

1 **Structure in the variability of the basic reproductive number**
2 **(R_0) for Zika epidemics in the Pacific islands**

3 Clara Champagne ^{*1,2}, David Georges Salthouse¹, Richard Paul^{3,4}, Van-Mai
4 Cao-Lormeau⁵, Benjamin Roche⁶ and Bernard Cazelles ^{†1,6}

5 ¹IBENS, UMR 8197 CNRS-ENS Ecole Normale Supérieure, 46 rue d'Ulm, 75230 Paris, France

6 ²Centre de Recherche en Economie et Statistiques (CREST), 15 boulevard Gabriel Péri, 92245 Malakoff cedex, France

7 ³Institut Pasteur, Unité de Génétique Fonctionnelle des Maladies Infectieuses, Department of Genomes and Genetics,
8 F-75724 Paris cedex 15, France

9 ⁴Centre National de la Recherche Scientifique, Unité de Recherche associée 3012, Paris, France

10 ⁵Unit of Emerging Infectious Diseases, Institut Louis Malardé, 98713 Papeete, Tahiti, French Polynesia

11 ⁶International Center for Mathematical and Computational Modeling of Complex Systems (UMMISCO), UMI 209
12 UPMC/IRD, Bondy cedex, France

*champagn@biologie.ens.fr

†cazelles@biologie.ens.fr

13 **Abstract**

14 Before the outbreak that reached the Americas in 2015, Zika virus (ZIKV) circulated in
15 Asia and the Pacific: these past epidemics can be highly informative on the key parameters
16 driving virus transmission, such as the basic reproduction number (R_0). We compare
17 two compartmental models with different mosquito representations, using surveillance
18 and seroprevalence data for several ZIKV outbreaks in Pacific islands (Yap, Micronesia
19 2007, Tahiti and Moorea, French Polynesia 2013-2014, New Caledonia 2014). Models are
20 estimated in a stochastic framework with state-of-the-art Bayesian techniques. R_0 for the
21 Pacific ZIKV epidemics is estimated between 1.5 and 4.1, the smallest islands displaying
22 higher and more variable values. This relatively low range of R_0 suggests that intervention
23 strategies developed for other flaviviruses should enable as, if not more effective control
24 of ZIKV. Our study also highlights the importance of seroprevalence data for precise
25 quantitative analysis of pathogen propagation, to design prevention and control strategies.

26 In May 2015, the first local cases of Zika were recorded in Brazil and by December of the
27 same year the number of cases had surpassed 1.5 million. On February 2016, the World
28 Health Organization declared Zika as a public health emergency of international concern¹
29 and in March 2016, local transmission of Zika was recognized in 34 countries. Previously
30 the Zika virus had circulated in Africa and Asia but only sporadic human cases had been
31 reported. In 2007 the outbreak on Yap (Micronesia) was the first Zika outbreak outside
32 Africa and Asia.² Since, Zika outbreaks have been also reported in French Polynesia and
33 in New Caledonia^{3;4} between 2013 and 2014 and subsequently, there have been cases of
34 Zika disease in the Cook Islands, the Solomon Islands, Samoa, Vanuatu, and Easter Island
35 (Chile) (see Fig. 1 in reference⁵).

36
37 Zika virus (ZIKV) is a flavivirus, mostly transmitted via the bites of infected *Aedes*
38 mosquitoes, although non-vector-borne transmission has been documented (sexual and
39 maternofetal transmission, laboratory contamination, transmission through transfusion).⁶
40 The most common clinical manifestations include rash, fever, arthralgia, and conjunctivitis⁶
41 but a large proportion of infections are asymptomatic or trigger mild symptoms that can
42 remain unnoticed. Nevertheless, the virus may be involved in many severe neurological
43 complications, including Guillain-Barre syndrome⁷ and microcephaly in newborns.⁸ These
44 complications and the impressive speed of its geographically propagation make the Zika
45 pandemic a public health threat.¹ This reinforces the urgent need to characterize the different
46 facets of virus transmission and to evaluate its dispersal capacity. We address this here by
47 estimating the key parameters of ZIKV transmission, including the basic reproduction
48 number (R_0), based on previous epidemics in the Pacific islands.

49
50 Defined as the average number of secondary cases caused by one typical infected individual
51 in an entirely susceptible population, the basic reproduction number (R_0) is a central
52 parameter in epidemiology used to quantify the magnitude of ongoing outbreaks and

53 it provides insight when designing control interventions.⁹ It is nevertheless complex to
54 estimate^{9;10}, and therefore, care must be taken when extrapolating the results obtained in
55 a specific setting, using a specific mathematical model. In the present study, we explore
56 the variability of R_0 using two state-of-the-art models in several settings that had Zika
57 epidemics in different years and that vary in population size (Yap, Micronesia 2007, Tahiti
58 and Moorea, French Polynesia 2013-2014, and New Caledonia 2014). These three countries
59 were successively affected by the virus, resulting in the first significant human outbreaks
60 and they differ in several ways, including population size and location specific features.
61 Hence, the comparison of their parameter estimates can be highly informative on the
62 intrinsic variability of R_0 . For each setting, we compare two compartmental models using
63 different parameters defining the mosquito populations. Both models are considered in a
64 stochastic framework, a necessary layer of complexity given the small population size and
65 state-of-the-art Bayesian inference techniques¹¹ are used for parameter estimation.

66 **Results**

67 We use mathematical transmission models and data from surveillance systems and seroprevalence
68 surveys for several ZIKV outbreaks in Pacific islands (Yap, Micronesia 2007², Tahiti and
69 Moorea, French Polynesia 2013-2014¹²⁻¹⁴, New Caledonia 2014¹⁵) to quantify the ZIKV
70 transmission variability.

