Bishop Lab Projects

Thaddeus Allen

The characteristics of tumor initiating cells in the lung remain ambiguous. One possibility is that lesions in different linage-restricted precursors give rise to distinct tumors. Alternatively, different genetic lesions could predetermine the final tumor phenotype. Both mechanisms may function coordinately. Classically lung cancer has been divided into two main histological groups, non-small cell lung cancer (NSCLC) and small cell lung cancer (SCLC). Unlike K-ras mutations, which are observed only in NSCLC, MYC activation has been observed in both subtypes. Currently, I am constructing multiple models of MYC induced lung cancer. Through analysis of these models I hope to determine the characteristics of cells transformed by MYC activation. Send an e-mail to Thaddeus Allen [1]

Jian Qu

I am interested in understanding the molecular circuitry governing self-renewal of normal and cancer stem cells. Through targeted gene deletion and transgenic over expression, I hope to identify and characterize novel proteins that are causal in controlling the homeostasis of normal stem cells and the pathogenesis of cancer stem cells in vivo. My ultimate goal is to exploit these proteins as stemness markers, and to provide knowledge helpful for the development of effective stem cell therapy and cancer therapy in the future. Send an e-mail to Jian Qu [2]

Dun Yang

Survivin is widely expressed in human cancers but undetectable in majority of differentiated adult tissues. Survivin expression correlates poor prognosis and resistance to some anticancer drugs. Consequently, understanding the function and expression regulation of survivin should provide valuable information. Survivin has been proposed to function in the inhibition of apoptosis during mitosis. There are also reports that survivin is a chromosome passenger that is involved in mitosis and cytokinesis. Although survivin is widely studied, there are many questions that remain about this important protein: How survivin could function as an inhibitor of apoptosis? What are the exact functions of survivin in cell cycle progression, cytokinesis and the mitotic checkpoint? Could survivin cause transformation by itself or by cooperating with a known oncoprotein? What are the interacting partners of survivin that are involved in survivin's many functions in cells? How differential expression of survivin between normal and tumor cells is regulated? The search for answers to these questions is the basis for much of the work currently under my investigation. An important approach is to use RNA interference to specifically silence gene expression in cultured mammalian cells. Cellular and molecular analysis of the altered phenotypes provides new information about the normal processes. Send an e-mail to Dun Yang [3]

UCSF Main Site

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