Glycosylation as new pharmacological strategies for diseases associated with excessive angiogenesis

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Résumé en anglais	Angiogenesis is a complex process describing the growth of new blood vessels from existing vasculature, and is triggered by local pro-angiogenic factors, such as vascular endothelial growth factor (VEGF), which increase the metabolism of endothelial cells (ECs). Angiogenesis takes part in various physiological conditions such as embryogenesis, placental growth, and in pathological conditions such as tumor growth, diabetic retinopathy, rheumatoid arthritis (RA) and ischemic diseases. Current therapies against excessive angiogenesis target vascular growth signaling. However, tumors often counteract these therapies through adaptive mechanisms, thus novel alternative anti-angiogenic strategies are needed. Targeting metabolism is a new anti-angiogenic paradigm, especially through the inhibition of energy metabolism and glycosylation, with the perspective of maintaining the delicate balance between the beneficial and deleterious effects of excessive angiogenesis in patients. Recent studies described a role for EC glycolysis and its main regulator 6-phosphofructo-2-kinase/fructose-2,6-bisphosphatase 3 (PFKFB3) in the regulation of angiogenesis, but only few studies are related to the role of the hexosamine biosynthesis pathway during angiogenesis. Glycosylation allows the formation of glycoproteins, glycolipids and proteoglycans and impacts many pathways. The addition of glycans to N-linked proteins is catalyzed by the enzymatic activity of N-acetylglucosaminyltransferases (GnTs), which regulates the glycosylation status of key angiogenic factors such as VEGF receptor 2 (VEGFR2) and Notch. In addition, glycan-galectin (Gal) interactions regulate vascular signaling programs and may contribute to tumor adaptations to anti-angiogenic strategies. Herein, we review novel pharmacological strategies targeting glycosylation, which could be used to decrease excessive angiogenesis in pathological conditions.

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