Title: The Role of Kv1.5 Channels in Maintaining Cardiac Function During Chronic Hemodynamic Stress: The Next Chapter.

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Objective: Mice null for Kv1.5 channels (Kv1.5-/-) show accelerated development of heart failure because of insufficient myocardial perfusion to meet the elevated metabolic requirements.

Abstract: Coronary blood flow is correlated to the production of metabolites which help with the tone of the vascular smooth muscle cells (VSMC) using redox reactions. Kv1.5 channels, some of which are redox sensitive, help with the membrane potential in VSMC. These channels play a key role in connecting metabolism to blood flow in the heart. In heart failure, left ventricular wall stresses and cardiac metabolic demands are elevated. The metabolic demands can be greatly affected by Kv1.5 channels. Kv1.5 null animas have increased lung weight and increased levels of fibrosis compared to WT mice post TAC. The Kv1.5-/- mice also have higher mortality after TAC compared to the WT mice. The study suggests that the presence of Kv1.5 channels (especially those in the vasculature) play a key role in maintaining myocardial blood flow during enhanced myocardial metabolic demands. If flow is insufficient to meet the metabolic demands, then heat failure occurs.