Title:	Investigating the role of Hdacs, Sox2 and Notch Signaling during Zebrafish Heart Regeneration
Authors:	Viswanathan, Praveen Kumar (/jspui/browse?type=author&value=Viswanathan%2C+Praveen+Kumar)
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Abstract:	Millions of people around the world die every year due to cardiovascular diseases, the most common one being myocardial infarction (MI). MI results in the irreversible loss of heart muscle cells caused by prolonged myocardial ischemia. Unlike humans, teleost fish like zebrafish can regenerate its heart completely, restoring structure and function. Human hearts cannot regenerate because of the inability of human cardiomyocytes to re-enter cell cycle and proliferate. Zebrafish cardiomyocytes can de-differentiate, proliferate and re-differentiate to replace the lost cardiomyocytes. By understanding how heart regeneration occurs in zebrafish, one can obtain valuable insights into how regeneration can be augmented in injured human hearts. The main aim of this study was to understand how pluripotency factors and epigenetic modifiers are regulated during heart regeneration. Our study shows that HDACs may play crucial roles in heart regeneration in zebrafish. Many pluripotency and epigenetic factors are regulated during heart regeneration. This study shows the upregulation of hdac1, sox2 and her4.1 after heart amputation. We have also shown that Notch signaling positively regulates the expression of Hdac1 and Sox2, while Hdacs/Hdac1 positively regulates the expression of Sox2. The pro-proliferative role of Hdac1 and Notch signaling is also shown. In addition, Oct4 is regulated by both Notch signaling and Hdacs, just like Sox2. We have hypothesized a short regulatory pathway, that is exclusively active in regenerating hearts.
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