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Research article

Two waves of COIVD-19 in Brazilian cities and vaccination impact

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Abstract: *Backgrounds:* Brazil has suffered two waves of Coronavirus Disease 2019 (COVID-19). The second wave, coinciding with the spread of the Gamma variant, was more severe than the first wave. Studies have not yet reached a conclusion on some issues including the extent of reinfection, the infection fatality rate (IFR), the infection attack rate (IAR) and the effects of the vaccination campaign in Brazil, though it was reported that confirmed reinfection was at a low level. *Methods:* We modify the classical Susceptible-Exposed-Infectious-Recovered (SEIR) model with additional class for severe cases, vaccination and time-varying transmission rates. We fit the model to the severe acute respiratory infection (SARI) deaths, which is a proxy of the COVID-19 deaths, in 20 Brazilian cities with the large number of death tolls. We evaluate the vaccination effect by a contrast of "with" vaccination actual scenario and "without" vaccination in a counterfactual scenario. We evaluate the model performance when the reinfection is absent in the model. *Results:* In the 20 Brazilian cities, the model simulated death matched the reported deaths reasonably well. The effect of the vaccination varies across cities. The estimated median IFR is around 1.2%. *Conclusion:* Overall, through this modeling exercise, we conclude that the effects of vaccination campaigns vary across cites and the reinfection is not crucial for the second wave. The relatively high IFR could be due to the breakdown of medical system in many cities.

Keywords: COVID-19; reinfection; breakthrough infection; vaccination effectiveness; mathematical modelling

1. Introduction

Brazil was greatly affected by the coronavirus 2019 (COVID-19) pandemic caused by the severe acute respiratory syndrome coronavirus 2 (SARS-COV-2). As of 01 February 2022, more than 628,000 people had died and 25.6 million had been infected. Many works on the impact of COVID-19 in Brazil have been published. The first case of COVID-19 was detected in Brazil on 25 February 2020. A study [1] reported and contextualized epidemiological, demographic and clinical findings for COVID-19 cases during the first 3 months of the epidemic. These findings provide a comprehensive description of the ongoing COVID-19 epidemic in Brazil and may help to guide subsequent measures to control virus transmission. Cotta et al. [2] employed a SIRU-type epidemic model to predict the COVID-19 epidemy evolution in Brazil, and analysed the influence of public health measures on simulating the control of this infectious disease. Castro et al. [3] used daily data on reported cases and deaths to understand, measure and compare the spatiotemporal pattern of the spread across municipalities in Brazil. Candido et al. [4] used a mobility-driven transmission model to show the impact of nonpharmaceutical interventions (NPIs) in Brazil. Their study provides the evidence that current interventions remain insufficient to keep virus transmission under control in the country. In response to the COVID-19 pandemic, governments worldwide have implemented social distancing policies with different levels of both enforcement and compliance. Silva et al. [5] conducted an interrupted time series analysis to estimate the impact of lockdowns on reducing the number of cases and deaths due to COVID-19 in Brazil. They suggested that the social distancing policies can be useful to reduce the number of new confirmed cases and flatten the epidemic curve. However, there have been some controversies. Buss et al. [6] based on blood donor data, found a high infection attack rate (76%) after the first wave in Manaus, Brazil, which was against other two independent serological studies [7,8]. The inconsistence is likely due to the difference in the samples they used. Blood donor data used in reference [6] could have been influenced by the prevalence of COVID-19, while the other two studies were based on general samples (randomly chosen or walk-in sample) [7,8]. The pandemic has caused huge amount deaths (in the peak more than hundred deaths per day in a city of two million populations), it is hard to believe the recruitment of the blood donors was not impacted. The conclusion of high infection attack rate in the first wave of the epidemic resulted in the occurrence of an even higher second wave difficult to explain, unless a substantially high reinfection rate was assumed. Based on epidemiological modelling, Coutinho et al. [9] estimated that 78% of people in Manaus were infected with SARS-COV-2 in the first wave of the epidemic and that 28% of cases in the second wave were attributed to reinfection with the P.1 variant. However, their work was only based on the data of the second wave without fitting the epidemic curve of the first wave (fitting two waves is much more difficult than fitting one wave) and the finding is against with the fact that only three confirmed reinfections were reported in the whole state of Amazonas where Manaus is the capital city with half of the population of the whole province. The reinfection was surely under-reported, but the under-reporting ratio (reported divided by the true number) is more likely to be 1/1000, or 1/10,000, rather than 1/100,000 or even more extreme which is needed to explain the second wave, given only three confirmed reinfections. Thus, the question is whether a model without reinfection or a minor extent of reinfection can explain the second wave reasonably well. Ignoring reinfection, Chagas et al. [10] studied the impact of population mobility on the spread of COVID-19 in Brazil using a SENUR model equipped with mobility information.

