Retrovirology

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Oral presentation

Inflammatory angiogenesis as a target for prevention and therapy: Kaposi's sarcoma and HIV tat as models

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Angiogenesis and an inflammatory infiltrate are conspicuous components Kaposi's sarcoma (KS). We found that this inflam-matory component may play a key role in angiogenesis, indicating that targeting inflammation is needed to completely block angiogenesis. Like many angiogenic molecules, HIV tat is also endowed with the capacity to recruit both endothelial cells and leukocytes. Angiogenesis is a common and key target of chemo-preventive molecules, a concept we termed "Angioprevention". Various chemoprevention molecules, such as flavonoids, antioxidants and retinoids, are able to repress growth of KS xenografts in vivo. These compounds act in the tumor micro-environment inhibiting the recruitment and/or activation of endothelial cells and innate immune cells, particularly granuolocytes and macrophages. N-acetyl-cysteine, the green tea flavonoid epigallocatechin-3-gallate (EGCG) and the chalchone Xantohumol (XN) all prevent angiogenesis in Matrigel sponges in vivo and inhibit the growth of Kaposi's sarcoma tumor cells (KS-Imm) in nude mice. Functional genomics analyses on the effects of these compounds in primary endothelial cells (HUVEC) in culture through Affymetrix GeneChip arrays identified overlapping sets of genes regulated by the anti-oxidants that centered on suppression the IkB/NF-kB signalling pathway. The repression of the NF-kB pathway demonstrates anti-inflammatory effects for the anti-oxidant compounds that indirectly inhibit angiogenesis, as exemplified by EGCG. Focusing on approaches that block inflammation will greatly enhance the effectiveness of anti-angiogenesis approaches in both therapy and prevention of cancer.