Empithelial membrane protein 2 (EMP2) as a lung epithelial protein that regulates PMN entry into the inflamed airspace. EMP2 knockout mice have reduced PMN accumulation and exhibit increased survival during bacterial infection. Inhibition of EMP2 can potentially reduce intra airway PMN accumulation and provide a specific treatment for various lung disorders.

**Potential Commercial Applications**

Development of EMP2 inhibitor for treatment of neutrophil-dependent lung disorders, such as:

- Acute lung injury
- Pneumonia (bacterial, viral, fungal)
- Bronchiectasis
- COPD and asthma
- Radiation- or chemotherapeutic-induced pneumonitis
- Idiopathic or induced interstitial lung disease
- Bronchopulmonary dysplasia
- Lung transplant rejection

**Competitive Advantages**

- EMP2 can selectively target PMN accumulation in the lung, rather than broadly affecting PMN trafficking through all tissues.

**Development Stage**

- Early stage
- *In vitro* and *in vivo* (animal) data available

**Inventors:** Michael Brian Fessler (NIH), Carmen J. Williams (NIH), and Wan-Chi Lin (NIH).

**Intellectual Property:**

- **Licensing Contact:** Brian W. Bailey, Ph.D.; 301–594–4094; bbailey@mail.nih.gov.
- **Dated:** September 25, 2020.
- **National Heart, Lung, and Blood Institute, Office of Technology Transfer and Development.**

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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, HHS.

**ACTION:** Notice.

**SUMMARY:** The inventions listed below are owned by an agency of the U.S. Government and are available for licensing to achieve expeditious commercialization of results of federally-funded research and development.

**FOR FURTHER INFORMATION CONTACT:** Licensing information may be obtained by communicating with Vidita Choudhry, Ph.D., National Heart, Lung, and Blood, Office of Technology Transfer and Development, 31 Center Drive, Room 4A29, MSC2479, Bethesda, MD 20892–2479; telephone: 301–594–4095; email: vidita.choudhry@nih.gov. A signed Confidential Disclosure Agreement may be required to receive any unpublished information.

**SUPPLEMENTARY INFORMATION:** Technology description follows.

**Reducing Bloodstream Neutrophils as a Treatment for Lung Infection and Inflammation**

During lung infection, bloodstream neutrophils (PMNs) responding to infection travel to the airspace lumen. Although successful arrival of microbicidal PMNs to the airspace is essential for host defense against inhaled pathogens, excessive accumulation of PMNs in the lung contributes to the pathogenesis of several prevalent lung disorders, including acute lung injury, bronchiectasis, and chronic obstructive pulmonary disease (COPD).

Unfortunately, there is no treatment for controlling PMN accumulation in the lung. The subject invention describes epithelial membrane protein 2 (EMP2)