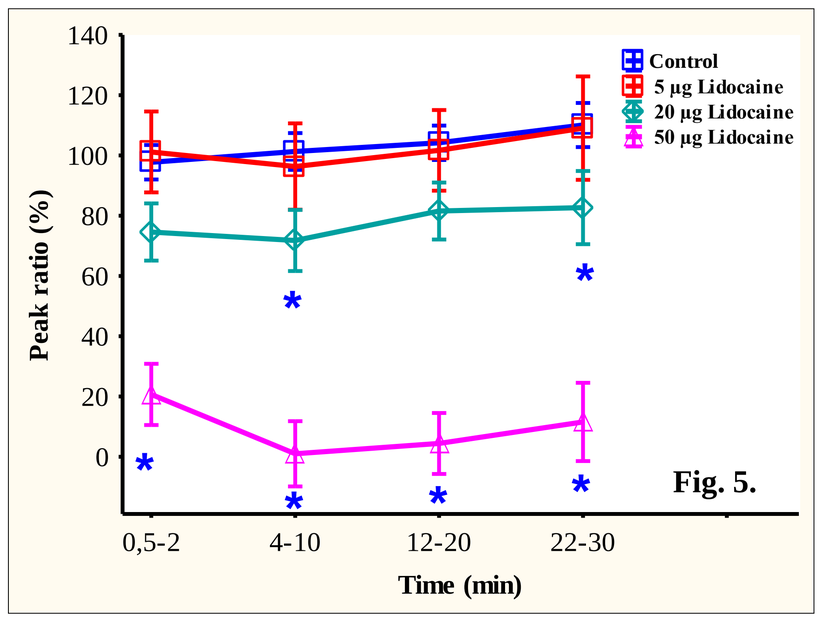


Fig. 5.

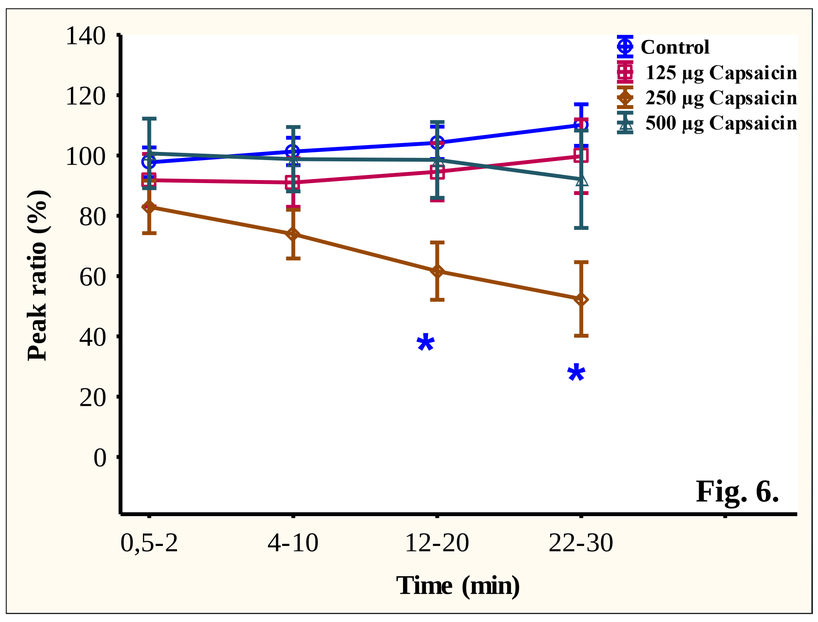


Fig. 6.

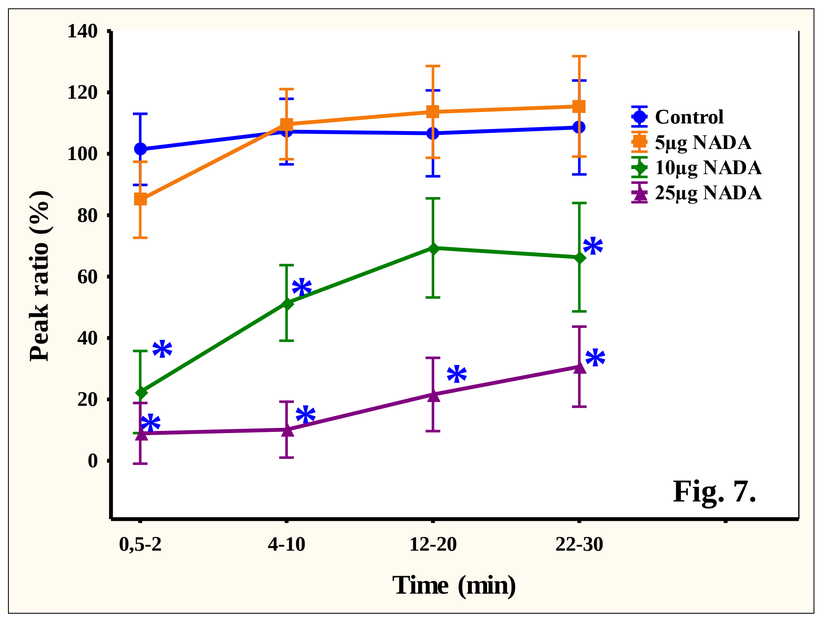


Fig. 7.

