

1 **Modeling COVID-19 epidemics in an Excel spreadsheet: Democratizing** 2 **the access to first-hand accurate predictions of epidemic outbreaks**

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13

14 **Abstract**

15 COVID-19, the first pandemic of this decade and the second in less than 15 years, has
16 harshly taught us that viral diseases do not recognize boundaries; however, they truly do
17 discriminate between aggressive and mediocre containment responses.

18 We present a simple epidemiological model that is amenable to implementation in Excel
19 spreadsheets and sufficiently accurate to reproduce observed data on the evolution of the
20 COVID-19 pandemics in different regions (i.e., Italy, Spain, and New York City (NYC)).

21 We also show that the model can be adapted to closely follow the evolution of COVID-19
22 in any large city by simply adjusting two parameters related to (a) population density and
23 (b) aggressiveness of the response from a society/government to epidemics. Moreover, we
24 show that this simple epidemiological simulator can be used to assess the efficacy of the
25 response of a government/society to an outbreak.

26 The simplicity and accuracy of this model will greatly contribute to democratizing the
27 availability of knowledge in societies regarding the extent of an epidemic event and the
28 efficacy of a governmental response.

29

30 Keywords: *COVID-19, coronavirus, SARS-CoV2, mathematical modeling, epidemic,*
31 *pandemic, Excel*

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33

34 **Introduction**

35 A SARS-CoV2 (COVID-19) pandemic was declared by the World Health Organization in
36 March 2020. More than 100,000 positive cases of COVID-19 infection had been declared
37 worldwide at that point, mainly in China, Italy, Iran, Spain, and other European countries.
38 By the end of March 2020, the official cumulative number of infected worldwide ascended
39 to more than 700,000, with a toll of death higher than 32,000 and a strong presence in Las
40 Americas, mainly in the USA¹. COVID-19, the first pandemic of this decade and the
41 second in less than 15 years, has harshly taught us that viral diseases do not recognize
42 boundaries; however, they truly do discriminate between aggressive and mediocre
43 containment responses.

44 Indeed, three months have passed since the emergence of COVID-19, and we have been
45 able to observe exemplary responses from some Asian countries (i.e., China², South
46 Korea³, Singapore⁴, and Japan), some highly aggressive responses in Europe and America
47 (i.e., Germany and USA), and several delayed or not so effective responses from other
48 regions (i.e., Italy and Spain)⁵. At this point, some territories in Latin America are just

49 experiencing the “lag phase” of the COVID-19 pandemic at home and do not appear having
50 yet implemented proper containment measures as rapidly as needed.

51 The gap between developed and developing countries may explain some of the differences
52 in the scale of the responses that we are observing. Countries that are better equipped than
53 others in terms of high-end scientific development, diagnostics technology, and health care
54 infrastructure may respond more efficaciously to a pandemic scenario. However, other
55 tools, such as mathematical modeling, are much more widely available and may be of
56 extraordinary value when managing epidemic events such as the COVID-19 pandemics. To
57 date, many papers have reported the use of mathematical models and simulators to evaluate
58 the progression of COVID-19 in local or more global settings^{63,7-9}. Predictions on the
59 possible evolution of COVID-19 based on mathematical modeling could therefore represent
60 important tools for designing and/or evaluating countermeasures^{8,10-12}.

61 However, mathematical modeling may (and probably should) become a much more
62 available tool in the case of public health emergencies—one ideally widely available to
63 practically any citizen in any of our societies. One decade ago, during the influenza
64 pandemics, mathematical modeling of epidemic events was the realm of privileged
65 epidemiologists who had (a) a fast computer, (b) programming experience, and (c) access
66 to epidemiological data. Today, those three ingredients are now reduced to a convectional
67 laptop, very basic differential equation-solving skills, and access to a website with reliable
68 online statistical information on the epidemics.

69 The main purpose of this contribution is to demonstrate that a simple mathematical model,
70 amenable to implementation in an Excel spreadsheet, can accurately predict the evolution
71 of an epidemic event at a local level (i.e., in any major urban area). This may be extremely
72 valuable for government officials who must predict, with high fidelity, the progression of

73 an epidemic event to better design their action strategies. Moreover, the democratization of
74 the modeling of complex epidemic events will empower citizens, enabling them to forecast,
75 decide, and evaluate. For instance, using this simple model, virtually any citizen could
76 assess, in real time, the efficacy of the actions of her/his society in the face of an outbreak.

77

78 **Rationale of the model formulation**

79 Here, we construct a very simple epidemiological model for the propagation of COVID-19
80 in urban areas. The model is based on a set of differential equations. The first equation of
81 the set (equation 1) states that the rate of accumulation of infected habitants in an urban
82 area (assumed to be a closed system) is given by the sum of the number of new infections
83 (positive contribution), the number of recovered patients (negative contribution), and the
84 number of deaths (negative contribution). A second differential equation states that the rate
85 of accumulation of the infected but asymptomatic population is proportional to the population
86 of infected and symptomatic subjects (equation 2). Two additional equations relate the
87 number of deaths and recovered patients with the number of newly infected ones (equation
88 3 and 4). Finally, the rate of depletion of the pool of the population susceptible to infection
89 is given by the sum of recovered patients, asymptomatic infected, and deaths (equation 5).
90 Recent experimental evidence suggests that rhesus macaques that recovered from SARS-
91 CoV-2 infection could not be reinfected¹³. However, at this point, the acquisition of full
92 immunity to reinfection has not been proved in humans, although it is well documented for
93 other coronavirus infections, such as SARS, and MERS^{14,15}. The analysis of sera of one
94 COVID-19 patient showed a peak production of specific IgGs against SARS-COV-2 by
95 two weeks after the onset of symptoms¹⁶. Based on immunological information on SARS

96 and MERS epidemiology and the limited evidence on the nature of the host immune
97 response to SARS-COV-2, we assume here that recovered patients become immune to
98 reinfection.

99

100 $dX_s/dt = R_{\text{Infected-s}} - R_{\text{Recovered}} - R_{\text{Death}}$ Equation (1)

101 $dX_{\text{as}}/dt = R_{\text{Infected-as}} = (2.5/1.0) * R_{\text{Infected-s}}$ Equation (2)

102 $dD/dt = R_{\text{Death}} = 0.023 * R_{\text{Infected-s}}$ Equation (3)

103 $dR/dt = R_{\text{Recovered}} = 0.977 * R_{\text{Infected-s}}$ Equation (4)

104 $dP_s/dt = -R_{\text{Infected-as}} - R_{\text{Recovered}} - R_{\text{Death}}$ Equation (5)

105

106 This system is equivalent to:

107 $dX_s/dt = R_{\text{Infected-s}} - R_{\text{Recovered}} - R_{\text{Death}}$ Equation (1)

108 where:

109 $R_{\text{Infected-as}} = (2.5/1.0) * R_{\text{Infected-s}}$ Equation (2')

110 $R_{\text{Death}} = 0.023 * R_{\text{Infected-s}}$ Equation (3')

111 $R_{\text{Recovered}} = 0.977 * R_{\text{Infected-s}}$ Equation (4')

112 $P_{s_n} = P_{s_{n-1}} - (X_{\text{as}} + R + D)$ Equation (5')

113

114 In this system, all equations depend on $R_{\text{Infected-s}}$. Here, we propose a simple formulation for
115 the evaluation of $R_{\text{Infected-s}}$ at the onset of a local epidemic event.

116

117 $R_{\text{Infected-s}} = dI_s/dt = \mu_o I_s$ Equation (6)

118

119 where μ_o is the specific rate of infection of a population in a large and vastly uninfected
120 urban area. We further propose that μ_o may be calculated from actual epidemiological data
121 corresponding to the first exponential stage of COVID-19 local epidemics. We determined
122 the appropriate ranges of values for μ_o by analyzing publicly available data from different
123 websites that continuously monitor the progression of confirmed cases of COVID-19 for
124 different nations (Table 1).

125

126 **Table 1.** Websites displaying COVID-19 data in practically real time.