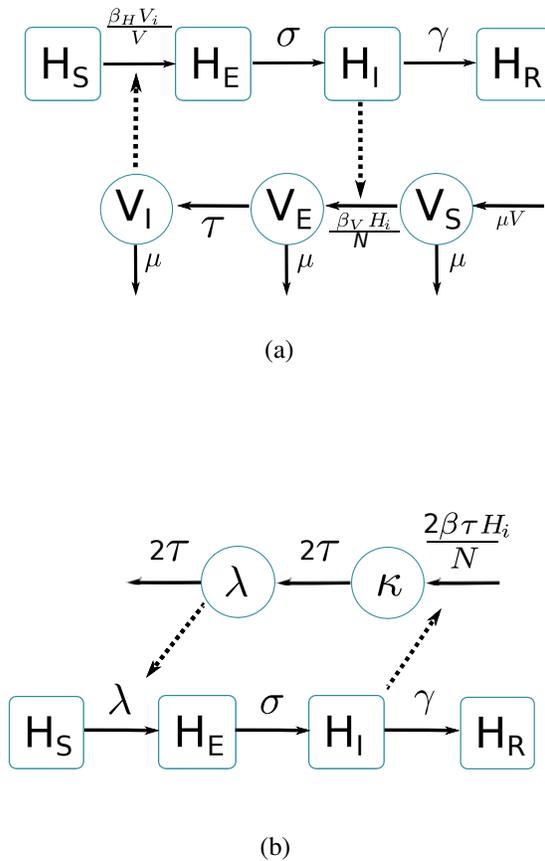


Figure 1 – Graphical representation of compartmental models. Squared boxes and circles correspond respectively to human and vector compartments. Plain arrows represent transitions from one state to the next. Dashed arrows indicate interactions between humans and vectors. a) Pandey model.¹⁶ H_S susceptible individuals; H_E infected (not yet infectious) individuals; H_I infectious individuals; H_R recovered individuals; σ is the rate at which H_E -individuals move to infectious class H_I ; infectious individuals (H_I) then recover at rate γ ; V_S susceptible vectors; V_E infected (not yet infectious) vectors; V_I infectious vectors; V constant size of total mosquito population; τ is the rate at which V_E -vectors move to infectious class V_I ; vectors die at rate μ . b) Laneri model.¹⁷ H_S susceptible individuals; H_E infected (not yet infectious) individuals; H_I infectious individuals; H_R recovered individuals; σ is the rate at which H_E -individuals move to infectious class H_I ; infectious individuals (H_I) then recover at rate γ ; implicit vector-borne transmission is modeled with the compartments κ and λ ; λ current force of infection; κ latent force of infection reflecting the exposed state for mosquitoes during the extrinsic incubation period; τ is the transition rate associated to the extrinsic incubation period.

71 Two compartmental models with vector-borne transmission are compared (cf. Figure 1).
 72 Both models use a Susceptible-Exposed-Infected-Resistant (SEIR) framework to describe
 73 the virus transmission in the human population, but differ in their representation of the
 74 mosquito population. Figure 1.a. is a schematic representation derived from Pandey et al.¹⁶

75 and formulates explicitly the mosquito population, with a Susceptible-Exposed-Infected
76 (SEI) dynamic to account for the extrinsic incubation period (time taken for viral dissemination
77 within the mosquito).

78

79 By contrast, in the second model (Figure 1.b.) based on Laneri et al.¹⁷ the vector is modeled
80 implicitly: the two compartments κ and λ do not represent the mosquito population but the
81 force of infection for vector to human transmission. This force of infection passes through
82 two successive stages in order to include the delay associated with the extrinsic incubation
83 period: κ stands for this latent phase of the force of infection whereas λ corresponds directly
84 to the rate at which susceptible humans become infected.

85

86 The basic reproduction number of the models (R_0) is calculated using the next Generation
87 Matrix method:⁹

$$R_0^{Pandey} = \sqrt{\frac{\beta_H \beta_V \tau}{\gamma \mu (\mu + \tau)}}$$

88

$$R_0^{Laneri} = \sqrt{\frac{\beta}{\gamma}}$$

89 In addition, we consider that only a fraction ρ of the total population is involved in the
90 epidemic, due to spatial heterogeneity, immuno-resistance, or cross-immunity. For both
91 models we define $N = \rho \cdot H$ with H the total size of the population reported by census.

92

93 The dynamics of ZIKV transmission in these islands is highly influenced by several sources
94 of uncertainties. In particular, the small population size (less than 7,000 inhabitants in Yap)
95 leads to high variability in transmission rates. Therefore all these models are simulated in
96 a discrete stochastic framework (Poisson with stochastic rates¹⁸), to take this phenomenon
97 into account. Stochasticity requires specific inference techniques : thus estimations are
98 performed with PMCMC algorithm (particle Markov Chain Monte Carlo¹¹).

99

100 Using declared Zika cases from different settings, the two stochastic models (Fig. 1) were
101 fitted (Figs 2-3). These models allow us to describe the course of the observed number
102 of cases and estimate the number of secondary cases generated, R_0 . Our estimates of R_0
103 lie between 1.6 (1.5-1.7) and 3.2 (2.4-4.1) and vary notably with respect to settings and
104 models (Figures 2-3 and Tables 1-2). Strikingly, Yap displays consistently higher values of
105 R_0 in both models and in general, there is an inverse relationship between island size and
106 both the value and variability of R_0 . This phenomenon may be explained by the higher
107 stochasticity and extinction probability associated with smaller populations and can also
108 reflect the information contained in the available data. However, the two highly connected
109 islands in French Polynesia, Tahiti and Moorea, display similar values despite their differing
110 sizes.

