Angiogenesis in Cancer; Pathogenesis and Drug Targets

Angiogenesis (also known as neovascularization) is the generation of new blood vessels from pre-existing vasculature. It is a hallmark of cancer and plays a key role in enabling tumor growth, progression, and metastasis. This poster discusses differences in normal angiogenesis physiology, and tumor vasculature, and highlights therapeutic targets aimed at suppressing angiogenesis. Researchers are now favoring a combination of strategies, which target different stages of angiogenesis in order to prevent tumor growth and expansion.

Angiogenesis

Angiogenesis is the generation of new blood vessels from pre-existing vasculature. It is a normal process in growth and development, which is required for the formation of arteries, veins, and capillaries in the embryo. Proliferation of new blood vessels also takes place in adults and is essential for the repair or regeneration of tissue during wound healing. Angiogenesis in normal tissues is a carefully regulated process, controlled by pro- and antiangiogenic factors such as vascular endothelial growth factor receptor (VEGFR) and endostatin respectively, to produce well-structured uniform vasculature.

Normal Angiogenesis

Angiogenesis is also a hallmark of cancer and plays a key role in enabling tumor growth, progression and metastasis. When a tumor develops, its size is limited by the availability of oxygen, glucose and other necessary nutrients from the existing vasculature. As a consequence of their genetic instability, tumors are heterogeneous in nature. Tumor angiogenesis can therefore differ significantly from physiological angiogenesis, producing poorly formed blood vessels with aberrant blood flow and differing permeability. In addition, factors such as a tumor’s p53 status, oxygen, glucose and other necessary nutrients from the existing vasculature. As a consequence of their genetic instability, tumors are heterogeneous in nature. Tumor angiogenesis can therefore differ significantly from physiological angiogenesis, producing poorly formed blood vessels with aberrant blood flow and differing permeability.

Tumor Vascularization

A primary trigger for the growth of new blood vessels in a tumor is hypoxia—tumor inducible factor (HIF-1) underpins a critical process of angiogenesis, which facilitates ubiquitination and its destruction. In a hypoxic environment the expression of HIF-1 α is stabilized, HIF-1 α associates with HIF-1 α, initiating transcription by binding to the response element of HIF-responsive genes, as well as binding the coactivators p300/CBP and p400 associate with HIF-1 α, increasing vasodilation and promoting angiogenesis.

Tumor Vascularization - Therapeutic Targets

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References:

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