- 127 • Our World in data:
128 <https://ourworldindata.org/coronavirus>
129 • El País
130 https://elpais.com/sociedad/2020/03/16/actualidad/1584360628_538486.html
131 • Coronavirus COVID-19 Global Cases by the Center for Systems Science and Engineering
132 (CSSE) at Johns Hopkins University (JHU).
133 <https://gisanddata.maps.arcgis.com/apps/opsdashboard/index.html#/bda7594740fd40299423467b48e9ecf6>
134 • Wikipedia, The Free Encyclopedia
135 https://en.wikipedia.org/wiki/2020_coronavirus_pandemic_in_Iran
136

137

138 This model correctly describes the evolution of the number of newly infected during the
139 initial stage of the epidemic episode. For later times, the rate of new infections is corrected
140 by a term that depends on the demographic density (D_d) of the region. Therefore:

141

142 $R_{\text{Infected-s}} = dI_s/dt = \mu_o I_s (D_d/D_{d_{\text{ref}}})$ Equation (7)

143

144 In equation (7), $D_d = P_s/A$, where A is the surface area of the region subject to analysis. In
145 this formulation, D_d is the total number of inhabitants of the region who are susceptible to
146 infection, while $D_{d_{\text{ref}}}$ is a value of demographic density in a densely populated urban area
147 that the model uses as a reference. In this work, the demographic density of the city of

148 Madrid is used as Dd_{ref} . Furthermore, since Dd is a function that considers only the
149 population susceptible to infection, a counter is needed to continuously update the number
150 of recovered patients, asymptomatic patients, and deaths. Therefore, at each time step
151 during the numerical integration, the susceptible population is updated by subtracting the
152 number of number of recovered patients, asymptomatic patients, and deaths. Note that in
153 our Excel spreadsheet, we use $Dd/Dd_{ref} = \text{density_Factor}$ (Supplemental Excel File 1).
154 Defining an expression for $R_{Infected-s}$ enables stepwise numerical integration, for example by
155 the Euler method. We have implemented this solution in a spreadsheet. To that aim,
156 differential equations (1) and (7) should be converted into their corresponding equations of
157 differences:

158

$$159 \quad \Delta X_s = \{R_{Infected-s} - R_{Recovered} - R_{Death}\} \Delta t \quad \text{Equation (8)}$$

$$160 \quad \Delta I_s = \{\mu_o I_s (Dd/Dd_{ref})\} \Delta t \quad \text{Equation (9)}$$

161

162 For all the simulation results presented here, we set $\Delta t = 1h = 1/24$ day. We have solved this
163 differential set, step by step, updating the values of $R_{Infected-s}$, $R_{Recovered}$, R_{Death} , and P_s ,
164 according to equations (2') to (5'). The ratio (Dd/Dd_{ref}) is also recalculated at each time
165 step using the updated value of P_s from equation (5').

166

167 **Selection of relevant epidemiological parameters for COVID-19**

168 The number of asymptomatic inhabitants was calculated under the assumption that only
169 ~30.0% of the infected population develops symptomatology (2.5 asymptomatic subjects
170 per 1.0 symptomatic subject). This assumption should be regarded as speculative, since

171 very limited information specific for the ration between symptomatic and asymptomatic
172 COVID-19 patients is available at this point.^{17,18} The percentage of asymptomatic
173 infections during pandemic Influenza A/H1N1/2009, based on epidemiology studies
174 founded in serological analysis in a vast range of geographical settings, has been estimated
175 has been between 65 and 85%¹⁹. These values are also consistent with the high number of
176 asymptomatic infected subjects estimated for other pandemic events. For instance, in the
177 context of pandemic influenza A/H1N1/2009, up to 20–40% of the population in urban
178 areas (i.e., Monterrey, México, and Pittsburgh, USA)^{20,21} exhibited specific antibodies
179 regardless of experiencing symptoms, while the fraction of confirmed symptomatic
180 infections was lower than less than 10%.

181 In addition, the average time of sickness was set at 14 days in our simulations, within the
182 range reported from 14 to 32 days²², with a median time to recovery of 21 days²³.
183 Therefore, the number of patients recovered (R) is calculated as a fraction of 0.977 of those
184 infected 14 days previously. Similarly, asymptomatic patients are only removed from the
185 pool of susceptible after full recovery. Note that, in the current version of our model,
186 asymptomatic patients are not considered part of the population capable of transmitting
187 COVID-19, despite recently reported evidence that suggests that asymptomatic subjects (or
188 minimally symptomatic patients) may exhibit similar viral loads²⁴ to those of symptomatic
189 patients and may be active transmitters of the disease^{2,25}. The number of deceased patients
190 was calculated as 0.023 of those infected 14 days before. This mortality percentage (case
191 fatality rate) lies within the range reported in recent literature for COVID-19^{9,26–28}. The
192 time lapse of 14 days between the onset of disease and death was statistically estimated by
193 Linton et al. in a recent report²⁹.

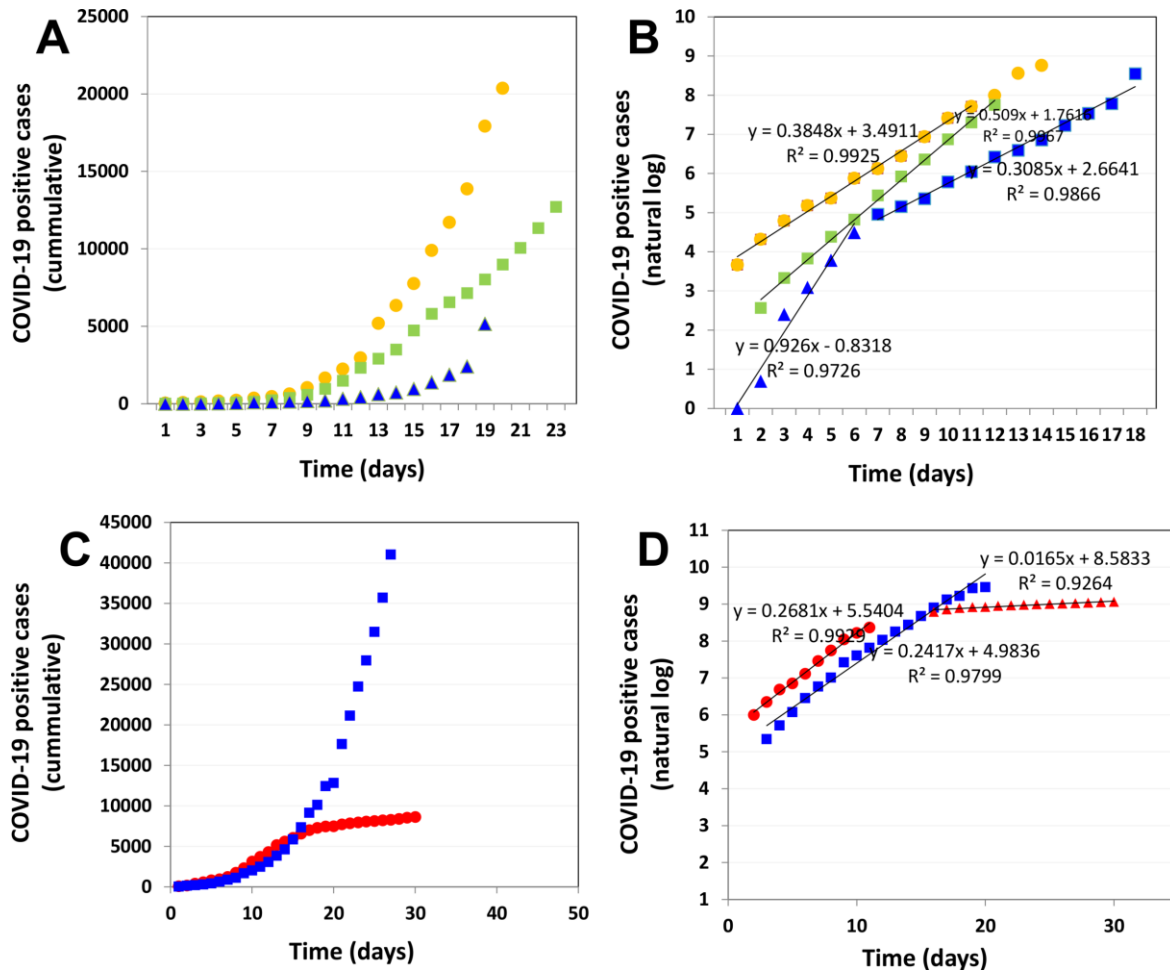
194 The straightforward implementation of the model in Excel (Supplemental Excel File 1),
195 using the set of parameters described before, allows the calculation of all populations (I_s , X_s ,
196 X_{as} , D , R , and P_s) every hour. Note that this model enables the description of the
197 progressive exhaustion of the epidemic, as expected by the progressive depletion of the
198 susceptible population. Next, we discuss criteria for selection of the values of μ_0 based on
199 the initial behavior of the COVID-19 Pandemic at different urban areas around the globe.

