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SARS-CoV-2 transmission and control in a hospital setting: an individual-based modelling study — [Source link](#)

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1 **SARS-CoV-2 transmission and control in a hospital setting: an individual-based**
2 **modelling study**

3 **Running title:** SARS-CoV-2 transmission and control in a hospital setting

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24 **Abstract**

25 **Background** Development of strategies for mitigating the severity of COVID-19 is now a top global
26 public health priority. We sought to assess strategies for mitigating the COVID-19 outbreak in a
27 hospital setting via the use of non-pharmaceutical interventions such as social distancing, self-
28 isolation, tracing and quarantine, wearing facial masks/ personal protective equipment.

29 **Methods** We developed an individual-based model for COVID-19 transmission among healthcare
30 workers in a hospital setting. We calibrated the model using data of a COVID-19 outbreak in a
31 hospital unit in Wuhan in a Bayesian framework. The calibrated model was used to simulate
32 different intervention scenarios and estimate the impact of different interventions on outbreak size
33 and workday loss.

34 **Results** We estimated that work-related stress increases susceptibility to COVID-19 infection among
35 healthcare workers by 52% (90% Credible Interval (CrI): 16.4% - 93.0%). The use of high efficacy
36 facial masks was shown to be able to reduce infection cases and workday loss by 80% (90% CrI:
37 73.1% - 85.7%) and 87% (CrI: 80.0% - 92.5%), respectively. The use of social distancing alone,
38 through reduced contacts between healthcare workers, had a marginal impact on the outbreak. A
39 strict quarantine policy with the isolation of symptomatic cases and a high fraction of pre-
40 symptomatic/ asymptomatic cases (via contact tracing or high test rate), could only prolong outbreak
41 duration with minimal impact on the outbreak size. Our results indicated that a quarantine policy

42 should be coupled with other interventions to achieve its effect. The effectiveness of all these
43 interventions was shown to increase with their early implementation.

44 **Conclusions** Our analysis shows that a COVID-19 outbreak in a hospital's non-COVID-19 unit can
45 be controlled or mitigated by the use of existing non-pharmaceutical measures.

46 **Keywords:** COVID-19, non-pharmaceutical interventions, hospital setting, individual-based model

47 **Background**

48 The world is in the midst of an unprecedented coronavirus outbreak caused by a novel virus recently
49 named COVID-19 by the World Health Organization (WHO). The outbreak which started in the city
50 of Wuhan, Hubei Province, China, in early December 2019 spread to many countries around the
51 world before being declared a pandemic by the WHO on March 11th, 2020.

52 Developing strategies for mitigating the severity of COVID-19 is now a top global health priority.

53 The range of containment strategies employed in different countries and regions varies from shelter-
54 in-place orders, the shutdown of public events, travel ban [1], and visitor quarantine, to intermediate
55 steps that involve partial closures (e.g. schools [2], workplaces, sporting, and cultural events) [3].

56 While such drastic steps can reduce infection spread, they exact a heavy toll on society and human
57 well-being. At present the only available means of containing COVID-19 spread is via the use of
58 non-pharmaceutical interventions [4, 5] such as social distancing, self-isolation [6], tracing and
59 quarantine [6, 7], wearing facial masks/ personal protective equipment (PPE) [8, 9].

60 Mathematical models of disease transmission are powerful tools for exploring this complex
61 landscape of intervention strategies and quantifying the potential benefits of different options [10-
62 13]. Traditional approaches in epidemiological modeling use compartmental models [14-16], which
63 assume a uniform population and simple mixing patterns with steady contact rates. Such models can
64 give qualitative answers for large-scale populations at best [17, 18], however, are not suitable to
65 account for the complexity and specifics of COVID-19 in local communities and small populations
66 (e.g., hospital, workplace, school). Such settings are characterized by heterogeneous populations,
67 multiple disease pathways, and complex social interactions.

68 Our focus here is COVID-19 transmission in a hospital setting, where healthcare workers (HCWs)
69 are at high risk to acquire infection through interactions with fellow HCW and with patients [19-22].
70 We developed a novel individual-based model (IBM) for COVID-19 transmission among HCWs,
71 and applied it to explore the efficacy of different control/ mitigation strategies via non-
72 pharmaceutical interventions. IBMs have been used extensively to model pathogens spread on
73 different scales, from global pandemics [23-25], to local social networks [26]. On the disease side,
74 our IBM features distinct infective stages and transitions, observed in Covid-19, with some hosts
75 recovering without any symptoms, while others undergoing mild or severe infection pathways. On
76 the social side, we take into account individual behavior, including mixing patterns among HCW,
77 their use of facemasks and PPE, and HCW-patient interactions. All of these factors play an
78 important role in COVID-19 transmission.

79 The IBM model was calibrated in a Bayesian framework using empirical data from a non-COVID
80 hospital unit. We used our calibrated model to simulate different intervention scenarios, including
81 adaptive behavior (social distancing in the workplace, individual protection, isolation of infected
82 individuals). In each case, we assessed the effect of interventions on outbreak outcomes: outbreak
83 size, outbreak duration, and workday loss.

84 **Methods**

85 *Individual-based modelling methodology*

86 In our model, an individual can undergo a sequence of infection stages, classified as susceptible (S),
87 pre-symptomatic/ asymptomatic (E), two symptomatic stages I_1 (upper respiratory stage) followed
88 by I_2 (advanced infection stage, lungs et al.), and recovered/immune state (R) (see Figure 1). These
89 states differ by their infectivity levels and stage duration. Unlike most other viral diseases, pre-

90 symptomatic/asymptomatic COVID-19 hosts (E -stage) are known to transmit pathogens [27-29]. So
91 we assign positive infectivity levels (b_0, b_1, b_2) to all three stages (E, I_1, I_2).

92 We modelled social mixing patterns by assuming that HCWs and ward patients interact on a daily
93 basis via aggregating in random groups of HCWs, and via patient visitation by HCWs (See SI in
94 details). The net outcome is a contact pool for each HCW-host, which varies randomly on a daily
95 basis. Each contact of a susceptible individual with infectious individuals (HCWs or patients) can
96 lead to infection (transition $S \rightarrow E$), with a probability that depends on infectivity levels of the
97 contact pool and the host susceptibility, a ($a = 0$ - fully protected, $a = 1$ - fully susceptible). The
98 latter depends on host health /immune status, individual behavior e.g. use of facial masks, and
99 environmental conditions. For instance, HCWs are supposed to use additional protection when
100 contacting patients. Then a probability of ‘surviving’ a single infective contact (b_i) for an S -host of
101 susceptibility a , is given by $1 - a b_i$. Combining all infective contacts of a given S -host, we get the
102 probability of infection ($S \rightarrow E$), $p_s = 1 - \prod_i (1 - a b_i)$.

103 We divided all HCWs staff into susceptibility strata based on the hospital data [30]: (i) normal pool,
104 60% of HCWs, have baseline susceptibility value, $a_N = 0.5$; (ii) high-risk (stressed) pool, 40% of
105 HCWs, with susceptibility level, $0.5 < a_S < 1$ (to be calibrated).

106 Two points of our setup require some clarification: (i) the proposed form of social mixing in random
107 clusters extends the conventional ‘social network’ transmission pathways (see Figures. S1-S2); (ii)
108 an infective ‘social contact’ in our context means an event of sufficient duration and proximity, to
109 allow transmission of pathogens from infected to susceptible host [31].

110 There is much uncertainty on disease progression of infective stages. Here we assume infected E -
111 hosts can undergo three different pathways: (A) asymptomatic ($E \rightarrow R$); (M) mild symptomatic
112 ($E \rightarrow I_1 \rightarrow R$); (S) severe symptomatic ($E \rightarrow I_1 \rightarrow I_2 \rightarrow R$), with population fractions ($v_A; v_M; v_S$).
113 In all cases, pre-symptomatic/ asymptomatic pool (E) can carry and transmit the virus, along with
114 (I_1, I_2). Each infective stage (E, I_1, I_2) has associated (mean) duration, $L_E; L_1; L_2$. The probability of
115 state transition ($E \rightarrow I_1; I_1 \rightarrow I_2$, etc.) depends on the time, d , spent in a given disease state, relative
116 to the mean stage duration, L . Specifically, we use a Bernoulli distribution $B(p)$ of parameter, $p =$
117 $\Phi(d/L)$, with sigmoid ($0 < \Phi(x) < 1$) of half-value, $x = 1/2$.

