"The tumor vasculature: functional reactivity and therapeutic implications"

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ABSTRACT

In the past decades, tumors have progressively been perceived as highly integrated systems in which the genetically unstable tumor cells and the genetically stable host cells cooperate to promote tumor growth. This view suggests that, beside tumor cells (that are targeted by conventional anticancer treatments such as radio- and chemotherapy), host cells within the tumor microenvironment can be targeted by antitumor therapy. Such alternative strategies are strongly supported by the need to overcome several limitations of the conventional therapies targeting tumor cells, such as collateral toxicity due to lack of tumor selectivity, limited tumor accessibility, and the selection of treatment-resistant variants. By contrast to tumor cells, the genetically stable host cells should not develop resistance to treatments. In this context, the observation that tumor growth is fundamentally dependent on the onset of a private tumor neovasculature (tumor angiogenesis) has revolutionized the field of cancer research. Several treatments have been developed aimed to prevent tumor angiogenesis (anti-angiogenic strategies) or to erase the existent tumor vasculature (anti-vascular approaches) supporting the survival and growth of thousands of tumor cells. However, although such therapies achieved cancer cure in animal models, they turned out to be rather inefficient when tested in patients. This can be attributed to differences in the angiogenic status between fast-growing animal tumors and slow-growing human tumors at the time of clinical detection. Another reading of the above-mentioned ...

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THE TUMOR VASCULATURE:
FUNCTIONAL REACTIVITY AND THERAPEUTIC IMPLICATIONS

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