Practice of Epidemiology

Estimating Variability in the Transmission of Severe Acute Respiratory Syndrome to Household Contacts in Hong Kong, China

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The extensive data collection and contact tracing that occurred during the 2003 outbreak of severe acute respiratory syndrome (SARS) in Hong Kong, China, allowed the authors to examine how the probability of transmission varied from the date of symptom onset to the date of hospitalization for household contacts of SARS patients. Using a discrete-time likelihood model, the authors estimated the transmission probability per contact for each day following the onset of symptoms. The results suggested that there may be two peaks in the probability of SARS transmission, the first occurring around day 2 after symptom onset and the second occurring approximately 10 days after symptom onset. Index patients who were aged 60 years or older or whose lactate dehydrogenase level was elevated upon admission to the hospital (indicating higher viral loads) were more likely to transmit SARS to their contacts. There was little variation in the daily transmission probabilities before versus after the introduction of public health interventions on or around March 26, 2003. This study suggests that the probability of transmission of SARS is dependent upon characteristics of the index patients and does not simply reflect temporal variability in the viral load of SARS cases.

Communicable diseases, emerging; disease outbreaks; disease transmission; Markov chains; models, statistical; SARS virus; severe acute respiratory syndrome

Abbreviations: CI, credible interval; DIC, deviance information criterion; LDH, lactate dehydrogenase; SARS, severe acute respiratory syndrome.

In attempts to limit the spread of an infectious disease, the timing of infectiousness is critical to understanding the effectiveness of control measures such as isolation and quarantine (1). Nevertheless, most investigators who have modeled the impact of proposed interventions against severe acute respiratory syndrome (SARS) have assumed that the probability of transmission is constant throughout the duration of illness (2–4). If the probability of transmission of an infectious agent such as SARS does not peak until a week or more after the onset of symptoms, then interventions such as the isolation of symptomatic individuals will be more effective than estimated. However, if a significant proportion of transmission occurs before or soon after symptom onset, it will be considerably more difficult to identify and isolate infected persons before they transmit the virus (1). Quarantine of contacts who were exposed but are not yet symptomatic, as was done during the 2003 outbreak in Hong Kong, China (4–6), will also be more difficult but may be of great importance in curbing the epidemic.

During the Hong Kong SARS epidemic, a large amount of data was collected that included (nearly) complete case-finding (1,755 cases), extensive contact tracing for all cases, and the ability to assume that the entire population was susceptible because of lack of prior immunity. We sought to...
Statistical analysis

**MATERIALS AND METHODS**

**Data sources**

Data from an integrated database linked by unique population-wide identification numbers (SARSID), derived from the Hong Kong Hospital Authority eSARS system and a master list containing information on reported contacts from the Hong Kong Department of Health, were analyzed. In this paper, we use the term “subject” or “contact” to refer to a person, infected or not, who was exposed to a SARS case patient—referred to as the “index case” or “index patient.” On the basis of virologic and clinical observations, we assumed that an index patient could have infected any reported contact whose date of symptom onset was at least 1 day after his or her own, and that no transmission occurred before symptom onset. Subjects who later became cases could be index patients for other contacts within the same household.

We included subjects whose exposure to an index patient included one or more “close contacts” (defined as caring for, living with, or directly contacting the body fluids of a SARS patient) but excluded “social contacts” (all other less intensive forms of contact). We also excluded subjects whose relationship to an index case was described as “roommate”—since these situations were primarily in nursing home settings—and subjects whose index case was part of the Amoy Gardens superspreading event (defined as living in or near the Amoy Gardens housing estate) or another putative point-source outbreak. To limit the data set to household exposures, we used two different methods to eliminate hospital- or clinic-based exposures (see the “Supplementary Data” section, which is posted on the Journal’s website (www.aje.oxfordjournals.org)).

**Statistical analysis**

**Preliminary analysis.** In a preliminary analysis, we sorted index cases on the basis of time to hospitalization (time from the date of symptom onset to the date of hospital admission) and examined the proportion of contacts infected and the average number of secondary cases generated by each index case. Here, contacts who were exposed to more than one index case were enumerated for each of their index cases.

