No Detectable Surge in SARS-CoV-2 Transmission Attributable to the April 7, 2020 Wisconsin Election

See also the AJPH COVID-19 section, pp. 1123–1172.

The April 7, 2020, Wisconsin election produced a large natural experiment to help understand the transmission risks of severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2). As of April 14, 2020, 1,551,711 total votes were cast (https://bit.ly/2yWPhlF), and 1,138,491 absentee ballots were returned as of April 21, 2020, suggesting that approximately 413,220 people voted in person. Waiting times in Milwaukee averaged 1.5 to 2 hours. Poll workers had surgical masks and latex gloves, hand sanitizer was made available to voters, isopropyl alcohol wipes were used to clean voting equipment, and painting tape and signs were used to facilitate social distancing.

Wisconsin tracks cases confirmed by testing (Figure 1a) and throughout April 2020 have restricted testing to frontline workers and those hospitalized with serious illness. We used a deconvolution-based method to reconstruct the SARS-CoV-2 epidemic curve by dates of infections rather than dates of reporting by health authorities and then used two different methods to estimate the instantaneous reproduction number $R_t$, which is the average number of secondary cases generated by one primary case with the time of infection on day $i$, from March 25 (the start of the safer-at-home order) through April 18 (Appendix, available as a supplement to the online version of this article at http://www.ajph.org).

As seen in Figure 1b, there is no detectable spike in $R_t$ on April 7. The number of SARS-CoV-2 tests performed in Wisconsin (https://bit.ly/2L13XYj) has been relatively stable throughout April (Figure 1c), suggesting that reduced testing capacity in the days after April 7, which could have censored some of the April 7 infections, did not occur. Moreover, new SARS-CoV-2 hospitalizations in Wisconsin have steadily declined throughout April (Figure 1d), from a high of 101 on April 3 to a low of 14 on April 18 (https://bit.ly/2L13XYj), suggesting that daily new hospitalizations are much less than testing capacity.

The lengths of the incubation period and the reporting delay imply that April 7 infections would not be reported until April 17 on average, with most cases being reported between April 11 and 22. Taken together, there is no evidence to date that there was a surge of infections attributable to the April 7, 2020 election in Wisconsin, which has a low level of SARS-CoV-2 transmission relative to the United States.

Finally, the Wisconsin Department of Health Services announced on May 15 that 71 people who either voted in person or worked at the polls on April 7 have tested positive for SARS-CoV-2. However, many of these people also experienced nonvoting exposures, and hence this fact is not necessarily inconsistent with our population-level analysis. To put this information into perspective, if we assume that the SARS-CoV-2 fatality rate among symptomatic patients who were physically capable of voting on April 7 (e.g., not including nursing home residents) is 1% (using the fatality rate of known cases for people younger than 60 years), then (in the worst case, in which all 71 cases were attributable to voting) we would expect 0.71 deaths out of 413,220 people, which is the fatality risk of driving an automobile approximately 140 miles (https://bit.ly/35mRMjq). However, in addition to the individual risk of voting on April 7, there is the community risk: how many downstream cases will these 71 original cases generate?

According to Figure 1b, the reproduction number in Wisconsin has been hovering near the value of one for all of April. If this value was much larger than one (as it was in, say, January) then these 71 cases would cause a lot of downstream damage, and if this value was clearly smaller than one then they would cause minimal damage. But a value near one, coupled with the small number of cases, means that it is very difficult to reliably predict the amount of downstream damage.

Taken together, it appears that voting in Wisconsin on April 7 was a low-risk activity.

Kathy Leung, PhD
Joseph T. Wu, PhD
Kuang Xu, PhD
Lawrence M. Wein, PhD

CONTRIBUTORS
The authors contributed equally to this editorial.

ABOUT THE AUTHORS
Kathy Leung and Joseph T. Wu are with the World Health Organization Collaborating Centre for Infectious Disease Epidemiology and Control, School of Public Health, University of Hong Kong, Hong Kong Special Administrative Region, China. Kuang Xu and Lawrence M. Wein are with the Graduate School of Business, Stanford University, Stanford, CA. Correspondence should be sent to Lawrence M. Wein, Professor, Graduate School of Business, Stanford University, 635 Knight Way, Stanford, CA 94305 (e-mail: lwein@stanford.edu). Reprints can be ordered at http://www.ajph.org by clicking the “Reprints” link.

This editorial was accepted May 7, 2020. doi: 10.2105/AJPH.2020.305779
CONFlicts of interest

The authors declare no conflicts of interest.

REFERENCES


Note. The thick bar in each panel depicts April 7, 2020, the date of the Wisconsin election. In generating the curve in panel C, a possible misentry in the original data set² led to the cumulative test count on March 29 being smaller than the day before; in response, we replaced the March 29 cumulative case count by the average value between March 28 and 30.
SUPPLEMENTARY APPENDIX

The instantaneous reproductive number $R_t$ was defined as the average number of secondary cases generated by one primary case with the time of infection on day $t$. If $R_t > 1$ the epidemic is expanding at time $t$, whereas $R_t < 1$ indicates that the epidemic size is shrinking at time $t$.

Since the epidemic curve of Wisconsin is based on the dates of test confirmation, we use a deconvolution-based method to reconstruct the SARS-CoV-2 epidemic curve by dates of infection [1-2]. Let $f_{\text{incubation}}$ be the probability density function (pdf) of the incubation period, and $f_{\text{onset}\text{-}\text{confirmation}}$ be the pdf of the time between symptom onset and test confirmation. We assume $f_{\text{incubation}}$ and $f_{\text{onset}\text{-}\text{confirmation}}$ are independent such that the pdf of the time between infection and confirmation is

$$f_{\text{infection}\text{-}\text{confirmation}}(t) = \int_0^t f_{\text{onset}\text{-}\text{confirmation}}(t - u)f_{\text{incubation}}(u)du$$

We use $f_{\text{infection}\text{-}\text{confirmation}}$ to deconvolute the time series of the daily number of confirmed cases to reconstruct an epidemic curve of daily number of new infections. We assume the incubation period distribution is gamma with mean and SD of 5.2 and 2.3 days [3]. We assume that the distribution of the time between symptom onset and confirmation is gamma with mean and standard deviation (SD) of 4.3 and 3.2 days, based on 186 cases reported in Jan-Feb 2020 in Beijing [4]. With the epidemic curve by dates of infection in hand, we applied two different methods -- developed by Wallinga and Teunis [5] and by Cori et al. [6] -- to estimate $R_t$ using the R package EpiEstim. We assume the generation
time distribution is approximately the same as the serial interval distribution, which was inferred to be gamma with mean 5.4 and SD 4.7 days from the dates of symptom onset of 56 infector-infectee pairs from mainland China [4].