Dana et al. [11] using a Bayesian model combining a Hamiltonian Monte Carlo algorithm,

predicted IFR of COVID-19 in Brazil to be about 0.3%. However, Mellan et al. [12] using a Bayesian model of semi-mechanistic COVID-19 transmission, predicted that IFR in 16 states of Brazil were between 0.7% and 1.2%. In reference [13], based on the random seroprevalence survey but not model simulation, the average IFR in Brazil was 1.03% (95% confidence interval [CI]: 0.88–1.22%).

Santos et al. [14] provided evidence for the presence of reinfection, although only one of 33 reported cases of COVID-19 recurrence was confirmed by genome sequencing. Two cases (that the first infection in the B.1.1.33 lineage and the second infection in the P.2 lineage harboring the E484K mutation) again confirmed the existence of reinfection [15,16]. Li et al. [17] used serological surveys based on random samples to estimate the infection attack rate (IAR, proportion of the population being infected) of COVID-19. Buss et al. [6], based on the sample of blood donors, estimated that over 75% of the population in Manaus had been infected with SARS-COV-2 by October 2020. However, with a sudden increase in COVID-19 hospitalizations in Manaus in January 2021, the second wave of epidemic turned to be more severe than the first wave, which implied that ~ 50% of the population were infected twice. Explanations for the resurgence of COVID-19 in Manaus included that the attack rate of SARS-COV-2 may be overestimated in the first wave of epidemic or the new variant of SARS-CoV-2 (Gamma variant) may evade immunity to previous infections [18].

In December 2020, SARS-COV-2 variant P.1 (Gamma) emerged in Brazil [19], which had been confirmed in six Brazilian cities as of 24 February 2021 [20], and accounted for more than 90% of COVID-19 cases in several Brazilian cities in August 2021 [21]. The spread of P.1 variant had been suggested as a contributing factor to the second wave of COVID-19 in Brazil. In the context of the spread of the P.1 variant, the odds of symptomatic SARS-CoV-2 infection were reduced by a factor of 0.5 following at least one dose of CoronaVac, one of two vaccines approved for emergency use in Brazil [22]. However, breakthrough cases infected with P.1 variant after CoronaVac vaccination were reported [23].

As of 19 October 2021, World Health Organization (WHO) concluded on the mortality of P.1 variant compared to the wild-type virus was still unclear [24]. Compared with wild-type virus, the risk of adjusted death associated with the P.1 variant was increased by 51% (95% CI 30–78%) [25]. The P.1 variant, compared with the original virus by multivariate analysis, did not show an increased risk of death but rather a decreased risk [26]. Given the increased transmissibility of P.1 variant, medical system crushed in many cities and the under-reporting of infections could be even severe, all these made the estimation of true IFR difficult. In this study, we assume that the IFR is constant through the two waves.

In order to study the change of the transmission rate, the effects of vaccination and reinfection on the two waves of epidemic in 20 Brazilian cities, we adopted and modified the classic Susceptible-Exposed-Infectious-Recovered (SEIR) model with additional classes for deaths and severe cases (or hospitalizations), and with vaccination and time-varying transmission rates.

2. Methods

The level of immune response of vaccination varies from person to person. High immune response implies high level of protection against infection and severe outcome after breakthrough infection (BTI). For simplicity, we adopt a dichotomy fashion where high immune response group (fully protection) enter the recovery class (R) after vaccination, and obtain long-term protection at least covering the study period (which will decay later). The low immune response group stay in the

susceptible pool (S) after vaccination, and is susceptible to BTI. The model has been used in previous works [27,28], for the sake of convenience of readers, we re-introduce it here.

The equations are given below:

$$S = -\frac{\beta SI}{N} - \eta\tilde{\nu}S \tag{1}$$

$$E = \frac{\beta SI}{N} - \sigma E \tag{2}$$

$$I = \sigma E - \gamma I \tag{3}$$

$$H = \pi\gamma I - \kappa H \tag{4}$$

$$D = \theta\kappa H \tag{5}$$

$$R = \eta\tilde{\nu}S + (1-\pi)\gamma I + (1-\theta)\kappa H \tag{6}$$

S, E, I, H, D and R denote the proportion of individuals which are susceptible, exposed, infectious, severe cases (or hospitalized), dead and recovered, respectively. η denotes the proportion of fully protected after vaccination (a proxy of the vaccination efficacy after second-dose). Parameters $\beta(t)$, σ, γ κ denote the rate of transmission, rate at which exposed showing infectiousness, rate at which infectious losing infectiousness (recovery rate), and rate at which severe cases (or hospitalized) leaving the severe (or hospitalized) stage. Parameters π and θ denote the ratios of infectious individuals entering H stage, and individuals in H entering death class. Thus, the IFR equals the product of π and θ. The exact definition of H is not crucial since we do not fit individual in H to data.

