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Bop controls ventricle formation

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Defects in cardiomyocyte differentiation and cardiac morphogenesis cause heart malformations affecting almost one in every 100 children, but the mechanisms that regulate such events remain largely unknown. In the April 1 online edition of Nature Genetics, Paul Gottlieb and colleagues from University of Texas at Austin, US, show that *Bop* encodes a muscle-restricted protein that is essential for cardiac differentiation and morphogenesis (*Nat Genet* 2002, DOI: 10.1038/ng866).

Gottlieb *et al.* used a modified subtractive hybridization approach and identified early cardiac-specific genes in chick and murine embryos. They observed that *Bop* was expressed specifically in cardiac and skeletal muscle precursors and in cardiomyocytes throughout organ development, beginning before cardiac differentiation. The protein m-Bop can interact with histone deacetylases and can function as a transcriptional repressor.

They also showed that targeted deletion of *Bop*in mice disrupted maturation of ventricular cardiomyocytes and interfered with formation of the right ventricle. In addition, normal expression in cardiomyocyte precursors of Hand2, a transcription factor essential for right ventricular development, was dependent upon m-Bop.

"Now with several genes - including Bop and Hand2 - identified as controllers of heart development, preventives [of pediatric heart problems] are finally conceivable," said Deepak Srivastava, the senior author of the paper. "The next major research steps to achieve this goal are already under way: to catalog and understand the mechanisms of all genes with critical roles in heart development and to correlate specific gene mutations with each specific heart defect in children."

References

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2. University of Texas at Austin, [http://www.utexas.edu]