2010-011 Peptides Inhibiting Surfactant Protein-D Assembly and Related Novel Targets

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Technology description

Rutgers scientists have discovered a novel mechanism of multimer assembly that relies on non-covalent linkages between cysteine residues. This mechanism has been shown to be critically important in the assembly of the innate immune regulatory protein surfactant protein D (SP-D). Peptides designed to disrupt these linkages have been shown to modify SP-D multimer formation. This mechanism is critically dependent on a particular cysteine containing sequence motif. This motif is found in other clinically relevant multimers such as HIV gp160, and those present in HCV. By designing peptides that can interact with specific structural cysteine containing motifs, one can interrupt assembly of specific multimeric proteins, such as gp 160. These peptides may be further developed for clinical use as a therapy and the target may be a unique drug discovery tool or a vaccine. This technology may find utility in bronchopulmonary dysplasia therapy, and in inflammation, emphysema, and chronic infections such as HIV and HCV.

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