According to the existing literature, $\sigma = 0.5$ per day, $\gamma = 0.33$ per day and $\kappa = 0.125$ per day, and the average generation time (sum of the mean latent period and the mean infectious period) equals 5 days [29,30], and the average time from infection to death is 13 days [21]. With only deaths data, without severe cases (or hospitalization) data, we cannot estimate π and θ simultaneously. Thus, we consider two alternatives, $\pi = \theta$ or $\pi = 0.15$ for all cities. The reasoning for the assumption $\pi = \theta$ is that in cities with low (or high) IFR, both π and θ could be low (or high), namely the two parameters are likely positively correlated. We estimate $\beta(t)$ and θ through fitting the model to reported SARI deaths which is a proxy of the true COVID-19 deaths since during the pandemic all SARI are likely due to SARS-COV-2.

The daily proportion of the whole population (including both vaccinated and unvaccinated population) being vaccinated, denoted as $v(t)$, is available from the Brazilian Health Ministry [31]. In our model, we use $\tilde{v}(t)$ to denote the daily proportion of the unvaccinated population being vaccinated on day t, where $\tilde{v}(t) = v(t)/(1 - \int_0^{t-1} v(s)ds)$. The unit of t is day.

We assume the time-varying $\beta(t)$ to be an exponential cubic spline [32–34] with the number of nodes $n_\beta = 8$ evenly spanning the study period, and an upper limit of $\beta(t) < 608$ then the basic reproduction was within a reasonable interval with an upper limit 5. In the previous studies on multiple waves of infections, cubic splines were widely used to model time-varying transmission rate [35–37]. Alternatively, Google mobility index or stringency index data can be used to drive the time varying transmission rate. However, Google mobility index data alone may not be sufficient in some cities. Previous studies propose to use a weekly autoregressive model was used to explain the additional

variability [38]. In this work, we argue that cubic spline with several nodes is a sensible choice compared with Google mobility index plus a weekly autoregressive model (which involves more parameters to be estimated).

The individual susceptibility is not uniform. It was believed that some proportion of the population (e.g., children) have low susceptibility. It is naïve to assume the whole people have uniform susceptibility. We assume that 5% of people have pre-existing immunity against COVID-19, which may or may not be reflected in the specific antibody [39]. We assume that the initial number of individuals in E is the same as I, which is selected from [100, 500] randomly. In addition, the individuals in H is 1/10 of those in I, and the individuals in D is 1/10 of those in H.

Our model is a partially observed Markov process (POMP) model, which allows us to adopt the maximum likelihood iterated filtering technology to fit the model to mortality data [36]. The detailed fitting procedure is well documented at https://kingaa.github.io/sbied/.