200

201 **Estimation of specific epidemic rate values**

202 Figure 1A shows the progression on the number of COVID-19 positive cases in different
203 regions, namely Spain (mainly Madrid), Iran (mainly Tehran), and New York City (NYC).
204 We have selected these three data sets to illustrate that the evolution of the epidemic has a
205 local flavor that mainly depends on the number of initial infected persons, the demographic
206 density, and the set of containment measures taken by government officials and society.
207 Figure 1B shows the natural log of the cumulative number of infections over time for the
208 same set of countries. This simple plotting strategy is highly useful for analyzing the local
209 rate of progression of the pandemic. If the local epidemic progression is consistent with a
210 simple first order exponential model where $dI/dt = \mu * I$, then the integral form of this
211 equation renders the linear equation: $\ln I/I_0 = \mu * t$. During the exponential phase, a straight
212 line should be observed, and the slope of that line denotes the specific rate (μ) of the
213 epidemic spreading. Note that COVID-19 has exhibited a wide range of spreading rates in
214 different countries (from ~ 0.3 to $\sim 0.9 \text{ day}^{-1}$). Note also that μ is related to the doubling time
215 (t_d), often reported in population and epidemiological studies, by the equation $t_d = \ln 2 / \mu$.

216 Therefore, ranges of doubling times between 0.75 and 2.45 days are observed just among
 217 these three regional cases.
 218



219
 220 **Figure 1. Epidemiological data related to the onset of a COVID-19 pandemic in different**
 221 **regions.** (A) Cumulative number of positive cases of COVID-19 infection in Spain (yellow
 222 circles), Iran (green squares), and NYC (blue triangles) during the first days after the outbreak. (B)
 223 Natural logarithm of the cumulative number of positive cases of COVID-19 infection in Spain
 224 (yellow circles), Iran (green squares), and NYC (blue triangles). (C) Cumulative number of positive
 225 cases of COVID-19 infection in Italy (blue squares) and South Korea (red circles). (D) Natural
 226 logarithm of the cumulative number of positive cases of COVID-19 infection in Italy (blue squares)
 227 and South Korea (red circles). Two clearly distinctive exponential stages are observed in the case
 228 of South Korean progression.

229
 230 Different exponential stages, perfectly distinguishable by their exhibition of different slopes
 231 (Table 2), may be observed within the same time series. For instance, the outbreak in NYC

232 (Figure 1B; blue symbols) was first described by an extremely high slope ($\mu_0 = 0.926 \text{ day}^{-1}$)
 233 ¹). However, after a series of measures adopted in NYC by the federal, state, and local
 234 governments, the specific growth rate of the epidemics fell to $\mu = 0.308 \text{ day}^{-1}$.
 235 The last point is extremely important, since two drastically different slopes can be observed
 236 before and after a package of adequate measures within the same territory. In addition, two
 237 localities that experienced similar initial specific epidemic rates may exhibit dramatically
 238 different evolutions as a function of the initial response of government and society (Figure
 239 1C,D). For instance, while the COVID-19 epidemics in Italy and South Korea exhibited
 240 practically equal μ_0 values, the Italian outbreak has maintained the same growth rate
 241 throughout 20 days, while South Korea has set an example by effectively and rapidly
 242 lowering the specific epidemic rate to nearly 0 in just two weeks.

243

244 **Table 2.** Specific infection rates (μ_0) and associated doubling times (t_d) for COVID-19
 245 infection in different geographic regions.

246

Territory	Temporality	μ	t_d
Spain (Madrid)	initial	0.358	1.937
Italy	initial	0.326	2.128
Italy	after stringent measures	0.119	5.849
Iran	initial	0.491	1.411
Iran (Tehran)	initial	0.506	1.370
Germany	initial	0.280	2.474
NYC	initial	0.591	1.173
NYC	after measures	0.293	2.362
South Korea	initial	0.293	2.362
South Korea	after stringent measures; massive testing	0.000	ND*
France	initial	0.379	1.828
France	after measures	0.161	4.311
Mexico	initial	0.209	3.324

247 (*) Not determinable

248

249 Validation and predictions

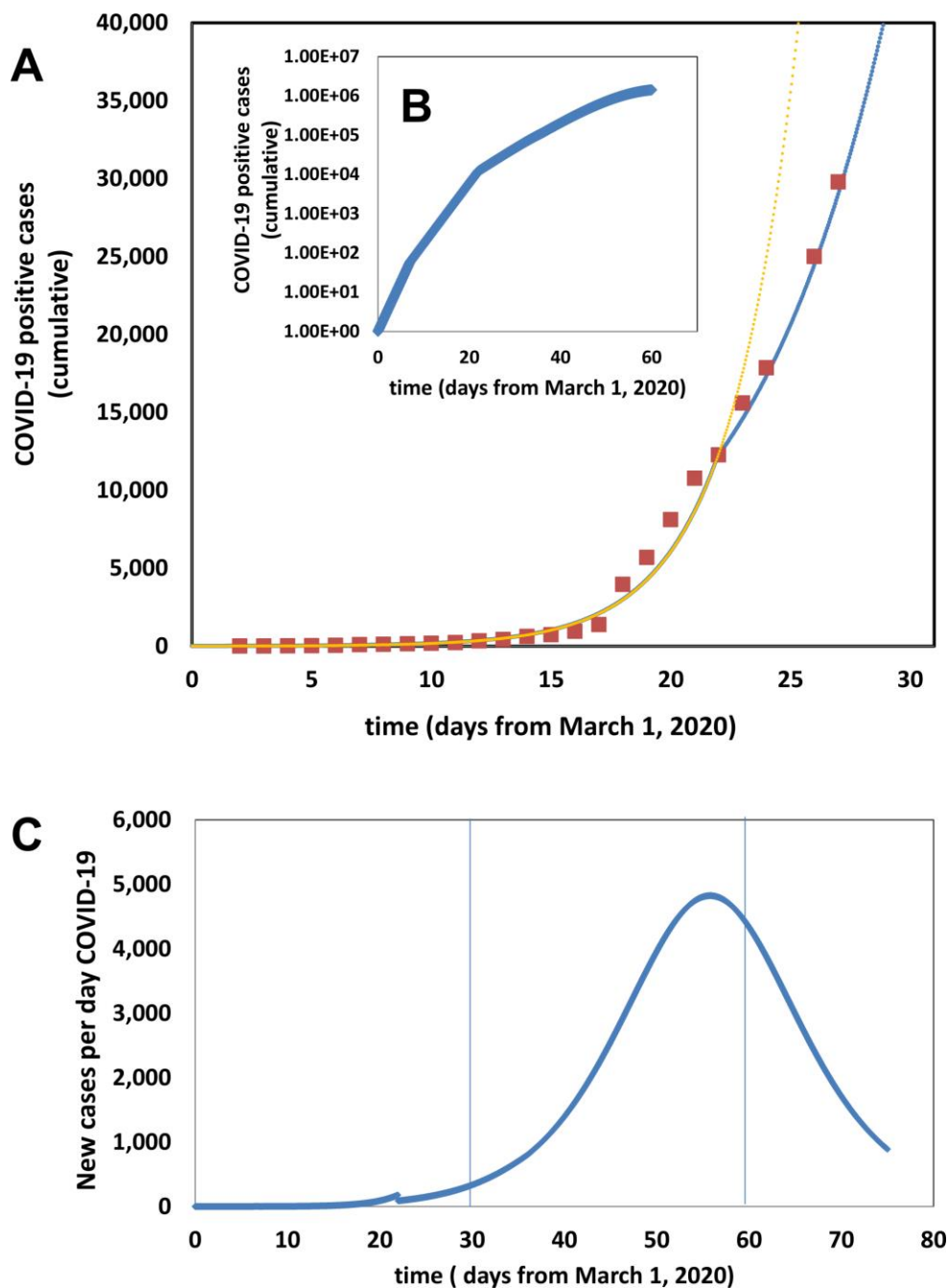
250 We have run different scenarios to validate the predictive capabilities of our epidemic
 251 model for COVID-19. Overall, the model is capable of closely reproducing the progression

252 of reported cases for urban areas of more than 5×10^6 inhabitants (i.e., Iran, the city of
253 Tehran in Iran, Spain, and NYC). We found that, adapting the model to a particular locality
254 is straightforward and only requires (a) the calculation of the population and the surface
255 area of the urban area, and (b) the selection of a t_d value (time to doubling the number of
256 infections). Note that our model is formulated in terms of values of the specific epidemic
257 growth rate (μ_o for the onset of the epidemic and μ for later times). However, expressing
258 the specific epidemic rate in terms of doubling time ($t_d = \ln 2 / \mu$) is more practical and
259 simpler to communicate and understand (Table 2).

