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Perspective

The Significance of Cell-Mediated Immunity (CMI) with Trivalent Inactivated Influenza Vaccines (TIV)

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DESCRIPTION

The Orthomyxoviridae family of enclosed negative-sense single-stranded RNA viruses with segmented genomes includes influenza viruses. Human respiratory illness is caused by three influenza genera-A, B, and C of which influenza A and B are the most significant clinically. According to the antigenicity of their main membrane glycoproteins, Haemagglutinin (HA) and Neuraminidase (NA), the influenza A viruses are further divided into subtypes. Since 1977, co-circulating influenza A/H1N1 and A/H3N2 strains have been responsible for yearly outbreaks of different intensity. Instead of being divided into subtypes, influenza B viruses are divided into two unique genetic lineages (B/Yamagata and B/Victoria). Since the 1980s, influenza B viruses from both lineages have co-circulated in the majority of influenza seasons. The primary target of host antibody reactions induced by inborn infection or immunisation that protect against human influenza infection is HA. Point mutations in the genes of the influenza virus are caused by the RNA-dependent RNA polymerase complex's lack of proofreading activity. The formation of variant viruses that may avoid immune detection is caused by the accumulation of minor alterations over time inside the antibody-binding regions of HA and NA, resulting in viruses that are antigenically distinct. Antigenic drift is a phenomenon that mostly explains why people might contract the flu more than once. Unlike another form of modification called antigenic shift, which is exclusively seen in influenza A viruses, antigenic drift is detected in both influenza A and B viruses. When several influenza viruses infect the same human or non-human (such as porcine) host cell at the same time, genetic reassortment of gene segments between the influenza viruses results in an abrupt, significant alteration known as an antigenic shift. As a result, a new, possibly pandemic influenza A virus with a novel HA (and NA) that humans have little to no protection against might be introduced. Trivalent Inactivated Influenza Vaccines (TIV), which are delivered intramuscularly and contain 15 g of each of the three chosen influenza strains (A/H1N1, A/H3N2, and one B lineage), were the standard for annual vaccination until recently. Based on predictions generated from monitoring data collected under the guidance of the WHO (Global Influenza monitoring and Response System, GISRS), the influenza strains are chosen for for inclusion in a seasonal vaccination. However, because there are two influenza B lineages currently circulating (B/Yamagata and B/Victoria), vaccine protection may be insufficient when the most common influenza B virus strain in circulation belongs to a different lineage from the B strain that is included in the vaccine. The same holds true when influenza A strains are mismatched.

It was anticipated that the recent release of inactivated Quadrivalent Influenza Vaccinations (QIV) including both B strains would solve the issue of B lineage mismatch. However, a recent study showed that the use of QIV is not connected to a higher level of protection against any influenza B illness. Additionally, the genetic diversity of influenza viruses has significantly decreased due to COVID-19 measures implemented as of March 2020 (such as social withdrawal, mask use, and travel restrictions), with no B/Yamagata lineage isolations being noted between April 2020 and August 2021. This may indicate the global extinction of this lineage and may be advantageous for next yearly vaccine reformulations (for example, include an additional A/H3N2 strain to reduce the chance of mismatch). By growing the viral strains in eukaryotic cells or embryonated eggs, or by using recombinant DNA technologies in which just the HA antigen is expressed in an insect cell line using a baculovirus expression system, inactivated vaccines can be produced.

The primary mechanism by which these vaccinations work is by the production of HA-specific antibodies that block viral entrance into respiratory epithelial cells. Less frequently, NA-specific antibodies are produced that prevent the release of developing viruses. There has been mounting evidence in recent years that vaccine-induced protection may be influenced by Antibody-Dependent Cellular Cytotoxicity (ADCC). A cell-mediated immune defence known as ADCC causes the target cell to be lysed by particular antibodies that bind to membrane-surface antigens expressed on target cells and also engage with Fc receptors on effector cells, such as Natural Killer (NK) cells. In both healthy and infected individuals, antibodies that cause ADCC frequently target internal viral proteins like M1 and NP (antigens that are not included in commercially available vaccinations). Thus, these antibodies might provide cross-reactive defense. The primary method by which antibodies against conserved (but subdominant) epitopes function may be ADCC.

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