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Targeting TNFR1 for enhanced therapy efficacy

A synthetic protein inhibitor targets TNFR1 to treat inflammatory diseases with higher specificity and fewer side effects.

IP Status: PCT Pending; Application No.: PCT/US2023/019264

Applications

- Inflammatory diseases
- Research tool

Key Benefits & Differentiators

- **Targeted Inhibition:** Specifically targets TNFR1, minimizing off-target effects on other natural ligand binding partners like TNFR2.
- **Enhanced Potency:** Utilizes an allosteric, non-competitive inhibition mechanism to increase functional potency.
- **Reduced Side Effects:** Directly targeting TNFR1 avoids the side effects associated with current therapies that target TNF.

Technology Overview

Inflammatory diseases are often driven by the overactivity of tumor necrosis factor receptor 1 (TNFR1), a crucial component in cellular signaling. Current therapeutic approaches typically inhibit the ligand TNF, which interacts with TNFR1, but this method can cause significant side effects due to interactions with other receptors, such as TNFR2. A more targeted approach that directly inhibits TNFR1 can potentially reduce these side effects and enhance treatment efficacy.

Researchers at the University of Minnesota have developed a synthetic protein ligand that binds to human TNFR1 with high affinity. This protein inhibits TNFR1 signaling through a non-competitive allosteric mechanism. Unlike existing FDA-approved therapies that target the ligand (TNF), this approach focuses on inhibiting the receptor itself, which prevents interference with other ligand interactions and reduces problematic side effects. The technology promises higher specificity and potency in treating various diseases involving TNFR1 overactivity.

Phase of Development

TRL: 3-4
The lead molecule, characterized in vitro, is now set for in vivo testing and further enhancement of its potency and pharmacokinetics.

Desired Partnerships

This technology is now available for:

- License
- Sponsored research
- Co-development

Technology ID
2022-053

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