Taurine improves *tibialis anterior* force production and muscle architecture in the 28 day old *mdx* mouse model of Duchenne muscular dystrophy

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Duchenne muscular dystrophy (DMD) affects approximately 1 in every 3,500 live male births making DMD the most common and lethal genetic disease experienced by humans. Those afflicted experience progressive muscle wasting and subsequent weakness with age resulting in a loss of functional mobility and ultimately a decrease in lifespan. Taurine supplementation has been found to increase force production of skeletal muscle in adult *mdx* mice, although in adulthood *mdx* mice undergo a natural reduction in the pathology that may contribute to this improvement. No studies have assessed taurine supplementation during the period of peak damage (21-28 days of age) in *mdx* mice. Investigating taurine supplementation during this peak damage period would more accurately reflect the potential for taurine as a therapeutic treatment for DMD.

Offspring from C57BL/10ScSn (WT), *mdx* and *mdx* taurine (Tau) supplemented mice (n=10, 13, 8 respectively) were obtained and utilized for experimentation at 28 (±1) days of age. Supplemented *mdx* offspring received taurine (dose 2.5%), in drinking water for 28 days. At 28 days of age, mice were anaesthetised with Nembutal (6mg/ml) in accordance with the La Trobe University Animal Ethics Committee. *Tibialis anterior* (TA) muscles were surgically exposed, and attached to an *in situ* muscle apparatus to measure force characteristics. Mice were euthanized by Nembutal overdose post experimentation. The Table summarises the findings. Specifically, there was no difference in either body or TA mass between all treatment types ($P > 0.05$, 1-way ANOVA). Peak twitch (P_t) and specific force (sPo) were significantly lower in both *mdx* groups compared to WT control ($P<0.001$), however, both Pt and sPo were significantly higher in the *mdx* Tau groups compared to *mdx* untreated ($P<0.001$). In general TA twitch characteristics, optimum length (L_0), time to peak (TPT) and half-relaxation time (1/2RT), were not different between groups (all $P>0.05$). Visualisation of haematoxylin and eosin (H&E); staining of the contracted TA of each mouse treatment (n=4) identified extensive damage in *mdx* mice, as seen by centralized nuclei and highly variant fibre sizes, that were less prominent in the taurine treated group which more closely emulated the WT.

These findings suggest that taurine supplementation acts to increase muscular force production and improves muscle architecture in the peak damage 28 day *mdx* mice. This is an exciting prospect for the future of DMD treatment.

Table. Mass and contractile data of WT, *mdx* and *mdx* taurine mice

	Body Mass (g)	TA Mass (mg)	sPo (kN/m^2)	Pt (mN)
WT	13.8 ± 2.4	20.6 ± 3.0	149.9 ± 30.0	126.9 ± 24.6
mdx	12.1 ± 2.2	18.1 ± 3.5	45.8 ± 14.2 *	54.1 ± 14.0 *
***mdx* Tau**	12.4 ± 0.9	17.8 ± 2.5	77.1 ± 13.1 *^	99.6 ± 15.6 *^

* p<0.001, different from WT group, ^ p<0.001, different than *mdx* group

(Mean ± SD; one-way ANOVA, Newman-Keuls post-hoc test)