

Inference of epidemiological parameters via model fitting: some examples from Covid-19

Renato Mendes Coutinho¹

Emanoel Fabiano Menezes Pereira²

SoLaBiMa 2026 - Asunción

October 08, 2026

¹Centro de Matemática, Computação e Cognição,
Universidade Federal do ABC, Brazil
renato.coutinho@ufabc.edu.br

²Instituto Federal de São Paulo, Brazil



Overview

We often build epidemiological models to answer very applied questions, and that usually involves

- (quantitative) prediction ability and/or
- comparison of outcomes in different (plausible) scenarios

To accomplish these goals, models need to:

- present appropriate structure, ie, include
 - relevant processes,
 - clear points of contact with data;
- have sufficiently accurate parameters.

The last one is far from simple, and a careless approach can completely derail results.

Overview

We can sometimes use “secondary” data¹ to fit models, and thus estimate key parameters.

I’m going to briefly present two works, both on models and model fitting for the Covid-19 pandemic, and at the end reflect on what ingredients makes such tasks feasible or challenging.

¹that is, data that doesn't measure directly the parameter of interest

Model-based estimation of transmissibility and reinfection of Sars-COV-2 P.1 variant

(2021)

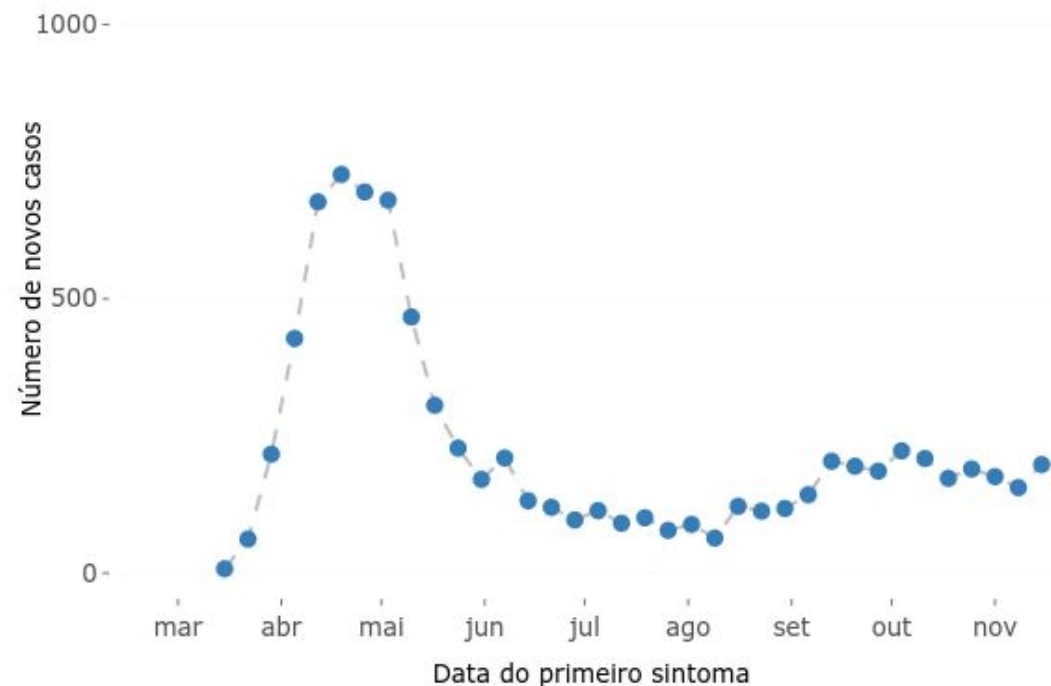
Context

Manaus already had a large epidemic with a huge number of cases, and hospital collapse, in April 2020

Three-quarters attack rate of SARS-CoV-2 in the Brazilian Amazon during a largely unmitigated epidemic

LEWIS F. BUSS ^{ID}, CARLOS A. PRETE JR. ^{ID}, CLAUDIA M. M. ABRAHIM ^{ID}, ALFREDO MENDRONE JR. ^{ID}, TASSILA SALOMON ^{ID}, CESAR DE ALMEIDA-NETO ^{ID},
RAFAEL F. O. FRANÇA ^{ID}, MARIA C. BELOTTI ^{ID}, MARIA P. S. S. CARVALHO ^{ID}, [...], AND ESTER C. SABINO ^{ID} [+26 authors](#) [Authors Info & Affiliations](#)

SCIENCE • 8 Dec 2020 • Vol 371, Issue 6526 • pp. 288-292 • DOI: [10.1126/science.abe9728](https://doi.org/10.1126/science.abe9728)