Methods: **Male Wistar rats were anesthetized with ketamine-xylazine, and facial nerve at both sides was exposed at buccal level (Fig. 1.). The nerve was wrapped around unipolar wire electrode, and electrically stimulated (1 mA for 250 µs). Action potentials of whisker muscles were recorded with unipolar needle electrodes placed into the whisker area of the rats, and the maximal amplitude was determined as differences between the highest positive and lowest negative peak (Fig. 2.). Amplitudes and peak-latencies (if there were any) were analyzed. After baseline measurement the substances were injected in the perineural sack in 10 µl volume, and repeated responses were detected for 30 min (Fig. 3.). The amplitude changes in percentage were analyzed.**

Results: **The single stimulus produced a visible whisker movement accompanied with EMG activity (Fig. 2.). Control recordings indicated that the magnitude of action potential was stabile at least for an hour, and the latency of the peaks appeared in consistent time (Fig. 4.). Injection of a vehicle did not produce significant changes in EMG activity; in contrast, injection of lidocaine (5-50 µg) evoked dose-dependent decrease in EMG activity (Fig. 5.). The highest dose of lidocaine (50 µg) produced a prolonged paralysis. NADA (5-25 µg) also produced a dose-dependent inhibitory effect on the EMG activity, and 25 µg produced a long-lasting inactivity (Fig. 6.). Regarding te effects of capsaicin, only 250 µg caused a partial decrease in the peak ratio (Fig. 7.). ED_{50} values revealed that NADA had the highest potency (37.6 nmol CI: 29.4-45.7) compared to lidocain (132.0 nmol CI: 110.1-154.0), while the ED_{50} dose of the capsaicin could not be calculated (Fig. 8.).**

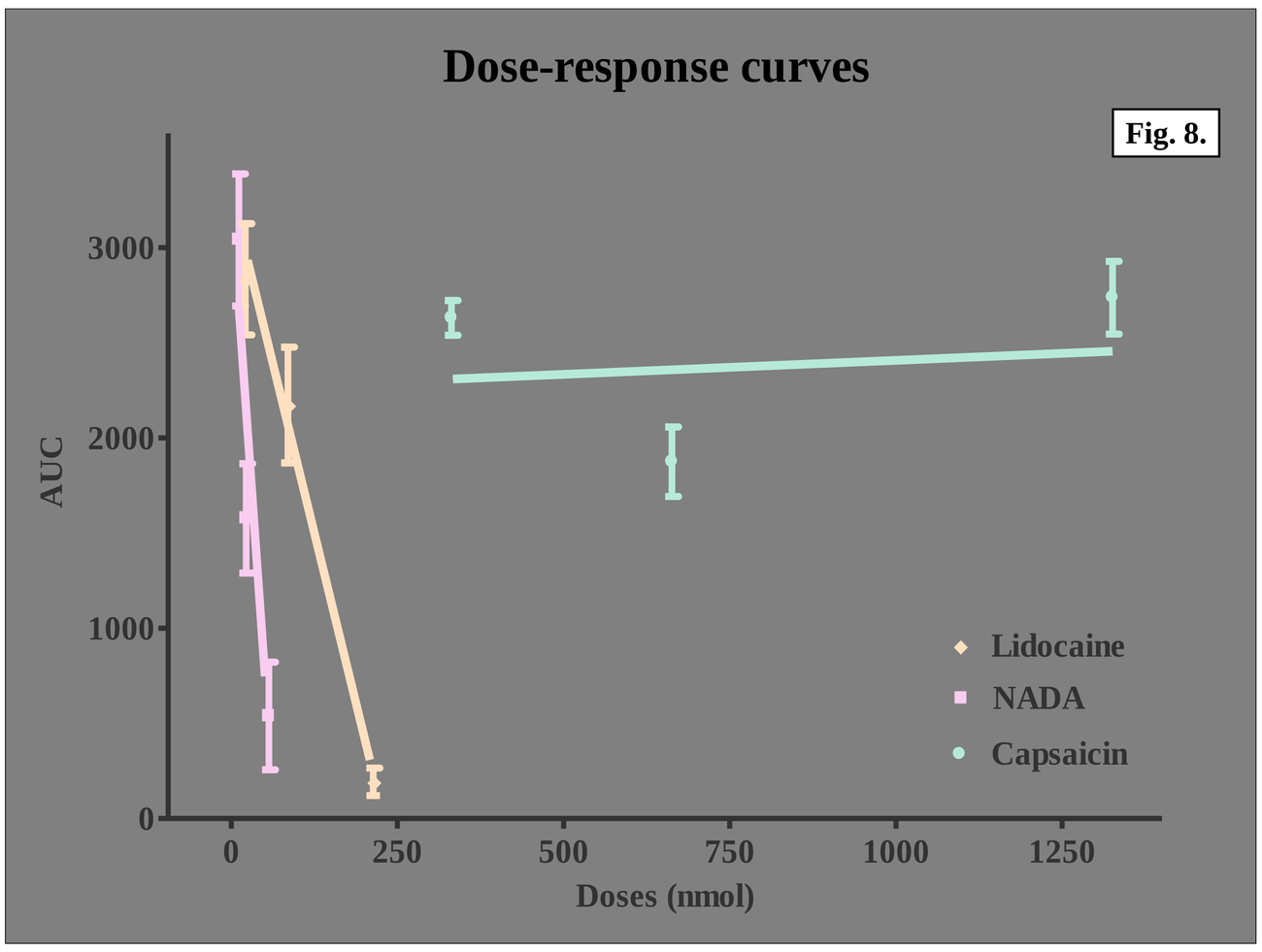


Fig. 8.

Conclusion: **These results show that intraneural injection of NADA but not capsaicin resulted in a sustained inactivation of vibrissae muscles, which may be related to the reported inhibitory effects of this ligand at the VGSC.**

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