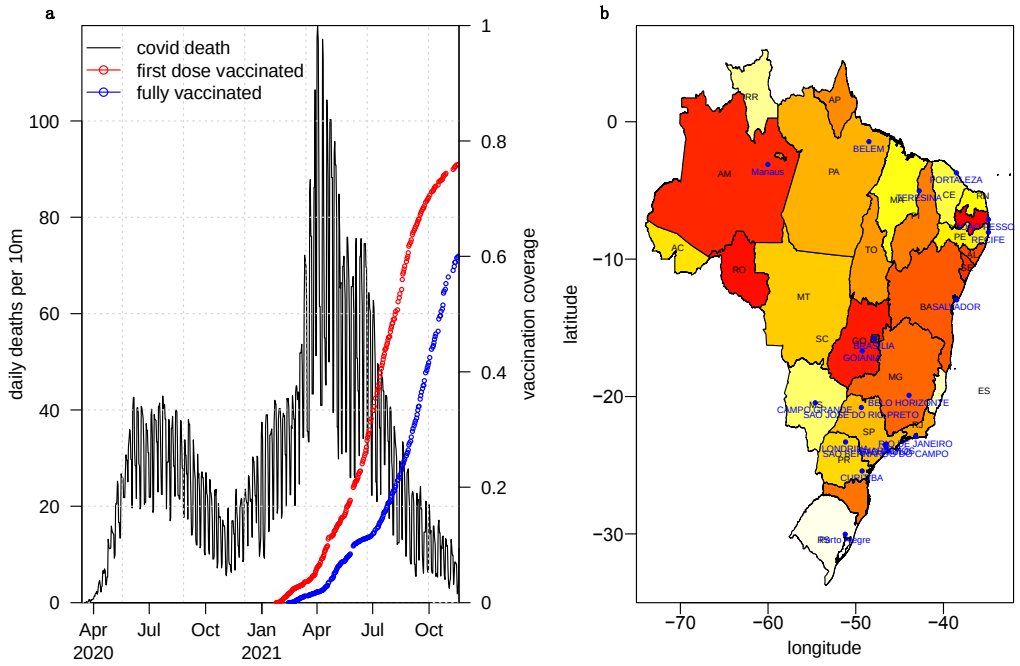

Figure 1. The daily reported COVID-19 deaths and vaccination coverage (one dose and two dose) in Brazil, obtained from WHO and the locations of the 20 cities studied in this work. We draw the map with the R package Shapefiles and boundary data from references [40,41]. The color of states is coded according to death per capita by 01 January 2021 showing the different state-level severity by then.

We assume a unified vaccine efficiency after two doses of vaccination, $\eta = 0.85$ against infection. This is a simplification of a very complicated scenario. In reality, individuals got partially protection after the first dose of vaccination, then got an improved protection after the second dose, possibly with a delay (e.g., two weeks) after the second dose. Vaccination leads to a reduce susceptibility against infection, and a reduced risk to severe cases or deaths after possible BTI. The protection (immunity) will decay over time. We synthesize all these complicities into a parameter $\eta = 0.85$, which takes into account the relatively high efficacy against severe outcome (> 95%) after BTI and a moderate efficacy against infection (~50%). To assess the population level impact of vaccination campaign, we compare the scenarios of "with" vaccination and "without" vaccination.

We first fit our model with actual vaccination data to reported SARI death and obtain the estimates of $\beta(t)$ and IFR. With these estimates, we set $v(t) = 0$, and rerun our model, we obtain the simulated deaths under the scenario of "without" vaccination.

Figure 1 shows the daily reported COVID-19 deaths and vaccination coverage (one dose and two dose) in Brazil and the locations of the 20 cities studied in this work.

3. Results

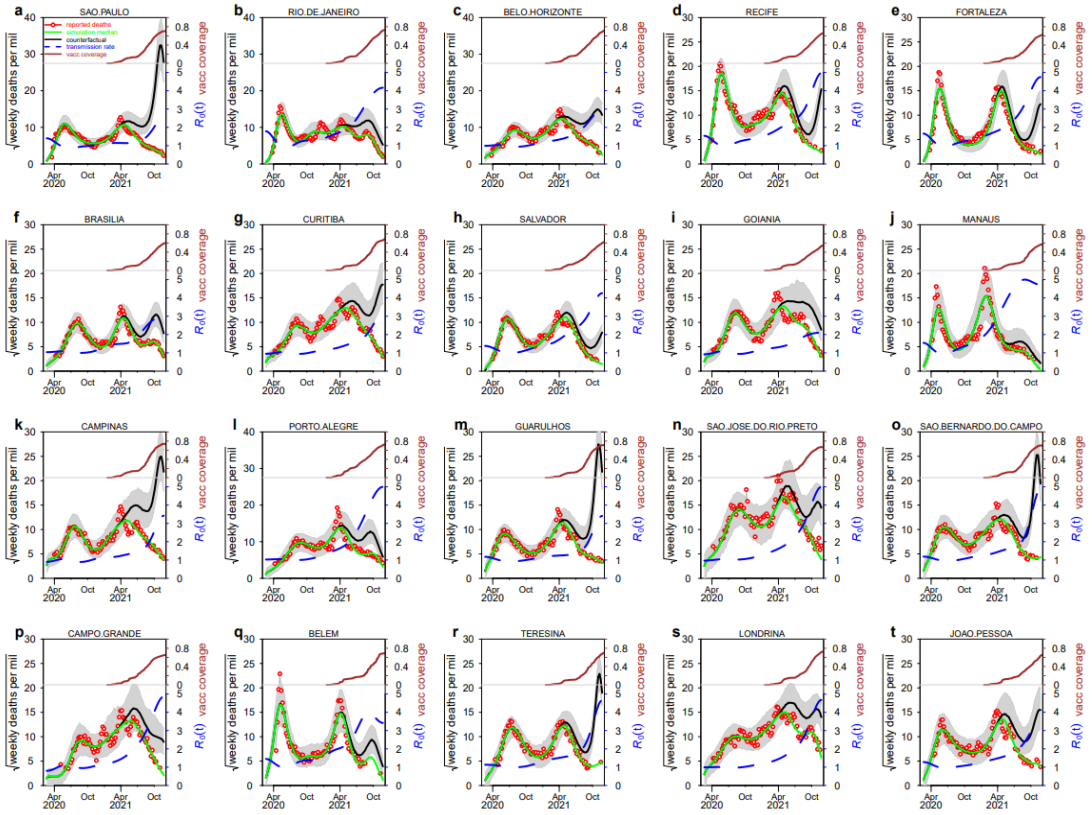

Figure 2. Model fitting and simulation results in 20 Brazilian cities. Red circles indicated the number of reported deaths from COVID-19. The green curves showed the median of the 1000 times model simulation with vaccination. The black curves (and gray region) showed the median of the 1000 times model simulation without vaccination (and the 95% confidence range of the model simulation). The blue dashed curve shows the estimated time-varying transmission rate in the form of $\beta(t)/\gamma$.