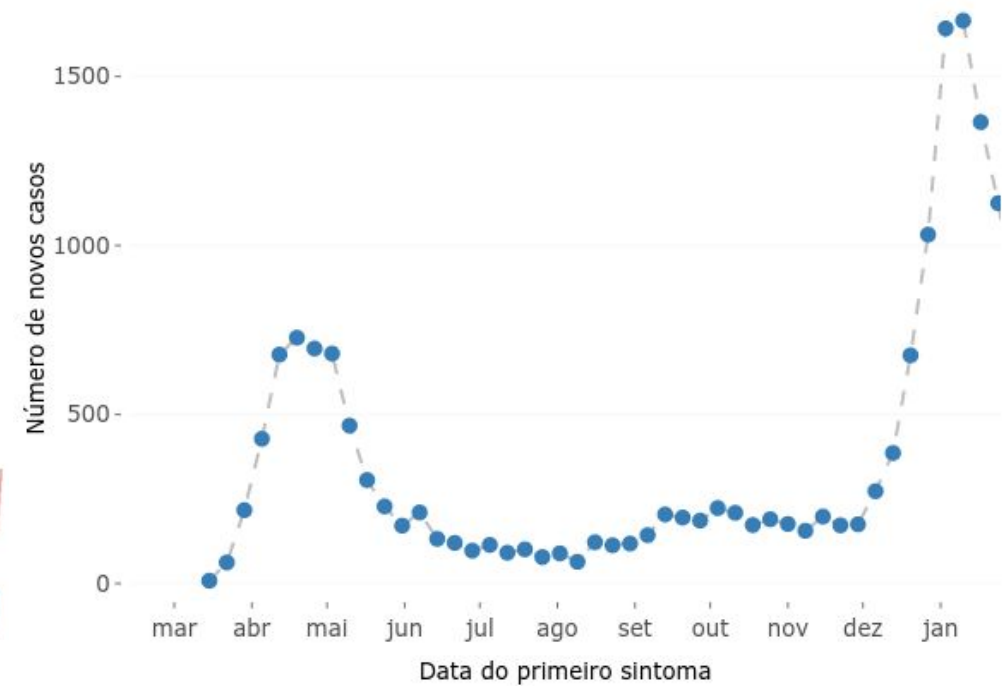
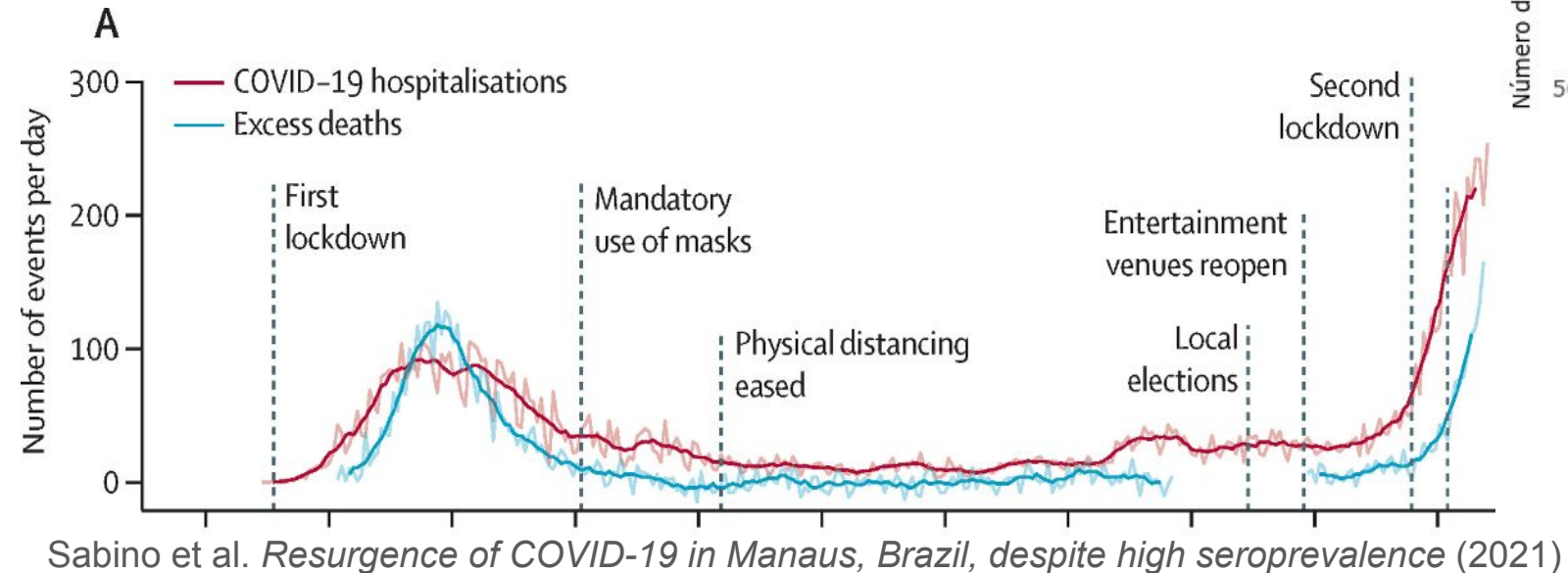


Could it have another?

Reinfection was defined in a way that made it almost impossible to detect - did that mean it was impossible?

The second epidemic in Manaus

A second epidemic, even larger than the first, happened - it was identified as a new variant of concern (P.1, later Gamma) in January 2021



Hospitalization data from SIVEP-Gripe. Observatório Covid-19 BR.

What's going on?

Main goal

The new variant will likely spread, and we want to estimate its impact.
For that, we need to know why the resurgence in Manaus occurred.

Was it due to very high transmissibility?

Or reinfection? (our main hypothesis)

Or relaxation of control measures?

Or can it be due to increased hospitalization rates?

Or maybe that estimate of seroprevalence was just wrong (as argued by some people at the time)

Main assumptions

- SEIR-like model structured by age, including severe (hospitalized) cases and deaths, and 2 variants
- original/wild type (1) and P.1 (2) with the same characteristics except:
 - Different **transmissibilities**
 - P.1 has a probability of **reinfection** of people recovered from the original variant
 - Different pathogenicity (changes IHR)

Model structure I

SIR or SEIR?

- Qualitatively, they are very similar!
- Quantitatively, we know from experience that SEIR models usually provide a better fit to data (with the same number of free parameters)

Which severity classes?

- We are going to use **hospitalization** data, so we certainly need that!
- Asymptomatic infections were already known to be important
- We may want to (eventually) compare the number of deaths

Model structure II

Why age structure?