**Primary analysis.** We used a Markov chain Monte Carlo method to simultaneously estimate the incubation period and the daily transmission probability from the day of symptom onset (day 1) to the day of hospital admission of the index patient (ranging from a minimum of day 1 to a maximum of day 19). This method accounts for the exposure of some contacts to multiple index cases as well as the likelihood of each generation interval. The prior probability of transmission on each day of illness, $p(d)$, followed an independent uniform(0,1) distribution. We assumed that the probability of transmission was the same for days 13–19. The incubation period was assumed to be gamma-distributed; information on 81 patients with a single known exposure was used to inform the prior distribution.

We updated our joint prior distribution using a discrete-time likelihood model similar to that of Rampey et al. (9). For each subject $i$, his or her household was followed for a period of $t_f$ days, labeled $t = 1$ through $t = t_f$, where this window differed in calendar time for different subjects. If $i$ became infected, $t_f$ was chosen as the day on which he or she became symptomatic. If not, exposure was tracked from time $t = 1$ and the window was chosen to cover all days on which $i$ was exposed to a symptomatic household member.

The probability that subject $i$ escapes infection at time $t$ (assuming that $i$ is susceptible at time $t$) is given by

$$e_i(t) = \prod_{j \in h_i} (1 - p(T_{i,j}(t))),$$

where $h_i$ is the set of index cases to whom subject $i$ is exposed and $T_{i,j}(t)$ is the day of illness of index case $j$ at time $t$.

The likelihood contribution of uninfected contact $i$ is simply the probability that subject $i$ escapes infection from all of his or her index cases at time $t = 1$ through $t_f$:

$$L(p|T_{i,j}(t), y_i = 0) = \prod_{t=1}^{t_f} e_i(t),$$

where $p$ is the vector of transmission probabilities and $y_i$ is the infection status (1 = case, 0 = noncase) of the contact.

The likelihood contribution of infected contact $i$ is slightly more complicated, since it takes into account the date of symptom onset of contact $i$ and therefore the incubation period. It is assumed that all infected contacts become symptomatic at time $t_f$. Thus, the likelihood contribution of infected contact $i$ is

$$L(p, \theta|T_{i,j}(t), y_i = 1) = \sum_{t=1}^{t_f} [1 - e_i(t)]g(t_f + 0.5 - t) \prod_{u=1}^{t-1} e_i(u),$$

where $g$ is the probability density function for the incubation period and $\theta$ is its parameters.

**Stratified analyses.** In further stratified analyses, we assessed how transmission probabilities varied for subjects whose index case 1) reported symptom onset before or after major public health interventions were imposed on March 26, 2003 (10); 2) had a high LDH level upon admission (defined as being above the predicted value—see “Supplementary Data”) versus a low LDH level (11); and 3) was aged 60 years or older versus younger, given the steep prognostic gradient change near this age threshold (6).
The contacts’ incubation period was assumed to be the same across strata. Date of onset, LDH level, and age of the index case were found to be significant predictors of nosocomial transmission (11) or household attack rates (12) and therefore could confound and/or modify the relation between duration of exposure and probability of SARS transmission. Stratifying on more than one index case characteristic at a time was not possible because of the limited sample size. The stratified models were compared with one another and with the unstratified model using deviance information criterion (DIC) statistics (13).

To determine whether the day-to-day variation in the transmission probability suggested by our models was significant, we compared both the unstratified and stratified models with simpler models which assumed that the probability of transmission was constant for the duration of exposure. In this case, the probability of transmission on each day was forced to be the same within strata and to follow a single uniform(0,1) prior distribution. We used DIC statistics to compare models.

Sensitivity analysis. The foregoing approach to estimating the daily transmission probabilities assumes that there is no variability in the infectiousness of index cases or the susceptibility of their contacts on a given day. To determine the sensitivity of the model to this assumption, we generated a hypothetical population consisting of highly infectious and normally infectious index cases whose probability of transmitting SARS to their contacts differed (see “Supplementary Data”). First, we assumed that level of infectiousness was unrelated to duration of exposure (“unconfounded heterogeneity”); we then assumed that highly infectious index cases were more likely to be isolated early on in their illness (“confounded heterogeneity”). We also explored whether the probability of transmission could be estimated in the stratified context for highly and normally infectious index cases when there was confounded heterogeneity. The probability of transmission on each day was estimated and compared with the “true” population mean.

RESULTS

Our final data set consisted of 120 infected contacts, 1,893 uninfected contacts, and 765 index cases; some contacts were exposed to multiple index cases. Index cases had a mean of 2.87 contacts (table 1). The mean number of secondary cases per index case varied with time to hospitalization, from a low of 0.13 to maximum of 0.50.