118 During an outbreak, the HCWs expressing symptoms are tested, and certain fractions ($f_1; f_2$) of
119 ($I_1; I_2$) are put in isolation, where they undergo their specific disease pathways, but do not mix and
120 transmit the pathogen. Two different types of diagnostic tests were used in the hospital, PCR for
121 light symptoms, and lung-scan for more severe conditions [30, 32]. Thus our assumed quarantine
122 fractions ($0 < f_i < 1$) account for limited test sensitivity, and a possible overlap of ‘COVID-like’
123 symptoms, expressed by non-COVID hosts. The recovered HCWs return to the work pool (see
124 Figure 1).

125 The model simulations were run on a daily basis and implemented in the Wolfram Mathematica
126 platform. The key inputs in the model include: 1) population makeup in terms of asymptomatic,
127 mild, and severe (A-M-S) progress groups, 2) initial infection status of HCW pool; 3) infectivity
128 levels (b_0, b_1, b_2) for (E, I_1, I_2) stages and susceptibility levels of individual hosts or host-pools; 4)
129 average duration of infective stages for (A-M-S) pathway; 5) daily social mixing patterns between
130 HCWs and infected patients; 6) daily isolation of symptomatic cases and recovery (See SI in details).

131 ***Model calibration***

132 Our model is calibrated to empirical data of a COVID-19 outbreak among HCWs in the department
133 of neurosurgery of Union Hospital, Wuhan, China from January 5th, 2019 to February 4th, 2020
134 [30]. A Bayesian method is used to calibrate the following important parameters in our IBM: (i)
135 mean infectivity (b_1, b_2) of symptomatic hosts (I_1, I_2), (ii) increased susceptibility level ($0.5 < a_s < 1$)
136 of the high-risk pool; (iii) fraction ν_A of HCWs going through the asymptomatic pathway ($E \rightarrow R$).

137 The Bayesian method uses the posterior probability distribution to quantify the uncertainties in these
138 model parameters using the observed data on the daily incidence of symptomatic cases and the daily
139 isolated cases. The prior distributions for all these parameters are taken to be uniform within
140 acceptable ranges. The likelihood for the observed data is assumed as a normal distribution with the
141 center at the predicted values from the IBM. The adaptive Metropolis algorithm [33] is used to
142 sample from the posterior distribution, where the jump size is adaptively chosen based on the sample
143 covariances. The chains are run for 10000 iterations, and after 5000 burn-in every 50th sample is
144 used as the final sample from the posterior distribution. To assess the convergence of the posterior
145 sampling, the Gelman-Rubin statistic [34] is computed for all the parameters. The statistics are found
146 to be very close to 1, the desired value in strong support of convergence. The calibration was
147 implemented using R statistical software.

148 ***Wuhan Hospital outbreak***

149 On December 26th, 2019, a patient later diagnosed with COVID-19 was admitted in the department
150 of neurosurgery of Union Hospital, Wuhan, China. No PPE was used by HCWs at that time. By
151 January 8th, HCWs started to show COVID-like symptoms (headache, cough, sore throat), and
152 screening and isolation were initiated among HCWs. From January 19th, patient's admission was

153 stopped in the department, and the hospitalized patient pool was gradually reduced from 200 to 20
154 by the beginning of February. Over the period from January 5th to February 4th, 92 out of the 171
155 HCWs of the department were suspected or confirmed COVID-19 cases and isolated. New patients
156 were only admitted in early March 2020 when the pandemic was declared under control in Wuhan.

157 *Intervention strategies*

158 We consider three types of interventions: (i) social distancing (reduced contact rates) among HCWs
159 and individual protection (facial masks), (ii) enhanced screening and isolation of infected HCWs,
160 (iii) patient-pool control (pool size and infection level), and individual HCW protection via PPE.

161 For our baseline case, we assumed 50% and 100% isolation (quarantine) fractions of symptomatic
162 cases ($I_1; I_2$), respectively, and fixed infection level of the patient pool (see Table S1). To account
163 for model uncertainties, we run each control simulations for 100 posterior parameter samples and 5
164 stochastic model realizations for each sample (500 histories altogether), over a six-month period.

165 For social distancing, we considered 50% and 75% contact-rate reduction relative to their baseline
166 values. The effect of face mask on inter-staff or staff-patient mixing, was simulated by reduced
167 susceptibility of individual HCWs, with several values of mask efficacy [9]. Screening and isolation
168 fractions (f_i) of HCWs were based on limited test sensitivity, combined with non-COVID
169 symptoms. An increase in targeted isolation assumes more intensive screening or test sensitivity. We
170 also studied the effect of isolating pre-symptomatic/ asymptomatic cases (E -pool). This task is more
171 challenging, as PCR tests have lower sensitivity for such hosts [32], so to identify a suitable E -
172 fraction would require intensive mass screening or contact tracing.

173 For quantitative assessment of control interventions and their impact, we use two measures: (1)
174 outbreak size = infection turnover (by the end of outbreak); (2) workday loss estimated from the

175 quarantine pool over the outbreak duration. The latter gives a simple economic measure of outbreak
176 impact and putative interventions. In each control experiment, we compare the ratio of two outputs
177 (outbreak size and workday loss) to their baseline values, and record these relative values and their
178 distribution.

179 Another important factor in the hospital setting is the in-patient pool. In our case (a non-COVID unit
180 in Wuhan), it varied from the full capacity to zero. The key inputs of the patient pool included (i)
181 infected prevalence, (ii) mean patient infectivity to HCWs. The former is controlled by patient
182 admission and screening/isolation procedures; the latter can be modulated by using PPE. We also
183 explored the effect of different timing of PPE implementation and its efficacy.

184 **Results**

185 *Model calibration*

186 The predictions from the calibrated IBM were very close to the observed data on daily symptomatic
187 and quarantine cases (see Figure 2). The fraction of asymptomatic disease-progress pool, ν_A , was
188 estimated at 0.308 (90% Credible Interval (CrI): 0.163 – 0.395). So a sizable part of transmission
189 was carried over by undetected cases (E -pool). Susceptibility level of the high-risk pool was
190 estimated at $a_S = 0.76$ (90% CrI: 0.582 – 0.965). We attributed a higher susceptibility level to work-
191 stress, and our results gave a quantitative measure to this increase at 52% (90% CrI: 16.4% - 93.0%)
192 above the normal level. The infectivity levels of pre-symptomatic and symptomatic infections were
193 estimated to be 0.124 (90% CrI: 0.111 – 0.144) and 0.225 (90% CrI: 0.202 – 0.262). See SI Figure.
194 S3 for the prior and posterior probability distributions.

195 ***Interventions***

196 The baseline scenario showed that almost all HCWs get infected, resulting in significant workday
197 loss, 1050 (90% CrI: 913-1282) over the six-month period (Table 1 and SI Figure. S4). The impact
198 of implementing social distancing through reduction of contact rates alone and wearing facemasks
199 alone, from the start of the outbreak, was evaluated (Table 1). The reduction of contact rates alone
200 has a marginal effect on mitigating the outbreak in the long run. The 50% - drop of contact rates
201 leads to about 4%-6% reduction of the outbreak size and workday loss, while 75% - drop leads to a
202 15%-17% reduction, relative to baseline values. The efficacy of facemasks is uncertain, and we
203 explored several values (50%, 67%, 75%, 85%, 95%), based on the previous studies [9]. We have
204 shown that wearing facemasks had a higher impact on mitigating the outbreak, than social distancing
205 (reduced contact rates). At 95% efficacy, we could achieve 80% (90% CrI: 73.1% - 85.7%)
206 reduction of outbreak size, and 87% (CrI: 80.0% - 92.5%) of workday loss, compared to the
207 baseline.

