Background:
Inappropriate complement activation may cause or aggravate several pathological conditions, such as age-related macular degeneration, asthma, adult respiratory distress syndrome, chronic obstructive pulmonary diseases, hemolytic anemia, rare kidney diseases, lupus, rheumatoid arthritis, rejection of xenotransplantation, stroke and heart attack. Regulating complement activation is important to control inflammation, autoimmune diseases, and infections. Peptides of the compstatin family inhibit complement system activation and are promising candidates to become therapeutics for the treatment of complement-related diseases and disorders.

Brief Description:
UCR researchers and their collaborators from Princeton University and the University of Cyprus have developed novel compstatin analogs that lead to the future development of therapeutics for age-related macular degeneration and other complement system-mediated diseases. The new peptides target the complement system protein C3, they are potent in terms of required binding affinity, and have improved solubility compared to previously known compstatin peptides.

Advantages:
• Targeting of protein C3, the converging point of complement activation pathways
• Potent binding affinity
• Higher solubility compared to previously known potent compstatin analogs

Applications:
• Therapeutics for age-related macular degeneration and rare diseases, e.g. paroxysmal nocturnal hemoglobinuria (PNH), atypical hemolytic uremic syndrome (aHUS), and C3 glomerulopathy (C3G)
• Therapeutics for other complement system-mediated inflammatory and autoimmune diseases, e.g. chronic obstructive pulmonary disease, systemic lupus erythematosus, rheumatoid arthritis, and ischemia reperfusion injuries

LEARN MORE:
Patent:
US Patent 9,512,180

Publications:

Keywords:
complement system, compstatin, C3, complement system-mediated diseases, autoimmune diseases, inflammatory diseases, rare diseases.

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