

**ACTION:** Notice.

**SUMMARY:** The inventions listed below are owned by an agency of the U.S. Government and are available for licensing in the U.S. in accordance with 35 U.S.C. 207 to achieve expeditious commercialization of results of federally-funded research and development. Foreign patent applications are filed on selected inventions to extend market coverage for companies and may also be available for licensing.

**ADDRESSES:** Licensing information and copies of the U.S. patent applications listed below may be obtained by writing to the indicated licensing contact at the Office of Technology Transfer, National Institutes of Health, 6011 Executive Boulevard, Suite 325, Rockville, Maryland 20852-3804; telephone: (301) 496-7057; fax: (301) 402-0220. A signed Confidential Disclosure Agreement will be required to receive copies of the patent applications.

**Methods and Compositions of Defensin-Antigen Fusion Proteins and Chemokine-Antigen Fusion Proteins as Vaccines for Tumors and Viral Infection**

Arya Biragyn (NCI).

PCT Application No. PCT/US01/43830 filed 19 Nov 2001 (DHHS Reference No. E-024-2002/0-PCT-02).

*Licensing Contact:* Catherine Joyce; (301) 435-5031; [joycec@mail.nih.gov](mailto:joycec@mail.nih.gov).

This invention relates to the development of a vaccine for increasing the immunogenicity of a tumor antigen, thus useful for the treatment of cancer, as well as a vaccine for increasing the immunogenicity of a viral antigen, thus allowing treatment of viral infection. In particular, the present invention provides a fusion protein comprising a defensin or chemokine fused to either a tumor antigen or viral antigen which is administered as either a protein or nucleic acid vaccine to elicit an immune response effective in treating cancer or effective in treating or preventing viral infection. In particular, the C-C chemokine macrophage inflammatory protein (MIP-3 $\alpha$ ) was shown to be particularly effective in increasing the immunogenicity of a tumor antigen. Aspects of this work have been published as a PCT patent application with publication number WO 03/025002.

This technology is available for licensing on an exclusive or a non-exclusive basis.

Dated: December 16, 2003.

**Steven M. Ferguson,**

*Director, Division of Technology Development and Transfer, Office of Technology Transfer, National Institutes of Health.*

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**DEPARTMENT OF HEALTH AND HUMAN SERVICES**

**National Institutes of Health**

**Government-Owned Inventions; Availability for Licensing**

**AGENCY:** National Institutes of Health, Public Health Service, DHHS.

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**Improved Endogenous Opioid Anti-Nociception With Reduced Neurodegeneration, Hyperalgesia, Allodynia and Tolerance**

Amina Woods, Toni Shippenberg, and Lawrence Sharp (NIDA).

U.S. Provisional Application No. 60/459,830 filed 01 Apr 2003 (DHHS Reference No. E-276-2001/0-US-01).

*Licensing Contact:* Norbert Pontzer; (301) 435-5502; [pontzern@mail.nih.gov](mailto:pontzern@mail.nih.gov).

Endogenous opioid peptides and receptors evolved to modulate nociceptive input in response to injury. One of those peptides, dynorphin, acts on the kappa opioid receptor subtype to produce analgesia without sedation, respiratory depression or constipation. Prior to this invention, dynorphin was not an acceptable analgesic because of certain severe toxic side effects, when given in doses higher than physiological concentrations, mainly NMDA mediated neurotoxicity. Dynorphin produces its

deleterious side effects by producing an NMDA mediated motor paralysis. In disease states such as stroke, spinal cord injury or neuropathic pain, activation of NMDA receptors by endogenous dynorphin may lead to neurodegeneration, hyperalgesia and allodynia. Tolerance to opiate drugs may also be mediated by the NMDA actions of dynorphin. This invention provides materials and methods to block NMDA receptor activation by dynorphin thus allowing the use of exogenous dynorphin as a beneficial nociceptive agent without side effects and preventing pathological actions of endogenous dynorphin in response to injury. Experimental data demonstrate: (1) attenuation of motor activity deficits, flaccid paralysis and mechanical allodynia produced by dynorphin administration; (2) reduction of infarct size and locomotor deficits after cerebral ischemia; (3) the reduction of morphine tolerization; and (4) so far no visible side effects.

**Pain Control by the Selective Local Ablation of Nociceptive Neurons**

Michael Iadarola and Zoltan Olah (NIDCR).

PCT/US01/09425 filed 22 Mar 2001, published as WO 02/076444 (DHHS Reference No. E-109-2000/0-PCT-02).

*Licensing Contact:* Norbert Pontzer; (301) 435-5502; [np59n@nih.gov](mailto:np59n@nih.gov).

The vanilloid receptor (VR) is a cation channel predominantly expressed on the peripheral processes and perikarya of nociceptive primary afferent neurons. Previous studies have shown that activation of the peripheral receptors by agonists such as capsaicin from hot peppers, or the much more potent resiniferatoxin, produces acute pain sensation which may be followed by desensitization. These inventors discovered that administration of VR agonists in the vicinity of neuronal cell bodies expressing the VR receptor can actually destroy those cells. To control pain and inflammatory disorders, the present invention provides methods and kits for the selective ablation of pain sensing neurons. For example, the intraganglionic administration of a VR agonist selectively ablates primary afferent nociceptive neurons without impairing other sensory modalities. This invention will greatly enhance the ability to control pain, inflammation and other conditions mediated by nociceptive neurons while sparing mental function and other sensations.

This research has been described, in part, in Olah et al., *J. Biol. Chem.*, 276, pp. 11021-11030, 2001.