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Functions of *CHD7*, the disease-causing gene for CHARGE syndrome, during mammalian heart development

CHD7 encodes an ATP-dependent nucleosome remodeling factor and haploinsufficiency for *CHD7* is the leading cause of charge syndrome. Congenital heart defects are major clinical features of CHARGE syndrome; however, the underlying molecular mechanisms of CHDs in CHARGE patients remain largely unknown. Our complementary yeast two-hybrid and biochemical assays reveal that *CHD7* is a novel embryonic-heart-interaction partner of BMP R-SMADs, which are nuclear mediators of BMP signaling pathways. *CHD7* is associated in a BMP dependent manner with the enhancers of Nkx2.5 that contains functional SMAD1 binding elements. *CHD7* is required for sustaining the active epigenetic signature of Nkx2.5 regulatory elements and its proper cardiac expression. Furthermore, inactivation of *CHD7* in mice impairs multiple BMP signaling-regulated cardiogenic processes at molecular, cellular, and morphological levels. Our results support the model that *CHD7* is recruited by BMP R-SMADs to the enhancers of BMP-targeted cardiogenic genes to epigenetically regulate their expression. Impaired BMP activities in embryonic hearts may have a major contribution to the heart defects in CHARGE syndrome.

Biography

Kai Jiao has acquired his MD from Beijing Medical University in 1992 and acquired his PhD from University of Iowa in 2000. He has completed his Postdoctoral training in Vanderbilt University Medical Center, Drs. Brigid Hogan and Scott Baldwin. He started his own lab in 2005 in Dept. of Genetics, UAB, where he was promoted to Associated Professor with tenure in 2010. He has published ~40 papers in peer reviewed journals. His major scientific interest is to reveal the molecular, genetic and epigenetic mechanisms that regulate heart development and their contribution to congenital heart diseases.

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