**GENOME-WIDE IDENTIFICATION AND CHARACTERIZATION OF CARDIAC HYPERTROPHY-RELATED LONG NONCODING RNAS (CH-LNCRNAS) IN MICE**

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Long noncoding RNAs (LncRNAs) are RNA transcripts longer than 200 nucleotides that lack protein-coding potential. Although thousands of lncRNAs have been identified, only a few have been linked to cardiac gene expression and function. In this study, we identified, from genome-scale RNA-seq data, 12 candidate lncRNAs associated with cardiac hypertrophy. The expression of these lncRNAs was altered in mouse models of cardiac hypertrophy induced by transverse aortic constriction (TAC)- or CnA transgene. To determine the function of these lncRNAs, we developed an adeno-associated virus serotype 9 (AAV9)-based functional screening in postnatal mice. An AAV9:cTNT vector, in which the cardiac troponin T (cTNT) promoter was used to direct cardiac-specific expression of target genes, was utilized to overexpress or knockdown candidate lncRNAs in mouse hearts. Postnatal day1 wild type or CnA transgenic pups were injected with AAV9 viruses and cardiac function was measured one and two months later. Thus far, we have tested 15 candidate lncRNAs for both gain- and loss-of-function studies. Among them, two lncRNAs were demonstrated regulating hypertrophy growth when knocked down. Finally, we identified the human homologues of CH-lncRNA through analyzing the conservation of the promoter regions of lncRNA genes. We showed that the expression of these human CH-lncRNA was dysregulated in human diseased hearts, suggesting the functional conservation of these lncRNAs in cardiac disease. Our study therefore demonstrated that lncRNAs are important regulator of cardiac hypertrophy and disease.