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A novel approach for inhibiting progression of flu virus infection at early stages of the disease by inhalation of α-Gal/SA liposomes

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The proposed therapy aims to accelerate the healing process in flu patients treated at early stages of the disease and accelerate the induction of a protective immune response thereby preventing further complications associated with flu infection. The therapy is based on inhalation of aerosolized biodegradable liposomes called a-Gal/SA liposomes that present multiple carbohydrate epitopes of two types: a-Gal epitopes with the structure Gala1-3Galb1-4GlcNAc-R and sialic acid (SA) epitopes with the structure SAa2-6Galb1-4GlcNAc-R. This treatment exploits the ability of flu virus to bind to SA epitopes and the ability of the natural anti-Gal antibody (Ab) to bind to a-Gal epitopes. Anti-Gal is the most abundant natural Antibody in humans, constituting ~1% of IgG, IgM and IgA. Inhaled a-gal/SA liposomes land in the mucus and the surfactant coating the respiratory tract. These a-gal/SA liposomes acting as a "decoy" bind flu virus via interaction between viral hemagglutinin and SA epitopes on the liposomes thereby slowing or preventing further infection of respiratory epithelial cells. Concomitant binding of the anti-Gal Ab to a-gal epitopes on a-Gal/SA liposomes activates the complement system and generates chemotactic complement cleavage peptides such as C5a that recruit multiple macrophages to the liposomes. The recruited macrophages internalize the liposomes and flu virus bound to their SA epitopes. This uptake is mediated by interaction between the Fc portion of anti-Gal IgA and IgG molecules on a-Gal/SA liposomes and Fc receptors on the macrophages. The internalized virus is destroyed by lysosomes within the macrophages. The recruited macrophages further function as antigen presenting cells that process immunogenic peptides of the internalized virus, transport them to the regional lymph nodes and present these processed peptides in association with MHC molecules for activation of CD4+ and CD8+ T cells specific to the infecting flu virus. The activated CD4+ T cells function as helper T cells that help B cells to produce Abs which neutralize the virus and thus inhibit its ability to infect respiratory epithelial cells. The activated CD8+ T cells differentiate into cytotoxic T cells (CTL) that kill virus infected epithelial cells thus preventing further progression of the viral infection. These combined effects of inhibiting viral infection of epithelial cells and induction of rapid anti-flu B cell and T cell protective immune responses may shorten the period of the disease, attenuate its severity and decrease complications in the respiratory system.

Biography

Uri Galili has completed his PhD at the Hebrew University Medical School (Jerusalem) and Post-doctorate at the Karolinska Institute (Stockholm). He conducted independent research as a Professor at UCSF, MCP-Hahnemann, Rush and UMass Medical Schools. He has published 199 papers.

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