"Genome Wide Mapping of FoxO1 Binding-sites *in vivo* and Functional Study of FoxO1 Target Genes in Mouse T Lymphocytes"

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Funding Category: A

Abstract: FoxO1 is a forkhead transcription factor that has important functions in the regulation of the expression of genes involved in diverse cellular processes including cell metabolism, proliferation, differentiation, and apoptosis. A recent study has revealed that FoxO1 and the other two FoxO-family transcription factors, FoxO3 and FoxO4, are redundant tumor suppressors; the absence of all three factors leads to the development of T cell lymphomas and hemangiomas in mice. We have found that FoxO1 is induced in T cells by transforming growth factor-beta (TGF-beta), a cytokine critically involved in the regulation of T cell transformation, homeostasis, and tolerance. Using a conditional knockout mouse model, we have shown that FoxO1 plays a non-redundant role in the control of mature T cell quiescence and homeostasis *in vivo*. To further understand the molecular mechanisms by which FoxO1 regulates T cells, we plan to define the genome-wide FoxO1 binding-sites and FoxO1 target genes in naïve T cells using the recently innovated ChIp-Seq/ChIp-chip, mRNA and microRNA array techniques, and advanced computational programs. In addition, we will investigate the functions of selected FoxO1 target genes in T cells using established immunological assays. These studies will help to generate insights on the genetic network by which FoxO1 regulates T cell homeostasis and tumorgenesis.