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Our research interest is relates to the role of androgen receptor (AR) in prostate cancer development and the cancer metastasis to bone. Prostate cancer is the most common carcinoma among male in western world except for skin cancer. Androgen, male hormone, plays a crucial role in the tumorigenesis and progress of prostate cancer. Androgen-regulated gene expression is mediated by the ligand-activated AR. Recurrent prostate cancers in patients have often AR amplification and mutations as well as are frequently metastasized to bone. The mechanisms of the tumor metastasis and cell survival in bone are unclear. We have found that bone morphogenetic protein-2 (BMP-2) necessary to bone cell proliferation and differentiation stimulates AR expression in human prostate cancer cells through a protein, heterogeneous nuclear ribonucleoprotein K (hnRNP K). HnRNP K enhances AR transcription in human prostate cancer cells and high levels of hnRNP K expression are observed in human prostate cancer specimen. Therefore, we are studying how BMP2 activates hnRNP K by protein kinase pathways and hnRNP K activates AR expression via modulating chromatin structure. Second, we are studying AR effects on dentin matrix protein 1 (Dmp1) expression. Dmp1 is an extracellular matrix protein and one of small integrin binding ligand N-linked glycoprotein (SIBINGs) family. RGD domain in Dmp1 interacts with integrins on cell membrane, stimulating cell proliferation and differentiation. Dmp1 is necessary for bone development and formation. We have found that AR activates Dmp1 expression via androgen response elements (AREs) in Dmp1 regulatory region. These signals between epithelial (prostate cancer) and mesenchymal (bone marrow) cells mutually regulate the growth of prostate cancer and osteoblast cells via BMP2-AR-Dmp1 pathways.