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IDENTIFYING POTENTIAL CROSS-TALK BETWEEN THE FASTED LIVER AND DYSFUNCTIONAL HEART

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Heart disease is the leading cause of death in the United States. The goal of our research is to discover pathways and potential targets for the prevention and reversal of heart disease. Previous work from our lab has shown that trehalose, a disaccharide sugar, triggers the fasting response in cells. Additionally, recent research has shown that the liver's fasting response is therapeutic in a heart failure context. We found that trehalose protects against heart failure in mice with transverse aortic constriction (TAC), a model of heart dysfunction. Now, we are studying which mechanisms trehalose may act through to prevent heart failure. Our current hypothesis is that crosstalk between the "fasted" liver and failing heart leads to the improved condition of the heart.

We took two approaches to discovering potential crosstalk between the liver and heart: studying particular factors released from the liver and identifying potential interactions between secreted factors from the liver and receptor factors in the heart. Identifying potential interactions between the organs using RNA-sequencing data is the most promising approach, and we have identified potential interactions between the organs. Looking forward, we will continue to identify factors and/or groups of factors of interest and conduct validating experiments on the factors we are identifying.