SUPPLEMENTARY INFORMATION:

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 reduced 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.

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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.


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