(Catalogue of Federal Domestic Assistance Program Nos. 93.306, Comparative Medicine; 93.333, Clinical Research, 93.306, 93.333, 93.337, 93.393–93.396, 93.837–93.844, 93.846–93.878, 93.892, 93.893, National Institutes of Health, HHS)

Dated: September 23, 2019.

Melanie J. Pantoja,

Program Analyst, Office of Federal Advisory Committee Policy.

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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: Chris Kornak at 240–627–3705 or *Chris.Kornak@nih.gov.* Licensing information may be obtained by communicating with 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 A signed Confidential Disclosure Agreement will be required to receive copies of unpublished information related to the invention.

SUPPLEMENTARY INFORMATION: Technology description follows:

Improvement of Broadly HIV-Neutralizing Antibodies; Anti-HIV–1 Antibody VRC01.23 for Prevention or Treatment of HIV Infection

Description of Technology:

Scientists at NIAID have developed broadly neutralizing antibodies (bNAbs) with enhanced neutralizing activity against HIV–1. Specifically, previously unknown gp120 interactions with a newly elucidated quaternary receptor (CD4)-binding site in the HIV–1 envelope have been discovered by engrafting the extended heavy-chain framework region 3 (FR3) loop of VRC03 onto several potent bNAbs (including VRC01, VRC07 and N6). The new antibodies show improved binding with CD4 by interacting with both binding sites and as a result show improved neutralization of various HIV–1 strains. Furthermore, they show reduced autoreactivity and, as a result, have prolonged *in vivo* half-life.

One of several antibodies that were developed using this technology is VRC01.23. It combines the VRC03 framework 3 alteration, with a G54W mutation in the heavy chain, and a 3 amino acid deletion in the light chain. The modifications improved the potency while reducing the autoreactivity. In particular, VRC01.23 is capable of neutralizing 96% of HIV–1 viruses tested at geometric mean IC50 =0.042 ug/ml, which is ~10-fold more potent than VRC01.

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:

- Improving human monoclonal antibodies for HIV treatment or prevention
- New candidates for use as a therapeutic or as a prophylactic

Competitive Advantages:

- Interaction with multiple HIV binding sites
- Reduced autoreactivity when using the VRC03 framework 3 region mutation
- Improved neutralization breadth and potency over existing antibodies
- Extended in vivo half-life

Development Stage:

- Pre-clinical

Inventors: Paolo Lusso, Qingbo Liu, Peter Kwong, Young Do Kwon, and John Mascola, all of NIAID.

Publications: Liu, Qingbo, et al. ''Improvement of antibody functionality by structure-guided paratope engraftment.'' Nature communications 10.1 (2019): 721.

Intellectual Property: HHS Reference No. E–034–2018–0–PCT–01—PCT Application No. PCT/US2019/019021 filed on 21 February 2019.

Licensing Contact: To license this technology, please contact Chris Kornak at 240–627–3705 or *Chris.Kornak@nih.gov,* and reference E–034–2018.

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 develop, evaluate or commercialize this technology. For collaboration opportunities, please contact Chris Kornak at 240–627–3705 or *Chris.Kornak@nih.gov.*

Dated: September 18, 2019.

Wade W. Green,

Acting Deputy Director, Technology Transfer and Intellectual Property Office, National Institute of Allergy and Infectious Diseases.

[FR Doc. 2019–20994 Filed 9–26–19; 8:45 am]

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 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. 209 and 37 CFR part 404 to achieve expeditious commercialization of results of federally-funded research and development.

FOR FURTHER INFORMATION CONTACT: Licensing information may be obtained by emailing the indicated licensing contact at the National Heart, Lung, and Blood, Office of Technology Transfer and Development Office of Technology Transfer, 31 Center Drive, Room 4A29, MSC2479, Bethesda, MD 20892–2479; telephone: 301–402–5579. A signed Confidential Disclosure Agreement may be required to receive any unpublished information.

SUPPLEMENTARY INFORMATION: Technology description follows.

Antagonists of Hyaluronan Signaling for Treatment of Airway Diseases, such as Asthma and Chronic Obstructive Pulmonary Disease (COPD), constitute a major health burden in the development word. It is estimated that nearly15.0% of the adult population in the US are affected with such diseases, and the economic cost burden is over $23 billion annually. Unfortunately, the current options for treatment of such diseases are quite limited, consisting only of bronchodilators and inhaled steroids. The need for a novel and more effective class of therapeutics agents is imperative. The subject invention provides for a potentially more specific and effective treatment of airway diseases as compared with existing treatments. It is based on the inhibition of Hyaluronan (HA), a structural polysaccharide that plays a role in the signaling pathway that leads to the