Derepression Of Interferon Stimulated Genes For Enhancing Protection In Autoinflammatory Reactions Including Those During Respiratory Infections.

Tech ID: 34488 / UC Case 2021-137-0

TECHNOLOGY DESCRIPTION

UNMET NEED: Severe COVID-19 patients experience immune system dysfunction, including suppression of protective interferon-stimulated gene (ISG)-expressing immune populations, which contributes to disease progression and mortality. Current treatments fail to address these dysregulated immune responses.

TECHNOLOGY: Researchers at UCSF have uncovered a novel mechanism wherein overactive antibody responses in severe COVID-19 patients inhibit interferon-stimulated protective immune states by engaging Fc receptors (FcRs) on immune cells. This discovery highlights the role of antibody-driven immune suppression in severe cases and offers new targets for immunotherapy to restore effective antiviral defense mechanisms. Developmental status: preclinical proof-of-concept achieved.

COMPETITIVE ADVANTAGE:

- **Identifies novel immune dysfunction:** Severe COVID-19 patients produce antibodies that inhibit ISG-expressing immune cells, critical for antiviral defense.
- **Fc receptor signaling mechanism:** Reveals that antibody-mediated signaling through inhibitory Fc receptors (CD32b) blocks interferon responses, leading to immune suppression.
- **Therapeutic potential:** Suggests targeted approaches to restore immune function, such as Fc receptor blockade, B cell-modulating therapies, or IVIG to compete with pathogenic antibodies.
- **Broad applicability:** Findings may extend to other viral infections with similar antibody-driven immune suppression, paving the way for new immunotherapy strategies.

PATENT STATUS

Patent Pending

CONTACT

Lei Wan

lei.wan@ucsf.edu

tel: .

OTHER INFORMATION

KEYWORDS

COVID, Antibody response, Immune suppression, CD32b, Fc receptor

CATEGORIZED AS

- **Biotechnology**
  - Health
- **Medical**
  - Disease: Infectious Diseases
  - Therapeutics

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600 16th St, Genentech Hall, S-272,
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