We show the results of the fitting and simulation for 20 Brazilian cities in Figure 2, with red circles, green curves and black curves representing the reported SARI deaths, simulated deaths in the 'with' vaccination scenario and the 'without' vaccination scenario, respectively. The blue dashed curve shows the estimates transmission rate $\beta(t)/\gamma$. The green curves closely matched the reported deaths in most of the cities. We estimate a median IFR at about 1.2%. The IAR equals the total SARI deaths divided by the estimated IFR. In a few cities, the green curves may be lower than the reported deaths which could be due to medical crush which was not modeled here (we discuss this later). Without consideration of reinfection, our model can capture the two waves. This demonstrated that

reinfection is not the crucial factor for the two waves (otherwise, one would encounter difficulty to achieve this). Our estimated IFR and IAR was in a reasonable range. A 10–20% reinfection rate may be slightly lower the estimated IAR. Because the IFR is a ratio of deaths over infections. A 10–20% reinfection rate means an increase in the size of the infections by a factor of 10–20%, which will result in a decrease in the estimates of IFR. In Recife and Fortaleza, where the epidemic was more severe than other cities in term of deaths per 1 million populations, it can be seen that the green and black curves were almost overlapped. However, in the cities that the deaths were relatively less severe, the differences between the green and black curves were much evident.