- We are going to run the model for a short interval (less than 3 months)
- Disease outcomes are strongly dependent on age
- Transmission is strongly dependent on age

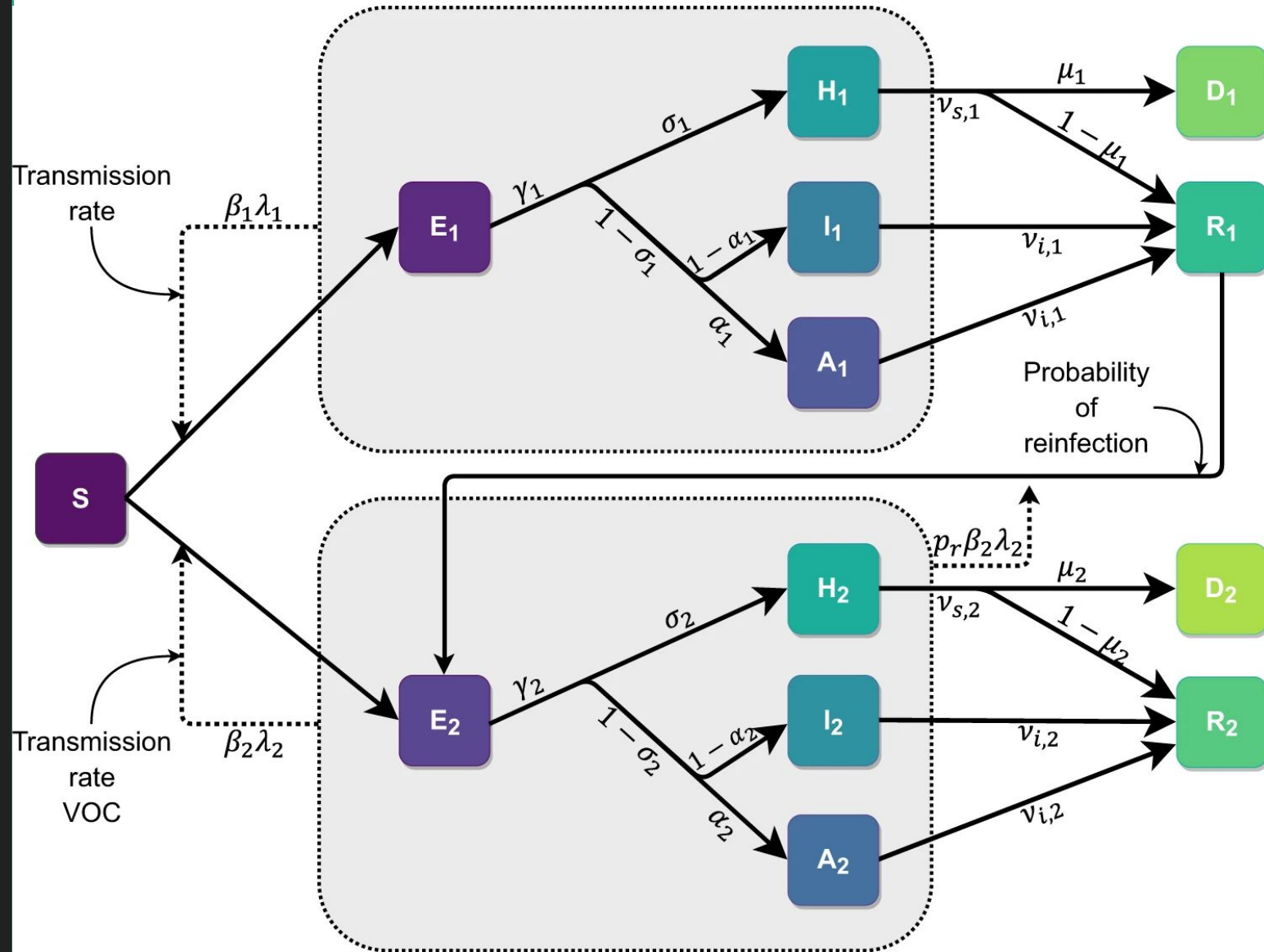
Model diagram

Variables:

(S)usceptible
(E)xposed (pre-symptomatic)
(A)symptomatic
(I)nfectious (mild case)
(H)ospitalized (severe case)
(R)ecovered
(D)eceased

Age structure:

- Young (<20 anos)
- Adults (20-60 anos) e
- Elderly (>60 anos)

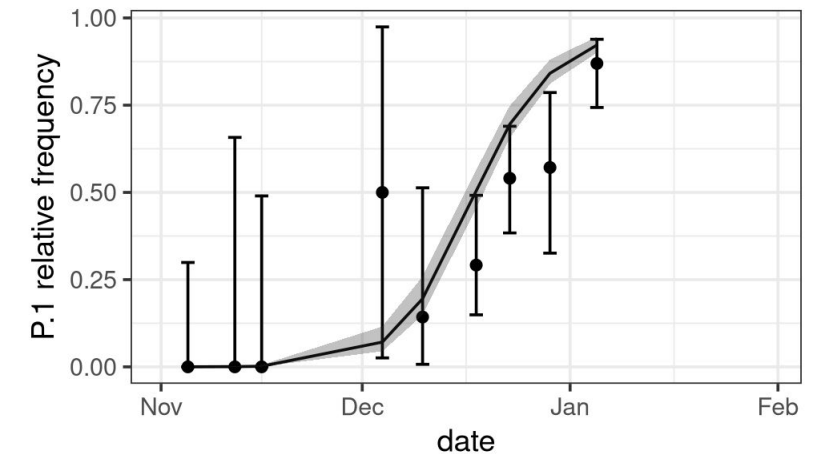
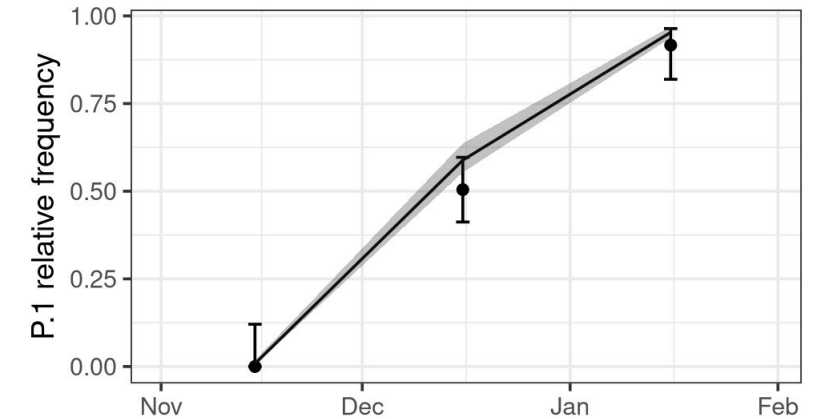