260

261 The selection of μ_o (t_d) can be easily done by fitting the prediction to the initial set of
262 reported cases of infection. In our experience, four to five reliable data points are needed
263 for a good fit. For instance, Figure 2 shows the predicted trend of the pandemic in NYC
264 during the first 28 days of March, 2020. In addition, we set ($Dd/Dd_{ref} = 1.90$), since the
265 population density in NYC is 1.90-fold higher than that in Madrid. A value of $t_d = 2.25$ was
266 also set for the first week of this simulation. Later, at day 7 (March 7), we reset the value of
267 t_d to 3.75 to reflect the modification of the slope of the local epidemic event in NYC (Figure
268 1d), due to the implemented measures of containment. Based on this exercise, we foresee
269 that this simple modeling tool can be used to evaluate the efficacy of containment
270 strategies. In other words, the value of μ_o required in the simulation to adapt the predicted
271 data to the actual trend of the local epidemic provides an indicator of the local rate of
272 spreading of the pandemic.

273



274

275 **Figure 2. Progression of the COVID-19 Pandemic in NYC.** (A) Initial evolution of the number
276 of positive cases of COVID-19 in NYC. Actual data points, as officially reported, are shown using
277 red circles. Simulation predictions are described by the blue dotted line. (B) Model prediction of
278 the total number of symptomatic patients through the months of March and April. (C) Model
279 prediction of new cases of COVID-19 during the period from March 1 to May 20, 2020 if no
280 further containment actions are adopted.

281

282 Therefore, the differences between μ_0 before and after interventions provide a real-time
283 quantitative measure of the effectiveness of that set of measures. This can be extremely
284 useful when assessing the efficacy of control of epidemics. For example, for NYC, this
285 simple model states that the set of containment measures adopted during the first week of
286 March in NYC diminished the specific rate of the epidemic by increasing the doubling time
287 of infections from a value of 2.25 to 3.75 days.

288 The ability to make close predictions of the progression of cases in a particular region has
289 profound and enabling implications. For example, in March 15th, our simulations predicted
290 that, in absence of more aggressive containment measures (yellow trend in Figure 2A), the
291 peak of infections in NYC will be reached by April 10, 2010, after reaching the
292 unprecedented value of 11,000 new cases per day, and a cumulative number of 1×10^6
293 citizens infected. However, we observed a deviation from this prediction by the third week
294 of March that we attribute to the stringent measures of social distancing established in NYC
295 earlier that week. Accordingly, we multiplied the value of (Dd/Dd_{ref}) in our simulations by
296 a factor of 0.50 to properly fit the new trend on actual cases (blue trend in Figure 2A). Note
297 that this suggests that the measures of social distancing imposed in NYC were equivalent to
298 decrease the effective demographic density to 50%. At the end of March, after this
299 adjustment, our model forecasts a peak of infections of nearly 5,000 new cases per day (less
300 than half than the prediction before social distancing), and a cumulative number of 1×10^6
301 citizens infected.

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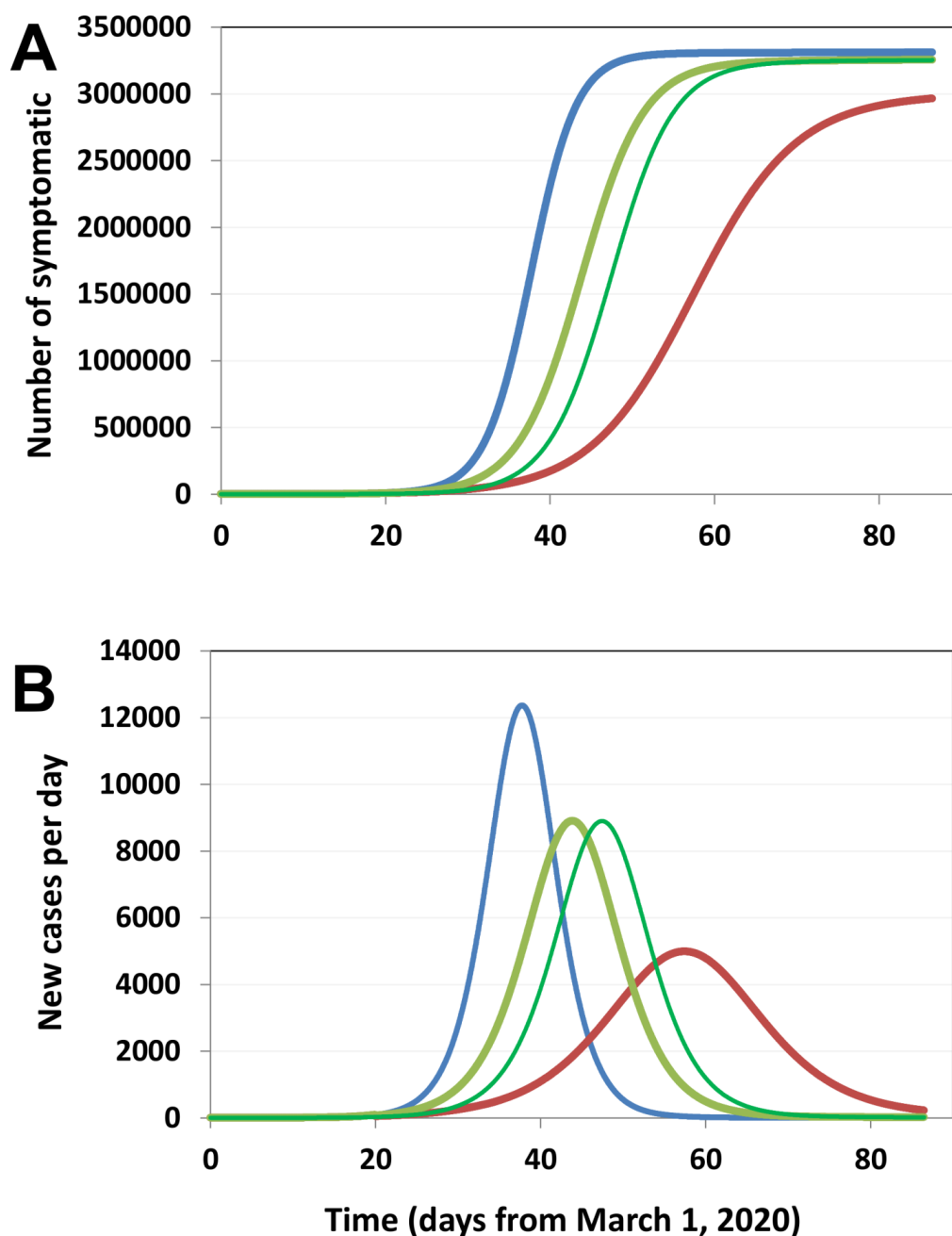
305 **Effect of social distancing**

306 Social distancing has been regarded as the one of the most effective buffering measures for
307 local COVID-19 epidemics^{30,31}. Next, we evaluate the effect of different degrees of social
308 distancing on the shape of the epidemic curve for NYC, one of the most densely urban
309 areas worldwide. This evaluation is straightforward, since the formulation of our model
310 explicitly considers the demographic density of the region as the most important modifier
311 of the rate of progression of the epidemics.

312 In the Excel implementation of the model, we multiply the demographic ration ($D_d/D_{d_{ref}}$)
313 by 0.75 to calculate the impact of distancing measures that would diminish social contact
314 by 25%. Similarly, we multiply ($D_d/D_{d_{ref}}$) by 0.50 to simulate the effect of a scenario of
315 social distancing that would diminish close social interaction by 50%. Figure 4 shows the
316 effect of three different degrees/levels of social distancing on the cumulative number of
317 infections (Figure 3A) and on the number of new cases of infection per day (Figure 3B).

318 Social distancing has a clear buffering effect on the epidemics, delaying the occurrence of
319 the peak of infections and distributing the number of cases across a longer time span. This
320 is remarkably important as it provides time for proper attention to patients with severe
321 symptomatology⁵.

322 For instance, our results suggest that, for an urban area such as NYC, imposing measures
323 that guarantee a social distance equivalent to a decrease in demographic density of 50%
324 will delay the peak of maximum number of infections by 25 days and will decrease its
325 intensity from 23000 to 9000 new cases of infection per day. In turn, this implies a lower
326 demand for hospital beds per day during the epidemics and may mark the difference
327 between a manageable crisis and a public health catastrophe^{5,30}.



