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

Friedman DA, Tait L, Vaughan AT. Influence of nuclear structure on the formation of radiation-induced lethal lesions. Int J Radiat Biol. 2016 May;92(5):229-40.

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