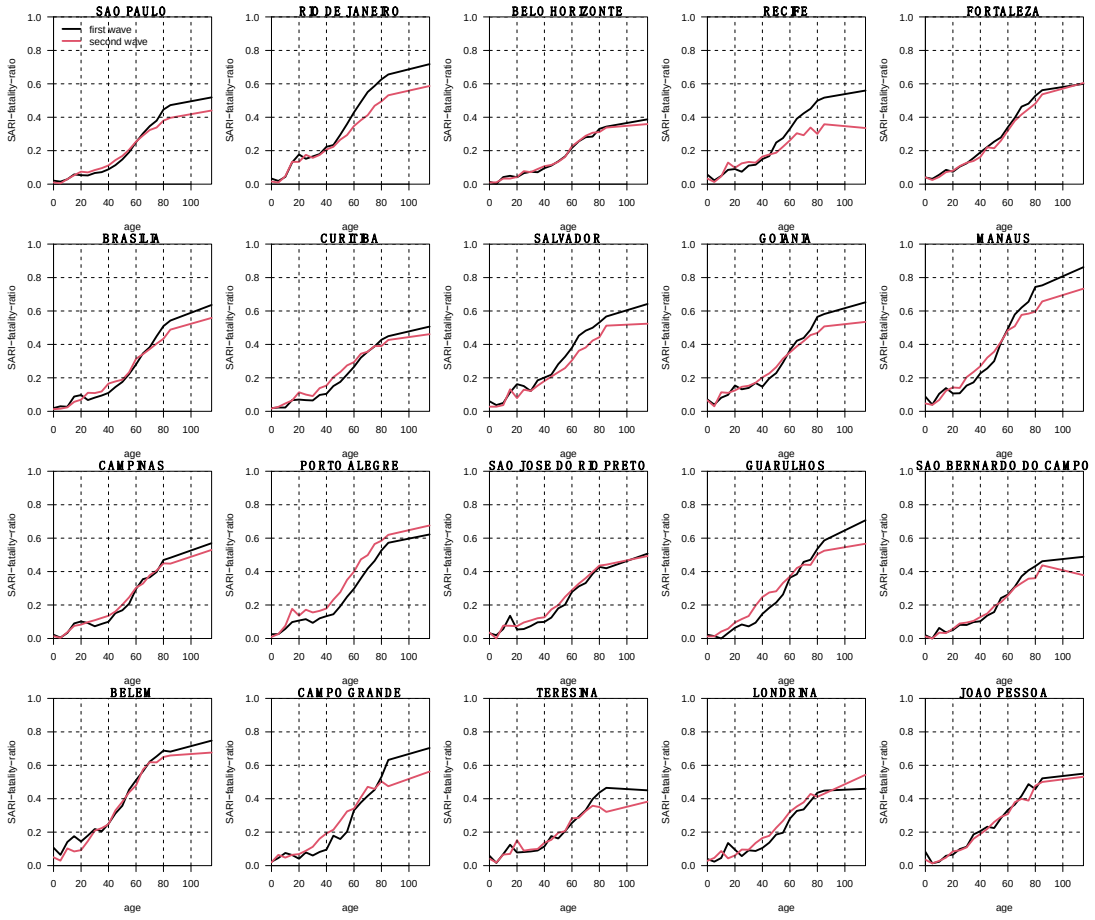

Figure 3. The SARI-fatality-ratio (death per SARI hospitalization) in 20 Brazilian cities of the two waves of the epidemic. We divide the two waves of hospitalizations before and after 23 November 2021, and the two waves of the deaths before and after 07 December 2021, namely allowing a two-week delay between the hospitalization start date and the date of the death, based on observation of this delay in the data. The black curve represented the first wave and the red curve represented the second wave. The difference between the black curve and the red curve indicated the change in the risk of death per hospitalization during the two waves of COVID-19 in different age groups.

We show the age profile of the SARI-fatality-ratios (risk of death per hospitalization of SARI) in Figure 3 for two waves (divided on 23 November 2020). The change in this mortality rate between the two waves was not significant, which justifies our assumption on a constant IFR in our model.

In supplementary, we show the total population of the 20 Brazilian cities in Table S1, and the prior ranges or values for the main parameters used in our model in Table S2. The detailed results of the model in two scenarios in Table S3. Model simulations for all cities are shown in Figures S1 where $\pi = 0.15, n_\beta = 8$. In the main text, we assume $\pi = 0, n_\beta = 8$, and there is little difference between the results of these two scenarios.

4. Discussion and conclusions

Brazil was severely impacted by the COVID-19 outbreak that began largely in March 2020 and subsequently experienced two large-scale deadly waves, the first dominated by the wild-type virus and the second by the Gamma P.1 variant. The Gamma P.1 variant was substantially more infectious than the wild-type virus, which led to the second wave with more severe cases and deaths. However, conclusions about the IFR, effect of reinfection and the effectiveness of vaccination of the two waves had not been reached.

Based on the seroprevalence survey of blood donors, and reported COVID-19 deaths and SARI deaths, Buss et al. [6] concluded that the IFR were 0.28% in Manaus, and 0.72% in São Paulo. However, the latest research found a different conclusion. In a large-scale sample and scope survey, Marra et al. [13] found that the average IFR for Brazil was 1.03% (95% CI 0.88–1.22%). The latter study was more convincing because of the larger sample and the consideration of effect of fading antibody levels. Based on model prediction, Mellan et al. [12] predicted the IFR was 0.7–1.2% in 16 states of Brazil. The median IFR in this study was predicted to be around 1.2%, which was slightly higher than the previous works that concluded to be about 1%. Here, we completely ignored reinfection and breakthrough infection, which could have led to a higher IFR. However, if we add a minor extent of reinfection (e.g., 10–20%), our model can also be fitted and we will get a slightly lower IFR. In addition, the IFR in this study was applied to two waves of COVID-19 covering both wild-type virus and the P.1 variant, and the later could have a higher risk of mortality and the medical system in many hardest hit cities had almost collapsed during both waves, in particularly the second wave. These factors explained a slightly higher IFR in this study.

Sabino et al. [18] put forward several hypotheses to explain why COVID-19 made a comeback in Manaus. One hypothesis was that P.1 variant may evade the immune response of previous infections other than P.1 variant, and may reinfect patients who were recovering from previous infection with the wild type virus. Based on epidemiological model, 28% of infections in the second wave of the COVID-19 were caused by reinfection with the P.1 variant [9]. However, the number of reported reinfection cases were still few [14–16], thus it is widely believed that the extent of reinfection was minor. Using the prevalence dynamics of the Bayesian model, it was estimated that the P.1 variant escaped immunity 25–61% [20]. Under the assumption that there was little reinfection, using phylogenetic methods, the infectivity of the P.1 variant was at least twice that of the parent strain [42]. In addition, He et al. [43] showed a two-strain SEIR model can fit the two wave in Manaus reasonably well without significant reinfection (the fitting in the sequence of P.1 proportion is much better than previous studies). This study re-confirms the argument that reinfection likely played weak role in causing any epidemic waves. Even if a significant proportion (e.g., 10–20%) of the population may be immunity boosted due to reinfection, their contribution to further transmission and to deaths are lower than the same amount of first time infection. Thus the attack rate of SARS-COV-2 in Manaus after the first wave was overestimated, and the IFR 0.28% is an

underestimate ignoring the medical system breakdown [6].

