

Molecular analyses of heart tissue in mice lacking mitochondrial protein p13

Satomi Hara¹, Norihito Shintani¹, Hiroki Ueno¹, Yohei Morota¹, Sae Ogura¹, Naoki Inoue¹, Hitoshi Hashimoto^{1,2,3,4,5}

¹Lab. Mol. Neuropharmacol., Grad. Sch. Pharmaceut. Sci., Osaka Univ., ²Mol. Res. Ctr. Children's Mental Dev., United Grad. Sch. Child Dev., Osaka Univ., ³Div. of Biosci., Inst. for Dataability Sci., Osaka Univ., ⁴Transdimensional Life Imaging Div., Inst. for Open & Transdisciplinary Res. Initiatives, Osaka Univ., ⁵Dept. Mol. Pharmaceut. Sci., Grad. Sch. Med., Osaka Univ.

p13 is mitochondrial protein highly expressed in heart tissue. Recently, our unbiased compound screen has shown that some cardiogenic drugs change p13 mRNA expression in vitro, suggesting p13 may play a role in cardiac function. To reveal the role of endogenous p13 in cardiac function, here, we investigated histological changes, mitochondrial complex 1 activity, and mRNA expression levels of mitochondria-related genes in the heart of p13 knockout (p13-KO) mice. Although no apparent abnormalities were observed in the weight and histology, complex 1 activity was significantly reduced in the p13-KO heart. In addition, mRNA expression levels of apoptosis-related genes, such as Bcl-xL, were significantly reduced in the p13-KO heart. These results suggest that endogenous p13 may be involved in energy metabolism and apoptosis in heart tissue.