The aim of this study was to compare the expression of transient receptor potential vanilloid type 1 (TRPV1) channels in hypertrophic hearts from transgenic mice showing overexpression of the catalytic subunit alpha of protein phosphatase 2A alpha (PP2Ac alpha) with wild-type mice and with TRPV1-/- mice. Transcripts of TRPV1, matrix metalloproteinase 9 (MMP9), discoidin domain receptor family, member 2 (DDR-2), atrial natriuretic peptide (ANP), GATA 4, and regulatory microRNA (miR-21) were analyzed using quantitative real-time PCR. Ventricle-to-body-weight-ratio was significantly higher in PP2Ac alpha transgenic mice compared to wild-type mice and TRPV1-/- mice (8.6±1.3mg/g; 5.4±0.3mg/g; and 5.4±0.4mg/g; respectively; p<0.05 by Kruskal-Wallis test). TRPV1 transcripts were significantly higher in PP2Ac alpha transgenic mice compared to wild-type mice (1.7±0.2 arbitrary units vs. 0.8±0.1 arbitrary units; p<0.05). TRPV1 protein expression was also significantly higher in PP2Ac alpha transgenic mice compared to wild-type mice. A significant linear correlation was observed between TRPV1 transcripts and the ventricle-to-body-weight-ratio (Spearman r=0.78; p<0.05). The expression of DDR-2 was significantly higher in PP2Ac alpha transgenic mice compared to wild-type mice and TRPV1 knockout mice. The expression of miR21 was significantly
higher in PP2Ac alpha transgenic mice compared with TRPV1-/− mice (0.103±0.018 (PP2Ac alpha transgenic mice); 0.089±0.009 (wild-type mice); and 0.045±0.013 (TRPV1-/− mice), respectively; p<0.05). Masson Goldner staining revealed that PP2Ac alpha transgenic mice showed increased heart fibrosis compared with TRPV1 knockout mice. The study suggests an important role of TRPV1 in the pathogenesis of genetically associated heart hypertrophy.