Source of data and parameters

- IHFR, recovery rate: SIVEP-Gripe
- IHR: Salje et al., 2020
- Population age structure: IBGE
- Contact matrices: Prem et al., 2020
- Hospitalizations during the previous week yield current incidence (at start of the simulation)

Fitted data:

- New cases per week: nowcasted from SIVEP-Gripe by Observatório
- Frequency of P.1: data from Fiocruz, and Faria et al. preprint data



Fitted parameters

- **Increase of transmissibility** of the new variant
- **Reinfection probability** (P.1 infecting recovered from original)

“Nuisance parameters”:

- Soroprevalence in Manaus about the time of emergence of P.1 (nov/2020)
- Frequency of P.1 at the beginning of November (accounts for time of emergence)
- Original variant's transmission rate (accounts for factors like relaxing of distancing measures, increasing contact rate)

Fitting by MLE

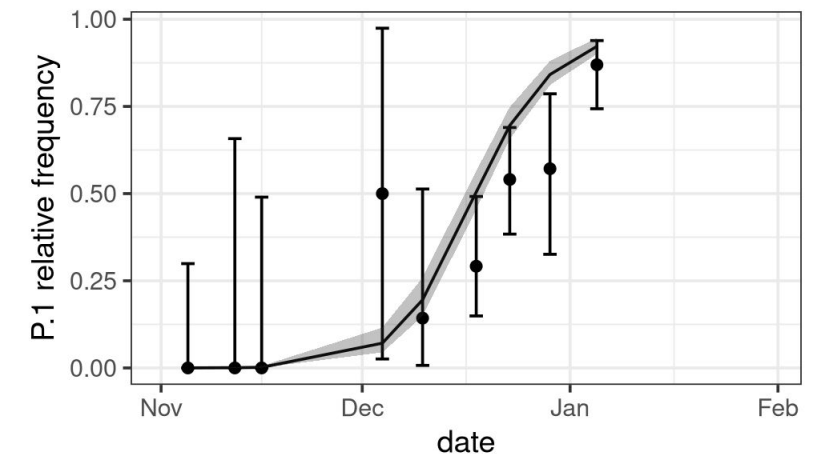
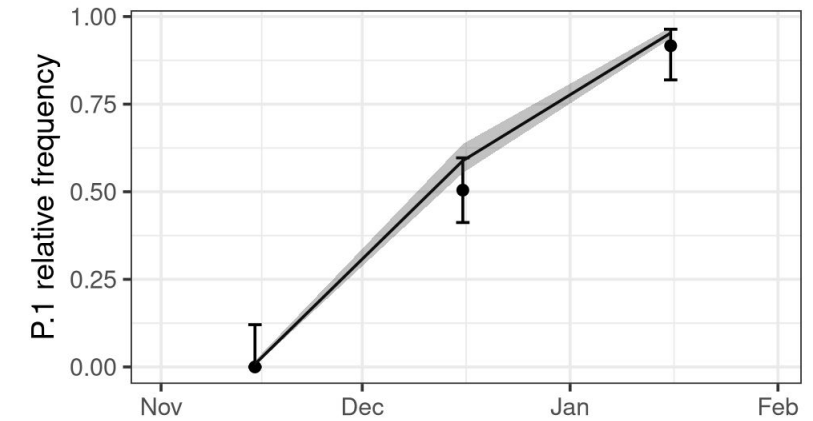
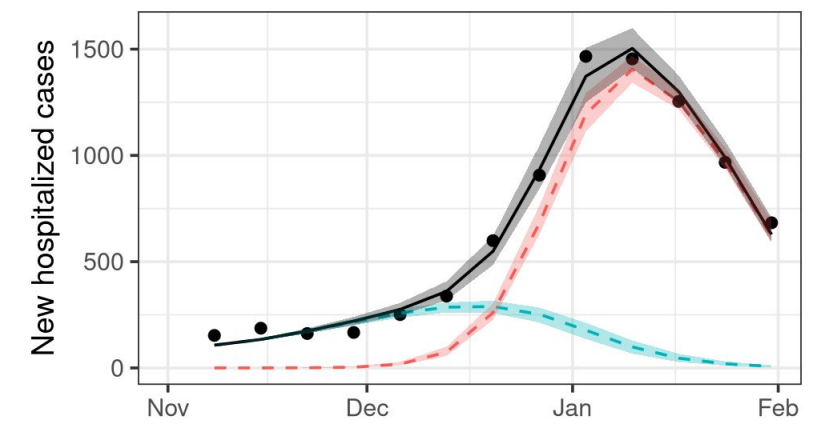
We use Maximum Likelihood Estimation (MLE)

Assuming hospitalized cases observed follow a Poisson distribution, and P.1 samples, a binomial distribution



Best fit

- Transmissibility increase of **2.61** (95% CI: 2.41-2.76)
- Relative force of reinfection 3.2% (2.6-4.0%)
- 28% of new infections were reinfections
- Initial prevalence of 78% (73-83%)
- Initial growth rate: 0.029 days^{-1} (0.024-0.035)
- Initial frequency of P.1: 30.4×10^{-5} (8.2-112.9 $\times 10^{-5}$)



Conclusions

- Transmissibility of VOC P.1 is **large**
- Reinfection probability was relatively low, but important
We estimated it accounted for 28% of cases - this was later found to be approximately correct!
- Prevalence in November was already high
- Potential for **very fast** spread - probably the current increase already reflects this
- It might spread even faster in places with lower prevalences
- Spreading control and mitigation measures are urgent