208 Figure 3 illustrates the combined effect of facemask and social contact. We used the same values of
209 facemask efficacy and contact rates as Table 1. For each value of facemask efficacy, we observed a
210 consistent reduction of the outbreak size with reduced contact rates. It varied from 13% - 34% drop
211 for low-efficacy facemask (50% protection), to 30% - 60% drop for high-efficacy facemask (95%
212 protection). We observed a similar percentage reduction for the workday loss. So the impact of
213 reduction of contact rates was much greater under the higher efficacy of facemasks.

214 We also explored the effect of timing of intervention by the following three scenarios: (1) at the
215 beginning (Figure. 3A); (2) after the first identified case (Figure. 3B); (3) after 10% of HCWs have
216 been identified as infected (Figure. 3C). Early interventions have made marked improvement under
217 different types of facemasks and contact rates. For instance, if control interventions (adoption of

218 high-efficacy facemasks and reduced contact rates) were implemented at the start of the outbreak,
219 we observed 80% - 90% reduction of the outbreak size (a near-complete control). A later
220 implementation (e.g. after the first identified case), gave 60% - 85% reduction. If the timing was
221 delayed to e.g. 10% identified cases, these numbers dropped to 40% - 60%. All intermediate cases
222 were shown in Figure. 3.

223 We next looked at the effect of HCWs screening and isolation via two scenarios. The first scenario
224 considered symptomatic cases only, by changing quarantine fraction (f_1) of I_1 , from its baseline
225 value (50%) to 60% – 100%; quarantine fraction (f_2) of I_2 was fixed at 100%. Figure. 4A shows
226 increased symptomatic isolation had only a marginal effect on the outbreak size, while raising
227 workday loss. A clue to low efficacy of symptomatic screening lies in (i) the role of pre-
228 symptomatic/ asymptomatic (E) pool in transmission, (ii) contribution of the patient source. To test
229 (i) we extended our quarantine strategy to pre-symptomatic/ asymptomatic cases (E). Of course,
230 such an extension requires intensive screening of the work pool. Under random selection, isolating f
231 -fraction of E , would require much more than f -fraction of HCWs tested. For numeric simulations,
232 we fixed (f_1, f_2) at (90%, 100%), and varied E -fraction from 10% to 60%. We still found the
233 effect of such a strategy was limited, it often prolongs the outbreak duration without affecting its
234 size. Besides, such a strategy can incur an economic burden by increased workday loss, though the
235 effect is subtler, as increased quarantine rate can slower transmission rate, hence fewer hosts would
236 be infected and need isolation. More significant progress was achieved by controlling the patient
237 source, via reduced patient prevalence (screening), or reduced infectivity (PPE) (see Figure. 4A-B).

238 We run several experiments with patient-pool control and PPE use (Figure. 4C). For PPE timing, we
239 made three choices: (i) the start of an outbreak, (ii) after the first identified HCW-case, (iii) no PPE

240 use. We assumed PPE provides 80% protection (via the reduced probability of transmission from an
241 infected patient). We also varied the infected prevalence level of the patient pool, from 0% to 5%
242 (baseline case was 2%). We found the control of patient infection (via e.g. PPE, screening and
243 isolation, particularly for new patients) can reduce outbreak size, even though the bulk of
244 transmission is carried over by inter-staff HCW contacts. We found the combined strategy (enhanced
245 HCW screening/isolation with patient control) could lead to marked improvement both in outbreak
246 size and in workday loss. This effect, however, is not observed for quarantine alone, under persistent
247 patient source.

248 Overall, we saw high-efficacy facemasks could provide the most effective control tool for reducing
249 COVID-19 transmission in HCW staff (Figure. 3).

250 **Discussion**

251 With the spread of COVID-19 in the world, the development of strategies for mitigating its severity
252 is a top public health priority. Large-scale population-level models of SARS-CoV-2 transmission
253 can give some qualitative answers for outbreak control on regional/country scales [35], however,
254 few studies have looked at the effects of interventions in a local community setting, such as hospital,
255 workplace, and school.

256 Using a novel individual-based modelling approach, we explored different scenarios for COVID-19
257 transmission and control in a non-COVID hospital unit. Our IBM methodology employed
258 conventional SEIR disease-stages with graded infectivity, extended to heterogeneous host makeup,
259 which includes multiple disease pathways, varying individual susceptibility, and behavioral patterns.
260 These factors can be affected by work stress, health status, use of face masks /PPE, and social
261 interactions. Social mixing was implemented in our model via inter-HCW contact pools and HCW-

262 patient interactions. Control interventions included test diagnostics and isolation of established
263 cases. Detailed data on the COVID-19 outbreak in the department of neurosurgery of Union
264 Hospital in Wuhan (China) [30] was used to calibrate the essential model parameters. It included the
265 onset of symptoms, diagnosis and isolation, and patient pool control, over the period January 5th,
266 through February 4th, 2020.

267 We allowed different disease-progress pathways: severe, mild, and asymptomatic (S-M-A), so the
268 HCW-pool was partitioned into (S-M-A) progress groups. One of the key uncertain parameters was
269 the asymptomatic-fraction, estimated at 31%, a relatively high proportion of undetectable infections.
270 Another uncertain input was individual susceptibility, which could be affected by health status or
271 work stress. Based on data analysis [30], we divided the work pool into a normal group (60% of
272 HCWs), and a high-risk group (40% of HCWs). We estimated the high-risk susceptibility level
273 relative to normal susceptibility and found work-related stress could increase the risk of COVID-19
274 infection by up to 52%.

275 The calibrated model was used to simulate a range of intervention scenarios, aimed at mitigating the
276 outbreak and examining its impact on the work pool. The baseline case, without interventions, gave
277 a large outbreak size, whereby almost all HCWs were infected over two months. It also incurred a
278 significant workday loss for the unit. Our results support early modeling findings of large-scale
279 populations, and subsequent empirical observations, that in the absence of control measures, a
280 COVID-19 epidemic could quickly overwhelm a region [12]. High-efficacy facemasks were shown
281 to be most effective for reducing infection cases and workday loss. The impact of social distancing
282 through the reduction of contact rates alone had an only marginal effect on mitigating the outbreak in
283 the long run. Reducing social contact rates to 50% (or 70%) resulted in a 4%-6% (or 15%-17%) drop

284 in the outbreak size, and a similar drop in the workday loss, compared to the baseline case. However,
285 the impact of reduction of contact rates was much greater under the higher efficacy of facemasks.

286 Implementing the quarantine policy (HCW screening and isolation) alone, even when all
287 symptomatic cases are included, would typically prolong the outbreak duration, but had a marginal
288 effect on its size, particularly under the existence of external (patient) source pressure. Our results
289 indicated that the low efficiency of symptomatic quarantine was due to a large share of transmission
290 being carried by pre-symptomatic/ asymptomatic (*E*) individuals [36], and to the patient source.
291 Hence, a quarantine policy for HCWs should be augmented with other interventions to achieve a
292 significant reduction. Efficient control of the patient source (via the use of PPE, their screening and
293 isolation, and/or admission) is one key to mitigating the HCW outbreak. The effectiveness of all
294 these interventions was shown to increase with their early implementation.

295 To our knowledge, this study is the first of its kind to provide quantitative modelled assessment and
296 projections for COVID-19 transmission in hospital settings. However, the IBM methodology
297 developed here, has a far broader scope, beyond healthcare facilities. Indeed, with proper
298 adjustment, it could be applied to many other local communities (workplaces, schools, city
299 neighborhoods, et al). The key feature of such IBM is a fine-scale resolution of community makeup,
300 social interactions, and disease pathways. Such information is essential for risk assessment and the
301 development of efficient control/ intervention strategies on a local scale.

302 The current model setup is subject to some limitations. First, it was designed for a single hospital
303 unit and simplified treatment of the patient pool, as the target group in our study was HCW-pool.
304 More realistic local communities could combine multiple units (e.g. large hospital), with refined
305 population structure (e.g. patients, visitors, staff), and more complex interactions (e.g. ‘random’ and

306 ‘scheduled’ contact pools). Empirical data on these interactions will be required to adequately
307 parameterize such models. Second, although we have made an effort to characterize the SARS-CoV-
308 2 transmission in a hospital setting, some parameters used in our setup were drawn from general
309 information sources, such as fractions of symptomatic mild and severe cases [37], disease stages and
310 durations [38], and associated infectivity levels [39], which may be adjusted in the future work.

