Non-technical summary of the project:

Cardiac diseases take the lives of 17.7 million people every year. Several congenital cardiac disorders, also called channelopathies, are linked to an alteration of potassium, calcium or sodium channel activity, such as: long QT syndrome (LQTS), Brugada syndrome (BrS), and Catecholaminergic polymorphic ventricular tachycardia (CPVT). These channelopathies are associated with cardiac arrhythmias and sudden death.

The main goal of our project is to find a specific therapeutic approach for the LQTS that results from a loss of function of the cardiac potassium channel, hERG. To find such a therapy, it is necessary to understand the molecular mechanisms governing ion channels activity.

In our previous work investigating hERG, using peptides which mimic two critical regions of the channel, we i) determined its opening and closing mechanism and also ii) identified hERG-specific activator peptides. We will initiate a collaboration with Stanford University’s Cardiovascular Institute through a visit of the junior researcher, Olfat Malak, to Dr. Wu’s lab. She will test the hypothesis that “similarly to genome editing, the hERG activator peptides will specifically correct the abnormal electrical activity of patient-specific induced pluripotent stem cells derived cardiomyocytes (iPSC-CMs), presented with LQTS”. This joint project will establish a successful long-term collaboration between both institutions.