

Novel Fibroblast Growth Factor 1-Derived Peptides for Therapy and Drug Discovery

Tech ID: 24919 / UC Case 2006-088-0

ABSTRACT

This technology relates to novel peptides derived from fibroblast growth factor 1 (FGF1). These peptides inhibit cross talk between integrins and FGF receptors in a dominant negative fashion. Since FGF signaling has been implicated in tumor progression and inflammation, this technology shows potential for therapies and drug discovery in cancer biology and inflammatory diseases.

FULL DESCRIPTION

Fibroblast growth factors (FGFs) regulate diverse cellular processes including growth and differentiation. In addition, aberrant FGF signaling has been implicated in tumor progression. FGFs are also pro-inflammatory growth factors that have been implicated in promoting pathological angiogenesis in chronic inflammatory disease. Therefore, the FGF signaling pathway is an attractive target for cancer and inflammatory disease therapy. However, specific antagonists of this pathway are not well characterised.

Researchers at the University of California, Davis have developed novel peptides derived from FGF1 that can bind to FGF receptors and block integrin-FGF receptor cross talk, inhibiting FGF signaling in a dominant negative fashion. These peptides show promise as therapeutic agents and tools for compound discovery for the advancement of cancer therapy and treatment of inflammation.

APPLICATIONS

- ▶ Inhibition of angiogenesis
- ▶ Inhibition of tumor growth
- ▶ Inhibition of inflammation
- ▶ Inhibition of excessive wound healing
- ▶ Reduction of resistance of tumor cells to chemotherapeutic agents
- ▶ Development of assays to discover new compounds that affect FGF signalling

FEATURES/BENEFITS

- ▶ Can bind to FGF receptors and block integrin-FGF receptor cross talk
- ▶ Inhibit FGF signaling in a dominant negative fashion

RELATED MATERIALS

- ▶ Mori, S. and Y. Takada, Crosstalk between Fibroblast Growth Factor (FGF) Receptor and Integrin through Direct Integrin Binding to FGF and Resulting Integrin-FGF-FGFR Ternary Complex Formation. Medical Sciences, 2013. 1(1): p. 20-36. - 08/13/2013
- ▶ Mori, S., V. Tran, K. Nishikawa, T. Kaneda, Y. Hamada, N. Kawaguchi, M. Fujita, Y.K. Takada, N. Matsuura, M. Zhao, and Y. Takada, A Dominant-Negative FGF1 Mutant (the R50E Mutant) Suppresses Tumorigenesis and Angiogenesis. PLoS One, 2013. 8(2): p. e57927. - 02/28/2013
- ▶ Yamaji, S., J. Saegusa, K. Ieguchi, M. Fujita, Y.K. Takada, and Y. Takada, A novel fibroblast growth factor-1 (FGF1) mutant that acts as an FGF antagonist. PLoS One, 2010. 5(4): p. e10273. - 04/21/2010
- ▶ Mori, S., C.Y. Wu, S. Yamaji, J. Saegusa, B. Shi, Z. Ma, Y. Kuwabara, K.S. Lam, R.R. Isseroff, Y.K. Takada, and Y. Takada, Direct Binding of Integrin $\alpha_v\beta_3$ to FGF1 Plays a Role in FGF1 Signaling. J Biol Chem, 2008. 283(26): p. 18066-75. - 06/27/2008

PATENT STATUS

CONTACT

Sharron J. Thompson
srthompson@ucdavis.edu
tel: 530-754-7661.



INVENTORS

- ▶ Mori, Seiji
- ▶ Takada, Yoshikazu

OTHER INFORMATION

KEYWORDS

fibroblast growth factor 1,
FGF1, derived peptides,
block, integrin-FGF
receptor cross talk,
dominant negative
inhibition

CATEGORIZED AS

- ▶ **Biotechnology**
 - ▶ Health
- ▶ **Medical**
 - ▶ Therapeutics

RELATED CASES

2006-088-0

Country	Type	Number	Dated	Case
---------	------	--------	-------	------

United States Of America	Issued Patent	8,796,209	08/05/2014	2006-088
United States Of America	Issued Patent	8,168,591	05/01/2012	2006-088

ADDITIONAL TECHNOLOGIES BY THESE INVENTORS

- ▶ [Suppression of sPLA2-Integrin Binding for Treating an Inflammatory Condition or Suppressing Cell Proliferation](#)
- ▶ [Novel Insight into Inhibiting IGF1 Signaling](#)
- ▶ [Tumor-Suppressing Growth Factor Decoy](#)
- ▶ [Modulating MD-2-Integrin Interaction for Sepsis Treatment](#)
- ▶ [Integrin Binding to P-Selectin as a Treatment for Cancer and Inflammation](#)
- ▶ [Novel IGF2 Signaling Inhibition](#)

University of California, Davis
InnovationAccess
1850 Research Park Drive, Suite 100, ,
Davis,CA 95618

Tel: 530.754.8649
innovationAccess@ucdavis.edu
research.ucdavis.edu/u/s/ia
Fax: 530.754.7620

© 2015 - 2017, The Regents of the University of California
[Terms of use](#)
[Privacy Notice](#)