328

329

330 **Figure 3. Prediction of the effect of social distancing on the progression of the COVID-19**
331 **pandemics in New York City (NYC).** (A) Model prediction of the total number of symptomatic
332 patients from March 1 to May 31, 2020 for different scenarios of social distancing: social
333 distancing as in March 20, 2010 (blue line, current prediction); social distancing effective on March
334 20, whereby the effective demographic density in NYC is reduced by 25% (light green line); social
335 distancing effective on March 20 whereby the effective demographic density in NYC is reduced by
336 50% (red line); and social distancing effective on March 10, whereby the effective demographic
337 density in NYC is reduced by 25% (dark green line). (B) Model prediction for the number of new
338 infections per day for each of the scenarios of social distancing described.

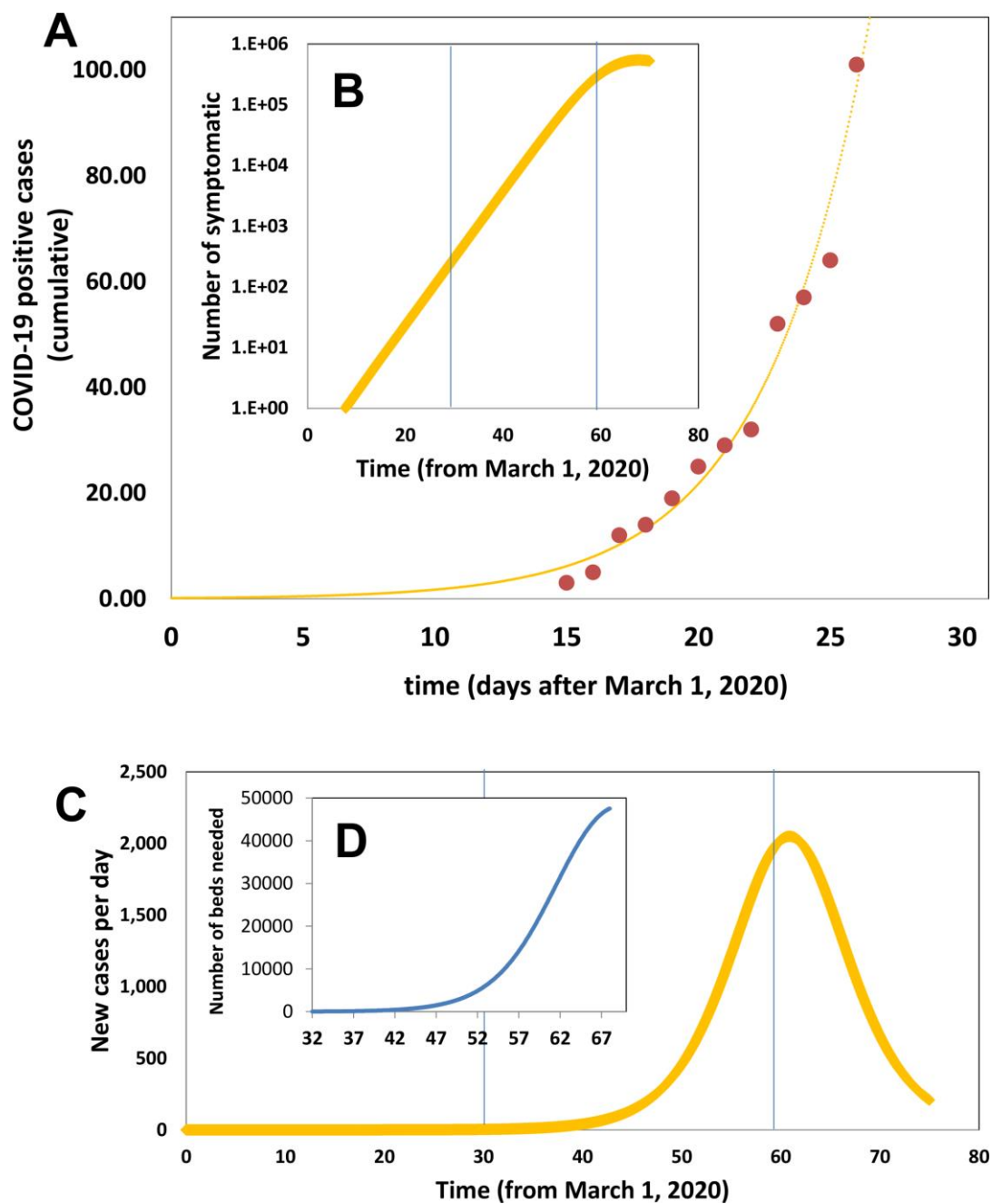
339 Interestingly, the effect of anticipating measures of social distancing has a moderate effect
340 on retarding the infection curve but not on decreasing the cumulative number of infections.
341 This moderate gain of time provides additional leeway for planning interventions or
342 allocating resources, with time being gold during pandemic events.

343

344 **Prediction in real time**

345 We are currently following the onset of the COVID-19 pandemic in Monterrey, the second
346 most industrialized city in México and the third most populated. Monterrey has a similar
347 demographic density to that of Madrid ($D_d/D_{d_{ref}}=0.95$). In addition, we set $t_d = 2.5$, based
348 on proper fitting to the first set of official values of COVID-19 infected announced for
349 Monterrey by the local authorities from March 15 to March 19, 2020. Remarkably, the
350 simulation results have accurately predicted the nine subsequent actual values, as officially
351 reported from March 19 to March 28 (Figure 4 A). Monitoring actual data, while
352 comparing with model predictions, enables real time assessment of the effectiveness of the
353 containment measures. In turn, this empowers officials, scientists, health care providers,
354 and citizens. Moreover, friendly and widely available mathematical modeling enables
355 rational planning. For instance, according to the pandemic scenario predicted for
356 Monterrey, in the absence of further containment measures and stricter social distancing,
357 the total number of symptomatic infected will surpass 650,000 persons (Figure 4B), and the
358 number of new infections per day (Figure 4C) will exhibit a peak of 2000 by the end of
359 April. The simulation may be used to forecast the demand of beds during the month of
360 April 2020, which is estimated to exhibit a peak of nearly 50000.

361



362
363

364 **Figure 4. Progression of the COVID-19 pandemic in the metropolitan area of**
365 **Monterrey, Nuevo León, Mexico.** (A) Initial evolution of the number of positive cases of
366 COVID-19 in the metropolitan area of Monterrey. Actual data points, as officially
367 reported, are shown using red circles. Simulation predictions are described by the blue
368 dotted line. (B) Model prediction of the total number of symptomatic patients from March
369 1 to May 20, 2020. (C) Model prediction of new cases of COVID-19 during the period
370 from March 1 to May 20, 2020 if no further containment actions are adopted. (D)
371 Estimation of the number of beds needed during the month of April 2020 in Monterrey,
372 based on the number of patients that will require hospitalization according to the model
373 predictions.

374 This estimate considers that only 10.0% of the symptomatic patients will require
375 hospitalization, which may be optimistic. Reports based in 44000 COVID-19 cases in
376 China indicate that the percentage of patients with severe symptoms may be 14%, with a
377 5% of critical cases³². In prospective, the total number of beds in the Mexican public health
378 sector is estimated in 20,000 (for the whole country).

379

380 **Concluding remarks**

381 We used a set of differential equations, recent epidemiological data regarding the evolution
382 of COVID-19 infection in a reduced set of regions (i.e., Spain, Iran, and NYC), and basic
383 information on the characteristics of COVID-19 infection (i.e., time from infection to
384 recovery, case mortality rate) to accurately recreate the onset of the COVID-19 in two
385 urban areas with different demographic characteristics (i.e., NYC and Monterrey, México).
386 We showed that the model can be adapted to closely follow the evolution of COVID-19 in
387 densely populated urban areas by simply adjusting two parameters related to (a) population
388 density and (b) aggressiveness of the response from a society/government to epidemics.
389 Scenarios such as those currently unfolding in Iran, Italy, or Spain emphasize the
390 importance of planning ahead during epidemic events. The availability of a simple model
391 may be highly enabling for local governments, physicians, civil organizations, and citizens
392 as they struggle in their endeavor to accurately forecast the progression of an epidemic and
393 formulate a plan of action. As previously stated, the use of simple/user-friendly models to
394 evaluate in (practically) real time the effectiveness of containment strategies or programs
395 may be a powerful tool for analyzing and facing epidemic events^{6,12}. This contribution

396 shows the prediction potential of an extremely simple simulation tool that can be used by
397 practically any citizen with basic training in Excel.

398

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402

403 **Author contributions**

404 MMA, EGG, and GTdS collected and analyzed epidemiology data. MMA formulated the
405 model and run the simulations. MMA and GTdS wrote the manuscript. All authors
406 reviewed and approved the manuscript.

