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Universal influenza vaccine research and development updates

Melvin Sanicas[1] and **Merlin Sanicas**[2]
[1]Bill & Melinda Gates Foundation, USA
[2]Ateneo School of Medicine and Public Health, Philippines

The frequent mutation of the flu virus strain has rendered the current influenza vaccines only viable for one season and the next season they become useless. The vaccines are reformulated every year in order to match the new form of strains that appear each season. These vaccines are also ineffective at preventing a pandemic flu in case of an outbreak. A pandemic flu is very unpredictable and it spreads quickly through the human population. Annual influenza epidemics are estimated to result in about 3 to 5 million cases of severe illness and about 250,000 to 500,000 deaths. These deaths could well rise into millions in case another influenza pandemic breaks out. The trivalent influenza vaccines are made with subtypes H3N2 and H1N1 and a virus in type B that matches the circulating strains. The current vaccines may sometimes have the capacity to provide protection from matching viruses but have limitations that presents the need to manufacture new vaccines each season. These concerns have led to research into producing a universal influenza vaccine that can protect against a wider range of influenza viruses. In the recent few years, many concepts of producing a universal vaccine that used the part of the virus that does not mutate as much hemagglutinin changes easily, whereas the stem remains relatively unchanged. The most advanced concepts are those that involve T cell stimulation (Matrix 1 and Nucleoprotein protein antigen) and M2e (ectodomain of M2). Several concepts have passed the phase two trials and this review presents an overview of the current research and development efforts.

melvin.sanicas@gmail.com

GPI-anchored CCL28 VLPs induce cross-protective immune response against H3N2 viruses

Teena Mohan, Compans R W and **Wang B**
Emory University, USA

Influenza infection typically initiates at respiratory mucosal surfaces. Induction of immune responses at the sites where pathogens initiate replication is crucial for the prevention of infection. We studied the adjuvant city of GPI-anchored CCL28 co-incorporated with influenza HA-antigens into virus like particles (VLPs), in boosting strong protective immune responses through an intranasal route in mice. We compared the immune responses to that from influenza VLPs without CCL28, or physically mixed with soluble CCL28 at systemic and various mucosal compartments. The cVLPs containing GPI-CCL28 showed in vitro chemotactic activity towards the cells expressing CCR3/CCR10 chemokine receptors. The GPI-CCL28-containing VLPs induced antigen specific endpoint titers and avidity index of IgG in sera and IgA in tracheal, lung and intestinal secretions, significantly higher (4-6 fold) than other formulations. Significantly higher (3-5 fold) hem agglutination inhibition titers with high serum neutralization against H3N2 viruses were also detected with CCL28-containing VLPs compared to other groups. The CCL28- containing VLPs showed complete and 80% protection, when vaccinated animals were challenged with A/Aichi/2/1968/H3N2 (homologous) or A/Philippines/2/1982/H3N2 (heterologous) viruses, respectively. Thus, GPI-anchored CCL28 in influenza VLPs act as a strong immunostimulator at both systemic and mucosal sites, boosting significant cross-protection in animals against heterologous viruses across a large distance.

mohan.teena@gmail.com