2007-08 Influenza Season Wrap-Up

The 2007-08 influenza season ended May 17, 2008 (week 20). This flu season was marked by an increase in the number of influenza cases and in the severity of cases, seen both locally and nationally when compared to recent influenza seasons.

The proportion of patient visits to sentinel providers for influenza-like illness (ILI), characterized by temperature of 100°F or greater and sore throat or cough, during the last week of the season was 0.9%; which is the same percentage as the start of the season (week 40, ending October 6, 2007)*. The peak for influenza in Clark County was during week 7, ending February 16, 2008, when the percent ILI was 2.33%*. Nationally, for week 20, the percent ILI was at 0.8% which is below the national baseline of 2.2%. Regionally, the percent ILI ranged from 0.2% to 1.4%, with all regions reporting below their region-specific baselines.

Also corresponding to the peak in ILI, the proportion of mortality from pneumonia and influenza (P&I) peaked during week 7 at 11.1%. The proportion of P&I mortalities remained high over the season with an overall average during the 32 weeks (week 40-week 20) at 7.8%. Nationally, during week 20, 7.3% of all deaths were P&I related; which is above the epidemic threshold of 6.3%. This marks the 19th consecutive week that the proportion of P&I mortalities has been above the epidemic threshold (1).

Laboyratory Surveillance

During the 2007-08 flu season, influenza A (H1), A (H3), and B have co-circulated throughout the United States. Influenza A (H3) dominated this season, however, this varied by week and by region. From week 40 through week 3, influenza A (H1) viruses were more frequently reported; from week 4 through week 12, influenza A (H3) viruses were more frequently reported; and from week 13 through week 20, influenza B viruses were more frequently reported. Regionally, Influenza A (H3) predominated in the East North central, East South Central, Mid-Atlantic, New England, South Atlantic, West North Central, and West South Central regions. While influenza A (H1) viruses predominated in the Mountain (including Nevada) and Pacific regions (1).

Influenza Vaccine

Viruses used to make influenza vaccine are chosen each year based on information gathered over the previous year about the strains of influenza virus that are affecting people and how the viruses are changing. Information on circulating influenza strains and disease trends are gathered by 122 national influenza centers in 94 countries. The collected viruses are further tested and analyzed by the four World Health organization (WHO) Collaborating Centers for Reference and Research on Influenza. Based on this vast amount of information, experts forecast which viruses are likely to circulate the following season and WHO recommends specific virus strains that can be used to make vaccines to protect against them. Each country then can use the recommendations to assist with national decisions about what viruses to use in the vaccines for their countries. In the United States, an advisory committee convened by the Food and Drug Administration (FDA) makes the final decision on the components of the vaccine in February. Manufacturers grow the vaccine strains and often begin the production process as early as January based on their assessments of which strains will be chosen for the vaccine (2).

The effectiveness of the vaccine depends in part on the match between the viruses in the vaccine and influenza viruses circulating in the community. If these are closely matched then vaccine effectiveness (VE) has been estimated as high as 70-90% in healthy adults. However, the VE is typically lower during influenza seasons when a suboptimal match between the vaccine strains and circulating influenza strains are observed (3).

Viral data reported to WHO and the National Respiratory and Enteric Virus Surveillance System (NREVSS) laboratories through April 5, 2008, demonstrated that of the influenza A viruses sub-typed , 27% were influenza A (H1N1) and 73% were influenza A (H3N2).
influenza A (H3N2). Antigenic characterization of a subset of these viruses by the Centers for Disease Control and Prevention (CDC) indicated that 69% of A (H1N1) viruses were A/Solomon Islands/3/2006-like, the A (H1N1) 2007-08 vaccine component, but 71% of A (H3N2) were A/Brisbane/10/2007-like, a recent antigenic variant of the A/Wisconsin/67-like virus, the A (H3N2) vaccine component. Additionally, 95% of antigenically characterized B viruses belonged to the B/Yamagata lineage. Viruses in this lineage are antigenically distinct from the B/Malaysia/2506/2004-like component of the 2007-08 vaccine. Preliminary studies suggest that the VE for the 2007-08 season in preventing influenza A was 58%. In contrast, no VE could be demonstrated against influenza B. However, it is important to note that influenza vaccination has been shown to provide measurable protection against influenza infection with viruses related to vaccine strains, even when the vaccine strains are not optimally matched to circulating strains (4).

Composition of the 2008-09 Influenza Vaccine

The FDA and WHO have recommended that the 2008-09 trivalent influenza vaccine for the Northern Hemisphere contain A/Brisbane/59/2007-like (H1N1), A/Brisbane/10/2007-like (H3N2), and B/Florida/4/2006-like viruses. All three components have changed from the 2007-08 Northern Hemisphere influenza vaccine. This recommendation was based on antigenic analyses of recently isolated influenza viruses, epidemiologic data, post-vaccination serologic studies in humans, and the availability of candidate vaccine strains and reagents (5).

This ends the influenza newsletter updates for the 2007-08 season. Look for the next newsletter in October of 2008 for the 2008-09 influenza season.

*Weighted average