311 **Conclusion**

312 Overall, our analysis shows that a COVID-19 outbreak among HCWs in a non-COVID-19 hospital
313 unit can be efficiently controlled /mitigated by non-pharmaceutical means. The most crucial factor of
314 success is high-efficacy facemasks for HCW contacts. It can be further augmented by social
315 distancing, screening/isolation, and patient source control.

316 **List of abbreviations**

317 World Health Organization (WHO); healthcare workers (HCWs); personal protective equipment
318 (PPE); Credible Interval (CrI).

319 **Declarations**

320 *Ethics approval and consent to participate*

321 The study protocol was approved by the institutional ethics board of Union Hospital, Tongji Medical
322 College, Huazhong University of Science and Technology, Wuhan, China (No. 20200029). Written
323 informed consent was required before the data collecting, and participants were informed that they
324 could refuse to answer any question. The questionnaire did not ask about infection status, and no
325 biological samples were collected.

326 *Consent for publication*

327 Not applicable.

328 *Availability of data and materials*

329 The computer codes, including the aggregated data, implemented in the Wolfram Mathematica
330 platform are available in Githubs <https://github.com/gimin-h/COVID-19-huang-et-al.->. All other
331 materials are available from the corresponding authors on reasonable request.

332 *Competing interests*

333 The authors declare that they have no competing interests.

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339 writing of the report, or the decision to submit for publication. The corresponding authors had full
340 access to all of the data and the final responsibility to submit for publication.

341 *Authors' Contributions*

342 QH, AM, and XJ contributed equally and shared the first authorship. QH, DG, AM, and MN
343 designed research, did model development, calibrations, and simulations, XJ, HZ, FF, PF, and WX
344 collected and provided hospital data, QH wrote the first draft, MN, DG, AM, and MAH made
345 critical revision of the manuscript. All the authors contributed to the interpretation of the study
346 results, read, comment, and approved the final version.

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349 to all the data in the study and took responsibility for the integrity of the data.

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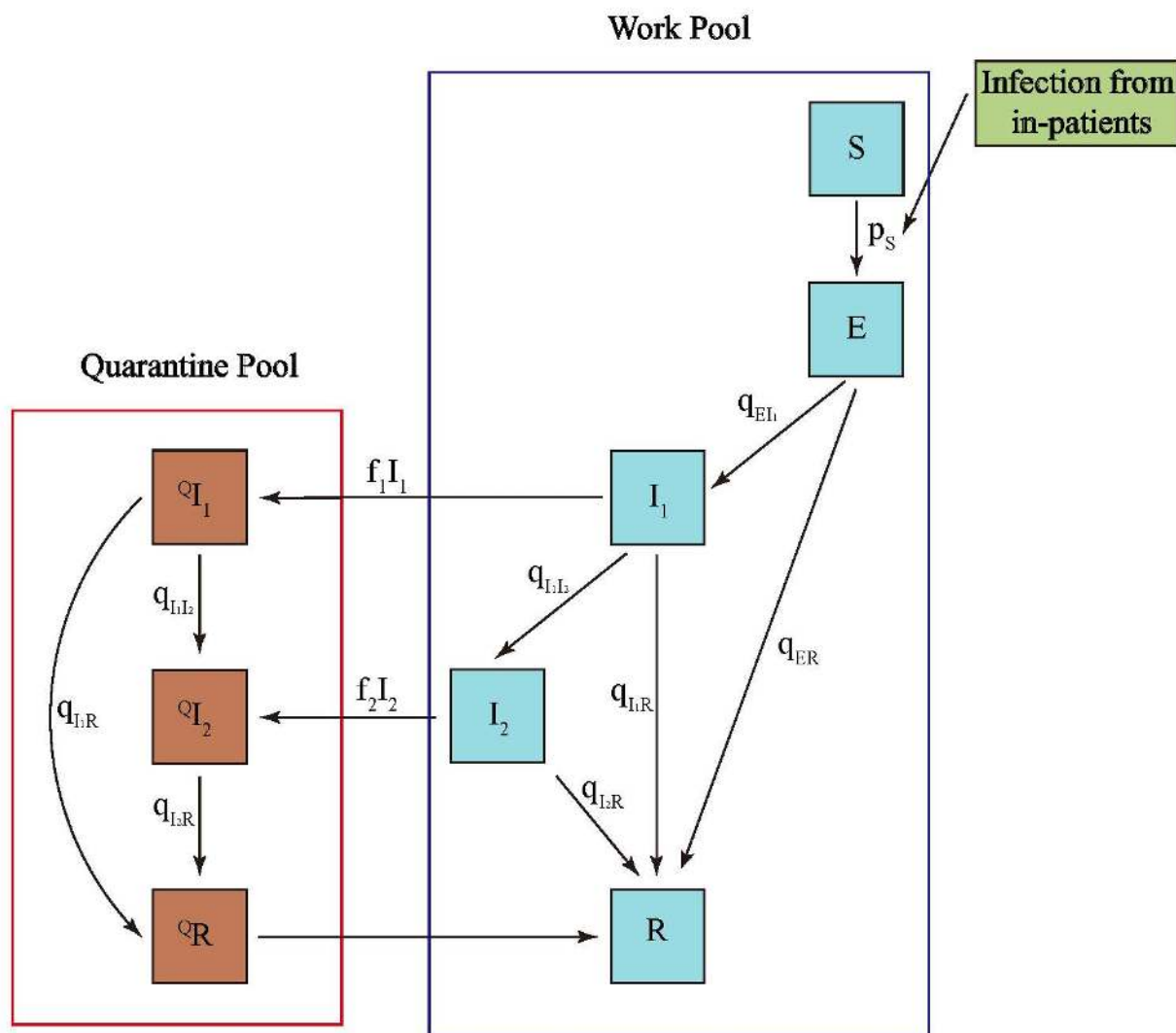
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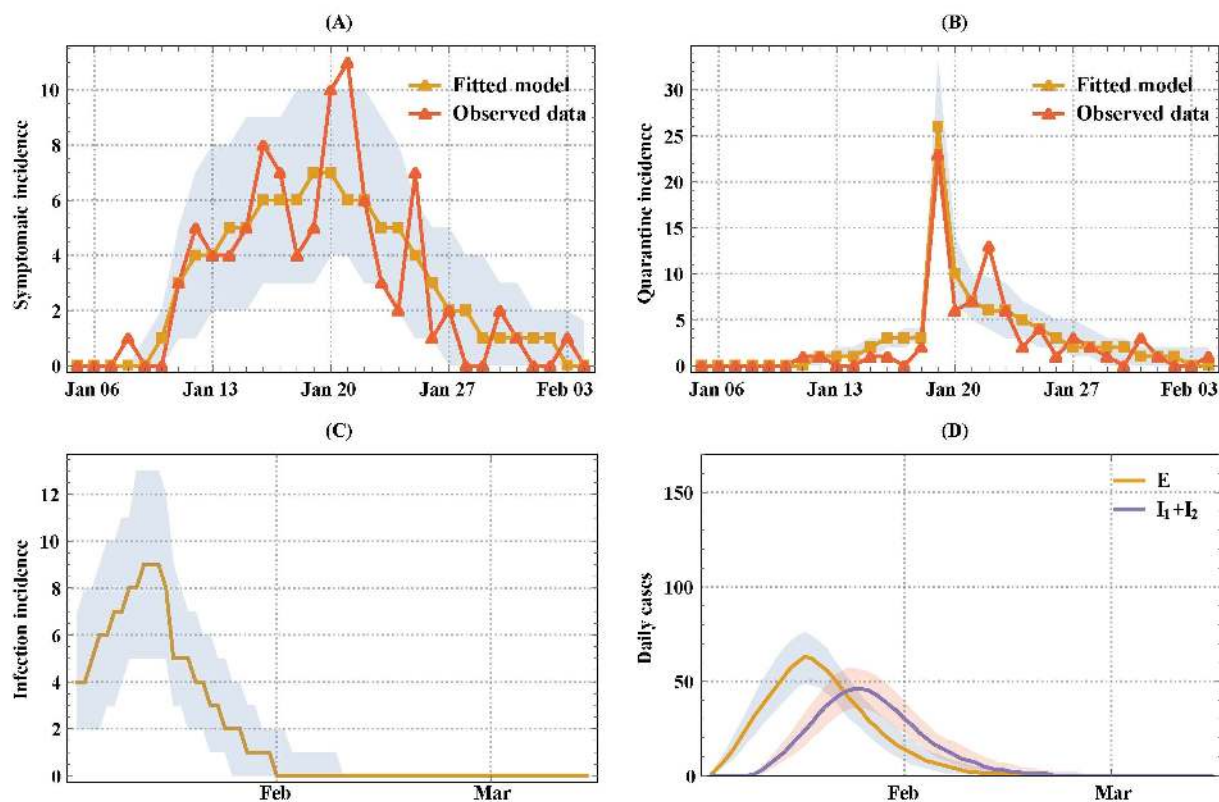
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- 468

