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Cardiac Development and Cell Cycle

Keum Nim Koh, Seog Jae Lee, Ho Keun Lee, Ji Eun Ahn, Jae Chol Kim, Ki Chan Ha, Soo Wan Chae, and Gou Young Koh*

Cardiac Regeneration Group and Institute of Cardiovascular Research, Chunbuk University

The molecular mechanisms that arrest cardiomyocytes in the cell cycle during postnatal period remain largely unknown. The activity of CDKs control cell cycle progression, and this activity is regulated positively and negatively by association of CDKs with cyclins and cyclin dependent kinase inhibitors (CKIs) respectively. In the first part, we will present the changes of expression of cyclins, CDKs and CKIs, and the activity of each CDK in cardiomyocytes during development. There were differential and dramatic decreases of mRNA and protein levels of cyclin, CDK and CKIs, and CDK activities in cardiomyocytes during the neonatal period. While the INK family of CKIs is not detectable in hearts, the KIP/CIP family of CKIs is detectable in most organs including the heart. The mRNA and protein levels of p21CIP1 and p57KIP2 were readily detectable in hearts at gestational and early postnatal periods and decreased thereafter. The protein levels of p27KIP1 increased significantly in the early postnatal period, then were expressed persistently, though levels decreased slightly in the adult period. Variable immuno-staining patterns of p27KIP1 were observed at different periods of development and in various locations in myocardium. In the second part, our application for studying cardiac development using zebrafish animal model will be presented. In addition, we will show our novel and efficient transgenic strategy in zebrafish for targeted gene expression into the heart.