

STANDARD DEVIATIONS: OINK

Okay and Oy vey,

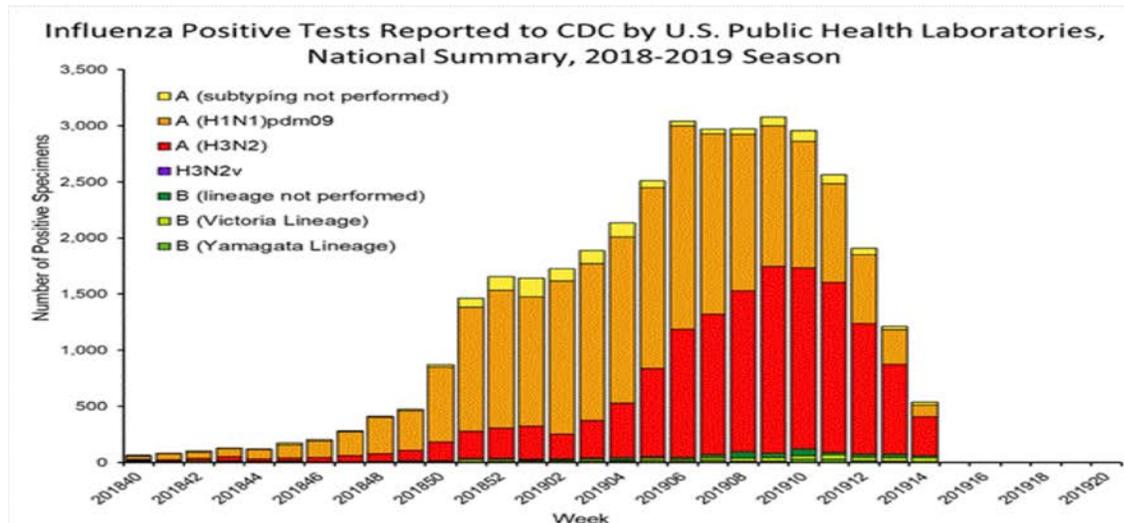
Our North American season for influenza is waning. Incidence and severity were relatively moderate for this cycle and the data accumulated will be interesting in respect to the efficiency of the vaccine used this time around. The CDC *FluView* is a great page to explore if you're into the fine details of surveillance (www.cdc.gov/flu/weekly/index.htm).

Back in November, I spoke about flu and how we mitigate Lab-Acquired-Infection (LAI) at the bench ([SD: Influenza for Dessert? 11/26/18](#)). Today, I throw some stats out re the number of samples Utah labs submitted to the State for follow-up and how those get tossed around.

At UPHL, we have tested and typed around 800 samples that your labs submitted from patients hospitalized with flu. A handful were forwarded to the CDC for confirmations and, routinely, we send samples tested by the State public health lab to CDC for the pool of samples used in determining vaccine choices for upcoming cycles of influenza.

Across the U.S., **clinical labs tested 987,000 samples for influenza** with a positive rate of about 16% (~160,000). What do your data show for test volumes with the flu season in your lab? Do these numbers seem in line with the national picture?

Of **37,000 positives** typed by public health, nationally, 97% have been Type A Influenza and 3% Type B. Of those A's, 60% were influenza **A (H1N1)pdm09** virus and the rest, H3N2. B influenza has been fairly evenly split by Victoria and Yamagata lineages with Victoria dominating the later part of the season.



I'm also taking up your time to mention Swine Influenza. Pigs are the mixing vessel for flu viruses to swap spit and the incubators of viral genetics that induce antigenic variants leading to pandemics. Understanding how influenzas change gives us a handle on pandemic evolution.

In the spring of 2009, a novel influenza A (H1N1) virus emerged. It was detected first in the United States and spread quickly across the United States and the world. This new H1N1 virus contained a unique combination of influenza genes not previously identified in animals or people. This virus was designated as influenza **A (H1N1)pdm09** virus. CDC estimated that between 151,700 and 575,400 people worldwide died from 2009 H1N1 virus infection during the first year the virus circulated as a pandemic event. This virus, as well as the 1918, 1957, and 1968 variants are suspected to have emerged as reassortment products from swine.

As seasons pass, our immunity to a circulating strain increases. Antigenic **drift** may weaken the vaccine response and our immunity, but we have some protection in the library of our history with exposure (note the strain that dominates this year). And yet, we still see illness and death.

When reassortment as antigenic **shift** unveils new antigenic variety, all bets are off. Our close interaction with swine and their role as incubators of avian, human and porcine viruses puts us in proximity to those new variants.

Reassortment is a dynamic and constant process. A new variant is coming. Whether an H2, H5, or H7, we will see a pandemic from swine emerge. The 2018-2019 season hasn't produced that virus, but the threat is real. This link to YouTube is a cute little refresher.



Click to play this short video on Antigenic Shift.



Laboratorians will always be at risk. Influenza is an infectious disease and our mitigation strategy must maintain a strong reliance on biosafety. Our diligence in testing and using safe laboratory technique must stay in mind and practice. This must continue in order to recognize and respond when the next pandemic arrives and keep us safe while we wait.

Have a great week and be safe,

Bryan

p.s. Porcine pandemonium persists in next week's pages!

References:

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