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Part III. Chemoprevention

Sanguinarine Inhibits VEGF-Induced Akt Phosphorylation

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Angiogenesis is the process of vascular growth by sprouting of preexisting vessels. This process impacts significantly on many important disease states including cancer, diabetic retinopathy, and arthritis. Endothelial cells receive multiple information from their environment, which leads them to progress along all stages of new vessel formation. Vascular endothelial growth factor (VEGF), in particular appears to be a master regulator of this process. This molecule interacts with cellular receptors and communicates with cell nucleus through a network of intracellular signaling, most of all by activating Akt pathway. This activation accounts for many of VEGF effects, including cell survival, migration, tube formation, and promotion of NO release. Sanguinarine (SA), an alkaloid isolated from *Sanguinaria canadensis*, is known for its antiangiogenic effects by suppressing basal and VEGF-induced new vessel growth. This article was aimed to evaluate the possible effect of SA (300 nM) on Akt phosphorylation in a porcine aortic endothelial cell line. The alkaloid significantly ($P < 0.001$) inhibited the VEGF-induced Akt increase, thus suggesting that this mode of action could be responsible, at least partially, for the antiangiogenic effect of SA.

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