3-P-061

Poster Sessions

Inhibition of TRPV2 prevents the progression of murine heart failure

Yuko Iwata¹, Madoka Hirayama¹, Shin Ito¹, Masafumi Kitakaze¹

¹*Dept. Clin. Res. and Dev., National Cerebral and Cardiovascular Center*

Transient receptor potential cation channel, subfamily V, member 2 (TRPV2) has been previously suggested as a principal candidate for Ca²⁺ entry pathways, and it seems to be a potential therapeutic target for heart failure, such as dilated cardiomyopathy (DCM). However, there are no known TRPV2-specific inhibitors to treat DCM. Here, we produced the antibodies against TRPV2, which interact with the TRPV2 extracellular site and inhibit the Ca²⁺ influx via TRPV2. Since these antibodies had no effects on the other members of the TRP family, such as TRPV1 and TRPC1, we tested the therapeutic efficacy of TRPV2 inhibition in the murine heart failure of the 4C30 DCM model or the transaortic constriction (TAC)-induced model. We observed that the treatment with an antibody by intraperitoneal injections at a dose of 0.25-1 mg/kg once a week for 2 weeks prevented the progression of cardiac dysfunction and myocardial injuries in DCM mice and TAC mice, as evaluated by echocardiography and tissue Masson's trichrome staining. We conclude that the cardioprotection afforded by the antibody against TRPV2 proves the importance of TRPV2 in the pathophysiology of heart failure and may be a promising treatment for patients with heart failure.