(SD2020-447) An anti-inflammatory peptide

Tech ID: 33137 / UC Case 2021-Z08-1

ABSTRACT

UC San Diego researchers synthesized a cell-penetrating NEMOActPep where the NEMO peptide was fused to a peptide known to penetrate cell membrane. They also synthesized the corresponding mutant version where all six critical amino acids within this NEMOActPep were mutated to glycines.Currently, UC San Diego is looking for a company interested in developing US Patent Rights.

TECHNOLOGY DESCRIPTION

In response to the importance of the IKK-NF-kB signalling pathways in many diseases, many pharmaceutical companies and academic research laboratories tried to develop inhibitors against IKK since its discovery in 1997. There are several highly potent inhibitors that have been developed but none turned out to be a drug because of toxicity. Indeed as of 2013 there are over 130 patents for IKK inhibitors. But nearly all these inhibitors are analogs of ATP, a substrate of all kinases that donate the terminal phosphate group to the substrate. It is thought that toxicity of those inhibitors might be due to 'off target' effects. Since several ATP analogs targeting other protein kinases are currently used as drug, it is puzzling why not a single IKK inhibitor passed even Phase I clinical trial. Perhaps, the architecture of IKK kinase domain prevents it from binding to putative 'non-toxic' ATP analog.

The invention is a strategy to block NF-kB transcription factors. These factors are essential for several physiological functions including immune and inflammatory responses, cell development and survival. However, unregulated activation of NF-kB can lead to chronic inflammation and blockade of normal cell death. It is therefore nor surprising that almost all inflammatory diseases are the direct outcome of constitutive nuclear NF-kB. In almost all cases, constitutive nuclear NF-kB is the result of constitutive IKK activation. Most often environmental hazards like infection, UV and toxic chemicals are responsible for constitutive IKK activity. In addition to the inflammatory diseases such as rheumatoid arthritis (RA), diabetes and heart diseases, unregulated IKK and NF-kB activity is seen in many cancers. We have found a way to inhibit IKK2, the primary activator of NF-kB. We believe that our invention will have real life application to cure or prevent these diseases.

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OTHER INFORMATION

KEYWORDS

NF-KB, enzyme inactivation, peptide interaction, polyubiquitin chain, protein kinase

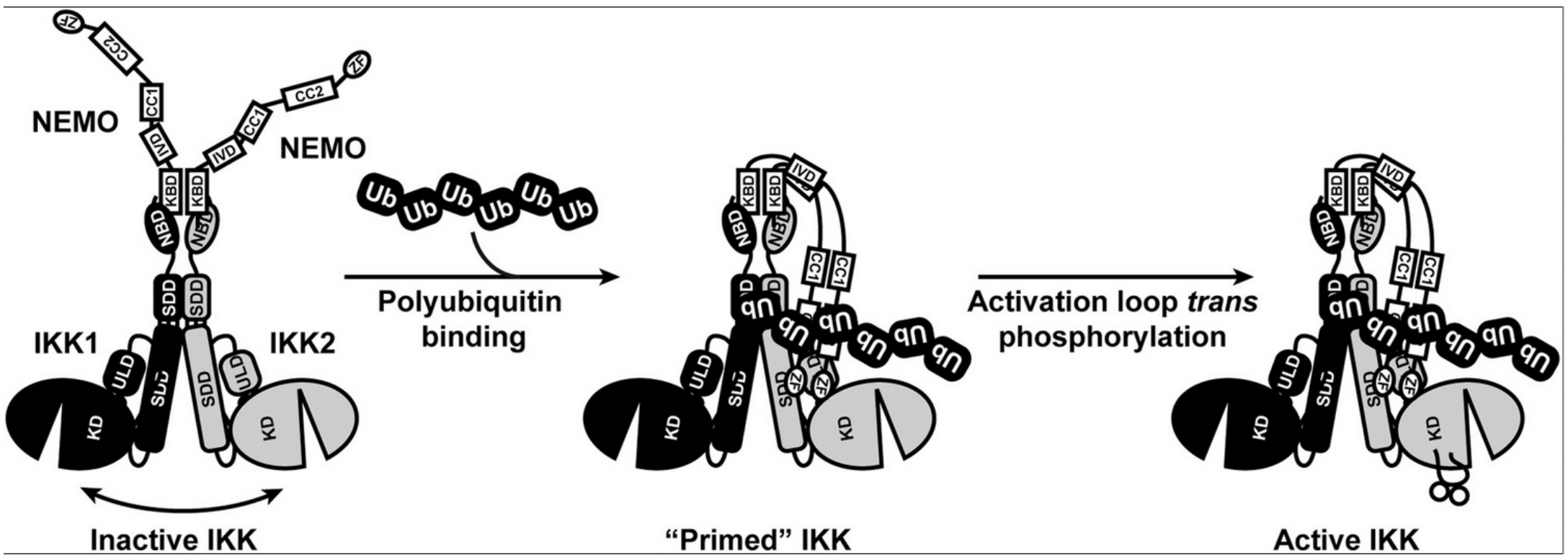
CATEGORIZED AS

- Medical
  - Disease: Autoimmune and Inflammation
  - Therapeutics

RELATED CASES

2021-Z08-1

APPLICATIONS



ADVANTAGES

STATE OF DEVELOPMENT

INTELLECTUAL PROPERTY INFO

UC San Diego is pursuing US patent rights (US 18,550,536) and welcomes interest from companies.

PCT Patent Application: 2022/022425

https://patents.google.com/patent/WO2022212428A1/en?oq=PCTUS2022%2f022425+

RELATED MATERIALS

- Ko MS, Cohen SN, Polley S, Mahata SK, Biswas T, Huxford T, Ghosh G. Regulatory subunit NEMO promotes polyubiquitin-dependent induction of NF-?B through a targetable second interaction with upstream activator IKK2. J Biol Chem. 2022 May;298(5):101864. doi: 10.1016/j.jbc.2022.101864. Epub 2022 Mar 24 - 03/24/2022

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