According to the NIH Cancer Seer data, approximately 6% of men and women will be diagnosed with lung cancer (LC) during their lifetime, and only 21.7% of these patients will survive for 5 or more years. Decades of research significantly increased our understanding of the clinical and molecular subtypes of this disease, resulting in development of the novel therapies that can improve the outcomes for some patients. Still, LC remains a leading cause of cancer deaths in the US; therefore, more research is needed for better prevention, diagnosis, and treatment of LC.

In our previous studies of the mechanisms that regulate cell division, we identified a group of proteins that assemble in a complex called the DREAM. This complex binds to regulatory elements on DNA, and suppresses the production of factors required for cell division. We hypothesized that if DREAM complex is disrupted, the cells could produce more of these factors, which could promote cancer growth. Indeed, there is evidence that components of the DREAM are targeted by genomic alterations in LC, and that disruption of DREAM can promote an aggressive LC growth. To test our idea, we created a genetically modified mouse lacking DREAM complex. Here, we propose to use this novel mouse model to determine the role of DREAM in lung cancer pathogenesis and progression. The anticipated outcomes of our pilot study include a better understanding of the early steps of LC pathogenesis, and characterization of an innovative animal model of LC for the future research.