469 **Figure**



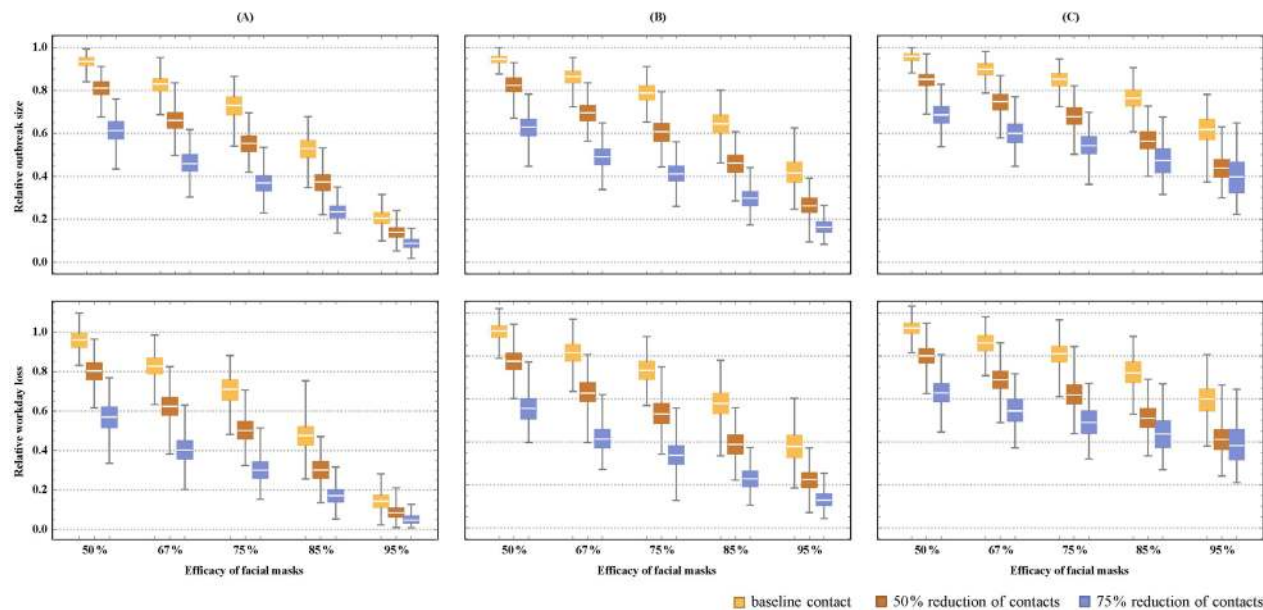
470
 471 **Figure 1: State transitions in the individual-based model.** The standard SEIR scheme is used to
 472 describe host states: S - susceptible, E - latent (pre-symptomatic/ asymptomatic but infectious), I_1 -
 473 first symptomatic (upper respiratory infection), I_2 - second stage (advanced lung infection), R -
 474 recovered/immune. Hosts can undergo three different pathways: asymptomatic ($E \rightarrow R$); mild
 475 symptomatic ($E \rightarrow I_1 \rightarrow R$); severe symptomatic ($E \rightarrow I_1 \rightarrow I_2 \rightarrow R$). Depending on the screening
 476 procedure, fractions of (I_1, I_2) are sent to quarantine, and released to the workpool upon recovery.

477 Infected patients are treated as an external source



478

479 **Figure 2: Model calibration and prediction.** Panel A shows the observed and fitted daily incidence
480 of symptomatic cases ($E \rightarrow I_1$). Panel B shows the observed and fitted daily quarantine cases among
481 HCWs. Panel C shows the predicted infection incidence ($S \rightarrow E$) from the calibrated model. Panel D
482 shows the corresponding predicted daily pre-symptomatic/ asymptomatic cases, E , and symptomatic
483 cases, $I_1 + I_2$, respectively. The grey shaded regions are 90% credible intervals.

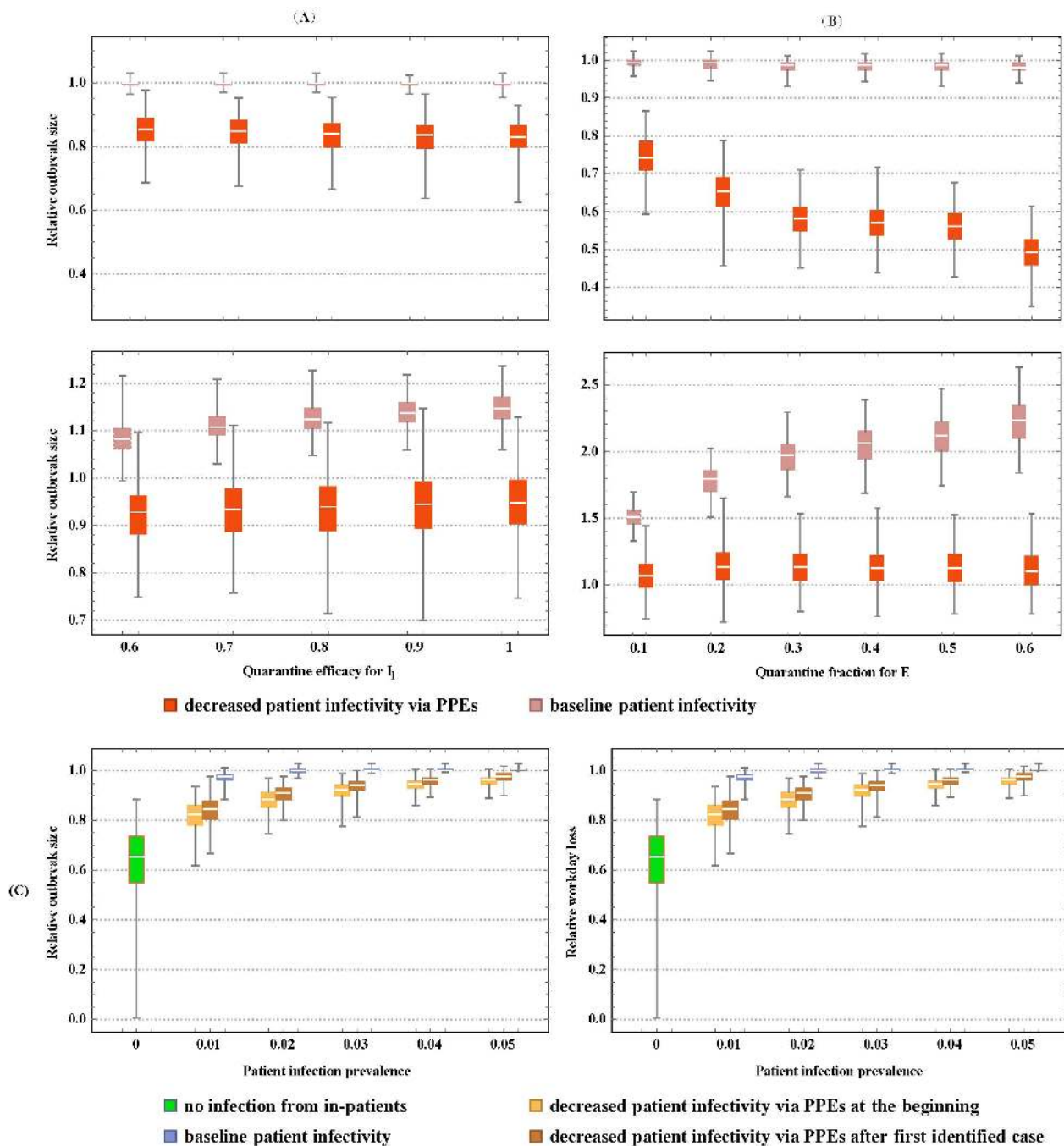


484

485 **Figure 3. The combined effect of facial masks and social distancing (reduced contact rate).**