Our primary analysis of the day-to-day variation in the transmission probability suggested that there might be a bimodal distribution in the infectiousness of index cases. The probability of transmission per contact was less than 0.06 on each day of illness but was estimated to be relatively high the day after symptom onset (0.029, 95 percent credible interval (CI): 0.014, 0.044). There was a larger second peak (0.058, 95 percent CI: 0.002, 0.169) that occurred approximately 10 days after symptom onset (figure 1). Estimates for later days were less robust because 1) there were fewer observations (i.e., only 25 percent of all exposures lasted more than 5 days) and 2) these estimates incorporated uncertainty from earlier days. The incubation period was estimated to follow a gamma distribution with a mean of 4.82 days (95 percent CI: 4.23, 5.50) and a variance of 15.8 days2 (95 percent CI: 11.56, 22.32) (figure 2).

Stratified analyses

The probability of transmission from index cases whose date of symptom onset occurred prior to public health
Interventions imposed on March 26, 2003, was slightly higher during the first 2 days after symptom onset and peaked again on day 9 after symptom onset (figure 3). Index cases whose symptoms began on or after March 26 had similar levels of transmission, with a slightly lower probability on days 1–5 post-symptom-onset and a higher peak on days 7–9. However, there were fewer index cases who were symptomatic for a week or more prior to isolation, so the estimates for these days were less precise. Stratifying by date of symptom onset did not improve the fit of the model to the observed data, based on the DIC statistics (table 2).

The probability of transmission from index patients whose LDH level was above the expected level was markedly higher than that from those whose LDH level was below the predicted level, peaking on days 8–9 of illness (figure 4). Index patients with low LDH levels were relatively unlikely to transmit infection to their contacts. The probability of transmission from low-LDH index patients was slightly increased the day after symptom onset and again during the second week of illness. The pattern of transmission from index patients whose LDH was not measured was similar to that of the unstratified data set. Stratifying by index-case LDH led to a considerably better model fit (table 2).

Similarly, index cases aged 60 years or older had a high probability of transmission compared with younger index patients. The probability of transmission from older index patients was approximately three times higher than in younger index patients during the first week of illness and remained high throughout the first 9 days of illness, decreasing slightly thereafter (figure 5). The probability of transmission from younger index cases was less than 0.022 prior to 1 week after symptom onset but appeared to be higher on days 8 and beyond, although there were limited data in this range. Again, stratifying by index patient age led to a better model fit (table 2).

**Comparison with simpler models**

The probability of transmission was estimated to be 0.013 (95 percent CI: 0.011, 0.016) per day for the unstratified model when transmission was assumed to be constant for the duration of exposure (table 2). A comparison of the DIC statistics suggested that allowing the probability of transmission to vary on each day offered a significant improvement in model fit for the unstratified model and most stratified models but did not improve the fit in comparison with stratifying by LDH level alone (table 2).

**Sensitivity analysis: effect of heterogeneity in simulated data**

For simulated data with infectiousness unrelated to duration of exposure (unconfounded heterogeneity), the model estimated the transmission probability relatively well (figure 6, part a). When infectiousness was negatively correlated with duration of exposure (confounded heterogeneity), the model performed slightly worse (figure 6, part b). However, the model was able to estimate the daily transmission probabilities within strata when we stratified on the infectiousness of the index case (normally infectious (figure 6, part c) vs. highly infectious (figure 6, part d)).

**DISCUSSION**

Previous efforts have been made to estimate the time course of infectiousness for SARS by examining the generation of secondary cases in mainland China (14) and
and by measuring temporal variation in viral load in prospectively identified cases from Hong Kong under the assumption that viral load is proportional to infectiousness (7). The results from these studies suggest that there is a single peak in infectiousness occurring approximately 7–10 days after symptom onset, with relatively little transmission occurring prior to 5 days post-symptom-onset. While the results of the present analysis support previous findings that an increase in infectiousness occurs approximately 1 week after symptom onset, they suggest that there

![FIGURE 3. Probability of transmission of severe acute respiratory syndrome (SARS) according to day of index case illness during the 2003 SARS epidemic in Hong Kong, China, stratified by date of symptom onset (before vs. on or after March 26). The solid lines represent the estimated mean transmission probabilities, while the dotted, dash-dotted, and dashed lines represent the 80%, 90%, and 95% credible intervals, respectively.](image)