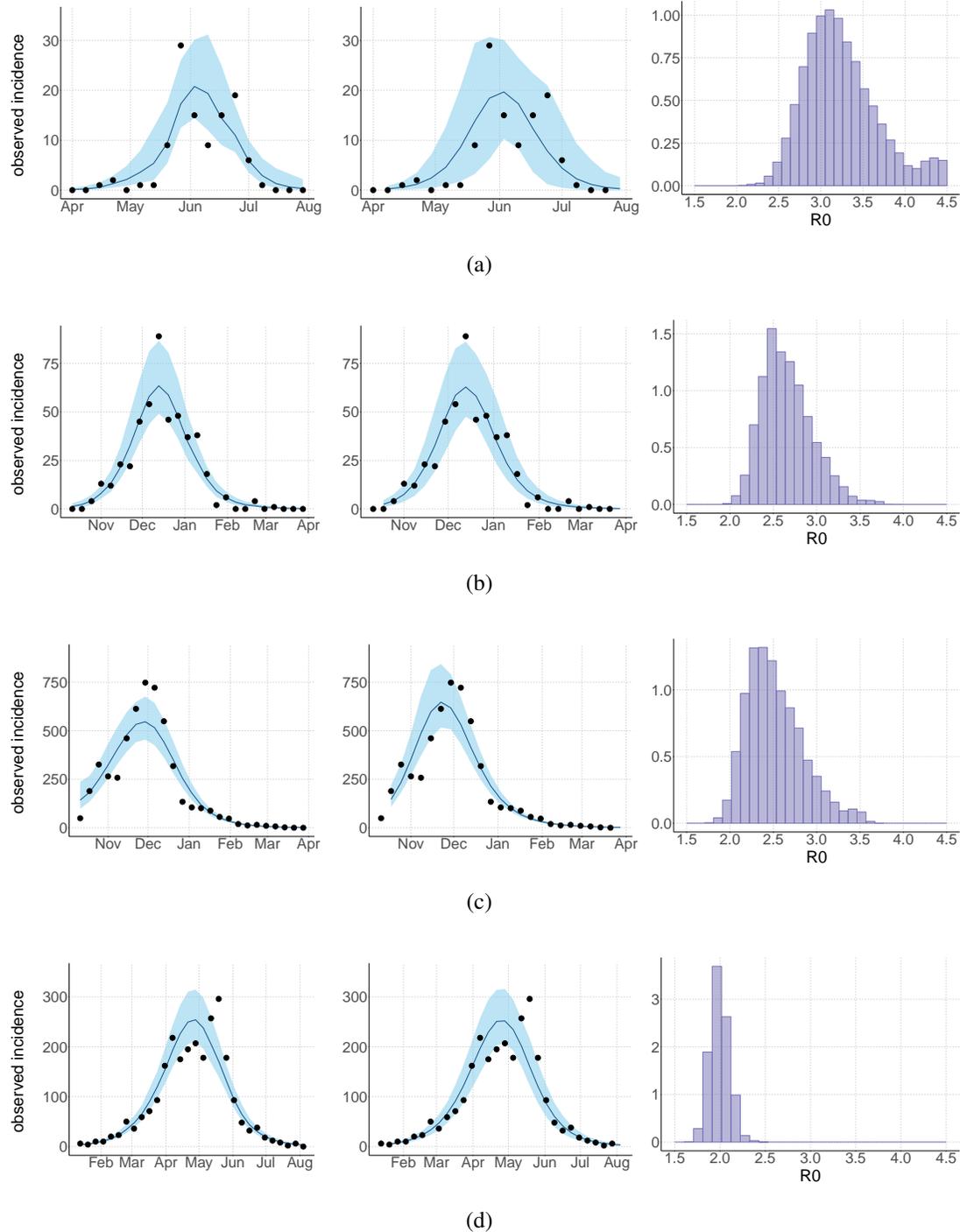


Figure 2 – Results using the Pandey model. Posterior median number of observed Zika cases (solid line), 95% credible intervals (shaded blue area) and data points (black dots). First column: particle filter fit. Second column: Simulations from the posterior density. Third column: R_0 posterior distribution. a) Yap. b) Moorea. c) Tahiti. d) New Caledonia. The estimated seroprevalences at the end of the epidemic (with 95% credibility intervals) are: a) 73% (CI95: 68-77, observed 73%); b) 49% (CI95: 45-53, observed 49%); c) 49% (CI95: 45-53, observed 49%); d) 39% (CI95: 8-92). See Figure 4.

