II. Abstract:

Redox homeostasis occupies a pivotal role during stress erythropoiesis. Selenium (Se), an essential micronutrient derived from the environment, functions as a redox gatekeeper through its incorporation into selenoproteins as selenocysteine (Sec) to exert broad redox regulatory activities. However, the influence of Se on stress erythropoiesis is not clear. In this study, we will investigate how Se deficiency affects erythropoiesis under stress condition in hemolytic anemia mouse model where we will closely evaluate hematologic kinetics of the recovery process. The first part of the study will focus on the intrinsic development of erythrocytes during anemic stress and Se deficiency. Both early progenitor stage and late erythroblast maturation stage will be examined phenotypically and mechanistically to identify when and where Se deficiency directly disrupts stress erythropoiesis. The second part of the study will investigate whether selenoproteins play a role in regulating erythropoietic microenvironment, which might indirectly affect stress erythropoiesis. **Our central hypothesis is Se deficiency impairs effective stress erythropoiesis through disrupting early progenitor stage and late erythroblast maturation process, for which selenoprotein deficient "erythropoietic niche" is partially responsible due to ineffective signaling by macrophages. The study will provide new insights into roles of Se/selenoproteins in stress erythropoiesis, as well as emphasize the importance of redox functioning in erythroid homeostasis.**