### TABLE 2. Mean probability of transmission of severe acute respiratory syndrome obtained when assuming that the probability of transmission is constant, as well as deviance information criterion statistics for unstratified and stratified models 1) assuming that transmission probability is constant and 2) allowing for day-to-day variation in transmission probability, Hong Kong, China, 2003

| Model                  | No. of index cases | Probability of transmission when transmission is assumed to be constant | Deviance information criterion statistic for Markov chain Monte Carlo model |
|------------------------|--------------------|-------------------------------------------------|--------------------------------------------------------------------------|
|                        |                    | Mean 95% credible interval                      | Constant transmission Daily variation                                     |
| Unstratified           | 765                | 0.013 0.011, 0.016                              | 1,712.7 1,711.6*                                                          |
|                        |                    |                                                |                                                                          |
| Stratified by:         |                    |                                                |                                                                          |
| Date of symptom onset  |                    |                                                |                                                                          |
| Before March 26        | 318                | 0.015 0.012, 0.019                              | 1,712.7 1,710.3                                                          |
| On or after March 26   | 447                | 0.012 0.009, 0.015                              |                                                                          |
| Index case lactate dehydrogenase level |                |                                                |                                                                          |
| Above predicted        | 279                | 0.024 0.019, 0.030                              | 1,679.9 1,687.4                                                          |
| Below predicted        | 375                | 0.007 0.005, 0.010                              |                                                                          |
| Not measured           | 111                | 0.010 0.005, 0.016                              |                                                                          |
| Age of index case      |                    |                                                |                                                                          |
| ≥60 years              | 129                | 0.027 0.019, 0.035                              | 1,697.5 1,694.9                                                          |
| <60 years              | 656                | 0.011 0.009, 0.013                              |                                                                          |

* Estimated for 13 days of index case illness.
† Estimated for 10 days of index case illness.
may also be an early peak in transmission, with a number of contacts being infected soon after symptom onset in the index case(s).

A Bayesian transmission model was used to infer “individual infectivity profiles” over the course of SARS infection in China (14). Variability in the infectivity of SARS cases was modeled using a gamma distribution, which was found to provide the best fit to the data compared with models which assumed that the transmission probability was constant or proportional to viral load. The estimated infectivity peaked on day 9 following symptom onset (14), similar to the timing of the second peak seen in our analysis. However, such a model would not accommodate a bimodal distribution in the transmission probabilities such as the one suggested here.

In Singapore, there were relatively few secondary cases resulting from index patients who were isolated prior to 5 days post-symptom-onset and a large increase thereafter (2). While the small number of secondary cases per primary case for persons isolated 0–4 days post-symptom-onset is consistent with what we observed among household contacts from Hong Kong, in our data set, index cases never generated more than 0.5 secondary cases, on average (table 1). Previous studies found similar household member attack rates in the two countries (12, 15). However, the analysis from Singapore included both hospital- and community-based exposures. Time from symptom onset to isolation declined markedly over the course of the Singapore epidemic (2), so the association between long time-to-isolation and transmission to secondary cases would have been strongly

FIGURE 4. Probability of transmission of severe acute respiratory syndrome (SARS) according to day of index case illness during the 2003 SARS epidemic in Hong Kong, China, stratified by the lactate dehydrogenase (LDH) level of the index case (high, low, or not measured). The solid lines represent the estimated mean transmission probabilities, while the dotted, dash-dotted, and dashed lines represent the 80%, 90%, and 95% credible intervals, respectively.

FIGURE 5. Probability of transmission of severe acute respiratory syndrome (SARS) according to day of index case illness during the 2003 SARS epidemic in Hong Kong, China, stratified by age of the index case (≥60 years vs. <60 years). The solid lines represent the estimated mean transmission probabilities, while the dotted, dash-dotted, and dashed lines represent the 80%, 90%, and 95% credible intervals, respectively.
confounded by any other control measures, including those for nonhousehold transmission, that became more effective over time. Primary-case patients who were isolated 5 or more days after symptom onset may have been the major contributors to nosocomial transmission—which accounted for 76 percent of SARS infections in Singapore (16)—either because such cases tended to occur earlier in the epidemic when hospitals had yet to institute a policy of isolation for SARS patients (2) or because such patients had a less severe clinical presentation and thus hospital employees adopted fewer precautions upon their admission.

