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Effect of an inhibitor of NO-synthase on the shortening velocity of mouse muscles

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Extensor Digitorum Longus (EDL) and soleus (SOL) muscles from C57 mice were dissected out and attached to a puller in oxygenated Krebs solution at 20° C. We measured the maximum tetanic force Po and the maximum velocity of unloaded contraction Vmax using the 'slack test' method of Edman. The muscles were then treated for 30 min by Nitro-L-arginine (NLA), an inhibitor of NO-synthase, which was added to the physiological saline at a final concentration of 10 mM. •

In EDL, a fast-twitch muscle, NLA had no effect on the maximal tetanic tension Po (mN/ mm^{-2}: controls: 245 ± 15; NLA-treated: 236 ± 14; *n = 9),* but markedly depressed Vmax (muscle fibre $lengths^{-1}$: controls: 8.1 ± 0.2; NLA-treated:5.9±0.3; Δ = 2.2 ± 0.2; p < 0.001; n = 9). The effect on SOL, a slow-twitch muscle, was similar: no effect on P_o (mN mm^{-2}: controls: 184 ± 18; NLA-treated: 189 ± 17; *n = 9)* and decrease of Vmax (muscle fibre $lengths^{-1}$: controls: 5.4 ± 0.3; NLA-treated: 3.9 ± 0.2; Δ =1.5 ± 0.3; *p* < 0.001; *n* = 9).

In separate experiments, we observed that the effect of NLA was fully reversible by bathing the muscle in oxygenated normal saline for 30 min.

Since there is NO-synthase bound to the plasma membrane of type II fibres (Kobzik *et al., Nature,* 372, 546-8, 1994), we conclude that NLA depresses Vmax by inhibiting this enzyme. Therefore, it is likely that NO is able to modulate the maximum shortening of contraction, through a mechanism that remains to be established.