manner and inhibits the ability of endothelin to produce phosphorylation of MyBP-C. These results suggest that phosphorylation of cMyBP-C may be a molecular component of the vascular endothelial cell - cardiac myocyte cross-talk. Coupled with already published work, the results also suggest that cMyBP-C phosphorylation may contribute to the regulation of the turnover of myofibrillar proteins.

2589-Pos
Comparative Effects of the Proline-Alanine Rich Regions of Human and Murine Cardiac Myosin Binding Protein-C
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The N-terminus of cMyBP-C can activate actomyosin interactions in the absence of Ca2+, but it is unclear which sequences mediate the activating effects. Herron et al. (Circ Res, 98:1290-8, 2006) found that the Pro-Ala rich region (P-A) of human cMyBP-C could activate tension in the absence of Ca2+, whereas Razumova et al. (J Gen Physiol, 132:575-85, 2008) found that murine C1 and M domains activated tension. The different results might be explained by isoform differences, especially in P-A which is only 46% identical between mouse and human cMyBP-C. The goal of this study was to determine if species-specific differences in P-A account for the different activating effects of human and murine cMyBP-C. Recombinant chimeric proteins containing the C0, P-A, and C1 domains from either human or murine cMyBP-C were engineered and their activating effects assessed using 

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