



Andrew Vaughan, Ph.D.

Clinical Interests	One of the earliest steps in either the generation of leukemia, or the genesis of a solid tumor is a break in DNA that is mis-repaired. In each case, the null hypothesis is that such breaks occur at random. In the case of leukemia, it is apparent that such breaks may be targeted to specific genes by aberrant action of normal gene products such as Activation Induced Cytidine Deaminase (AID). For the generation of solid tumors, the deletion of large tracts of DNA containing tumor suppressor genes may also be regulated by local DNA secondary structure and/or epigenetic modification. Proof of cellular control over such early events in tumorigenesis would disprove the null hypothesis and open up new areas for suppressing such tumor promoting events.
Title	Professor
Specialty	Cancer, Radiation Oncology
Department	Radiation Oncology
Division	Radiation Oncology
Center/Program Affiliation	UC Davis Comprehensive Cancer Center
Education	Ph.D., Birmingham University, Birmingham, UK, 1978 M.Sc., Birmingham University, Birmingham, UK, 1975 B.Sc., Portsmouth Polytechnic, Portsmouth, UK, 1974
Professional Memberships	American Association for Cancer Research American Society for Therapeutic Radiology and Oncology Institute of Biology: Chartered Biologist Radiation Research Society of North America Royal Society for Chemistry: Chartered Chemist
Select Recent Publications	Ho B, Baker PM, Singh S, Shih SJ, Vaughan AT. Localized DNA cleavage secondary to genotoxic exposure adjacent to an Alu inverted repeat. <i>Genes Chromosomes Cancer</i> . 2012 Feb 15. doi: 10.1002/gcc.21938. [Epub ahead of print] Klein EA, Guiou M, Farwell DG, Luu Q, Lau DH, Stuart K, Vaughan A, Vijayakumar S, Chen AM. Primary radiation therapy for head-and-neck cancer in the setting of human immunodeficiency virus. <i>Int J Radiat Oncol Biol Phys</i> . 2011 Jan 1;79(1):60-4. Epub 2010 Apr 10. Chen AM, Chen LM, Vaughan A, Farwell DG, Luu Q, Purdy JA, Vijayakumar S. Head and neck cancer among lifelong never-smokers and ever-smokers: matched-pair analysis of outcomes after



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