486 Three levels of social distancing were considered (normal, reduced by 50%, reduced by 75%). For
487 face-mask efficacy, we considered five putative values (50%, 67%, 75%, 85%, 95%). The efficacy is
488 measured via reduced host susceptibility per contact ($a \rightarrow 0.5 * a$; $a \rightarrow 0.33 * a...$). We also
489 considered different timing of preventive measures: (1) start of the outbreak (Panel A); (2) after the
490 first identified HCW case (Panel B); (3) after 10% of HCW-staff got infected (Panel C). In each
491 case, we estimated the posterior distribution of the relative outbreak size, and the workday loss over
492 baseline values.

493



494

495 **Figure 4. The effect of the quarantine and patient sources on relative outbreak size and**
 496 **workday loss.** The patient sources (infection prevalence and infectivity) were controlled via
 497 screening/isolation and the use of PPE by HCWs. We considered two quarantine strategies for
 498 HCWs: symptomatic cases only (column A), adding pre-symptomatic/ asymptomatic cases (column

499 B). We used the following marking: (pink) baseline patient infection level, (red) reduced patient
500 infection by 80% via PPE use by HCWs. In column A, the quarantine fraction of moderate/severe
501 cases (I_2) was fixed at 100%, and the quarantine fraction of mild cases (I_1) was varied from 60% to
502 100%. In column B, we fixed symptomatic ($I_1; I_2$) quarantine fractions at (90%, 100%), and varied
503 the quarantine fraction of pre-symptomatic/ asymptomatic E -pool from 10% to 60%.

504 Panel C shows the effect of patient infection and different timing of PPE use: i) start of the outbreak,
505 ii) after the first identified HCW infection, iii) no PPE use. We considered different levels of
506 prevalence of the infected patient pool: 0%, 1%, 2% (baseline value), 3%, 4%, 5%.

Tables

Table 1: Effects of implementing social distancing through reduction of contacts alone and wearing face masks alone, from the start of the outbreak. We simulated a six-month intervention-regimen for the calibrated model. The progress was measured in terms of outbreak size, workday loss, and cumulative quarantine incidence. Reasonable (50% and 75%) reduction of contact rates and levels of efficacy of facial masks (50%, 67%, 75%, 85%, 95%) were chosen. The results shown are predicted median (90% Credible Interval).

Progress Measure	Intervention							
	Baseline Results (no intervention)	Reduction of contact rates		Efficacy of facial masks				
		50%	75%	50%	67%	75%	85%	95%
Outbreak size	170	164	146	159	141	124	90	35

	(168-171)	(159-169)	(135-156)	(150-165)	(128-151)	(108-137)	(75-104)	(24-46)
Workday loss	1050 (913-1282)	992 (855-1202)	853 (721-1044)	950 (817-1145)	819 (678-997)	698 (562-876)	469 (342-605)	142 (77-212)
Cumulative quarantine incidence	112 (98-136)	107 (93-130)	93 (80-113)	103 (89-123)	90 (76-108)	78 (63-96)	53 (40-68)	16 (9-24)

Supplementary information

Setup and parameters of the individual-base model

The individual-based model we developed features a heterogeneous host population with distinct infective stages and transitions, different patterns of disease-progression (asymptomatic, mild, severe), individual risk factors (health status, work stress, et al), individual behavior including social mixing patterns among HCWs, use of PPE/ facial masks, and HCW-patient interactions.

The key inputs in IBM are described below:

- 1) Host community consists of three disease progress groups, asymptomatic, mild, severe (A-M-S), i.e., population fractions (v_A, v_M, v_S) that undergo three pathways of Figure 1: (A) asymptomatic ($E \rightarrow R$); (M) mild symptomatic ($E \rightarrow I_1 \rightarrow R$); (S) severe symptomatic ($E \rightarrow I_1 \rightarrow I_2 \rightarrow R$). Based on the WHO report [1], we assumed that for symptomatic cases, 80% are mild symptomatic, i.e., $v_M = 0.8 * (1 - v_A)$, $v_S = 0.2 * (1 - v_A)$, Fraction v_A was estimated at 0.3080 (90% Credible Interval (CrI): 0.1628 – 0.3948) using the Bayesian method, consequently, we get (v_M, v_S) equal to (0.5536; 0.1384);
- 2) Initial infection state. We assume that only one HCW was in the latent E stage at the beginning of the simulation, while the rest 170 HCWs were susceptible;
- 3) Stage-specific infectivity levels (b_0, b_1, b_2) for (E, I_1, I_2). We assume $b_1 = b_2$, for symptomatic groups (I_1, I_2), while asymptomatic infectivity $b_0 = 0.55 * b_1$ [2]. Then we calibrated b_1 to be 0.2245 (90% CrI: 0.202 – 0.262) using the Bayesian method, consequently, $b_0 = 0.124$ (90% CrI: 0.111 – 0.144);
- 4) Individual susceptibility level (a). In general, a may depend on host health /immune status, individual behavior e.g. use of facial masks or PPE, and environmental conditions, $a = 0$ (fully protected), $a = 1$ (fully susceptible). Work stress is another factor that can affect susceptibility. According to [3] around 40% HCWs claimed stress during the outbreak. Hence, we allowed 60% HCWs to have a baseline susceptibility, $a_N = 0.5$, while high-risk (stressed) pool, 40% of HCWs, with susceptibility level $0.5 < a_S < 1$ (to be calibrated by the Bayesian method).
- 5) The average duration of infective stages for each pathway. According to the published studies [1, 4-8], we fixed the average duration L_{ER} of asymptomatic path ($E \rightarrow R$) to be 10 days; the average durations (L_{EI_1}, L_{I_1R}) of mild symptomatic path ($E \rightarrow I_1 \rightarrow R$) to be (5;9) days; and the average durations ($L_{EI_1}, L_{I_1I_2}, L_{I_2R}$) of severe symptomatic path ($E \rightarrow I_1 \rightarrow I_2 \rightarrow R$) to be (5; 3; 14) days.

6) Social mixing patterns between HCW, and external patient sources; Intra-HCW contacts were simulated as daily aggregation in small groups of 2,3, or 4 hosts, randomly drawn from the current working HCW pool. The basic case (full HCW working pool) assumes 60 pair-contacts, 30 triple-contacts, 8 quadruple-contacts per day. It approximately gives a 2.2 contact rate per HCW-host per day from hospital data (Figure S2) [3]. Larger host aggregations are possible, but we ignore them here. Besides internal mixing, each HCW-host visited patients at a prescribed rate = 20/per day. Unlike HCW staff, the patients were not individualized, but a random patient cluster (determined by mean visitation number per day) was drawn from the total patient pool (200) with a prescribed infected fraction (0.02), using a hypergeometric distribution. According to hospital data, no new patients were admitted after January 19th, and their pool was discharged after January 19th, at a rate of 5% /day, and HCWs will wear PPEs in face of patients after Jan 19th, so we assume patient infectivity, b_{PPE} , is decreased to be 0.02, i.e., the efficacy of PPE is about decreasing risk of infection by 80%, when we calibrated our IBM.

