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Annual seasonal influenza vaccination and cross-neutralization to the pandemic 2009 H1N1 influenza virus

Annual seasonal influenza vaccination (TIV) induces the production of neutralizing antibody to the hemagglutinin (HA) surface protein. Mutations that change antigenicity occur predominantly in the globular head (HA1 subunit) of HA, while the stem region of the HA transmembrane subunit (HA2) is more conserved. Pandemic 2009 H1N1 influenza virus differs from seasonal H1N1 strains that circulated in the past 50 years and resembles a strain that did not circulate but was used in the 1976 swine influenza vaccine. Emergence of the novel pandemic influenza A virus (pdm2009 H1N1) raised questions about whether immunization with prior influenza vaccines could confer any protection. We investigated whether persons immunized with the 1976 swine flu or recent seasonal influenza vaccines, or both, harbor neutralizing antibodies to pdm2009 H1N1. We found that archived sera from the 1976 swine influenza vaccine trials cross-neutralized the pdm2009 H1N1 and to a lesser extent the A/New Caledonia/20/1999 H1N1 strain, which was used in vaccines during the 2000/01-2006/07 influenza seasons. Sera from persons who received several seasonal influenza vaccines containing A/New Caledonia/20/1999 H1N1 cross-neutralized the pdm2009 H1N1, regardless of whether they received the 1976 swine influenza vaccine. Cross-neutralization between pdm2009 H1N1 and A/New Caledonia/20/1999 frequently mapped to HA2. A conservative mutation in HA2 corresponding to a residue at 89 in the A/Solomon Islands/3/2006 and A/Brisbane/59/2007 H1N1 strains that circulated in the 2006/07 and 2007/08 influenza seasons, respectively, abrogated this neutralization. These findings highlight a cross-neutralization determinant influenced by a point mutation in HA2 and suggest that HA2 may be evolving under direct or indirect immune pressure.

Biography

Dr. Wei Wang received his Ph.D. in 2000 from the University of Saskatchewan, Canada, and completed postdoctoral studies from National Cancer Institute. He joined US Food and Drug Administration in 2005.