407

408 **Competing interest**

409 The authors declare no competing interests.

410

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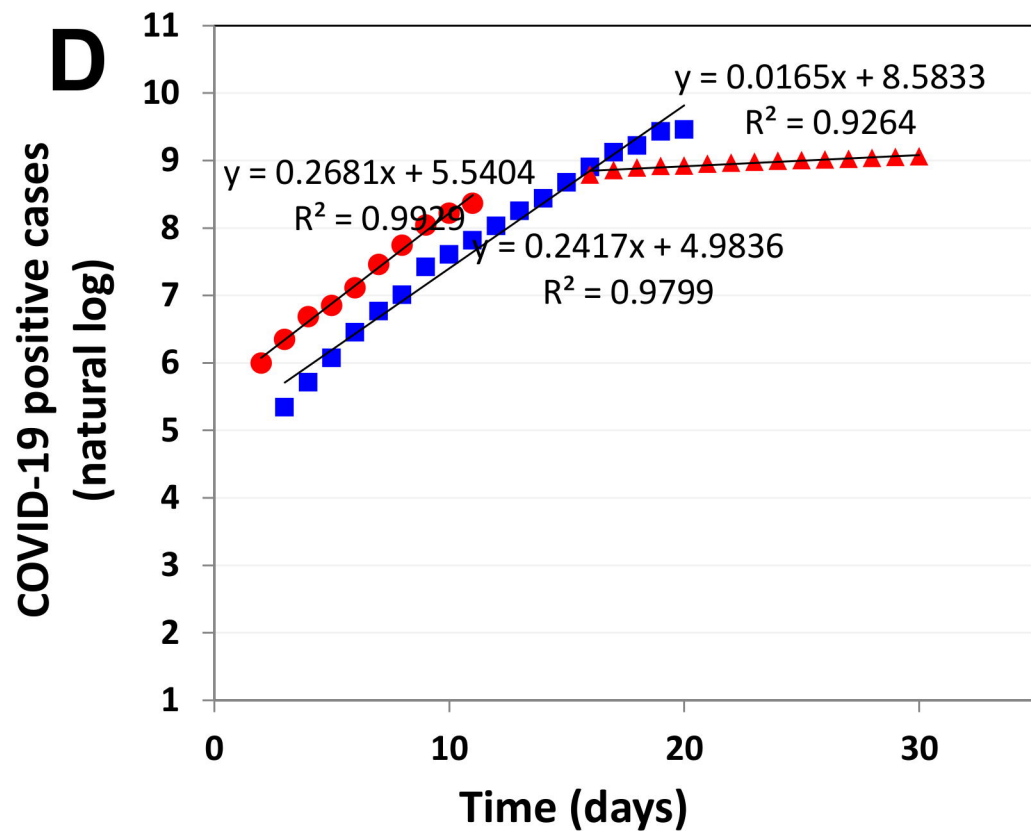
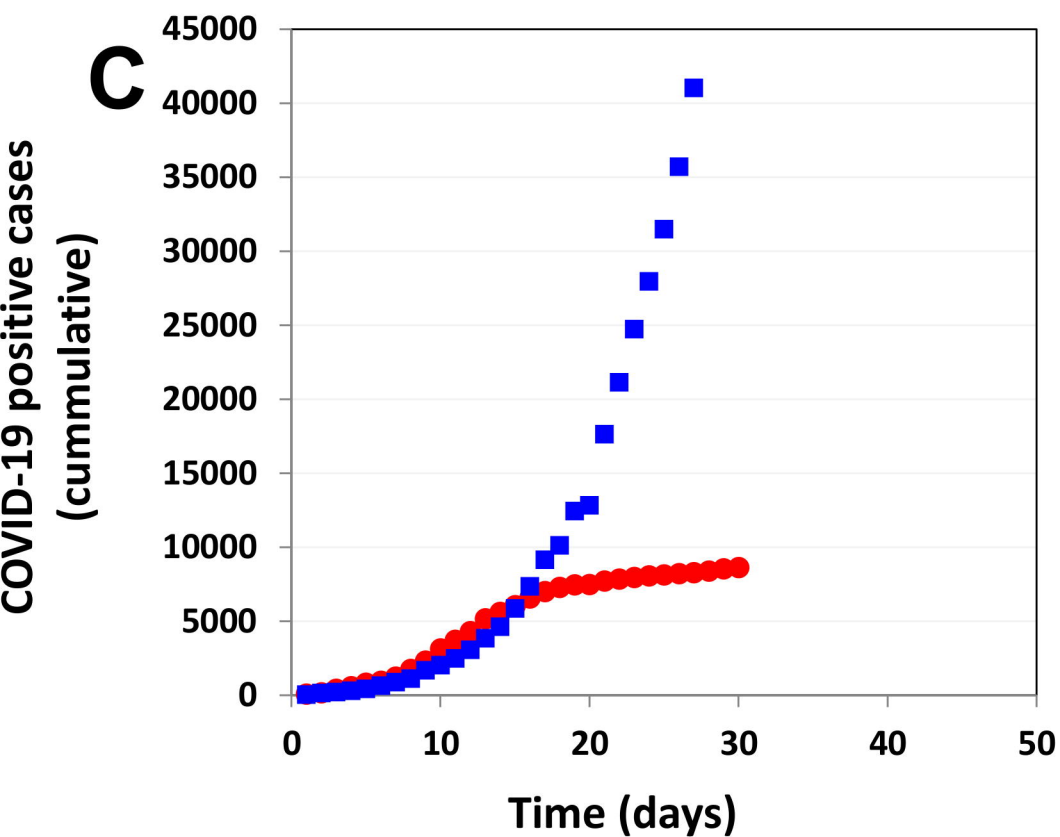
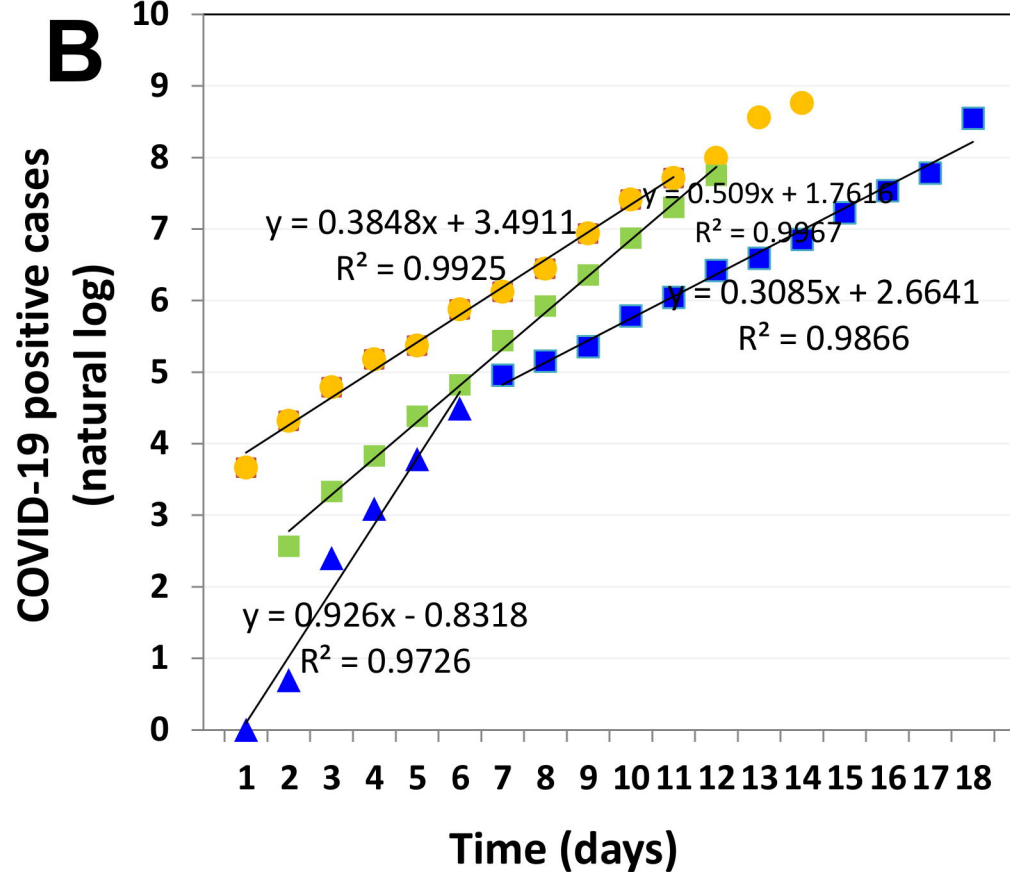
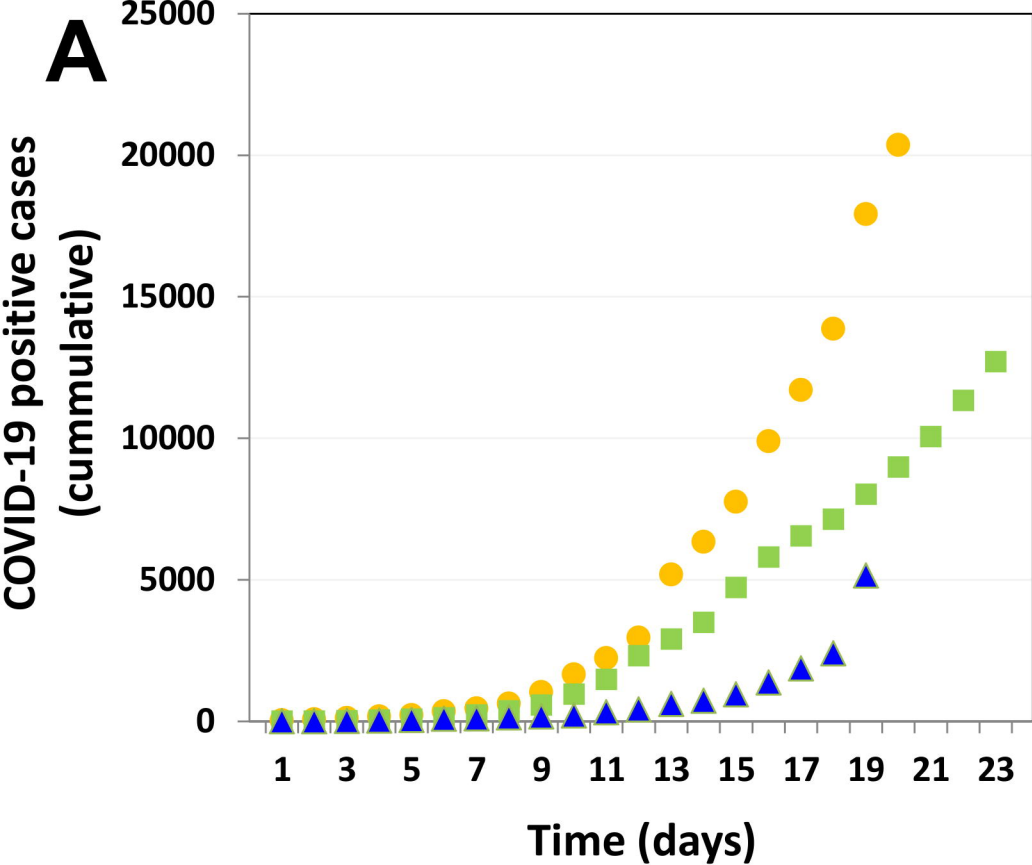
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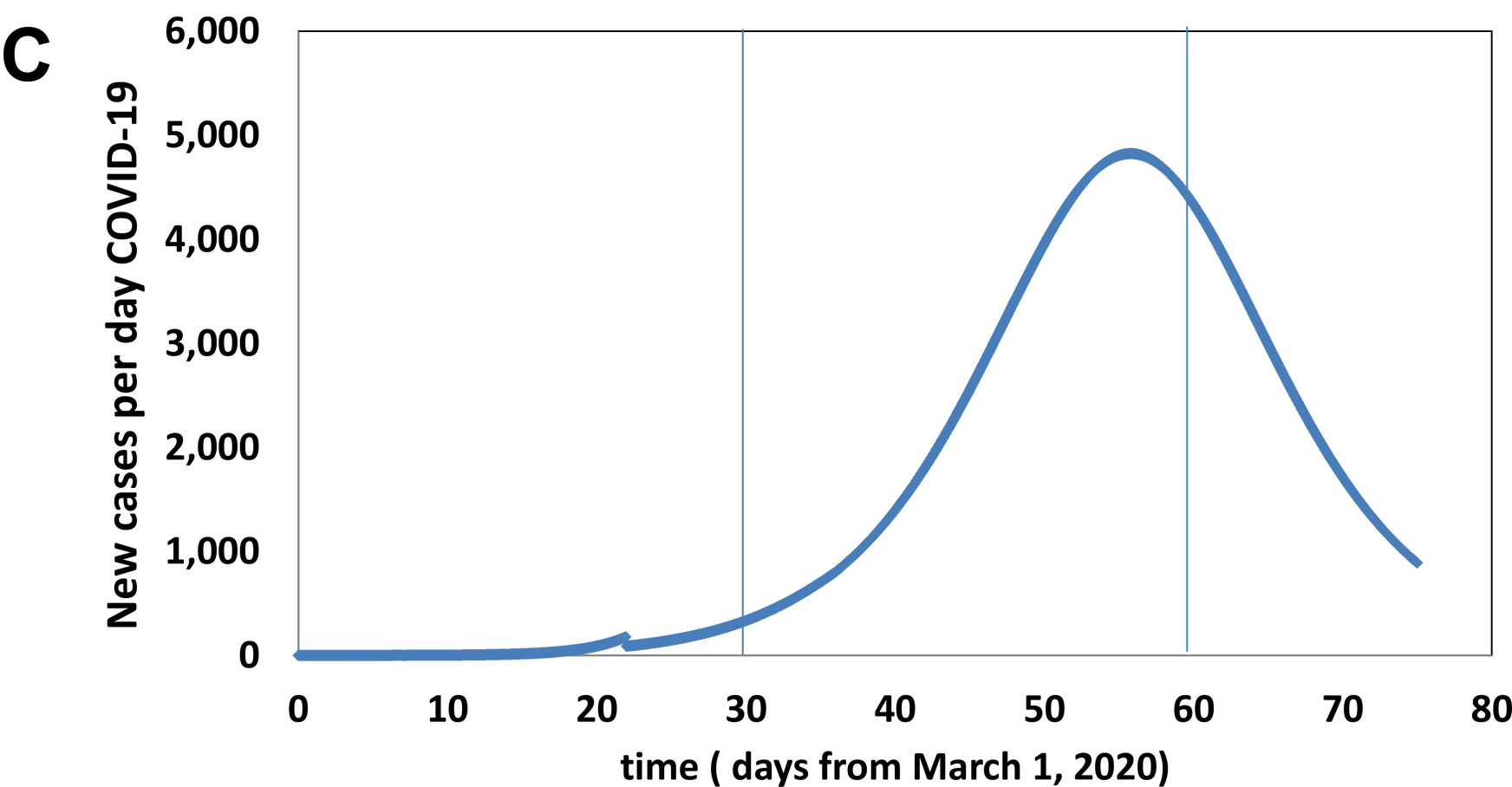