Published paper: <https://www.nature.com/articles/s43856-021-00048-6>

About the group

Renato Mendes Coutinho (UFABC)

Flávia Maria Darcie Marquitti (IFGW e IB - Unicamp)

Leonardo Souto Ferreira (IFT - UNESP)

Marcelo Eduardo Borges (Observatório Covid-19 BR)

Rafael Lopes Paixão da Silva (IFT - UNESP)

Otávio Canton (IFT-UNESP)

Tatiana P. Portella Zenker (IB-USP)

Silas Poloni Lyra (IFT-UNESP)

Caroline Franco (IFT-UNESP)

Mateusz M. Plucinski (CDC - USA)

Fernanda C. Lessa (CDC - USA)

Antonio Augusto Moura da Silva (UFMA)

Roberto Kraenkel (IFT-UNESP)

Maria Amélia de Sousa Mascena Veras (FCMSCSP)

Paulo Inácio Prado (IB-USP)



Estimation of infection and waning immunity parameters of Covid-19 using an SEAIHR model with age structure and immunological status

First author: **Emanoel Fabiano Menezes Pereira**

(Ph.D. thesis at UFABC, now at IFSP)



Context and goal

- End of 2022: large uncertainty over the duration of immunity after infection or vaccination
- Epidemics with lower hospitalization burden, but infections never go to 0 (endemic state?)

Essential knowledge needed to determine vaccination schedule:
do we need to take vaccines every 6 months? Every year? Never again?

The main question then is to estimate the waning immunity rate, or equivalently: how long on average does the immunity last?

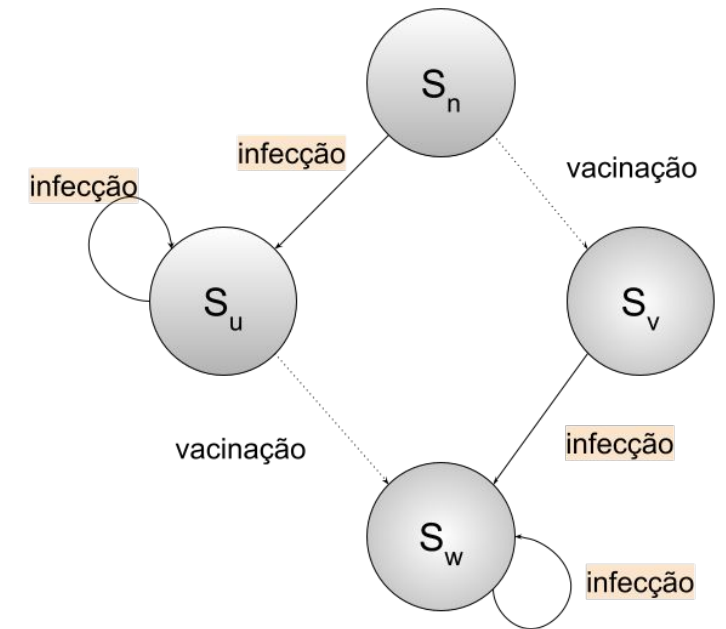
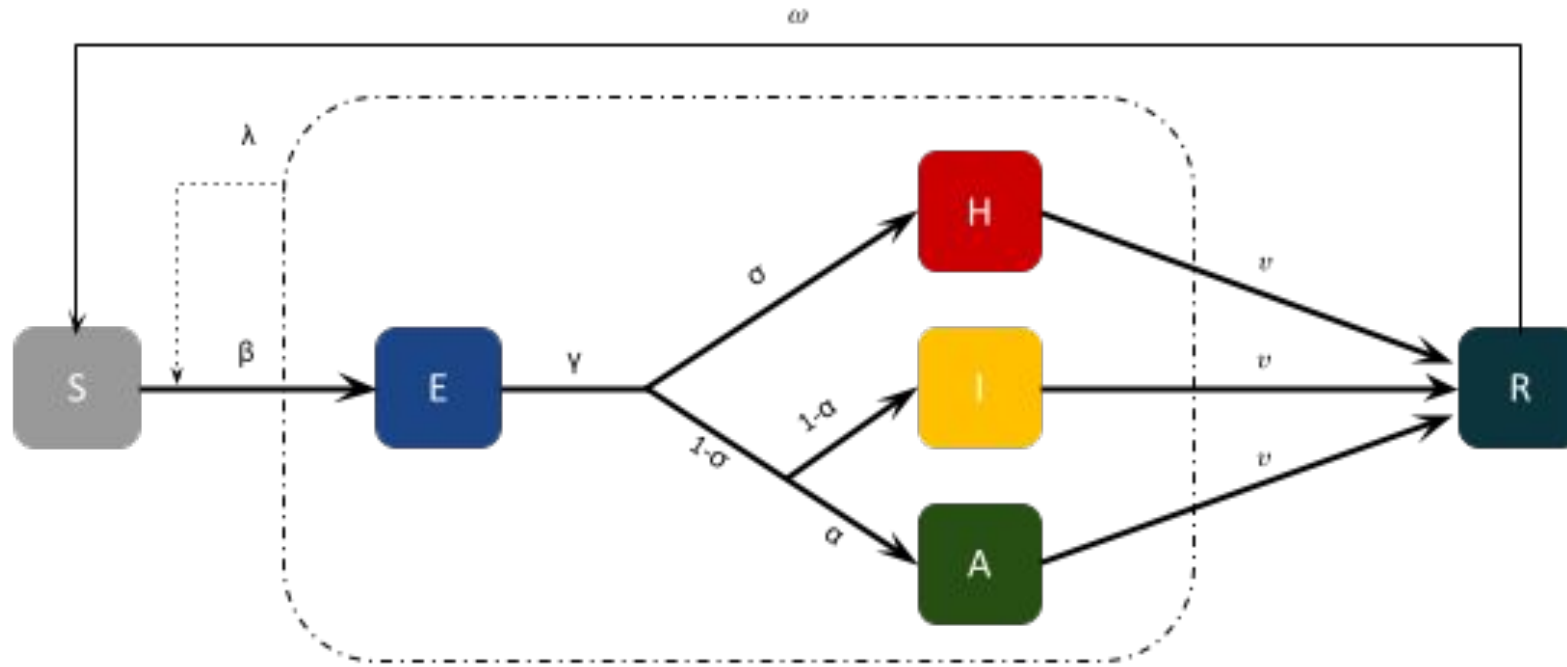
We probably also need to estimate the transmission rate, once again.

