Surveillance triggers for community-based mitigation of pandemic influenza

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OBJECTIVE

In response to an Institute of Medicine (IOM) report[1] recommending community-based pandemic influenza mitigation strategies be informed by surveillance and disease modeling, we aimed to assess the feasibility of using emergency department (ED) data to identify model derived threshold triggers for initiating intervention efforts in the event of a 1918-like pandemic [2,3].

BACKGROUND

Four waves of pandemic influenza from 1918-1920 in New York City (NYC) caused ~40,000 deaths, primarily of young-adults and children[4]. The explosiveness of the autumn 1918 wave has led many to believe that in the event of a similar pandemic today early detection and intervention strategies may not be effective. Recent historical studies of the 1918 pandemic, however, provide evidence of controllable transmissibility[3,5], of a limited early wave[4], and of social distancing measures significantly reducing pandemic impact in many US cities[2,3]. Importantly, mitigation efforts initiated after the beginning of community-wide transmission (even up to the point of 3-6% of a population being infected[2]) significantly reduced the total impact in 1918.

METHODS

We use historical estimates of excess mortality[4], case fatality proportion and observed lag from illness to death in the 1918 pandemic[5], and current estimates of influenza-attributable excess ED visits and deaths in NYC[6]. From these estimates, we extrapolate 1918-like scenarios onto the 2003/04 A/H3N2-Fujian influenza epidemic season, and for both real and extrapolated data, identify the weeks when excess ED visits clearly exceed Serfling model-based thresholds. We then calculate the cumulative attack rate (cAR) proportion at which detection occurred, as the proportion of incident excess visits to total excess visits during the 8-12 week epidemic wave. We compare these to recently published historical modeling studies[2,3] to evaluate whether identified surveillance triggers provide sufficient warning consistent with model derived thresholds.

RESULTS

During the 2003/04 influenza season, influenza-attributable ED visits exceeded 3.2 standard deviations above model predicted visits at a cAR of 6% (see Figure). For the extrapolated 1918-like scenario, based on a 1-week lag from illness to death, using ED visits as a surrogate measure of illness, and a conservative assumption of a ~5:1 excess ED visit to death ratio, we estimate that community-wide transmission would be evident at a cAR of 0.7% (Figure).

CONCLUSION

While predictions of the emergence and trajectory of pandemic influenza are uncertain, our analysis suggests that the community-wide phase of a 1918-like pandemic would be evident in the NYC ED system before 1% of total visits had occurred. Research and modeling studies indicate that while regional pandemic occurrence may be unavoidable, local detection and intervention initiated at a point <1% to 6% of total incident impact may be early enough for non-pharmaceutical social distancing efforts to be at least partially effective[2,3]. This provides evidence in support of delaying initiation of costly and controversial efforts, such as school and mass transit closure, until after community-wide transmission has been confirmed—and not simply after suspected, sporadic, or geographically distant cases are identified.

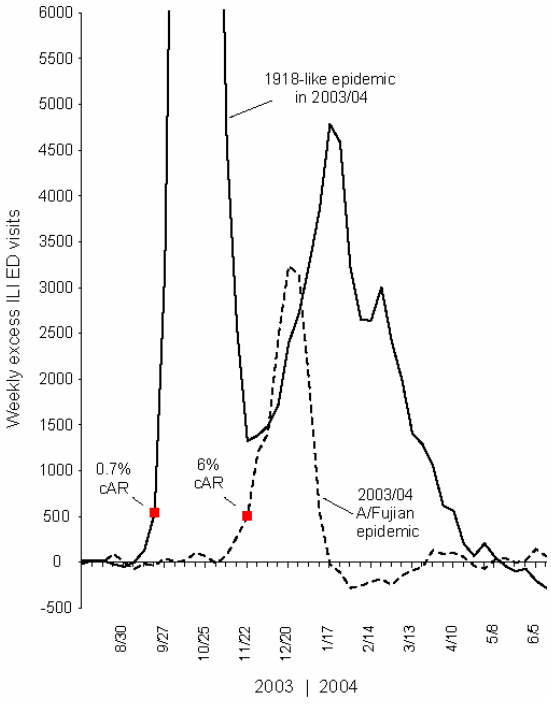

Figure Weekly excess influenza-attributable ED visits (obs – exp) are shown for the 2003/04 A/Fujian season and a 1918-like epidemic based on 1-wk illness to death lag and ~5:1 excess ED visit/deaths$_{1918}$. Red squares indicate %cAR for week of detection of community-wide epidemic >3.2 sd (Serfling model).

1. IOM (2006) *Modeling Community Containment for Pandemic Influenza* (National Academies Press, DC). http://www.nap.edu/catalog/11800.html
2. Hatchett RJ, Mecher CE & Lipsitch M (2007) *PNAS* 104:7582-7.
3. Bootsma MCJ & Ferguson NM (2007) *PNAS* 104:7588-93.
4. Olson DR, Simonsen L, Edelson PJ & Morse SS (2005) *PNAS* 102:11059-63.
5. Mills CE, Robins JM & Lipsitch M (2004) *Nature* 432:904-6.
6. Olson DR, Heffernan RT, Paladini M, Konty K, Weiss D & Mostashari F (2007) *PLoS Med* 4:e247.