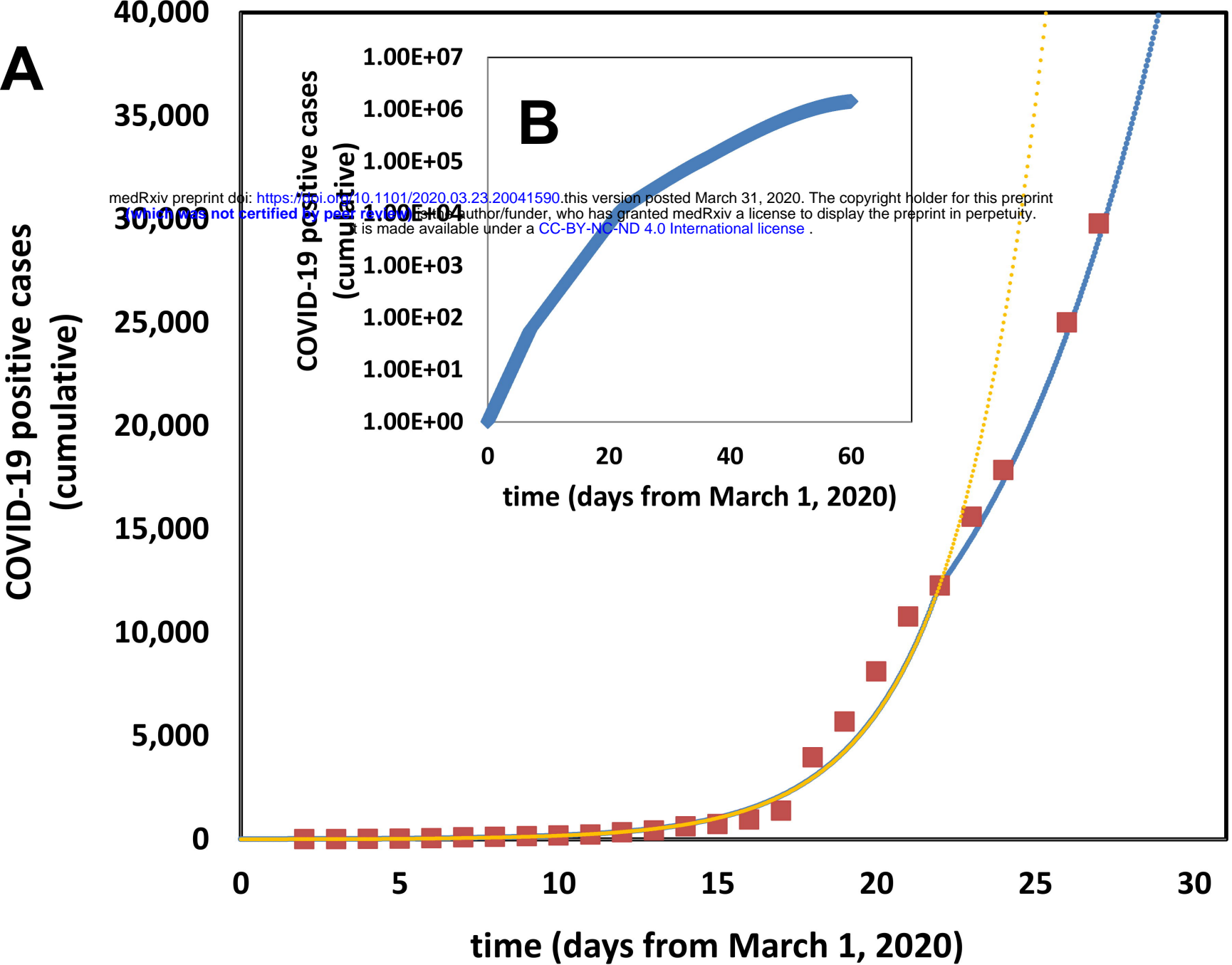
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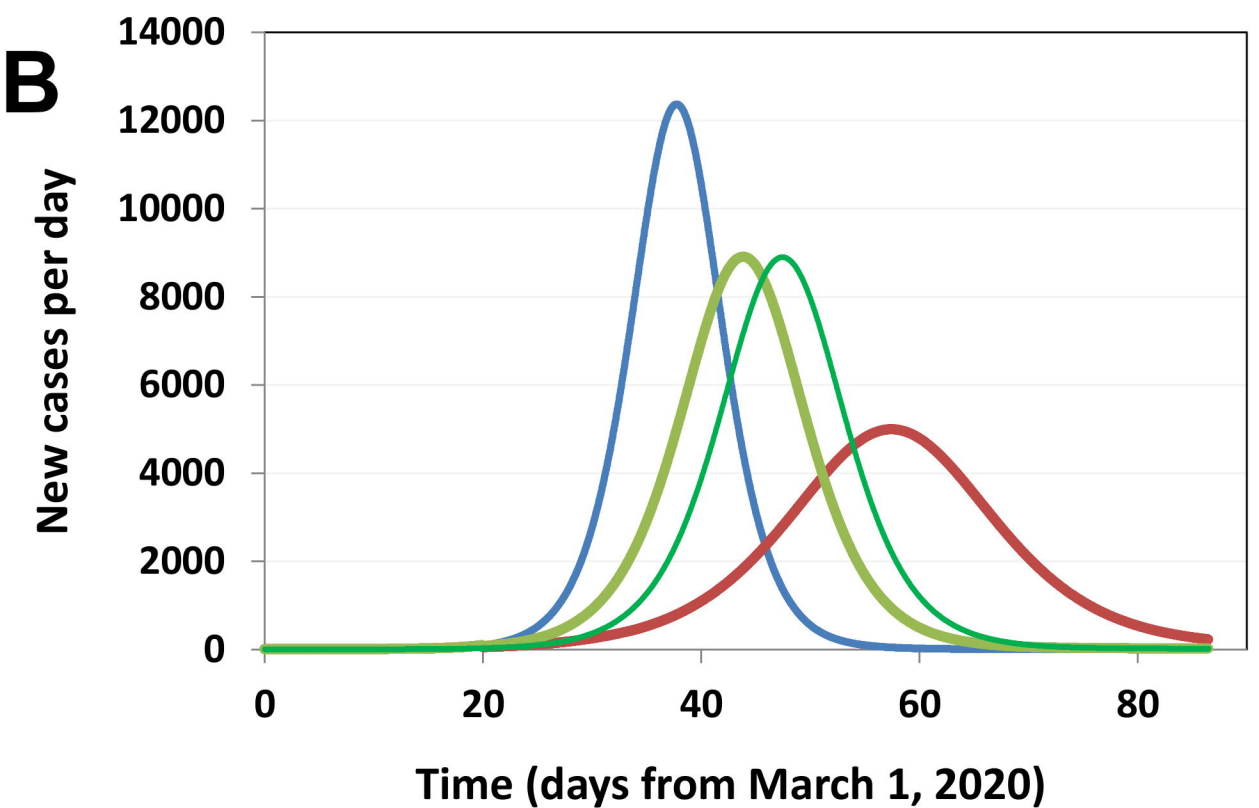
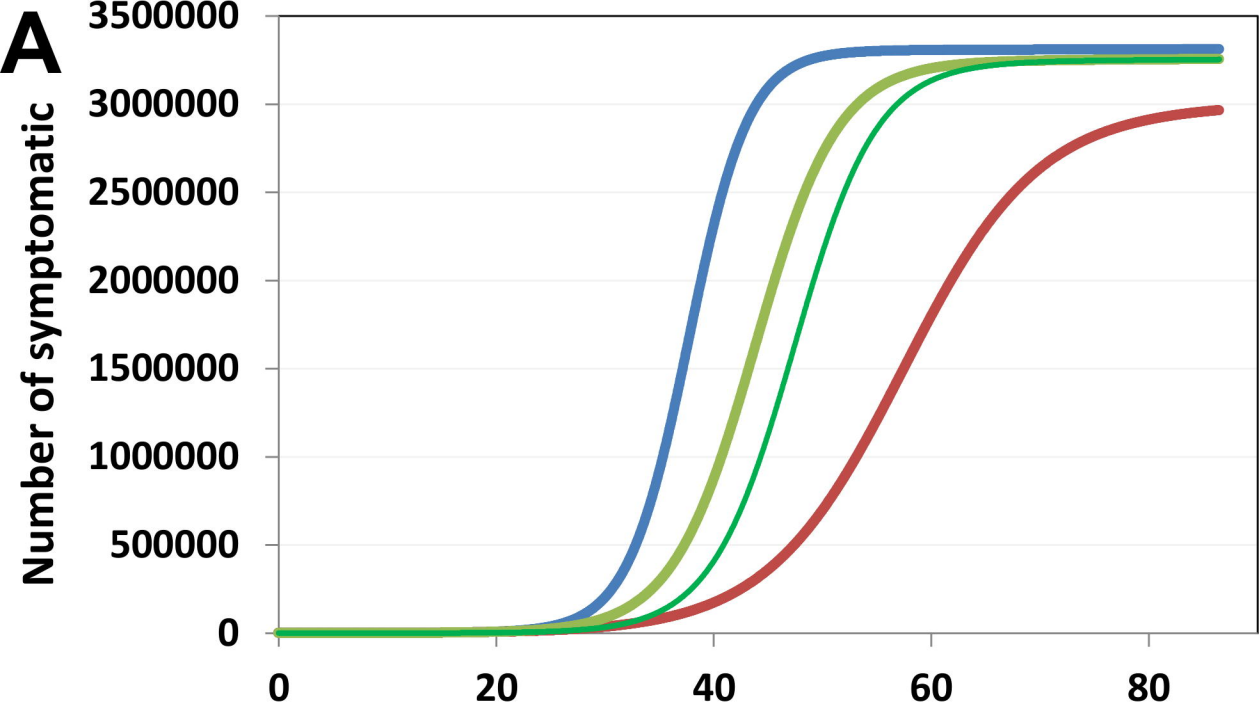
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499







COVID-19 positive cases
(cumulative)

100.00
80.00
60.00
40.00
20.00
0.00

B
Number of symptomatic

1.E+06
1.E+05
1.E+04
1.E+03
1.E+02
1.E+01
1.E+00

0 20 40 60 80
Time (from March 1, 2020)

0 5 10 15 20 25 30
time (days after March 1, 2020)

C

New cases per day

2,500
2,000
1,500
1,000
500
0

D
Number of beds needed

50000
40000
30000
20000
10000
0

32 37 42 47 52 57 62 67

0 10 20 30 40 50 60 70 80
Time (from March 1, 2020)