7) Daily isolation of symptomatic cases and recovery. We assumed that HCWs expressing symptoms are tested, and prescribed fractions ($f_1; f_2$) of ($I_1; I_2$) put in isolation. The assumed quarantine fractions ($0 < f_i < 1$) combine limited test sensitivity, and overlapping 'COVID-like' symptoms, expressed by other (non-COVID) hosts. According to data, the Union Hospital in Wuhan has much more strict quarantine policy after January 19th, when they were aware of the seriousness of COVID-19, so we fixed f_1 to be 0.1 and 0.8 before and after January 19th, respectively, and fixed f_2 to be 0.15 and 0.85 before and after Jan 19th, respectively, when we calibrated our IBM.

Supplementary tables and figures

Table S1. IBM inputs and parameters.

Parameter	Descriptions	Value	Source
$(a_N; a_S)$	Susceptibility of a susceptible individual without stress and with stress	(0.5; 0.7608)	Calibrated
	Percentage of HCWs without and with stress	(0.6,0.4)	Data [3]
(b_0, b_1)	Infectivity of an asymptomatic and symptomatic infectious individual, respectively	(0.1235, 0.2245)	Calibrated [2]
(b_0, b_{PPE})	Infectivity of patient before January 19th and after January 19th (because of PPE), respectively	(0.1235, 0.02)	Estimated based on hospital policy
(n_2, n_3, n_4)	Number of random pair, triple, quadruple contacts per day among HCWs	(60;30;8)	Typical contacts/day
L_{ER}	The average duration of asymptomatic ($E \rightarrow R$)	10	[1, 4-8]
(L_{EI_1}, L_{I_1R})	The average duration of mild symptomatic ($E \rightarrow I_1 \rightarrow R$)	(5;9)	[1, 4-8]
$(L_{EI_1}, L_{I_1I_2}, L_{I_2R})$	The average duration of moderate/severe symptomatic ($E \rightarrow I_1 \rightarrow I_2 \rightarrow R$)	(5;3;14)	[1, 4-8]
q	Transition rate of disease progression based on average duration and days	$\frac{(d/L)^m}{1 + (d/L)^m}$	

f_1	Screening/ diagnostic fraction of mild symptomatic infectious population before and after January 19 th , 2020, respectively;	(0.1;0.8)	Estimated based on hospital policy.
f_2	Screening/ diagnostic fraction of severe symptomatic infectious population before and after January 19 th , 2020.	(0.15;0.85)	Estimated based on hospital policy.
(v_A, v_M, v_S)	Fraction of populations goes through three paths of disease progression (1) asymptomatic ($E \rightarrow R$); (2) mild symptomatic ($E \rightarrow I_1 \rightarrow R$); (3) moderate/severe symptomatic ($E \rightarrow I_1 \rightarrow I_2 \rightarrow R$)	(0.3080; 0.5536; 0.1384)	Calibrated [1]
External source from patients			
	average number of visits to patients per HCW per day; the number of infectious patients, the total number of patients	(20;4;200)	Data
Initial symbols	Description	Values	Source
(S_0, E_0)	initial susceptible population; initial exposed population	(170;1)	Data

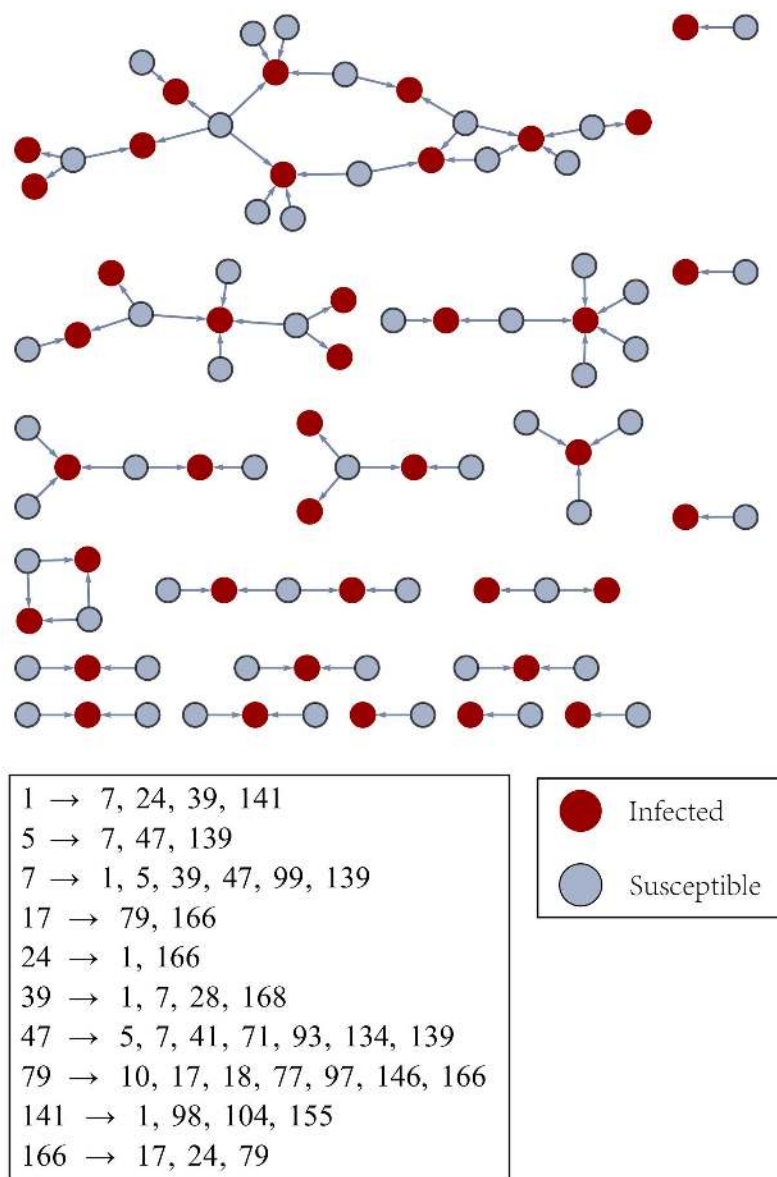


Figure S1. A typical daily snapshot of transmission pathways resulting from random mixing. Table at the bottom shows selected contact pools, and the graph arrows indicate which susceptible hosts (grey) were potentially in contact with infective ($E - I$) hosts (red). In our scheme, a single 'red' can infect multiple susceptibles, and a susceptible (grey) can be linked to multiple infectives.

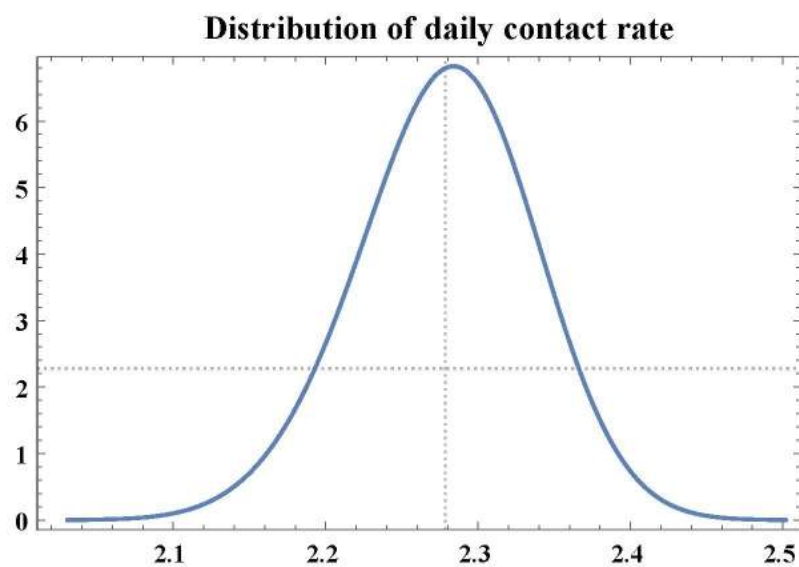


Figure S2. Distribution of baseline contact. The basic case (full HCW working pool) assumes 60 pair-contacts, 30 triple-contacts, 8 quadruple-contacts per day. It approximately gives a 2.2 contact rate per HCW-host per day.

