Comment on Dehning et al (Science, 15 May 2020, eabb9789: Inferring change points in the spread of COVID-19 reveals the effectiveness of interventions)

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Abstract

Dehning et al. (Science, 15 May 2020: eabb9789) report change points in the growth rate of SARS-CoV-2 infections that promptly respond to non-pharmaceutical interventions (NPIs). We challenge their findings on methodological and empirical grounds.

Comment on "Inferring change points in the spread of COVID-19 reveals the effectiveness of interventions"

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Abstract: Dehning et al. (Research Article, 15 May 2020: eabb9789) report change points in the growth rate of SARS-CoV-2 infections that promptly respond to non-pharmaceutical interventions (NPIs). We challenge their findings on methodological and empirical grounds.

Main Text: In many countries, NPIs against the spread of the coronavirus SARS-CoV-2 have been adopted that had strong negative side effects on the economy as well as on physical, mental, and social health conditions (*1-4*). Given these side effects, it is important to know whether these measures were successful in curbing the spread of the virus. To examine this issue, Dehning et al. (3) tried to model the growth rate of SARS-CoV-2 infections in Germany using a Susceptible-Infected-Recovered (SIR) model combined with Bayesian parameter inference. The authors find change points in the growth rate that respond closely to three NPIs that became effective 9 March (prohibition of large public events), 16 March (closing of schools and other educational institutions along with the closing of non-essential stores), and 23 March (extensive lockdown, including a contact ban). They conclude that the full extent of interventions was necessary to stop the virus spread.

There are several fundamental methodological issues that cast serious doubt on the conclusions drawn by Dehning et al. Accounting for these issues suggests that actually the opposite of their principal inference is correct: Neither of the governmental interventions could have had any effect on the virus spread because the number of new infections declined much earlier than estimated in their study. Furthermore, they ignore direct empirical evidence showing that the adopted countermeasures did have very low or even no effects. Taken together, we consider the study of Dehning et al. (*5*) as seriously flawed.

In order to assess potential effects of NPIs on the spread of a virus, it is crucial to determine the date of infection as exactly as possible. With misspecified infection dates, any conclusions about the effect of NPIs are meaningless. Dehning et al. estimate the date of infection based on the date when a confirmed case was reported, according to the Johns Hopkins University Center for Systems Science and Engineering (JHU CSSE) dashboard. In order to infer the infection date from the reporting date, they include a parameter in their SIR model that aims at determining the so-called 'reporting delay', i.e., the delay between infection date and reporting date. Critically, their parameter estimate is constrained by an

informative prior that, in turn, is based on the assumption of an incubation period (5–6 days) and a test delay. Using their priors, the authors estimate a reporting delay of 8.6 days (simple model, initial phase from March 2 to March 15) and 11.4 days (full model with change points, phase from March 2 to April 21).

This procedure is faulty. A first general problem is that Dehning et al. model the spread of the coronavirus based on data from the JHU CSSE dashboard. As Robert Koch Institute (RKI), Germany's federal health agency, point out in their official FAQ section on the coronavirus (*6*), data provided by the JHU CSSE dashboard allow only limited conclusions about the virus spread because these data stem from various internet sources (e.g., authorities, media reports, social media) which vary in reporting guidelines and do not provide more detailed information on the cases.

The second and even more fundamental problem is that the reporting delay estimated by Dehning et al. based on informative priors is much too short. An official RKI publication documents that the actual delay between infection date and reporting date amounted to 2-3 weeks, or 14-21 days (*7*). This figure exceeds the delays of 8.6 (11.4) days inferred from the model by Dehning et al. by far. The delay established by RKI is the sum of the incubation period, the lag until the patient visits a doctor after symptoms become more pressing, the time for conducting a test and returning its result, and administrative lags for data collection, processing, and publication. Crucially, test laboratories were overwhelmed during March 2020 and could not return their results in a timely manner.

A third problem of the approach to infer infection dates from published confirmed cases is that during weekends substantially fewer cases were reported. Dehning et al. try to account for this problem by adapting confirmed cases using a reporting fraction that is based on an estimation of additional parameters. However, the resulting stepwise pattern in the estimated effective growth rate within a week may reflect the artificial setting of parameters rather than the true course of the virus spread.

Finally, inferring infection dates from reporting dates would only make sense if reporting dates varied systematically with infection dates. However, the intervals between dates of actual infections, diagnostic testing, and reporting differ vastly across people. For instance, many suspicious people were tested even before symptom onset, whereas true patients were partly tested more than 20 days after symptom onset (*8*). Therefore, it is hardly possible to conclude anything meaningful from modeling the spread of infections using reporting dates. Moreover, employing raw numbers of confirmed cases without accounting for increasing test numbers introduces a strong distortion into the model because test numbers accelerated enormously during March.