Main assumptions

- SEIR-like model structured by age, including severe (hospitalized) cases
- Individuals can be either:
 - n : Naïve (never infected, never vaccinated)
 - u : Infected, but unvaccinated
 - v : Vaccinated, but never infected
 - w : Both infected and vaccinated

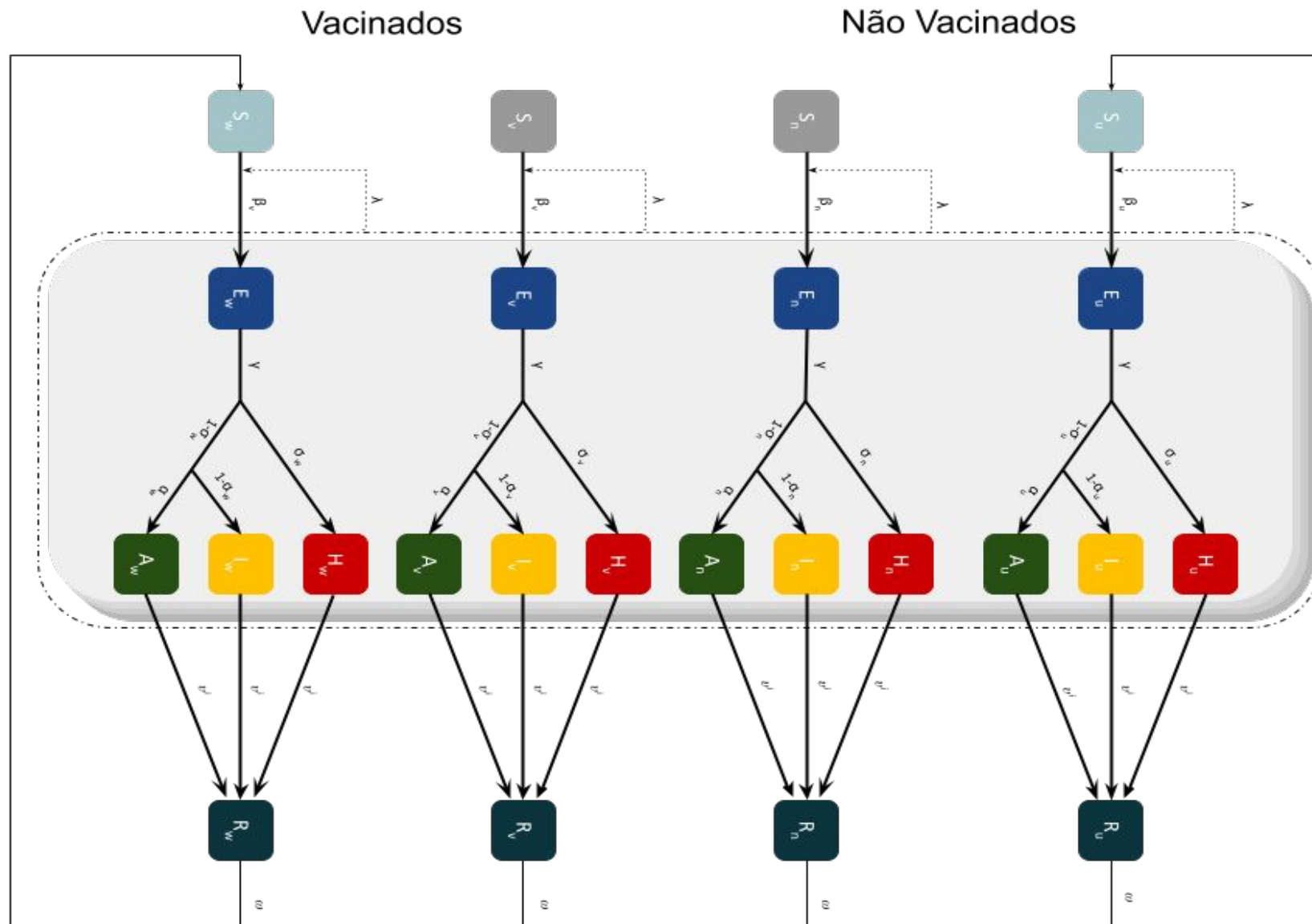
Different immunity status will lead to different levels of protection, but for simplicity, we assume waning immunity rate ω is the same for all.

Model diagram



Waning immunity with rate ω may lead to different S classes.

Full model diagram



4 immunity
status classes

4 age classes

Some more parameters....

- $1 - \epsilon\beta_j$: protection against infection
- $1 - \epsilon\alpha_j$: protection against clinical symptoms
- $1 - \epsilon\sigma_j$: protection against hospitalization

where j denotes immunity status

These values come from vaccine efficacy, which were estimated during clinical trials for vaccine approval, but the first one ($\epsilon\beta_j$) has huge uncertainty.

Protection against infection was not discussed much since the focus was on reducing severe cases, and it was already known that vaccines couldn't *completely* prevent infection (but *completely* does not mean no effect!)