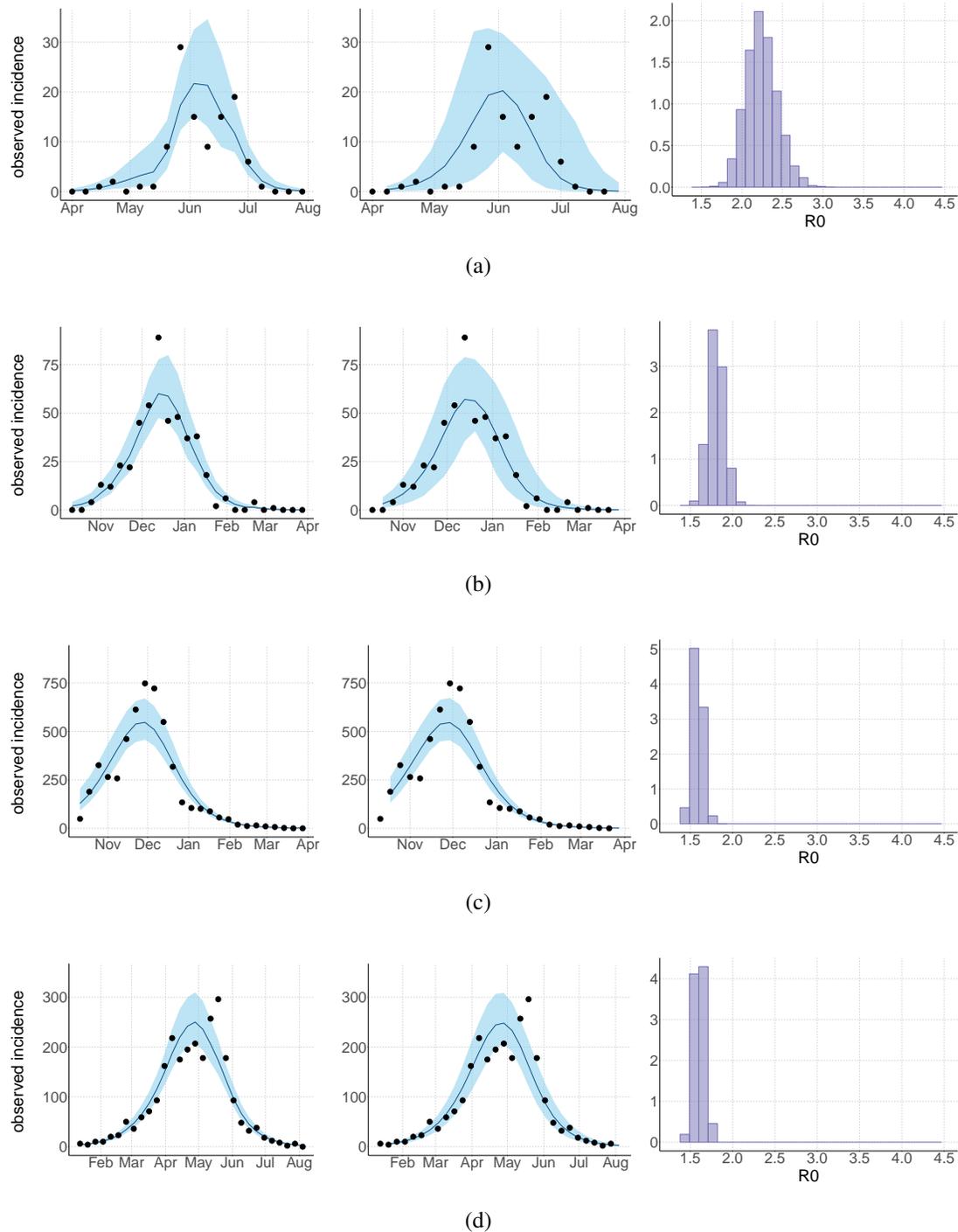


Figure 3 – Results using the Laneri model. Posterior median number of observed Zika cases (solid line), 95% credible intervals (shaded blue area) and data points (black dots). First column: particle filter fit. Second column: Simulations from the posterior density. Third column: R_0 posterior distribution. a) Yap. b) Moorea. c) Tahiti. d) New Caledonia. The estimated seroprevalences at the end of the epidemic (with 95% credibility intervals) are: a) 72% (CI95: 68-77, observed 73%); b) 49% (CI95: 45-53, observed 49%); c) 49% (CI95: 45-53, observed 49%); d) 65% (CI95: 24-91). See Figure 5.

111 Regarding model variability, R_0 estimates are always higher and coarser with the Pandey
 112 model than with the Laneri model (cf. Tables 1-2). The Pandey model has two additional
 113 estimated parameters (in particular, the mosquito lifespan), which can explain the higher
 114 variability of the output. It is worth noting that these parameters are very sensitive (see
 115 Materials and methods). The difference in R_0 may also be linked to the difference in the
 116 estimated initial number of infected individuals (H_{I0}), which is higher in the Laneri model
 117 than in the Pandey model. Because of the high proportion of asymptomatic cases (the
 118 ratio of asymptomatic:symptomatic is estimated to be 1:1.3, V.-M Cao-Lormeau personal
 119 communication), it is hard to determine which scenario is more realistic, the time between
 120 introduction of the disease into the island and the first reported symptomatic case being
 121 unknown in most settings.

PANDEY MODEL		Yap	Moorea	Tahiti	New Caledonia
Population size	H	6,892	16,200	178,100	268,767
Basic reproduction number	R_0	3.2 (2.4 – 4.1)	2.6 (2.2 – 3.3)	2.5 (2.0 – 3.3)	2.0 (1.8 – 2.2)
Observation rate	r	0.024 (0.018 – 0.031)	0.058 (0.047 – 0.071)	0.060 (0.048 – 0.072)	0.029 (0.009 – 0.10)
Fraction of population involved	ρ	74% (69-79)	50% (46-54)	50% (46-54)	34% (8-91)
Initial number of infected individuals	H_{I0}	2 (1-8)	5 (0-21)	304 (16-1145)	37 (1-386)
Infectious period in human (days)	γ^{-1}	5.2 (4.0 – 6.5)	5.2 (4.0 – 6.6)	5.2 (4.0 – 6.5)	5.4 (4.1 – 6.7)
Extrinsic incubation period in mosquito (days)	τ^{-1}	10.5 (8.6 – 12.4)	10.5 (8.6 – 12.4)	10.4 (8.5 – 12.5)	10.7 (8.9 – 12.5)
Mosquito lifespan (days)	μ^{-1}	15.4 (11.8 – 19.3)	15.3 (11.6 – 19.2)	14.9 (10.9 – 18.8)	15.3 (11.5 – 19.4)

Table 1 – Parameter estimations for the Pandey model. Posterior median (95% credible intervals). All the posterior parameter distributions are presented in Figures 6-9.

