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Cardiohepatic Interactions in the Pathogenesis of Heart Failure

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In heart failure, pump function is impaired, blood flow is reduced, and energy metabolism in tissues and organs throughout the body becomes dysfunctional. The liver is the largest organ in the human body and plays a central role in lipid and sugar metabolism, protein synthesis, detoxification, and bile acid production. It has long been known that there is an interaction between the heart and liver via blood circulation and the autonomic nervous system, especially in acute and chronic heart failure, which can cause acute ischemic hepatitis (shock liver) and chronic congestive liver injury. In recent years, basic research has accumulated, indicating the possibility of further interorgan communication between the heart and liver (heart-liver interaction). Hepatokine is a liver-derived hormone synthesized and secreted by hepatocytes. The liver secretes hepatokine to regulate glucose and lipid metabolism throughout the body, and its effects on the heart have recently been reported as a target organ of hepatokine. Selnoprotein P (SeP), a hepatokine, is rich in selenocysteine and has been reported to function as a transport protein for the trace element selenium.

In a mouse model of myocardial ischemia-reperfusion, we found that myocardial infarct size was significantly reduced in SeP-KO mice. Mice overexpressing SeP in the liver showed an increase in myocardial infarct size, indicating the action of SeP as an aggravating factor for myocardial infarction.

In a mouse model of transverse aortic constriction (TAC), a pressure overload-induced heart failure model, we found that hepatic expression of SeP in WT was significantly increased by TAC. TAC-induced cardiac hypertrophy and pulmonary congestion were significantly attenuated in SeP KO compared to WT. In addition, myocardial upregulation of fetal-type genes such as atrial and brain natriuretic factor and interstitial fibrosis of the heart were significantly less in SeP KO than in WT mice after TAC. These results suggest that the absence of endogenous SeP attenuated cardiac hypertrophy, dysfunction and fibrosis in response to pressure overload in mice. SeP possibly plays a maladaptive role against progression of heart failure through the liver-heart axis.

SeP secreted from the liver is expected to elucidate new mechanisms involved in the pathogenesis of cardiac disease and to develop therapies based on the cardiohepatic interaction.

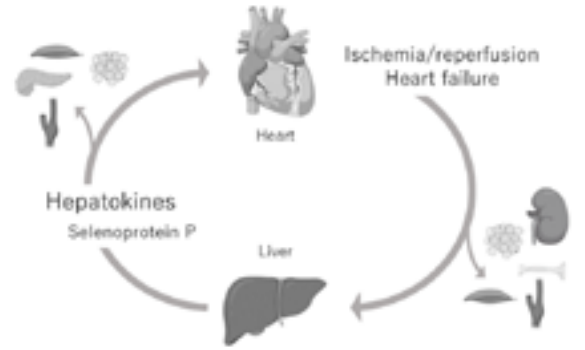

Figure 1. Cardiohepatic interactions

Biography

Soichiro Usui is an Associate Professor in Cardiovascular medicine in Kanazawa University, Japan. He is also Fellow of the European Society of Cardiology and the Japanese Circulation Society. He obtained his PhD in Medical Science from Kanazawa University, Japan. He held a postdoctoral position at the University of Medicine and Dentistry-New Jersey Medical School, USA. He is interested in molecular mechanisms of cardiac remodeling in heart failure and adipose-derived regenerative cell research. He published over 90 refereed journal papers.

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