Before this study, the population level effect of vaccines against SARS-CoV-2 variants in Brazil is unclear. A study claimed that at least one dose of CoronaVac had been shown to be effective against symptomatic SARS-COV-2 infection in the context of P.1 variant transmission [22]. The IAR was relatively high and vaccination rates were relatively low comparing to other developed countries [21]. Gazit et al. [44] concluded that compared with two doses of BNT162b2 vaccine induced immunity, infection induced immunity had a longer duration and stronger protective effect on symptomatic diseases caused by delta variant. Although it was not a study of the P.1 variant, one would expect similar effects. The limitation of vaccination was highlighted in a recent study that showed vaccination was not enough to prevent transmission in household settings exposed to the delta variant, but there was no mention of P.1 variant [45].

By setting the extreme situation of without reinfection, the simulated death toll curve matched the actual reported deaths. Thus, we demonstrate that reinfection was not the key to these two waves of COVID-19. The difference between the green and black curve for each city in Figure 2 was evident and varied across cities. These hit-hardest cities show smaller population effects of vaccination, since most of the population had naturally immunized before vaccination. In cities where the epidemic was relatively mild, the effect of vaccination was better.

The strengths of our study include that this is a large-scale multiple city study in Brazil. Prior to this, most studies are limited to a single city. In addition, we further elucidated the impact of time varying transmission rate and vaccination on the two waves of COVID-19 in Brazil bases on model simulation. Even though reinfection was ignored, our model can reproduce the two waves with slightly higher IFR than previous believed. But the breakdown of medical systems in many cities could have led to such an IFR. In addition, we only focused on overall protection of the second-dose of vaccination, but in fact the first-dose also provided partial protection although it was much weaker [22].

Overall, we conclude that reinfection was not the key factor in the two waves of COVID-19 in Brazil, and vaccination saved substantial number of lives. Correctly assessing the risk of reinfection and the scale of reinfection-led mortality is very important and very challenging, we argue that to achieve this goal, large-scale control-case study or household study is needed. Assessing the effectiveness of vaccination campaign is also very important and challenging. In this work, we demonstrated that reinfection-led mortality is unlikely a major factor in Brazil and vaccination prevented a large number of deaths, and the effects are not uniform across cities.

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Conflict of interest

This work does not involve any conflict of interest.

