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Hypoxia is a prominent feature of inflamed tissues; including tumors, myocardial infarcts, atherosclerotic plaques, lung of asthmatics, healing wounds and sites of bacterial infections. Certain of these conditions are also associated with increased number of mast cells [6]. In contrast to the effect of hypoxia on macrophage functions that is well documented [7], the effect of hypoxia on mast cell functions is poorly investigated. Hypoxia may regulate mast cell activation through a number of mechanisms. (1) Since mast cell activation can induce angiogenesis and mast cell precursors are found in peritumoral and tumoral capillaries [6] hypoxic conditions in tumours may provide a permissive environment for angiogenesis and mast cell function. (2) Hypoxia regulates expression of vascular endothelial growth factor (VEGF) and basic fibroblast growth factor (bFGF) by up-regulating hypoxia inducible factor 1 alpha (HIF1alpha) expression. This effect is primarily mediated by VEGF but is not restricted to VEGF. (3) Mast cells have receptors for thrombospondin (TSP) and can degranulate by exposure to this glycoprotein or a fragment of it. Thrombospondin is a major component of the extracellular matrix and a potent inducer of angiogenesis. (4) Mast cells can release ATP, a potent activator of angiogenesis. (5) Mast cells secrete vasoactive amines and can, therefore, affect nearby vessel function. Hyaluronanase is released by mast cells during degranulation [6] that can degrade hyaluronan and in turn promote angiogenesis by degrading hyaluronan in the extracellular matrix. 5ec8ef588b

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