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Canonical Wnt Signaling and Its Role in Cardiac Arrhythmogenesis

Tiankai (Kevin) Yin

Mentor: Stacey Rentschler

The Canonical Wnt signaling pathway is a key component of cardiac development, and our lab has previously seen that manipulation of this developmental pathway can lead to phenotypes of arrhythmogenic cardiomyopathy, with a slowed right ventricular conduction velocity and increased susceptibility to ventricular tachycardia. Although the adult left and right ventricular myocytes have different developmental origins, the differences in their physiological properties at baseline and in response to stress have not been well studied. We aimed to look at the role of Wnt in the development and maturation of the heart and differential effects through analysis of adult left and right ventricles where Wnt has been inactivated during development (Wnt Loss of Function (LoF) mice: *Mlc2v*$^{Cre/+}$*; Ctnnb1* $^{DM/Flox}$). In electrophysiology experiments performed on these mice, we see a slowed conduction velocity in the right ventricle, and an increased susceptibility to ventricular tachycardia. Previous studies have shown that connexins, sodium channels, myocyte cell size, and fibrosis determine cardiac conduction velocity. I performed immunostaining and Western Blots, and found that myocyte cell size and fibrosis were not affected, while there is a decrease in the Connexin 43 protein in the right ventricle of the Wnt LoF mouse hearts. Interestingly, connexins levels were unchanged in the left ventricle, and conduction velocity was also unaffected. These results suggest that changes in the electrical substrate in the right ventricle is likely responsible for the slowed conduction velocity and therefore could possibly increase susceptibility to ventricular tachycardia.