LANERI MODEL		Yap	Moorea	Tahiti	New Caledonia
Population size	H	6,892	16,200	178,100	268,767
Basic reproduction number	R_0	2.2 (1.9 – 2.6)	1.8 (1.6 – 2.0)	1.6 (1.5 – 1.7)	1.6 (1.5 – 1.7)
Observation rate	r	0.02 (0.018 – 0.032)	0.057 (0.047 – 0.071)	0.057 (0.048 – 0.068)	0.015 (0.009 – 0.033)
Fraction of population involved	ρ	73% (69 – 78)	51% (46 – 54)	54% (49 – 59)	70% (31 – 100)
Initial number of infected individuals	Hi_0	2 (1 – 10)	9 (1 – 28)	667 (22 – 1570)	82 (2 – 336)
Infectious period in human (days)	γ^{-1}	5.3 (4.0 – 6.6)	5.2 (4.0 – 6.6)	5.1 (4.0 – 6.5)	5.4 (4.0 – 6.7)
Extrinsic incubation period in mosquito (days)	τ^{-1}	10.6 (8.8 – 12.7)	10.6 (8.6 – 12.6)	10.5 (8.5 – 12.4)	10.8 (8.9 – 12.7)

Table 2 – Parameter estimations for the Laneri model. Posterior median (95% credible intervals). All the posterior parameter distributions are presented in Figures 10-13.

122 For the duration of infectious and intrinsic incubation (in human) and extrinsic incubation
 123 (in mosquito) periods, the posterior density resembles the informative prior (cf. Figures
 124 6-13), indicating the models' incapacity to identify properly these parameters without more
 125 informative data. Moreover, these parameters have a clear sensitivity (see Materials and
 126 methods) and precise field measures are therefore crucial for reliable model predictions.

127

128 The fraction ρ of the population involved in the epidemic and the observation rate r display
 129 very large credible intervals when seroprevalence is unknown (New Caledonia). They are
 130 highly correlated with one another (cf. Figures 17 and 21) and therefore unidentifiable
 131 without precise information on seroprevalence.

132 Discussion

133 The reproduction number R_0 is a key parameter in epidemiology that characterizes the
 134 epidemic dynamics and the initial spread of the pathogen at the start of an outbreak in a
 135 susceptible population. R_0 can be used to inform public health authorities on the level
 136 of risk posed by an infectious disease, vaccination strategy, and the potential effects of
 137 control interventions¹⁹. In the light of the potential public health crisis generated by the
 138 international propagation of ZIKV, characterization of the potential transmissibility of this

139 pathogen is crucial for predicting epidemic size, rate of spread and efficacy of intervention.

140

141 Using data from both surveillance systems and seroprevalence surveys in four different
142 geographical settings across the Pacific,^{2;12-15} we have estimated the basic reproductive
143 number R_0 (see Figs 2-3 and Tables 1-2). Our estimate of R_0 obtained by inference based
144 on Particle MCMC¹¹ has values in the range 1.6 (1.5-1.7) - 3.2 (2.4-4.1). Our R_0 estimates
145 vary notably across settings. Lower and finer R_0 values are found in larger islands. This
146 phenomenon can at least in part be explained by large spatial heterogeneities and higher
147 demographic stochasticity for islands with smaller populations, as well as the influence
148 of stochasticity on biological and epidemiological processes linked to virus transmission.
149 This phenomenon can also be specific to the selection of the studied island or can reflect
150 a highly clustered geographical pattern, the global incidence curve being the smoothed
151 overview of a collection of more explosive small size outbreaks. However, it is notable that
152 the two French Polynesian islands yield similar estimates of R_0 despite differing population
153 sizes. Indeed, other important factors differ among French Polynesia, New Caledonia and
154 Yap, such as the human genetic background and their immunological history linked to
155 the circulation of others arboviruses. Moreover, whilst both New Caledonia and French
156 Polynesia populations were infected by the same ZIKV lineage and transmitted by the same
157 principle vector species, *Aedes aegypti*, the epidemic in Yap occurred much earlier with a
158 different ZIKV lineage²⁰ and vectored by a different mosquito species *Aedes hensilli*.²¹ In
159 French Polynesia, the vector *Aedes polynesiensis* is also present and dominates in Moorea
160 with higher densities than in Tahiti. Finally, different vector control measures have been
161 conducted in the three countries.

162

163 To date, studies investigating Zika outbreaks in the Pacific have always estimated R_0 using a
164 deterministic framework. Using a similar version of the Pandey model in French Polynesia,
165 Kucharski et al.²² estimated R_0 between 1.6 and 2.3 (after scaling to square root for

166 comparison) for Tahiti and between 1.8 and 2.9 in Moorea. These estimates are slightly
167 lower and less variable than ours. This difference can be explained firstly by the chosen
168 priors on mosquito parameters and secondly because our model includes demographic
169 stochasticity. Moreover, they predicted a seroprevalence rate at the end of the epidemic
170 of 95-97%, far from the 49% measured. In Yap island, a study²³ used a very detailed
171 deterministic mosquito model, and estimated an R_0 for Zika between 2.9 and 8. In this
172 case, our lower and less variable estimates may come from the fact that our model is more
173 parsimonious in the number of uncertain parameters, especially concerning the mosquito
174 population. Finally, a third study²⁴ relied on another method for R_0 calculation (based only
175 on the early exponential growth rate of the epidemic) in French Polynesia as a whole and
176 in Yap. Again, the obtained parameters are lower than ours in French Polynesia and higher
177 in Yap. In all these studies a deterministic framework is used excluding the possibility of
178 accounting for the high variability of biological and epidemiological processes exacerbated
179 by the small size of the population. In these three studies, like in ours, it is worth noting
180 that little insight is obtained regarding mosquito parameters. Posterior distribution mimics
181 the chosen prior (cf. Figures 6-13). Both the simulation of the epidemics and the estimated
182 R_0 are highly sensitive to the choice of priors on mosquito parameters, for which precise
183 field measures are rare.

