Controlling Tumor Growth And Malignancy

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ABSTRACT

Researchers at the University of California, Davis have developed a novel, non-toxic, and water soluble peptide that inhibits MARCKS activity and retains PIP2 pool to suppress PIP3 production. As a result, the peptide treatment is capable of suppressing PIP3 -mediated signaling, shrinking tumor growth, reducing metastasis to other organs and enhancing cancer cell sensitivity to therapeutic reagents including tyrosine kinase inhibitors (TKIs) and other chemotherapeutic agents.

FULL DESCRIPTION

Myrisolyated alanine-rich C kinase substrate (MARCKS), a substrate of protein kinase C, is a key regulatory molecule for cancer cell migration, invasion, and metastasis. Studies have shown elevated levels of MARCKS (as well as its signaling activity) in highly invasive lung cancer cell lines and lung cancer specimens from non-small-cell cancer patients. These associations are also found in breast and colon cancers, making MARCKS and its signaling activity a potential therapeutic target for cancer treatment.

Researchers at the University of California, Davis have shown how inhibiting MARCKS activity can shrink tumor growth and metastases. Researchers have developed a novel, non-toxic, and water soluble peptide which has been shown to shrink tumor growth and repress metastasis to other organs, as well as restore cancer cell sensitivity to therapeutic targets. Mechanistically, this peptide can reduce PIP3 pools and phospho-MARCKS levels through trapping membrane PIP2, leading to blocking PIP3-mediated signaling networks such as PI3k/AKT, PDK1, GRP1 and ARNO signaling.

In-vitro tests were performed in various cell lines derived from lung cancer, such as CL1-0/F3, CL1-5, PC9, A549 and several TKIs resistant cell lines (H1975,H1650), and cell lines derived from colon and breast cancers, including those triple negative breast cancer cell lines. These results showed anti-tumor, anti-motility, anti-invasive and synergistic effect with TKIs on cancer cells without causing toxicity to normal lung epithelial cells. *In vivo* tests were performed using subcutaneously grown tumors, or an orthotopic lung injection xenograft model. Tissue treated with this novel peptide at 50 nmoles level showed a significant decrease of metastatic nodules in the contralateral lung and other organs, essentially blocking all metastasis from the tumor to other lung sites as well as to other organs.

APPLICATIONS

- Therapeutic to suppress tumor growth and malignancy
- Enhance cancer cell sensitivity to chemotherapeutic agents, particularly enhancing TKIs sensitivity of drug resistant cells

FEATURES/BENEFITS

- Non Toxic
- Water Soluble
- Inhibition of tumor growth
- Effectiveness is at nano mole level
- Tumor shrinkage
- Decreases tumor metastasis
- Enhance therapeutic potential

RELATED MATERIALS

- American Journal of Respiratory and Critical Care Medicine, 2014, Vol.190: 1127-1138, 10.1164/rccm.201408-1505OC Targeting Myristoylated Alanine-Rich C Kinase Substrate Phosphorylation Site Domain in Lung Cancer Mechanisms and Therapeutic Implications - 10/15/2014

PATENT STATUS

Country	Type	Number	Dated	Case

INVENTORS

- Chen, Ching-Hsien
- Wu, Reen

OTHER INFORMATION

KEYWORDS

chemotherapeutic, tyrosine kinase inhibitors, TKIs, MARCKS, Myrisolyated alanine-rich C kinase substrate, protein kinase C, non-toxic, water soluble, tumor, cancer, oncology

CATEGORIZED AS

- **Biotechnology**
  - Genomics
  - Health
  - Proteomics
- **Materials & Chemicals**
  - Nanomaterials
- **Medical**
  - Disease: Cancer
  - Therapeutics

RELATED CASES

2013-698-0

United States Of America	Issued Patent	10,189,881	01/29/2019	2013-698	

ADDITIONAL TECHNOLOGIES BY THESE INVENTORS

- Disease Markers: Mucin 5B Monoclonal Antibodies
- Peptide Inhibitors of Idiopathic Pulmonary Fibrosis
- Suppression of Allergic Lung Inflammation and Hyperactivity
- Mucin-Specific Monoclonal Antibodies

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