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New genetic discovery improves understanding of congenital heart disease

Reviewed by Emily Henderson, B.Sc.Sep 21 2022

A new study led by the Masonic Medical Research Institute published in the journal Cells shows for the first time that a particular gene, called VGLL4, is required for embryo development but is dispensable for myocardial growth. This fact was previously unknown, and with this discovery, medical researchers now have useful new information about heart cell development, helpful for our understanding of congenital heart defects and heart failure.

Why this matters: Congenital heart disease is one of the leading causes of pediatric morbidity and mortality, which is why it is important to decipher the molecular mechanisms that control heart development. Cardiovascular development has become a crucial element of understanding congenital heart diseases, and the more we know about this, the better we can treat heart malformations.

Study summary: To understand the VGLL4 function in the heart, the authors generated two VGLL4 loss-of-function mouse lines: a germline VGLL4 depletion allele and a cardiomyocyte-specific VGLL4 depletion allele. The analysis of the embryos revealed that VGLL4 knockout embryos had reduced body size, malformed tricuspid valves, but normal myocardium and normal heart function. Read the full article from Cells, linked here: https://doi.org/10.3390/cells11182832

What's new here: This is a newly discovered function about VGLL4; this protein is needed for embryonic development, but that function is independent from and isolated from the growth of the heart's myocardial wall.

Team authors: Caroline Sheldon, Aaron Farley, Qing Ma, William Pu, and Zhiqiang Lin. Dr. Zhiqiang Lin led the team, he is a Principal Investigator at the Masonic Medical Research Institute. The research team includes colleagues from Harvard Stem Cell Institute and Boston Children's Hospital. Dr. Lin co-authored a previous study about VGLL4 in 2016, also with Dr. Pu and Dr. Ma.

Source:
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Sheldon, C., et al. (2022) Depletion of VGLL4 Causes Perinatal Lethality without Affecting Myocardial Development. Cells. doi.org/10.3390/cells11182832.