

ANGPT1 Human

Angiopoietin-1 Human Recombinant

CYK0029

Product Overview

Name	ANGPT1 Human
Accession(Primary)	Q15389
Description	Angiopoietin-1 Human Recombinant

Target information (Q15389)

Gene Name

About Gene Symbol

Background

This factor was found in the conditioned medium of the human neuroepithelioma cell line SHEP1 and the mouse myoblast cell line C2C12ras. The cDNA encoding a protein of 498 amino acids was isolated by using a secretion-trap expression cloning procedure exploiting the presence of a signal sequence present in growth factors that are secreted by producer cells (Davis et al, 1996). Murine and human factors show 97 % identity at the amino acid level. For a related factor see: CDT6. The human gene has been mapped to chromosome 8q22.3-q23 (Cheung et al, 1998). The expression of angiopoietin-1 mRNA is downregulated by PDGF, EGF, IL1-beta, and TGF-beta (Enholm et al, 1997). Angiopoietin-1 is a ligand for the receptor-like tyrosine kinase designated TIE-2 (Davis et al, 1996). Binding of Angiopoietin-1 to its receptor induces tyrosine phosphorylation of the cytoplasmic receptor domain. A naturally occurring antagonist of Angiopoietin-1 binding to TIE-2 is Angiopoietin-2. Angiopoietin-1 also binds to the TIE-1 receptor and this interaction appears to be critical for the development of the right-hand side venous system. It is dispensable for the left-hand side venous system, suggesting that right-hand and left-hand side vascular networks are established early before asymmetrical features of the network become morphologically discernible (Loughna and Sato, 2001). Angiopoietin-1 does not directly promote the

growth of cultured endothelial cells. Angiopoietin-1 is chemotactic for endothelial cells (Witzenbichler et al, 1998). Excess soluble TIE-2 receptors abolish the chemotactic response of endothelial cells toward angiopoietin-1. Angiopoietin-1 has been shown to counteract cell death by apoptosis in cultured endothelial cells (Holash et al, 1999). Angiopoietin-1 also acts as an apoptosis survival factor for endothelial cells and this effect is augmented by the presence of VEGF (Kwak et al, 1999).

Angiopoietin-2 dose-dependently blocks directed migration toward Angiopoietin-1. Carlson et al (2001) have shown that Ang-1 binds rather selectively to vitronectin and that Ang-1 can directly support adhesion of human umbilical vein endothelial cells and fibroblasts in a process mediated by integrins. The physiologic roles of Angiopoietin-1 and its receptor are limited to angiogenic processes that occur subsequent to the earlier vasculogenic and angiogenic actions of the VEGF family and their receptors. However, it is unlike most of the known angiogenesis factors such as VEGF and other classical endothelial cell growth factors in that addition of the factor to cultures of endothelial cells does not directly promote cell growth even though the TIE-2 receptor becomes activated. Angiopoietin-1 also appears to be incapable of inducing the formation of tubules by endothelial cells. Angiopoietins can potentiate the effects of other angiogenic cytokines. An investigation of the impact of angiopoietins on neovascularization in vivo in the cornea micropocket assay of neovascularization demonstrates that neither Angiopoietin-1 nor Angiopoietin-2 alone promote neovascularization. Holash et al (1999) have shown that a subset of tumors initially grows by coopting existing host vessels. Regression of these vessels via a process that involves disruption of interactions between endothelial cells and smooth muscle cells as well as cell death by apoptosis of endothelial cells first causes loss of tumour cells before angiogenesis begins at the tumor margin and the tumor is rescued under the influence of VEGF, Angiopoietin-1, and probably other angiogenic stimuli. The embryonic expression pattern of Angiopoietin-1 suggests that it plays a particularly important role in the developing heart. Angiopoietin-1 is expressed highly in the myocardial wall surrounding the endocardium expressing TIE-2. Expression of Angiopoietin-1 becomes much more widespread later in development.