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Supplementary

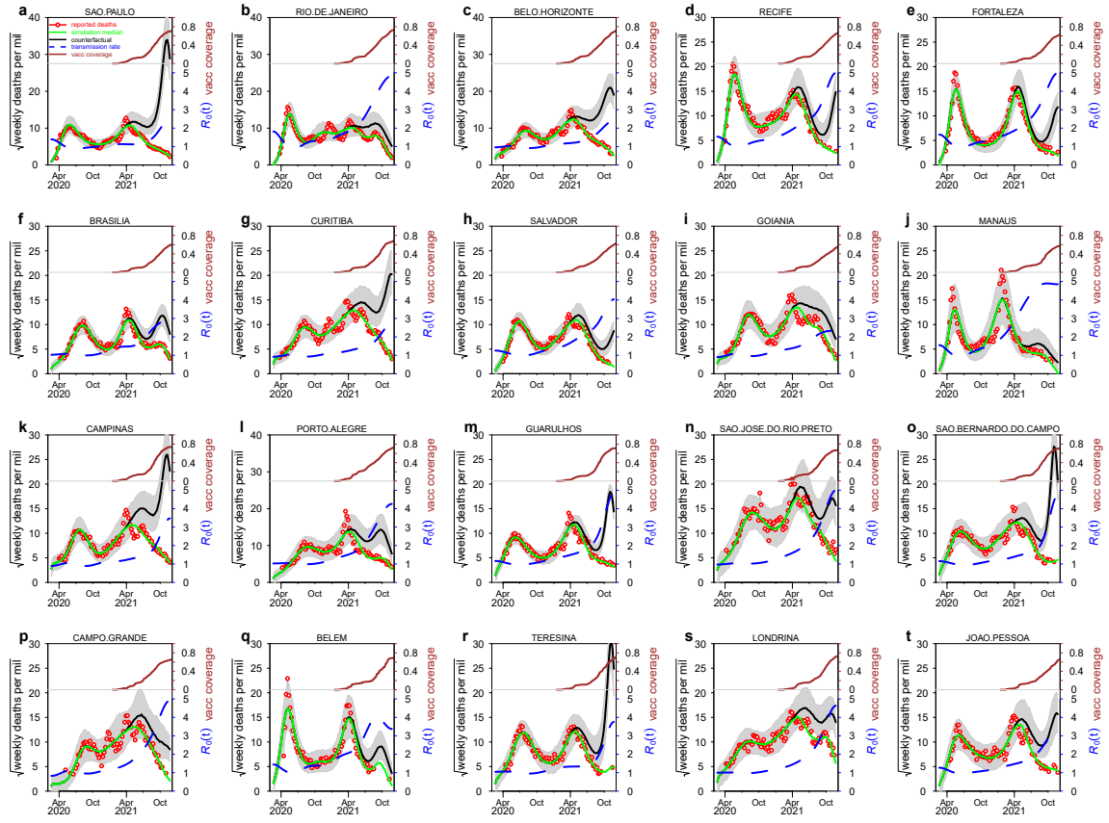

Figure S1. Reproducing Figure 2 with $\pi = 0.15, n_\beta = 8$.

Table S1. Total population of 20 Brazilian cities in 2020.

city	Pop (2020)
SAO PAULO	12325232
RIO DE JANEIRO	6747815
BELO HORIZONTE	2521564
RECIFE	1653461
FORTALEZA	2686612
BRASILIA	3055149
CURITIBA	1948626
SALVADOR	2886698
GOIANIA	1536097
MANAUS	2219580
CAMPINAS	1213792
PORTO ALEGRE	1488252
SAO JOSE DO RIO PRETO	464983
GUARULHOS	1392121
SAO BERNARDO DO CAMPO	844483
BELEM	1499641
CAMPO GRANDE	906092
TERESINA	868075
LONDRINA	575377
JOAO PESSOA	817511

Table S2. Prior ranges/values for the main parameters used in our model.

Parameter	Symbol	Prior range/value
Transmission rate	$\beta(t)$	<608
Rate at which exposed showing infectiousness	σ	0.5/day
Rate at which infectious losing infectiousness	γ	0.33/day
Rate at which hospitalized leaving the hospitalized stage	κ	0.125/day
Ratio of infectious individuals entering hospitalized stage	π	θ or 0.15
Ratio of hospitalized individuals entering death stage	θ	(0.090664, 0.145647) or (0.0548, 0.141421)
Vaccination efficacy after second-dose	η	0.85
Number of nodes of an exponential cubic spline	n_β	8

Table S3. The detailed results of determined parameters of the model corresponding to 20 Brazilian cities in two scenarios.

	loglik.1	loglik.2	diff_loglik	IFR.1	IFR.2
SAO.PAULO	-553.4918	-553.4808	-0.011	0.0212	0.0212
RIO.DE.JANEIRO	-531.5078	-531.5882	0.0804	0.0099	0.01
BELO.HORIZONTE	-416.3607	-416.8723	0.5117	0.0207	0.0129
RECIFE	-385.3031	-385.346	0.0429	0.0157	0.0157
FORTALEZA	-396.84	-396.8842	0.0442	0.0098	0.0098
BRASILIA	-403.0766	-403.0966	0.02	0.0083	0.0082
CURITIBA	-420.2897	-420.3855	0.0958	0.0185	0.0158
SALVADOR	-366.712	-366.1312	-0.5808	0.0082	0.0082
GOIANIA	-410.2133	-410.668	0.4547	0.0156	0.015
MANAUS	-397.4874	-398.2648	0.7774	0.0082	0.0082
CAMPINAS	-371.4368	-371.3761	-0.0608	0.018	0.0203
PORTO.ALEGRE	-394.5856	-395.424	0.8384	0.0113	0.0112
GUARULHOS	-338.6575	-338.1193	-0.5382	0.0092	0.0097
SAO.JOSE.DO.RIO.PRETO	-350.3752	-350.8219	0.4467	0.0212	0.0212
SAO.BERNARDO.DO.CAMPO	-315.6685	-315.7307	0.0622	0.0139	0.0134
CAMPO.GRANDE	-321.0268	-320.6764	-0.3504	0.0116	0.012
BELEM	-336	-336.1543	0.1543	0.01	0.0102
TERESINA	-287.7254	-287.191	-0.5345	0.0109	0.0111
LONDRINA	-331.6809	-331.4184	-0.2625	0.0167	0.0171
JOAO.PESSOA	-320.6502	-320.657	0.0068	0.0121	0.0123
Min.	-553.4918	-553.4808	-0.5808	0.0082	0.0082
1st Qu.	-404.8608	-404.9894	-0.1112	0.0098	0.0099
Median	-378.37	-378.361	0.0436	0.0119	0.0122
Mean	-382.4545	-382.5143	0.0599	0.0135	0.0132
3rd Qu.	-334.9202	-334.9703	0.2274	0.0171	0.0157
Max.	-287.7254	-287.191	0.8384	0.0212	0.0212