The results of our analysis also seem to be inconsistent with the viral shedding data for 14 patients from the Amoy Gardens housing estate (7). The slight increase in the transmission probability approximately 10 days after symptom onset does not reflect the 100-fold increase in viral load that occurred between days 5 and 10 in patients from that case series (7); nor is the early peak in transmission we find reflected in the viral load data. While viral load was not reported for nasopharyngeal swabs collected prior to 5 days after symptom onset in the Hong Kong case series, Tang et al. (17) found that the proportion of samples collected from possible and probable SARS cases in Toronto, Canada, that tested positive by reverse-transcriptase polymerase chain reaction increased steadily, from 35.4 percent for specimens collected on days 0–2 of illness to more than twice that for samples collected 9–11 days after symptom onset.

Unlike the studies on viral load, the current study took into account both the infectiousness of index cases and the susceptibility of their contacts. Any discrepancy between our findings and the viral load data may be due solely to this difference. The early peak in transmission may represent infection of highly susceptible contacts, while the second peak may reflect infection of persons who were only susceptible when exposed to a high cumulative viral dose. Alternatively, the early peak may represent infection of primary caregivers, who had a greater degree of exposure to the index case than other household members. Day-to-day differences in the precautions taken by household members exposed to SARS cases could account for the early peak if household members took fewer precautions during the first few days of symptoms than they did later on (18).

Similarly, the presence of highly infectious persons in the population could contribute to this early peak. If a significant proportion of contacts were infected during the first few days of exposure (presumably by highly infectious index

**FIGURE 6.** Sensitivity of statistical analyses to heterogeneous infectiousness in simulated data for a hypothetical population. Plots show the estimated transmission probabilities for (a) unconfounded heterogeneity, (b) confounded heterogeneity, and confounded heterogeneity when data are stratified on (c) normally infectious index cases and (d) highly infectious index cases. The solid lines represent the estimated mean transmission probabilities, while the dotted, dash-dotted, and dashed lines represent the 80%, 90%, and 95% credible intervals, respectively. The thick black line represents the “true” population mean probability.
cases), then fewer susceptible contacts would remain in the household on later days, leading to lower estimates of the transmission probability. This would be even more problematic if highly infectious persons tended to be hospitalized earlier in their illness, as suggested by our sensitivity analysis.

Stratification by index case LDH level or age should have reduced bias in our estimates by controlling for potential confounders. Persons with more severe disease (as indicated by a high LDH level) were more likely to transmit SARS to their contacts and tended to do so earlier in their illness. The slight increase in transmission from low-LDH index patients around day 10 is consistent with the timing of peak viremia (17). Similarly, the higher probability of transmission from elderly index patients may be due to the increased infectiousness of these persons or the increased susceptibility of their contacts. Older index patients were twice as likely to have high LDH levels (unpublished data). Furthermore, assessment of the age distribution of SARS cases revealed an excess of cases in the elderly (6, 19), suggesting that they were more susceptible to infection. The infected contacts of older index patients were more than twice as likely to also be aged 60 years or older than the infected contacts of younger index patients.

It is also possible that the early peak in transmission was due to the presence of co-primary infections within households. If members of the same household were infected simultaneously in the community but had slightly different incubation periods, persons with longer incubation periods might have been misclassified as having been exposed in the household. This could have led to overestimation of infectiousness soon after symptom onset. However, when we excluded all infected contacts with serial intervals of less than 3 days and reanalyzed the data, the results were similar (data not shown), suggesting that this was not a major source of bias.

Several other assumptions were made in our analysis. Based on prior findings that fewer than 0.2 percent of asymptomatic contacts of SARS patients were seropositive in this same population (20), we assumed no asymptomatic infections. Further, we assumed that all SARS index patients faithfully reported their date of symptom onset and identified all of their contacts. Incomplete contact tracing might have been a problem early in the epidemic, and some SARS patients might have underreported their contacts because they were too sick to answer questions or because they were reluctant to do so after policies such as quarantine were instituted. However, the average number of contacts per index case did not appear to vary with time to hospitalization (table 1), suggesting that such bias would have had a minimal effect on the observed pattern of transmission. Finally, the possible tendency of family members to report to the hospital only after more than one family member had become ill might have led to a spurious association between time to hospitalization and infectiousness. However, the fact that we see a similar pattern being repeated across strata suggests that this pattern of infectiousness is real and not a result of bias.

