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### ***Research Interests***

Our laboratory discovered that inducible cAMP early repressor (ICER) acts as a tumor suppressor gene product that inhibits cell growth, anchorage-independent cell growth, tumor formation in nude mice, and the expression of several growth-related genes. ICER functions as a transcriptional repressor and is the product of the cAMP responsive element modulator gene. Recently, our laboratory has found that ICER protein expression is regulated by phosphorylation. The MAPK ERK directly phosphorylates and targets ICER for ubiquitin-mediated degradation by the proteasome. The growth and focus-forming potential is severely diminished in cells ectopically expressing the unphosphorylatable mutant form of ICER. Results from our laboratory demonstrate that ICER protein is down-regulated at S and mitosis. Several other laboratories as well as ours showed that MAPK pathway is strongly activated at mitosis. Hence, ICER regulation during the cell cycle by the MAPK pathway may have important implications in cell growth and tumorigenesis. These results lead us to postulate the following hypothesis: *The deregulation of ICER protein by the Ras/MAPK pathway leads to abnormal cell cycle and cell transformation.* To test this hypothesis the following five specific aims are currently studied in my laboratory: (1) to determine if the MAPK pathway regulates ubiquitin-mediated degradation of ICER protein during mammalian cell cycle, (2) to determine how ICER regulates the mammalian cell cycle, (3) to determine if ICER affects cellular growth and development in mice, (4) to determine if the RAS oncogene targets ICER for ubiquitin-mediated degradation in cells and in a mouse cancer model, and (5) to study the regulation of ICER expression in human prostate cancer cells.

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