184

185 In the absence of sufficient data, the modeling of mosquito-borne pathogen transmission
186 is a difficult task due to non-linearity and non-stationarity of the involved processes.²⁵
187 This work has then several limitations. First, our study is limited by the completeness and
188 quality of the data, with regard to both incidence and seroprevalence, but, above all, by the
189 scarcity of information available on mosquitoes. Incidence data is aggregated at the island
190 scale and cannot disentangle the effects of geography and observation noise to explain
191 bimodal curves observed in Yap and New Caledonia. Moreover, although all data came
192 from national surveillance systems, we had very little information about the potential degree

193 of under-reporting. Seroprevalence data were gathered from small sample sizes and were
194 missing in New Caledonia, which leads to strong correlation between the observation rate
195 and the fraction of the population involved in the epidemic. Because of the high proportion
196 of asymptomatic or mildly symptomatic cases, the magnitude of the outbreaks is difficult
197 to evaluate without precise seroprevalence data²⁶ or detection of mild, asymptomatic or
198 pre-symptomatic infections.²⁷ Considering vectors, no demographic data were available
199 and this partly explains the large variability of our R_0 estimations.

200 Secondly, incidence and seroprevalence data were difficult to reconcile; the use of incidence
201 data led to higher infection rates than those observed in the seroprevalence data. This
202 difficulty has been overcome by considering that only a fraction of the population (ρ)
203 is involved in the epidemic and then our model manages to reproduce the observed
204 seroprevalence rate. This exposed fraction could be the result of spatial heterogeneity and
205 high clustering of cases and transmission, as observed for dengue. Finer scale incidence
206 and seroprevalence data would be useful to explore this. Another explanation for higher
207 predicted than observed infection rates could be due to interaction with other flaviviruses.
208 The Zika outbreak was concomitant with dengue outbreaks in French Polynesia^{12;13} and
209 New Caledonia.¹⁵ Examples of coinfection have been reported⁴ but competition between
210 these close pathogens may also have occurred. Finally, mathematical models with vectorial
211 transmission may tend to estimate high attack rates, sometimes leading to a contradiction
212 between observed incidence and observed seroprevalence. Assumptions on the proportionality
213 between infected mosquitoes and the force of infection, as well as the density-dependence
214 assumption in these models could be questioned. Indeed even if these assumptions are at the
215 heart of the mathematical models of mosquito-borne pathogen transmission^{28;29} a recent
216 review,³⁰ and recent experimental results^{31;32} question these important points.

217

218 On a final note, the estimates of R_0 for ZIKV are similar to but generally on the lower side of
219 estimates made for two other flaviviruses of medical importance, dengue and Yellow Fever

220 viruses^{33–35}, even though caution is needed in the comparison of studies with differing
221 models, methods and data sources. Interventions strategies developed for dengue should
222 thus enable as, if not more effective control of ZIKV, with the caveat that ZIKV remains
223 principally a mosquito-borne pathogen with little epidemiological significance of the sexual
224 transmission route.

225

226 In conclusion, using state-of-the art stochastic modeling methods, we have been able to
227 determine estimates of R_0 for ZIKV with an unexpected relationship with population
228 size. Further data from the current Zika epidemic in South America that is caused by
229 the same lineage as French Polynesia will enable us to confirm this relationship. Our
230 study highlights the importance of gathering seroprevalence data, especially for a virus
231 that often leads to an asymptomatic outcome and it would provide a key component for
232 precise quantitative analysis of pathogen propagation to enable improved planning and
233 implementation of prevention and control strategies.

234 **Materials and methods**

235 **Data**

236 During the 2007 outbreak that struck Yap, 108 suspected or confirmed Zika cases (16 per
237 1,000 inhabitants) were reported by reviewing medical records and conducting prospective
238 surveillance between April 1st and July 29th 2007.² In French Polynesia, sentinel surveillance
239 recorded more than 8,700 suspected cases (32 per 1,000 inhabitants) across the whole
240 territory between October 2013 and April 2014.^{12;13} In New Caledonia, the first Zika
241 case was imported from French Polynesia on 2013 November 12th. Approximately 2,500
242 cases (9 per 1,000 inhabitants) were reported through surveillance between January (first
243 autochthonous case) and August 2014.¹⁵

244

245 For Yap and French Polynesia, the post-epidemic seroprevalence was assessed. In Yap, a
246 household survey was conducted after the epidemic, yielding an infection rate in the island
247 of 73%.² In French Polynesia, three seroprevalence studies were conducted. The first one
248 took place before the Zika outbreak, and concluded that most of the population was naive
249 for Zika virus.³⁶ The second seroprevalence survey was conducted between February and
250 March 2014, at the end of the outbreak, and reported a seroprevalence rate around 49%.¹⁴
251 The third one concerned only schoolchildren in Tahiti and was therefore not included in the
252 present study.

253

254 Demographic data on population size were based on censuses from Yap², French Polynesia³⁷,
255 and New Caledonia.³⁸

256 **Models and inference**

257 **Model equations**

258 Although the models are simulated in a stochastic framework, we present them with ordinary
259 differential equations for clarity. The reactions involved in the stochastic models are the
260 same as those governed by the deterministic equations, but the simulation process differs
261 through the use of discrete compartments. It is described in the next section.