This analysis demonstrates the importance of effective disease surveillance, complete case-finding, and extensive contact tracing during an epidemic. Doing so not only allows for isolation of cases and quarantine of contacts, but also permits timely analysis of transmission dynamics so that the effectiveness and utility of certain interventions can be evaluated. More complete analyses following the epidemic allow investigators to better understand the factors contributing to transmissibility.

The lessons learned from this analysis do not apply only to SARS, which may or may not reemerge from its animal reservoir; some of these findings can probably be extrapolated to other diseases as well. The early peak in transmission that we observed may not be specific to SARS but rather may be due to variability in the susceptibility of contacts to any infection.

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REFERENCES

1. Fraser C, Riley S, Anderson RM, et al. Factors that make an infectious disease outbreak controllable. Proc Natl Acad Sci U S A 2004;101:6146–51.
2. Lipsitch M, Cohen T, Cooper B, et al. Transmission dynamics and control of severe acute respiratory syndrome. Science 2003;300:1966–70.
3. Lloyd-Smith JO, Galvani AP, Getz WM. Curtailing transmission of severe acute respiratory syndrome within a community and its hospital. Proc Biol Sci 2003;270:1979–89.
4. Riley S, Fraser C, Donnelly CA, et al. Transmission dynamics of the etiological agent of SARS in Hong Kong: impact of public health interventions. Science 2003;300:1961–6.
5. Donnelly CA, Ghani AC, Leung GM, et al. Epidemiological determinants of spread of causal agent of severe acute respiratory syndrome in Hong Kong. Lancet 2003;361:1761–6.
6. Leung GM, Hedley AJ, Ho LM, et al. The epidemiology of severe acute respiratory syndrome in the 2003 Hong Kong epidemic: an analysis of all 1755 patients. Ann Intern Med 2004;141:662–73.
7. Peiris JS, Chu CM, Cheng VC, et al. Clinical progression and viral load in a community outbreak of coronavirus-associated SARS pneumonia: a prospective study. Lancet 2003;361:1767–72.
8. Yu IT, Li Y, Wong TW, et al. Evidence of airborne transmission of the severe acute respiratory syndrome virus. N Engl J Med 2004;350:1731–9.
9. Rampey AH Jr, Longini IM Jr, Haber M, et al. A discrete-time model for the statistical analysis of infectious disease incidence data. Biometrics 1992;48:117–28.
10. Leung GM, Lam TH, Ho LM, et al. The impact of community psychological responses on outbreak control for severe acute
respiratory syndrome in Hong Kong. J Epidemiol Community Health 2003;57:857–63.

11. Chen MI, Chow AL, Earnest A, et al. Clinical and epidemiological predictors of transmission in severe acute respiratory syndrome (SARS). BMC Infect Dis 2006;6:151. (Electronic article).

12. Lau JT, Lau M, Kim JH, et al. Probable secondary infections in households of SARS patients in Hong Kong. Emerg Infect Dis 2004;10:235–43.

13. Spiegelhalter DJ, Best NG, Carlin BP, et al. Bayesian measures of model complexity and fit (with discussion). J R Stat Soc B 2002;64:583–640.

14. McBryde ES, Gibson G, Pettitt AN, et al. Bayesian modelling of an epidemic of severe acute respiratory syndrome. Bull Math Biol 2006;68:889–917.

15. Goh DL, Lee BW, Chia KS, et al. Secondary household transmission of SARS, Singapore. Emerg Infect Dis 2004;10:232–4.

16. SARS Investigation Team from DMERI. Strategies adopted and lessons learnt during the severe acute respiratory syndrome crisis in Singapore. Rev Med Virol 2005;15:57–70.

17. Tang P, Louie M, Richardson SE, et al. Interpretation of diagnostic laboratory tests for severe acute respiratory syndrome: the Toronto experience. CMAJ 2004;170:47–54.

18. Leung GM, Ho LM, Chan SK, et al. Longitudinal assessment of community psychobehavioral responses during and after the 2003 outbreak of severe acute respiratory syndrome in Hong Kong. Clin Infect Dis 2005;40:1713–20.

19. Anderson RM, Fraser C, Ghani AC, et al. Epidemiology, transmission dynamics and control of SARS: the 2002–2003 epidemic. Philos Trans R Soc Lond B Biol Sci 2004;359:1091–105.

20. Leung GM, Chung PH, Tsang T, et al. SARS-CoV antibody prevalence in all Hong Kong patient contacts. Emerg Infect Dis 2004;10:1653–6.