Taurine supplementation increases rat cardiac calsequestrin 2 protein content, while decreasing the taurine transporter protein

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Taurine (Tau) is a conditionally essential beta-amino acid with diverse physiological roles. Tau supplementation has been used to treat a range of cardiac conditions, including congestive heart failure but mechanisms remain unclear. This study examined the effect of Tau supplementation on calcium handling protein contents and on the Tau transporter (TauT) protein in rat cardiac muscle. Twelve 8 wk Sprague Dawley rats were fed Tau (2.5% w/v) in drinking water *ad libitum* and standard chow for 2 wk while 10 rats (Con) were given normal drinking water and chow. Animals were killed by anesthetic overdose (Nembutal; ~85 mg/kg I.P.) in accordance with Victoria University Animal Ethics procedures and hearts rapidly dissected. There was no difference in body mass, whole heart or left ventricular masses, nor the amount of muscle water, as indicated by the left ventricle dry mass/wet mass ratio, between Con and Tau treated animals after supplementation. Tau supplementation resulted in an increase in total protein (Con 10.6 ± 0.4 *vs* Tau 12.4 ± 0.5 μg protein/mg wet muscle, p=0.013). Western blot analysis showed that Tau supplementation increased calsquestrin 2 protein (40%; p=0.005) and decreased TauT protein (34%; p=0.0013). There was no change in SERCA2, RyR2 or NCX proteins. In conclusion, Tau supplementation resulted in an increase total protein content and calsquestrin 2, which may help explain some of the benefits of Tau in heart failure. The observed decrease in TauT protein might suggest regulation of the total Tau content that the cardiac muscle can acquire.