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Meis1 regulates of post-natal cardiomyocyte cell cycle arrest

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We recently showed that the neonatal mammalian heart is capable of complete regeneration following resection of the entire ventricular apex. This regenerative response is mediated by proliferation of preexisting cardiomyocyte, and subsides after the first week of life. In an effort to determine the mechanism of cardiomyocytes cell cycle arrest after the first week of life, we performed a gene array after cardiac injury at multiple post-natal timepoints. This enabled us to identify Meis1 as a potential regulator of neonatal cardiomyocyte proliferation. Meis1, which belongs to the TALE family of homeodomain transcription factors, is required for normal hematopoiesis and cardiac development. While Meis1 has been extensively studied in the hematopoietic system, little is known about its role in the heart. Our results indicate that Meis1 expression and nuclear localization in the post-natal cardiomyocytes coincides with cell cycle arrest. To further explore this pattern, we generated a cardiomyocyte-specific Meis1 knockout mouse, and showed that loss of Meis1 results in robust cardiomyocyte proliferation in the adult heart. Moreover, we determined that INK4b–ARF–INK4a locus genes and p21; synergistic cyclin dependent kinase inhibitors that induce arrest at all three cell cycle checkpoints, require Meis1 for transcriptional activation. These results identify Meis1 a key regulator of post-natal cardiomyocyte cell cycle arrest, and a potential therapeutic target for cardiac regeneration

Biography

Sadek obtained his medical degree from Ain Sham University in Egypt, and his PhD in Cardiovascular Physiology from Case Western Reserve University in Ohio. He completed clinical training in Internal Medicine and cardiology at the University Hospitals of Cleveland, and a post doctoral-fellowship in stem cell biology at UT Southwestern Medical Center. Dr. Sadek is currently an Assistant Professor at UT Southwestern Medical Center in the Division of Cardiology. His research focuses on the interplay between metabolism and cell cycle regulation. The Sadek lab is funded by grants from the NIH and the AHA

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