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T cell immune response in influenza infection

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Influenza infection invokes innate and adaptive immune responses in order to protect host against viral/bacterial infection. Upon infection virus-specific T cell responses are induced, including CD4+ T helper cells and CD8+ cytotoxic T cells,Memory CD4 and CD8 T cells are generated in response to viral infection. Influenza virus evade antibody-based vaccines, and memory CD4 T cells in anti-viral immunity helps in the development of broad-based T cell-mediated immunity that will provide protection against influenza infection with variant or new influenza virus strains that affect the public health. In the periphery and lung of children and adults memory CD4 T cells directed against influenza infection with the potential to mediate "first-line" immunity to viral challenge, particularly at the site of infection. CD4 T cells are typically classified into different subsets based upon cytokine expression. By analyzing pre-committed population of T helper cells can lead to a wider understanding of the mechanisms and it might control the regulation of heterogeneity in cytokine patterns displayed because of pathogen challenges. The influenza virus induces chemokine and cytokine production by infected epithelial cells, monocytes and macrophages,which leads these chemokine to attract immune cells to the site of infection, including macrophages, neutrophils and natural killer (NK) cells.

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Molecular biomarkers of the susceptible population with influenza viruses: α2-3 and α2-6 siaylated glycans in human saliva

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Recent studies have elucidated that expression of certain glycoproteins in human saliva are increased or decreased according to age, meanwhile, human saliva may inhibit viral infection and prevent viral transmission. We find that seven lectins (e.g., MAL-II and SNA) show significant age differences in both females and males, and seven lectins (e.g., WFA and STL) show significant sex differences in children, adults and elderly people. Interestingly, we observe that healthy elderly individuals have the strongest resistance to influenza A virus (IVA) mainly by presenting more terminal α2-3/6-linked sialic acid residues in their saliva, which bind with the influenza viral hemagglutinations. However, it is often noted that hospitalizations and deaths after an influenza infection mainly occur in the elderly population living with chronic diseases, such as diabetes and cancer. We observed that the expression level of the terminal α2-3-linked sialic acids of elderly individuals with type 2 diabetes mellitus and liver disease (hepatitis B, hepatic cirrhosis, hepatocellular carcinoma) were down-regulated significantly, and the terminal α2-6 linked sialic acids were up-regulated slightly or had no significant alteration. But, in the saliva of patients with gastric cancer, neither sialic acid was significantly altered. These findings may reveal that elderly individuals with chronic diseases, such as diabetes and liver disease, might be more susceptible to the avian influenza virus due to the decreased expression of terminal α2-3-linked sialic acids in their saliva.

Our findings imply that the expression level alterations of terminal α2-3/6-linked sialic acids is a risk factor that could be a biomarker to distinguish those patients who are at a greater risk for infection with IAV, and may provide pivotal information to recommend strongly routine vaccination for them with influenza vaccines.

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