262 The equations describing Pandey model are:

$$\begin{aligned}\frac{dH_S}{dt} &= -\beta_H v_I H_S \\ \frac{dH_E}{dt} &= \beta_H v_I H_S - \sigma H_E \\ \frac{dH_I}{dt} &= \sigma H_E - \gamma H_I \\ \frac{dH_R}{dt} &= \gamma H_I \\ \frac{dv_S}{dt} &= \mu - \frac{\beta_V H_I}{N} v_S - \mu v_S \\ \frac{dv_E}{dt} &= \frac{\beta_V H_I}{N} v_S - \tau v_E - \mu v_E \\ \frac{dv_I}{dt} &= \tau v_E - \mu v_I\end{aligned}$$

263 where $v_S = \frac{V_S}{V}$ is the proportion of susceptible mosquitoes, $v_E = \frac{V_E}{V}$ the proportion of
264 exposed mosquitoes, and $v_I = \frac{V_I}{V}$ the proportion of infected mosquitoes. Since we are
265 using a discrete model, we cannot use directly the proportions v_S , v_E and v_I whose values
266 are smaller than one. Therefore, we rescale using $V = H$, which leads to $V'_S = v_S \cdot H$,
267 $V'_E = v_E \cdot H$, and $V'_I = v_I \cdot H$.

268 In this model, the force of infection for humans is $\lambda_H = \beta_H v_I$, and the force of infection for
269 mosquitoes is $\lambda_V = \beta_V \frac{H_I}{N}$

270

271 The equations describing Laneri model are:

$$\begin{aligned}\frac{dH_S}{dt} &= -\lambda H_S \\ \frac{dH_E}{dt} &= \lambda H_S - \sigma H_E \\ \frac{dH_I}{dt} &= \sigma H_E - \gamma H_I \\ \frac{dH_R}{dt} &= \gamma H_I \\ \frac{d\kappa}{dt} &= \frac{2\beta H_I \tau}{N} - 2\tau\kappa \\ \frac{d\lambda}{dt} &= 2\tau\kappa - 2\tau\lambda\end{aligned}$$

272 In this model, the role of mosquitoes in transmission is represented only through the
273 delay they introduce during the extrinsic incubation period (EIP, incubation period in the
274 mosquito). For modeling reasons, this delay is included by representing the force of
275 infection from infected humans to susceptible humans with two compartments κ and λ :
276 in this formalism, the duration between the moment when an exposed individual becomes
277 infectious and the moment when another susceptible individual acquires the infection has a
278 gamma distribution of mean τ^{-1} .^{17;39;40} Therefore, λ represents the current force of infection
279 for humans $\lambda_H = \lambda$. The compartment κ represents the same force of infection but at a
280 previous stage, reflecting the exposed phase for mosquitoes during the extrinsic incubation
281 period. As an analogy to Pandey model, the force of infection for mosquitoes is $\lambda_V = \frac{\beta H_I \tau}{v_s N}$,
282 and therefore, the parameter β can be interpreted as the product of a transmission parameter
283 β' by the proportion of susceptible mosquitoes: $\beta = v_s \beta'$. The force of infection for
284 mosquitoes is then similar to Pandey's: $\lambda_V = \beta' \tau \frac{H_I}{N}$.
285 Again, since we are using a discrete model, we cannot use directly the proportions λ and κ
286 whose values are smaller than one. Therefore, we rescale up to a factor N , which leads to
287 $L = \lambda N$ and $K = \kappa N$.

288

289 Stochastic framework

290 Both models are simulated in a stochastic and discrete framework, the Poisson with
 291 stochastic rates formulation,¹⁸ to include the uncertainties related to small population size.
 292 In this framework, the number of reactions occurring in a time interval dt is approximated
 293 by a multinomial distribution. In a model with m possible reactions and c compartments,
 294 z_t being the state of the system at time t and θ the model parameters, the probability that
 295 each reaction r^k occurs n_k times in dt is calculated as follows:⁴¹

$$p(n_1, \dots, n_m | z_t, \theta) = \prod_{i=1}^c \left\{ M_i \left(1 - \sum_{X(k)=i} p_k \right)^{\bar{n}_i} \prod_{X(k)=i} (p_k)^{n_k} \right\} + o(dt)$$

296 with, $z_t^{(i)}$ being the number of individual in compartment i at time t ,

297 • $p_k = \left(1 - \exp - \sum_{X(l)=i} r^l(z_t, \theta) z_t^{X(l)} dt \right) \frac{r^k(z_t, \theta)}{\sum_{X(l)=i} r^l(z_t, \theta)}$

298

299 • $\bar{n}_i = z_t^{(i)} - \sum_{X(k)=i} n_k$ (the number of individuals staying in compartment i in dt)

300

301 • $M_i = \binom{z_t^{(i)}}{n_k}_{X(k)=i} \bar{n}_i$ (multinomial coefficient)

302 Observation models

303 The only observed compartments are the infected humans (incidence measured every week)
 304 and the recovered humans (seroprevalence at the end of the outbreak when data is available).
 305 In order to link the model to the data, two observation models, for both incidence and
 306 seroprevalence data, are needed.

307

308 **Observation model on incidence data**

309 The observed weekly incidence is assumed to follow a negative binomial distribution¹⁸
310 whose mean equals the number of new cases predicted by the model times an estimated
311 observation rate r .

312

313 The observation rate r accounts for non observed cases, due to non reporting from medical
314 centers, mild symptoms unseen by health system, and asymptomatic infections. Without
315 additional data, it is not possible to make a distinction between these three categories of
316 cases. We also implicitly make the assumption that these cases transmit the disease as
317 much as reported symptomatic cases.