Hypothesis: (quasi-)endemic state

Casos Diários de Covid-19 (por 100.000 habitantes) nas Cidades Seleccionadas



Analysis and fitting

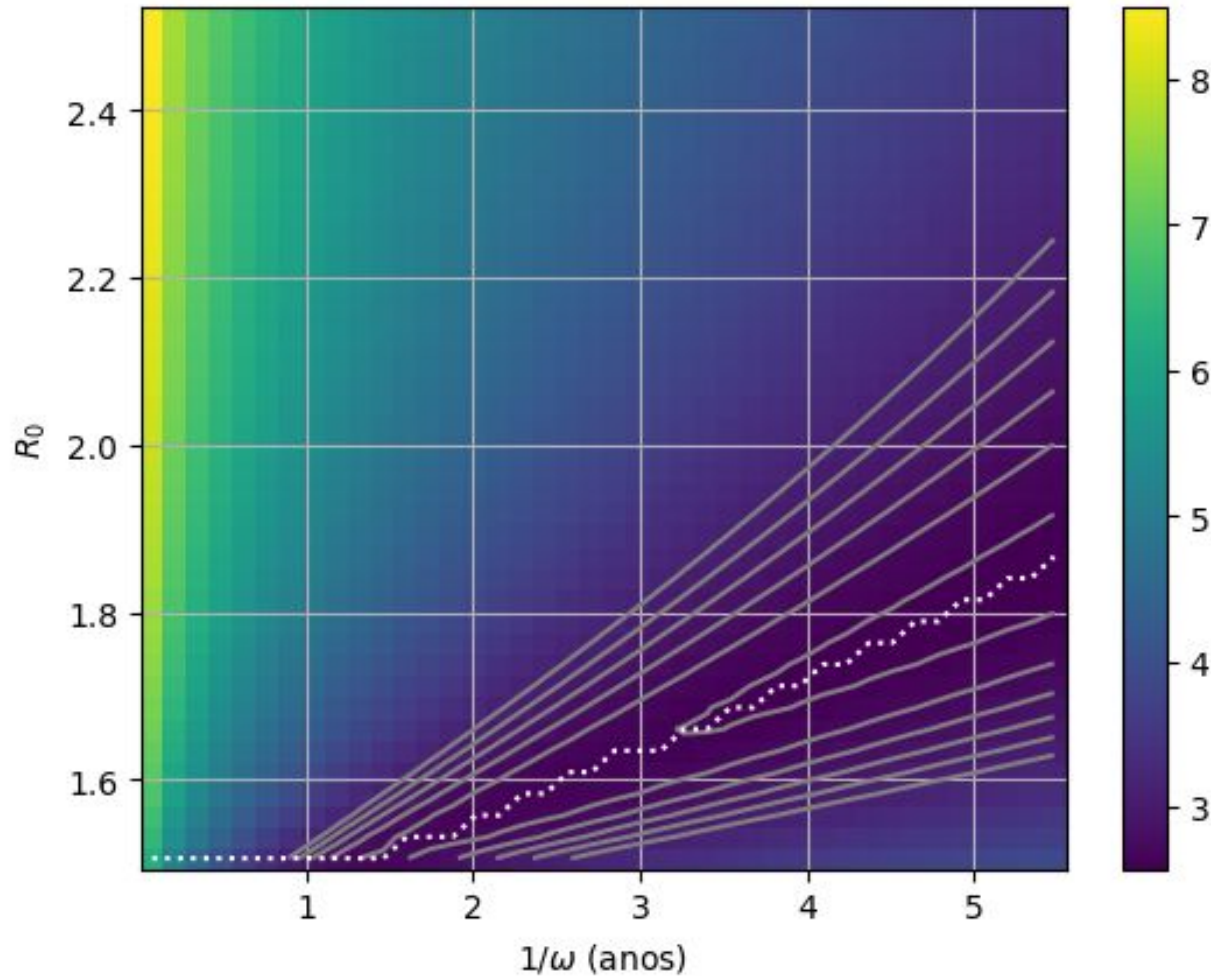
We are going to assume that the approximately stationary state between peaks is close to the *endemic state* - this is borne out by model simulations

We solve for the endemic fixed point of the model to obtain a system of (only!) 4 algebraic equations* which we numerically solve, and use it to determine total number of hospitalizations

We use this solution to fit β and ω using maximum likelihood using a Poisson probability model for hospitalizations

We repeat this procedure for 6 Brazilian state capitals (large cities with many cases)

