Revealing Vitamin D Pathways to Prostate Cancer Prevention and Treatment

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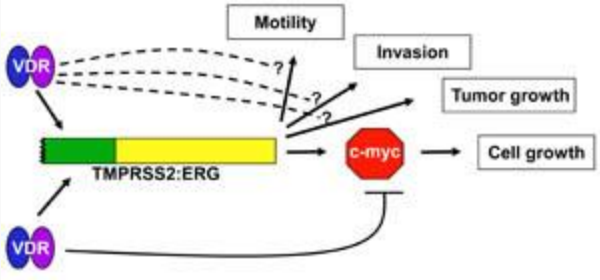
Supported by Fiscal Year 2003 (FY03) and FY08 PCRP Idea Development Awards, Dr. Nancy Weigel is discovering the mechanisms by which vitamin D, well-recognized to inhibit growth of prostate cancer cells in culture and in some animal tumor models, can best be exploited for prostate cancer prevention and treatment.

Higher exposure to sunlight (the major contributor to vitamin D production in the body) correlates with decreased risk for prostate cancer. Phase II/III clinical trials are already in progress to test the potential of vitamin D and its derivatives to prevent or delay the progression of early- and late-stage prostate cancer. Although the precise mechanisms through which vitamin D inhibits prostate cancer growth are still unclear, it has been shown that the active metabolite of vitamin D, 1,25-dihydroxyvitamin D_3(1,25D), and a highly potent analog, EB1089 (seocalcitol), bind the vitamin D receptor (VDR) in cells and subsequently turn on the expression of genes that inhibit prostate cancer cell growth. A better understanding of specific changes in gene expression and their role in prostate cancer development will provide insight toward maximizing the efficacy of vitamin D in preventing and treating the disease.

With her PCRP funding, Dr. Weigel sought to specifically identify the genes regulated by vitamin D in both normal and cancerous prostate cells. One of the genes whose expression was suppressed in non-transformed (RWPE-1), androgen-dependent (LNCaP and LAPC-4), and androgen-independent (C4-2) prostate cancer cells was c-Myc, an oncogenic transcription factor frequently overexpressed in prostate cancer. Using small inhibitory RNA (siRNA) knockdown experiments of c-Myc expression in C4-2 cells, Dr. Weigel showed that the reduction in c-Myc expression by siRNA treatment was similar to that observed with 1,25D treatment. In both 1,25D and siRNA-treated cells, there was a comparable decrease in cell proliferation. Using androgen-dependent prostate cancer cells (LNCaP and LAPC-4) treated with or without EB1089, she showed that about 25 genes were upregulated and 7 genes were downregulated by EB1089 in both cell lines. One of the upregulated genes was TMPRSS2. As this androgen regulated gene plays a role in the TMPRSS2-ETS gene fusions found in over 50% of prostate tumors and is associated with more aggressive disease, Dr. Weigel examined the effect of 1,25D on the TMPRSS2-ERG fusion gene

present in the androgen-dependent prostate cancer cell line VCaP. She found that 1,25D and EB1089 both increased the expression of the TMPRSS2-ERG fusion gene, but reduced the growth and proliferation of VCaP cells. Furthermore, vitamin D decreased the expression of c-Myc, a TMPRSS2-ERG target gene and transcription factor that is overexpressed in a variety of cancers and is a critical component for cell proliferation. Thus, 1,25D may be beneficial in prostate cancers that express the TMPRSS2-ETS gene fusions, but additional studies including studies in vivo are needed.

Dr. Weigel, a dedicated teacher who has also received FY05 and FY08 PCRP Collaborative Undergraduate Historically Black Colleges and Universities Student Summer Training Program Awards, directs the Prairie View A & M/Baylor College of Medicine SMART Summer Undergraduate Prostate Cancer Research Project, which is a 9-week summer prostate cancer research experience designed to encourage minority college students to pursue careers in biomedical science.



The vitamin D receptor (VDR) induces TMPRSS2:ERG expression, but down-regulates c-Myc, an ERG target. The TMPRSS2:ETS factor fusions, present in greater than 50% of prostate cancers, stimulate motility, invasiveness, and cell and tumor growth. Dr. Weigel's research team's finding that VDR induces TMPRSS2 led them to test the response of VCaP cells expressing the TMPRSS2:ERG fusion to VDR activation. Treatment with the active metabolite of vitamin D, 1,25-dihydroxyvitamin D3, induced TMPRSS2:ERG, but reduced cell growth and expression of c-Myc, an ERG target gene in this cell line. Whether there are additional actions of 1,25-dihydroxyvitamin D3 that counteract ERG stimulated motility, invasiveness, and growth of tumors in vivo remains to be determined.

Publication:

Rohan JN and Weigel NL. 2009. 1alpha,25-dihydroxyvitamin D3 reduces c-Myc expression, inhibiting proliferation and causing G1 accumulation in C4-2 prostate cancer cells. *Endocrinology* 150(5):2046-54.

Washington MC and Weigel NL. 2010. 1alpha,25-dihydroxyvitamin D3 inhibits growth of VCaP prostate cancer cells despite inducing the growth promoting TMPRSS2:ERG gene fusion. *Endocrinology* 151(4):1409-1417.

Links:

Public and Technical Abstracts: TMPRSS2-ERG Gene Fusions and Vitamin D Action in Prostate Cancer

Public and Technical Abstracts: Prairie View A&M/Baylor College of Medicine SMART Summer Undergraduate Prostate Cancer Research Project

Public and Technical Abstracts: Prairie View A & M/Baylor College of Medicine SMART Summer Undergraduate Prostate Cancer Research Project

Public and Technical Abstracts: Vitamin D Receptor Target Genes as Predictors of Responsiveness to Treatment with a Vitamin D Receptor

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ORNG Applications

Verify Publications

Research

research activities and funding

R01HD022061 (WEIGEL, NANCY LYNN)Jul 1, 1986 - Jun 30, 1995
NIH/NICHD
REGULATION OF PROGESTERONE RECEPTORS BY PHOSPHORYLATION
Role: Principal Investigator

R55CA057539 (WEIGEL, NANCY LYNN)Sep 30, 1992 - Mar 31, 1993
NIH/NCI
REGULATION OF HUMAN STEROID RECEPTORS BY PHOSPHORYLATION
Role: Principal Investigator

R01CA057539 (WEIGEL, NANCY L)Apr 15, 1993 - Mar 31, 2006
NIH/NCI
Regulation of Human Steroid Receptors by Phosphorylation
Role: Principal Investigator

R01CA075337 (WEIGEL, NANCY LYNN)Dec 24, 1998 - Nov 30, 2004
NIH/NCI
INHIBITION OF PROSTATE CANCER CELL GROWTH BY VITAMIN D
Role: Principal Investigator

R01DK065252 (WEIGEL, NANCY LYNN)Aug 1, 2003 - May 31, 2008
NIH/NIDDK
Coactivators and Androgen Receptors in Prostate Cancer
Role: Principal Investigator

R01CA107691 (WEIGEL, NANCY LYNN)Jun 2, 2004 - May 31, 2009
NIH/NCI
Targets of Vitamin D Receptor Action in Prostate
Role: Principal Investigator

R13CA112924 (WEIGEL, NANCY LYNN)Feb 18, 2005 - Jan 31, 2006
NIH/NCI
Conference on Hormonal Regulation of Tumorigenesis
Role: Principal Investigator

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selected publications

Publications listed below are automatically derived from MEDLINE/PubMed and other sources, which might result in incorrect or missing publications. Faculty can login to make corrections and additions.

List All | Timeline

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