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Physiological role of gp130 receptor in newborn mouse cardiomyocyte development

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Mammalian ventricular cardiomyocytes (VCM) are still premature at birth and continue to proliferate and differentiate by maturating mitochondria and excitation-contraction coupling system for a certain period after birth. In the heart at this period, the concentration of various humoral factors changes. However, it is still unknown which of them are responsible for the VCM development. Here, we examined a role of gp130, a main subunit of receptors for the IL6-family of cytokines in this process. A specific gp130 inhibitor, SC144 (3 mg/kg) or its vehicle was subcutaneously injected to mice daily from day 1 to 20 after birth. SC144 significantly increased the heart and lung weights as compared with vehicle. Although SC144 did not cause arrhythmia, it induced a significant decrease in the contractility of the left ventricle (LV) with thinning of the LV wall without dilation of the inner diameter as assessed with echocardiogram and histological analysis. In isolated VCM, SC144 significantly shortened their longitudinal and disorganized T-tubular structure. SC144-treated VCM exhibited a significantly reduced peak amplitude of L-type Ca^{2+} channel currents and twitch Ca^{2+} transients compared with the control. Taken together, these results suggest that gp130 plays crucial roles in the VCM development in a mouse perinatal stage.