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Endothelial cell metabolism: parallels and divergences with cancer cell metabolism

Dries Verdegem12, Stijn Moens12, Peter Stapor12 and Peter Carmellet12*

Abstract:

The stromal vasculature in tumors is a vital conduit of nutrients and oxygen for cancer cells. To date, the vast majority of studies have focused on unraveling the genetic basis of vessel sprouting (also termed angiogenesis). In contrast to the widely studied changes in cancer cell metabolism, insight in the metabolic regulation of angiogenesis is only just emerging. These studies show that metabolic pathways in endothelial cells (ECs) importantly regulate angiogenesis in conjunction with genetic signals. In this review, we will highlight these emerging insights in EC metabolism and discuss them in perspective of cancer cell metabolism. While it is generally assumed that cancer cells have unique metabolic adaptations, not shared by healthy non-transformed cells, we will discuss parallels and highlight differences between endothelial and cancer cell metabolism and consider possible novel therapeutic opportunities arising from targeting both cancer and endothelial cells.

Keywords: Endothelial cell metabolism, Cancer cell metabolism, Angiogenesis, Metabolic pathways, Metabolism, Antiangiogenic therapy, Cancer, Glycolysis

Why target endothelial metabolism?

Cancer cells are metabolically highly active and require large supplies of nutrients. Blood vessels are vital to their survival, as they not only supply oxygen and nutrients but also remove metabolic waste. Tumors excessively stimulate blood vessel growth to meet their metabolic needs. Thus, a better understanding of how blood vessels nourish tumors can offer novel therapeutic opportunities to prevent or reverse tumor progression. In the past decades, antiangiogenic strategies were primarily based on starving the tumor by destroying the vascular supply through inhibition of key pro-angiogenic molecules. Vascular endothelial growth factor (VEGF) is one of the key regulators of angiogenesis and the prime target of antiangiogenic drug development for the treatment of multiple cancers [1,2]. However, insufficient efficiency and resistance to VEGF-signaling blockade strategies limit their overall success [2-5], necessitating the development of alternative-mechanistically distinct-

strategies. We therefore proposed a new paradigm to inhibit tumor growth or other angiogenic pathologies, one that is based on starving the pathological (tumor) vessels themselves from critical metabolic fuel and energy [6,7]. We postulated that when quiescent endothelial cells (ECs) switch to rapid vessel sprouting (the so-called angiogenic switch), they adapt their metabolism to generate additional energy and biomass for growth and division. More simply, the angiogenic switch also requires an 'angiogenic metabolic switch.' We further hypothesized that metabolism might be an attractive therapeutic target, since signals from VEGF and other pro-angiogenic factors would centrally converge onto metabolism. In the likely event that cancer cells overcome inhibition of VEGF by upregulating other pro-angiogenic signals to bypass anti-VEGF therapy. metabolism remains the downstream target to increase EC proliferation for angiogenesis (Figure 1). Hence, targeting EC metabolism might offer unprecedented opportunities for the development of alternative antiangiogenic

In contrast to the vast literature on angiogenesis (nearly 50,000 papers have been published on VEGF alone), only a handful of papers have been published on how ECs adapt their metabolism when shifting from quiescence to rapid growth during vessel sprouting.

[&]quot;Laboratory of Angiogenesis and Neurovascular link, Vesalius Research Center, VB, K.U.Leuven, Campus Gasthursberg, Herestsant 49, box 912, 3000 Leuven, Belgium.



^{*} Correspondence pere connelletativiti kuleuvenibe:

[&]quot;Laboratory of Angiogenesis and Neutovancular link, Vesalius Research Center, Department of Oncology, University of Leuven, Leuven 3000, Belgium

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