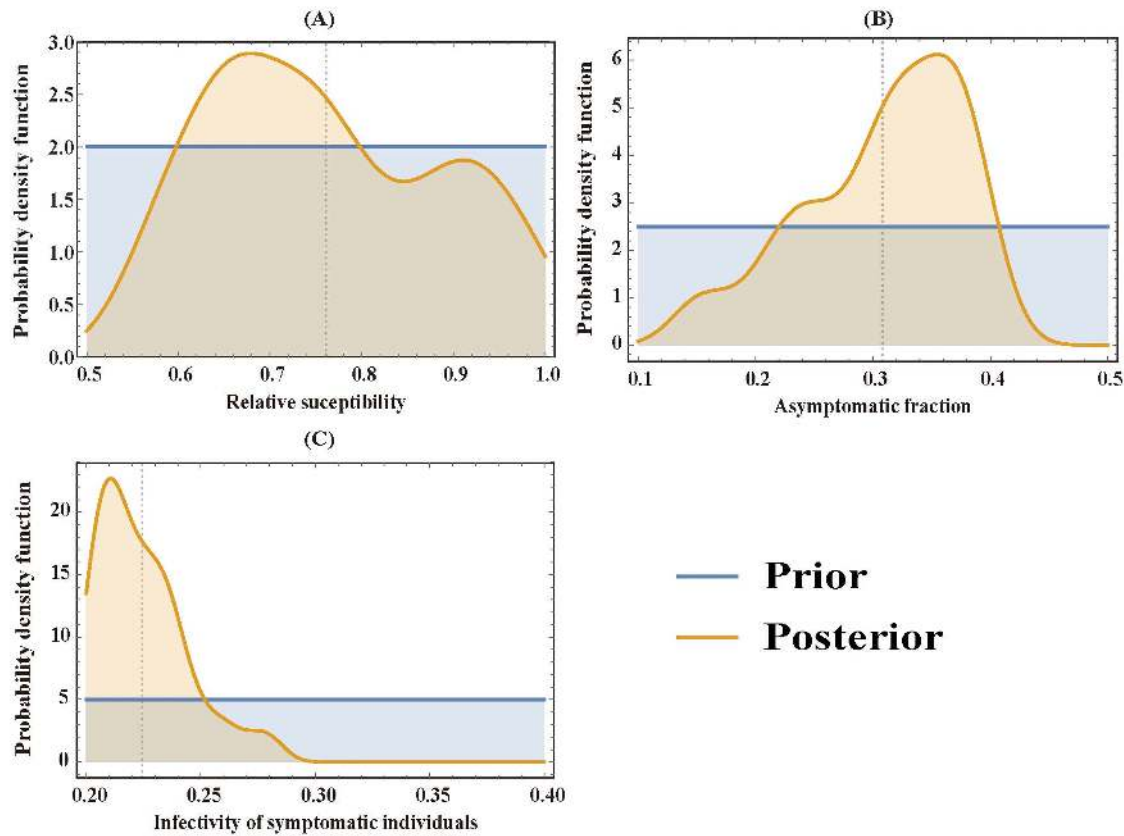


Figure S3. The prior and posterior distribution for key parameters. Panel (A): relative susceptibility, a_S , of the stressed HCWs when compared to a baseline susceptibility of $a_N = 0.5$; The prior distribution of a was uniform in $(0.5, 1)$ and the posterior was estimated to be 0.7608 (90% CrI: 0.5822 – 0.9653). Panel (B): asymptomatic fraction, v_A , i.e., the fraction of the HCWs which goes through the asymptomatic pathway ($E \rightarrow R$). The prior distribution of v_A was uniform in $(0.1, 0.5)$ and the posterior was estimated to be 0.3080 (90% CrI: 0.1628 – 0.3948). Panel (C): infectivity of a symptomatic individual, b_1 . The prior distribution of b_1 was uniform in $(0.2, 0.4)$ and the posterior was estimated to be 0.2245 (90% CrI: 0.202 – 0.262).

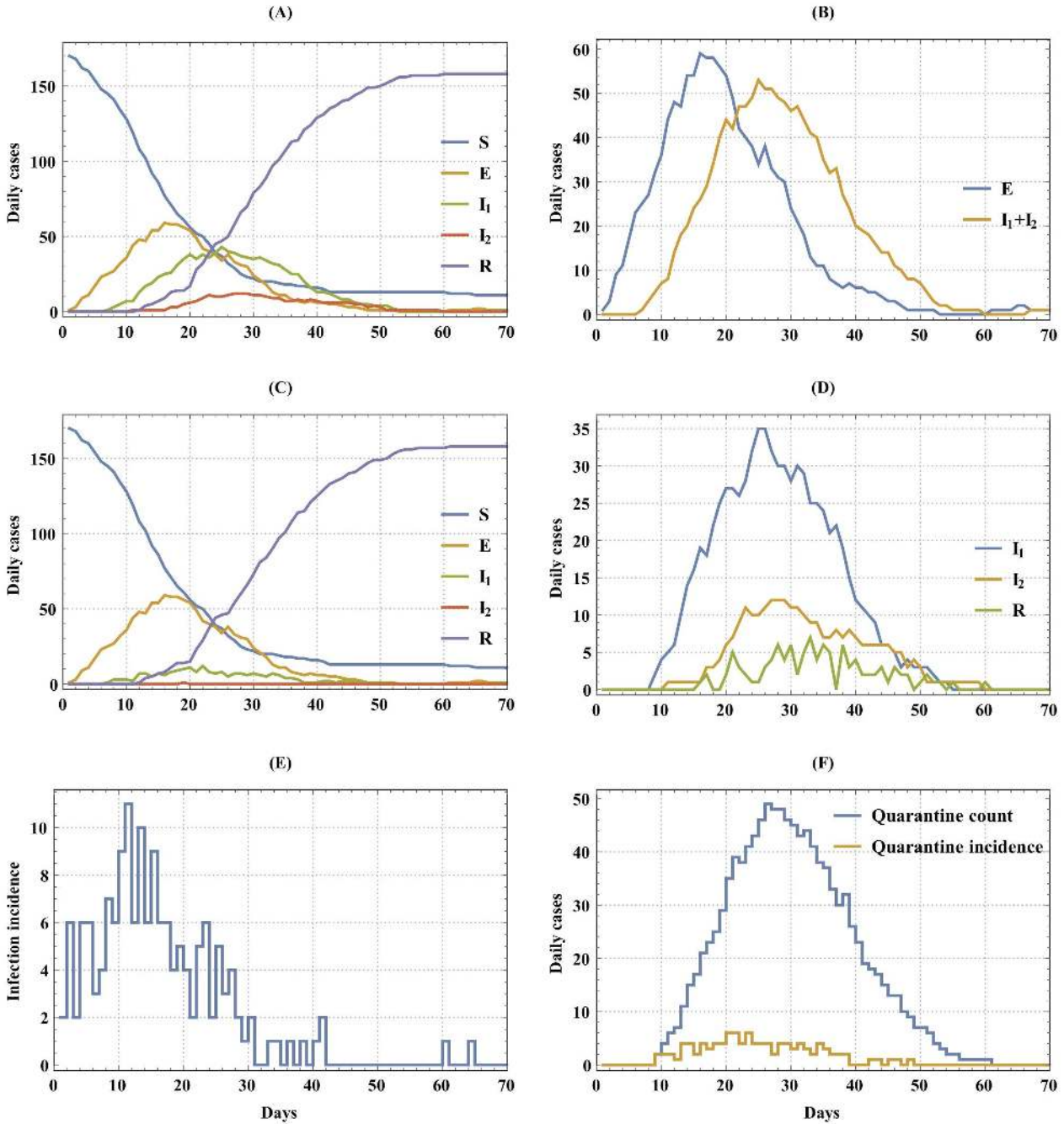


Figure S4. Typical outbreak history over a 70-day period for a baseline case. Panel (A) shows the outbreak history of the combined pool (work pool+quarantine pool); Panel (B) shows the simulation of asymptomatic, E , and symptomatic cases, $I_1 + I_2$, in the combined pool. Panels (C) and (D) show the history of the work pool and

quarantine pool, respectively. Panel (E) shows daily infection incidence ($S \rightarrow E$). Panel (F) shows daily quarantine incidence (yellow) and quarantine count (blue), respectively.

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