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The active form of vitamin D, Calcitriol, induces vasodilation in the rat aorta

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Increasingly the rapid, non-genomic effects of vitamin D are being explored, rather than the classical genomic effects that are usually induced by chronic exposure[1]. This study aimed to investigate the acute direct effects of the active form of vitamin D, Calcitriol, on contractility of isolated rat aortas.

Arterial rings from the thoracic-abdominal aorta of female Wistar rats (4 rings per rat) were equilibrated in Krebs solution (pH 7·4) gassed with 95 % O_2 and 5 % CO_2, and maintained at 37 °C and 1·5 g of initial tension for 60 min. To check contractility, each arterial ring was initially exposed to 60 mM KCl, and then washed with Krebs solution to return to the rested state. In all experiments, 3 arteries were exposed to increasing concentrations of Calcitriol, while 1 artery was used as control and the vehicle (DMSO) added instead of Calcitriol. The role of endothelial nitric oxide (NO) in the vitamin D-induced relaxation was investigated by adding the endothelial NO synthesis inhibitor, L-NAME (10^{-4}M) for 20 min prior to the addition of vehicle (DMSO) or increasing concentrations of Calcitriol (10^{-13}M to 10^{-7}M). Statistical analysis was performed using Students T-test (KCl and L-NAME responses only) or repeated measures ANOVA. $p < 0{\cdot}05$ was considered statistically significant.

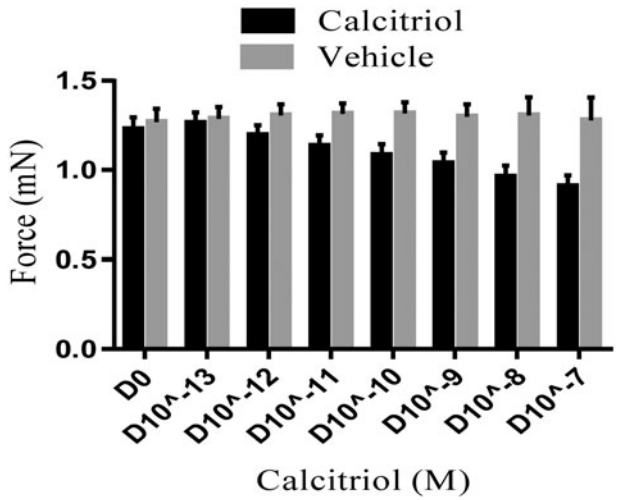

Figure 1. Dose dependent vasorelaxation induced by Calcitriol (n = 5 rats).

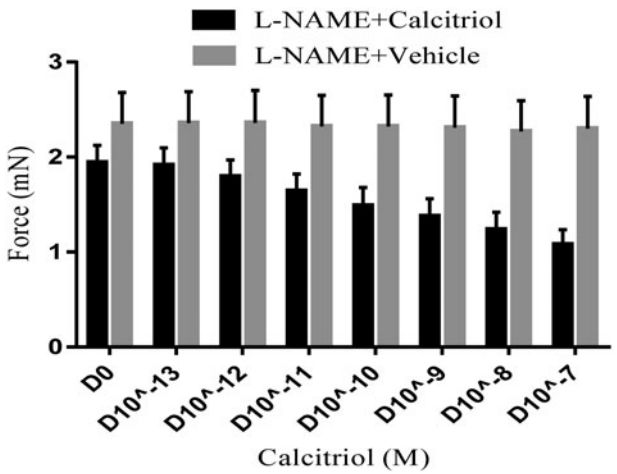

Figure 2. L-NAME does not block the Calcitriol- induced relaxation (n = 5 rats).

All vessels responded to KCl ($p < 0{\cdot}001$). Increasing concentrations of Calcitriol (10^{-13}M to 10^{-7}M) induced a dose-dependent vasorelaxation of rat arteries compared to vehicle (DMSO) ($p < 0{\cdot}001$, Figure 1), with the maximal relaxation being reached at the highest concentration (10^{-7}M). Pre-incubation of arteries with L-NAME (10^{-4}M) significantly increased contraction from the resting tension ($p < 0{\cdot}001$), but did not block the vitamin D-induced vasorelaxation ($p < 0{\cdot}001$, Figure 2).

These results provide evidence that Calcitriol elicits an acute, dose-dependent relaxation of the rat aorta. The fact that this vasorelaxation was not altered by L-NAME, indicates that this effect is not via the formation of NO.

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1. Falkenstein E, Tillmann H-C, Christ M, *et al.* (2000) *Pharmacol Rev* **52**(4), 513–556.

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