Results

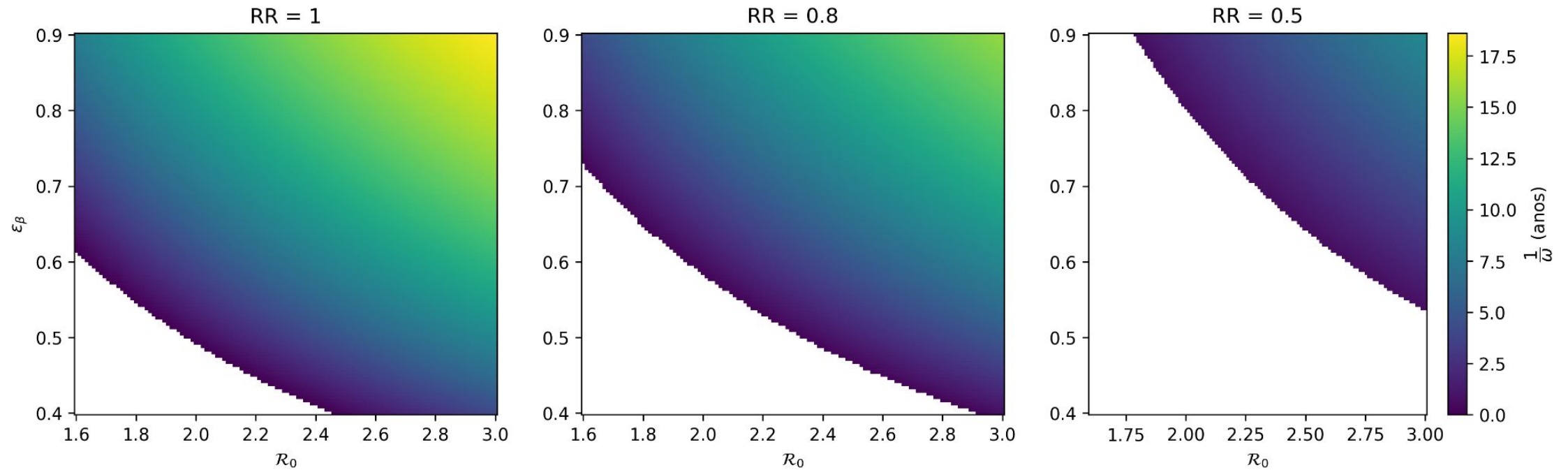


Small changes in β (related directly* to R_0) lead to large changes in $1/\omega$

The log-likelihood has a valley in the R_0 - $1/\omega$ plane

Different combinations of R_0 and $1/\omega$ fit equally well the hospitalization data.

Results



We set $\epsilon\beta w = RR \cdot \epsilon\beta u$ and find the best fit for ω depending on R_0 .

For many parameter combinations, there is no endemic solution (the infection goes to zero).

Conclusions

- We are unable to estimate the rate of waning immunity ω independently from transmission rate β (related to R_0) and
- Vaccine protection against infection has a large effect, even for low values of protection
- One way to look at it: we have exchanged uncertainty on ω for uncertainty in R_0 and $\varepsilon\beta$
- For many parameters, immunity duration is much larger than previously expected
- A plausible interpretation is that the observed epidemics post-vaccination, with the Omega variant and descendants, is mostly due to immunological escape, not waning

Outlook

- Could we have “guessed” these results before working everything out?
- This is related to identifiability, but in practice it's hard to take everything into account, especially uncertainty in other parameters
- Model structure is important, but does not guarantee good fitting or accurate estimates
- Simpler models may help,
- but not at the cost of hiding/ignoring key information

Thank you!

Contact me at: renato.coutinho@ufabc.edu.br



Equations and variables

Completely Susceptible

$$\frac{dS}{dt} = -\lambda_1 \frac{S}{N} - \lambda_2 \frac{S}{N} \tag{1a}$$

Original Strain

$$\frac{dE_1}{dt} = \lambda_1 \frac{S_1}{N} - \frac{E_1}{\gamma_1} \tag{1b}$$

$$\frac{dA_1}{dt} = \frac{(1 - \sigma_1)\alpha_1 E_1}{\gamma_1} - \frac{A_1}{\nu_{i,1}} \tag{1c}$$

$$\frac{dI_1}{dt} = \frac{(1 - \alpha_1)(1 - \sigma_1)E_1}{\gamma_1} - \frac{I_1}{\nu_{i,1}} \tag{1d}$$

$$\frac{dH_1}{dt} = \frac{\sigma_1 E_1}{\gamma_1} - \frac{H_1}{\nu_{s,1}} \tag{1e}$$

$$\frac{dR_1}{dt} = \frac{A_1}{\nu_{i,1}} + \frac{I_1}{\nu_{i,1}} + \frac{(1 - \mu_1)H_1}{\nu_{s,1}} - p_r \lambda_2 \frac{R_1}{N} \tag{1f}$$

$$\frac{dD_1}{dt} = \frac{\mu_1 H_1}{\nu_{s,1}} \tag{1g}$$

P.1 Strain

$$\frac{dE_2}{dt} = \lambda_2 \frac{S_2}{N} - \frac{E_2}{\gamma_2} + p_r \lambda_2 \frac{R_1}{N} \tag{1h}$$

$$\frac{dA_2}{dt} = \frac{(1 - \sigma_2)\alpha_2 E_2}{\gamma_2} - \frac{A_2}{\nu_{i,2}} \tag{1i}$$

$$\frac{dI_2}{dt} = \frac{(1 - \alpha_2)(1 - \sigma_2)E_2}{\gamma_2} - \frac{I_2}{\nu_{i,2}} \tag{1j}$$

$$\frac{dH_2}{dt} = \frac{\sigma_2 E_2}{\gamma_2} - \frac{H_2}{\nu_{s,2}} \tag{1k}$$

$$\frac{dR_2}{dt} = \frac{A_2}{\nu_{i,2}} + \frac{I_2}{\nu_{i,2}} + \frac{(1 - \mu_2)H_2}{\nu_{s,2}} \tag{1l}$$

$$\frac{dD_2}{dt} = \frac{\mu_2 H_2}{\nu_{s,2}} \tag{1m}$$

Parameter	Description	Value	Source
γ	Average time in days between being infected and developing symptoms	5.8	Wei et al 2020 (13)
ν_i	Average time in days between being infectious and recovering for asymptomatic and mild cases	9	Cevik et al 2021 (2)
ν_s	Average time between being infectious and recovering/dying for severe cases	8.4	SIVEP-Gripe for the São Paulo State
ξ_i	Reduction on the exposure of symptomatic cases (due to symptoms/quarantining)	0.1	Assumed
ξ_s	Reduction on the exposure of severe cases (due to hospitalization)	0.9	Assumed
ω	Relative infectiousness of pre-symptomatic individuals	1	Assumed
α	Proportion of asymptomatic cases	[0.67,0.44,0.31]	Young Adult and Elderly (3) (12)
σ	Proportion of symptomatic cases that require hospitalization	[0.001, 0.012, 0.089] *	Salje et al. (10)
μ	In-hospital mortality ratio	[0.417,0.188,0.754]	Portella et al. (7)
χ	Case report probability	1.0	Assumed

$$\lambda_k = \beta_k \hat{C}[A_k + \omega E_k + (1 - \xi)I_k + (1 - \xi_{sev})H_k]$$