[Billing Code 4140-01-P]

DEPARTMENT OF HEALTH AND HUMAN SERVICES

National Institutes of Health

Government-Owned Inventions; Availability for Licensing

AGENCY: National Institutes of Health, HHS.

ACTION: Notice.

SUMMARY: The invention listed below is owned by an agency of the U.S. Government and is available for licensing 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.

FOR FURTHER INFORMATION CONTACT: Licensing information and copies of the patent applications listed below may be obtained by communicating with the indicated licensing contact at the Technology Transfer and Intellectual Property Office, National Institute of Allergy and Infectious Diseases, 5601 Fishers Lane, Rockville, MD, 20852; tel. 301-496-2644. A signed Confidential Disclosure Agreement will be required to receive copies of unpublished patent applications.

SUPPLEMENTARY INFORMATION: Technology description follows.

CD300b expression exacerbates endotoxemia and septic peritonitis

Description of Technology:

The innate immune system is the first line of host defense against invading pathogens. Lipopolysaccharides (LPS), present in gram-negative bacteria membranes, cause strong immune responses following detection by the Toll-like receptor 4 (TLR4) on immune cells. This detection results in the release of pro-inflammatory cytokines, such as tumor necrosis factor alpha, interleukin-6, and interferon gamma, to assist with clearance of the infectious insult. In parallel, interleukin-10 (IL-10), an anti-inflammatory cytokine, is induced to limit the immune response. This is because unchecked immune activation leads to a more severe immunopathology, such as septic shock and subsequently death. Current therapies to treat sepsis are ineffective, and clinical trials based on neutralization of specific inflammatory cytokines have failed.

The inventors, listed below, have discovered that CD300b is a LPS binding receptor. This interaction results in a reduced IL-10 production, leading to an amplification of lethal inflammation. *In vitro,* anti-CD300b antibodies block LPS binding to CD300b, stopping association with TLR4 and CD14 and increases IL-10 levels. *In vivo,* administration of anti-CD300b antibodies protects animals from septic shock, due to a reduce level of pro-inflammatory cytokines but subsequent increase in the anti-inflammatory cytokine, IL-10.

This technology is available for licensing for commercial development in accordance with 35 U.S.C. 209 and 37 CFR Part 404, as well as for further development and evaluation under a research collaboration.

Potential Commercial Applications:

As a means of treating endotoxemia and septic peritonitis

Competitive Advantages:

No current therapeutics are available to treat septic shock

Development Stage:

Pre-clinical

Inventors:

John E. Coligan, NIAID, NIH

Oliver H. Voss, NIAID, NIH

Konrad Krzewski, NIAID, NIH

Publications: Voss, Oliver H., et al. "Lipopolysaccharide-induced CD300b receptor binding to toll-like receptor 4 alters signaling to drive cytokine responses that enhance septic shock." Immunity 44.6 (2016): 1365-1378.

Intellectual Property: HHS Reference No. E-112-2016/0 - US Patent Application No. 62/308,144 filed 03/14/2016

Licensing Contact: Chris Kornak, 240-627-3705, chris.kornak@nih.gov

Collaborative Research Opportunity: The National Institute of Allergy and Infectious Diseases is seeking statements of capability or interest from parties interested in collaborative research to further co-develop CD300b antagonists. For collaboration opportunities, please contact Chris Kornak, 240-627-3705, chris.kornak@nih.gov.

Dated: February 16, 2017

Suzanne Frisbie,

Deputy Director

Technology Transfer and Intellectual Property Office

National Institute of Allergy and Infectious Diseases

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