RKI (*8*) employs a more reasonable approach. Their model is not based on reporting dates but on the dates of symptom onset, which are known for about 62.5% of the infected people in Germany. The benefit of such a modeling strategy is that dates of symptom onset reflect infection dates much more accurately and do not show weekend-related effects. To indicate the true coronavirus spread, RKI uses the effective reproduction number, R. Fig. 1 contrasts the spread of the coronavirus according to the flawed estimation by Dehning et al. with the estimation of the effective R-value by RKI.

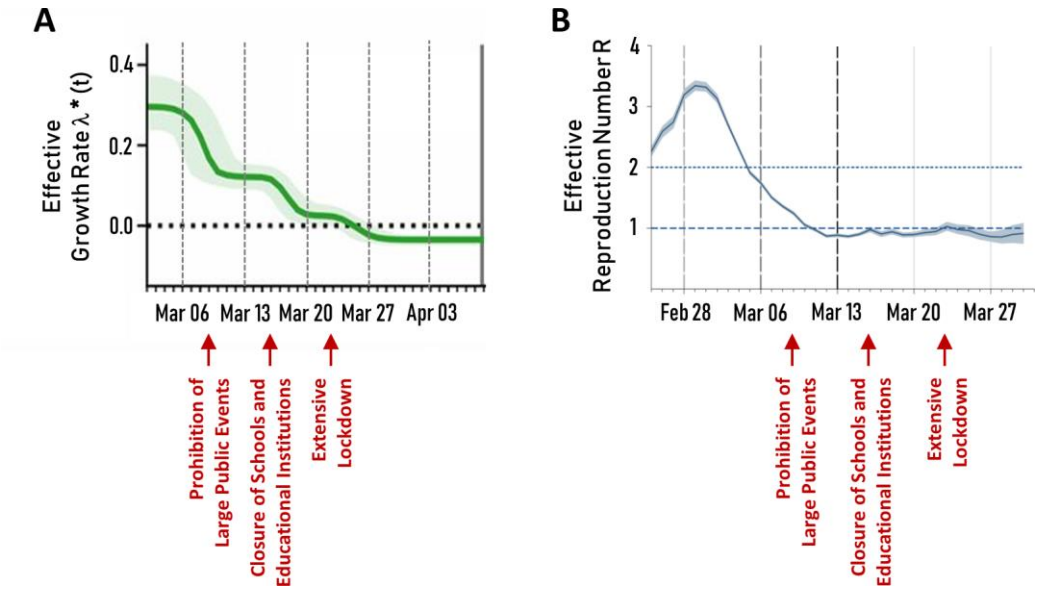

Fig. 1. **Illustration of the invalid (date of reporting) and valid (date of symptom onset) estimation of the spread of new infections**. (**A**) Estimation of the spread of new infections (effective growth rate) based on Dehning et al. (taken from (*5*), *Fig. 3A*). (**B**) Estimation of the spread of new infections (effective reproduction number) based on RKI (taken from (*8*), *Fig. 4*). Note: Since the timeline in the original RKI figure shows the spreading rate with a delay of 8 to 13 days (incubation period of 4-6 days, serial interval of 4 days, moving average of 4 days; for details, see (*9*)), for the purpose of visual comparison, the timeline in the original RKI figure has been shifted forwards by 10 days.

As can be seen in Fig. 1, when estimating the spread of the coronavirus based on the date of symptom onset (Fig. 1B), the spreading rate of the virus already starts to decrease on March 1, and a R-value of below one is reached already on March 10, just a single day after inhibition of large gatherings. Considering the variance in latency between infection and symptom onset, it is obvious that none of the NPIs could have had any effect; the virus spread was already in decline. Several reasons for such an autonomous decline have been suggested recently. One reason is that differences in host susceptibility and behavior can result in herd immunity at much lower prevalence of infection in the population. Accounting for individual variation in susceptibility or exposure to the coronavirus through a realistic parameter in modeling yields a maximum of 17% to 20% of the population that needs to be infected to reach herd immunity (*10*), an estimate that is empirically well supported by the cohort of the Diamond Princess cruise ship (*11*). Another reason is that seasonality may also play a role in the dissipation (*12*).

Finally, the ineffectiveness of some of the NPIs the study by Dehning et al. focuses on is also supported by empirical studies that examine their effects more directly. For instance, recent studies have shown that children are less involved in the transmission of SARS-CoV-2 than adults (*13*,*14*), suggesting that the closure of schools and kindergartens does little to contain the spread of SARS-CoV-2. This is supported by studies of previous pandemics. For instance, a recent review of the effects of school closures found only marginal effects regarding the spread of SARS-1 in China, Hongkong, and Singapur in 2003 (*15*).

To sum up, the inferences regarding the effectiveness of non-pharmaceutical measures by Dehning et al (*5*) are invalid for a number of reasons. In particular, their model implies counterfactual lags between infection dates and reporting dates. Conversely, RKI publications

suggest that the pandemic receded autonomously in Germany before any governmental measures were taken.

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