Maternal Coxsackievirus B Induced Dysregulation of SUMOylation Processes as a Potential Cause of Hypoplastic Left Heart Syndrome

Bailey Kemp
University of Minnesota, Morris, kempx192@morris.umn.edu

Sarah Severson
University of Minnesota, Morris, sever534@morris.umn.edu

Follow this and additional works at: http://digitalcommons.morris.umn.edu/urs_2018

Part of the Cardiovascular Diseases Commons, and the Congenital, Hereditary, and Neonatal Diseases and Abnormalities Commons

Recommended Citation
http://digitalcommons.morris.umn.edu/urs_2018/1
We hypothesize that a maternal asymptomatic coxsackievirus B infection causes alteration of SUMOylation patterns in the fetus, which results in functional repression of the transcription factor Nkx2.5, leading to underdevelopment of the fetal heart and ultimately Hypoplastic Left Heart Syndrome.

Coxsackievirus infection represses the function of Nkx2-5 via patterns of SUMO-1 and the related enzymes involved in SUMOylation evidence that the function of Nkx2-5 is dependent on modification by small heart, and has been linked to several congenital heart defects. Notch1. Because of this, SUMO should be explored further as a possible congenital cardiac malformation.

Coxsackievirus-induced dysregulation of SUMOylation of the vital cardiac transcription factor, Nkx2-5, is not fully defined, it is clear that alteration of the SUMOylation processes could contribute to disrupted Nkx2-5 transcriptional activity. The underlying cause for an interruption of SUMOylation is proposed to be coxsackievirus B-induced alterations to SUMOylation processes. We previously associated with CHD's were identified to have a 50-80% decrease in SUMOylation, but the mechanism underlying this is still unknown. The present study proposes a possible mechanism by which coxsackievirus-induced alterations could contribute to HLHS.

Coxsackievirus-induced dysregulation of SUMOylation processes could play a significant role in the development of HLHS.