318

319 The observation model for incidence data is therefore :

$$Inc_{obs} = NegBin(\phi^{-1}, \frac{1}{1 + \phi r Inc})$$

320 Inc_{obs} being the observed incidence, and Inc the incidence predicted by the model. The
321 dispersion parameter¹⁸ ϕ is fixed at 0.1.

322 **Observation model on seroprevalence data**

323 Seroprevalence data is fitted for Tahiti, Moorea, and Yap settings. It is assumed that the
324 observed seroprevalence at the end of the epidemic follows a normal distribution with fixed
325 standard deviation, whose mean equals the number of individuals in the H_R compartment
326 predicted by the model.

327

328 The observation model for seroprevalence data is therefore :

$$H_R^{obs} = Normal(H_R, \Lambda)$$

329 at the last time step, with notations detailed for each model in Table 3.

Island	Date	Standard deviation Λ	Observed seroprevalence H_R^{obs}
Yap	2007-07-29	150	5005 ²
Moorea	2014-03-28	325	$0.49 \times 16200 = 7938$ ¹⁴
Tahiti	2014-03-28	3562	$0.49 \times 178100 = 87269$ ¹⁴

Table 3 – Details of the observation models for seroprevalence

330 **Prior distributions**

331 Informative prior distributions are assumed for the mosquito lifespan, the duration of
332 infectious period, and both intrinsic and extrinsic incubation periods. The initial numbers of
333 infected mosquitoes and humans are estimated, and the initial number of exposed individuals
334 is set to the initial number of infected to reduce parameter space. We assume that involved
335 populations are naive to Zika virus prior to the epidemic and set the initial number of
336 recovered humans to zero. The other priors and associated references are listed in Table 4.

337

Parameters		Pandey model	Laneri model	References		
R_0^2	squared basic reproduction number	Uniform[0, 20]	Uniform[0, 20]	assumed		
β_V	transmission from human to mosquito	Uniform[0, 10]	.	assumed		
γ^{-1}	infectious period (days)	Normal(5, 1) in [4, 7]	Normal(5, 1) in [4, 7]	13		
σ^{-1}	intrinsic incubation period (days)	Normal(4, 1) in [2, 7]	Normal(4, 1) in [2, 7]	42-44		
τ^{-1}	extrinsic incubation period (days)	Normal(10.5, 1) in [4, 20]	Normal(10.5, 1) in [4, 20]	45;46		
μ^{-1}	mosquito lifespan (days)	Normal(15, 2) in [4, 30]	.	47;48		
ρ	fraction of population involved	Uniform[0, 1]	Uniform[0, 1]			
Initial conditions (t=0)		Pandey model	Laneri model			
H_{I0}	Infected humans	Uniform[10^{-6} , 1]·N	Uniform[10^{-6} , 1]·N			
H_{E0}	exposed humans	H_{I0}	H_{I0}			
H_{R0}	Recovered humans	0	0			
	Infected vectors	$V_{I0} = \text{Uniform}[10^{-6}, 1] \cdot H$	$L_0 = \text{Uniform}[10^{-6}, 1] \cdot N$			
	exposed vectors	$V_{E0} = V_{I0}$	$K_0 = L_0$			
Local conditions		Yap	Moorea	Tahiti	New Caledonia	References
r	Observation rate	Uniform[0, 1]	Uniform[0, 1]	Uniform[0, 0.3]	Uniform[0, 0.23]	13;15;49
H	Population size	6,892	16,200	178,100	268,767	2;37;38

Table 4 – **Prior distributions of parameters.** "Uniform[0,20]" indicates a uniform distribution between in the range [0,20]. "Normal(5,1) in [4,7]" indicates a normal distribution with mean 5 and standard deviation 1, restricted to the range [4,7].

338 The range for the prior on observation rate is reduced for Tahiti and New Caledonia, in
339 order to reduce the parameter space and facilitate convergence. In both cases, we use the
340 information provided with the data source. In French Polynesia, 8,750 cases we reported,
341 but according to local health authorities, more than 32,000 people would have attended
342 health facilities for Zika¹³ ($8750/32000 \leq 0.3$). In New Caledonia, approximately 2,500
343 cases were reported but more than 11,000 cases were estimated by health authorities⁴⁹
344 ($2500/11000 \leq 0.23$). In both cases, these extrapolations are lower bounds on the real
345 number of cases (in particular, they do not estimate the number of asymptomatic infections),
346 and therefore can be used as upper bounds on the observation rate.

347 **Estimations**

348 **Inference with PMCMC**

349 The complete model is represented using the state space framework, with two equation
350 systems: the transition equations refer to the transmission models, and the measurement
351 equations are given by the observation models.

352

353 In a deterministic framework, this model could be directly estimated using MCMC, with a
354 Metropolis-Hastings algorithm targeting the posterior distribution of the parameters. This
355 algorithm would require the calculation of the model likelihood at each iteration.

356

357 In our stochastic framework, the model output is given only through simulations and the
358 likelihood is intractable. In consequence, estimations are performed with the PMCMC
359 algorithm (particle Markov Chain Monte Carlo¹¹), in the PMMH version (particle marginal
360 Metropolis-Hastings). This algorithm uses the Metropolis-Hastings structure, but replaces
361 the real likelihood by its estimation with Sequential Monte Carlo (SMC).

Algorithm 1 PMCMC¹¹ (PMMH version, as in SSM⁴¹)

In a model with n observations and J particles.

$q(\cdot|\theta^{(i)})$ is the transition kernel.

- 1: Initialize $\theta^{(0)}$.
 - 2: Using SMC algorithm, compute $\hat{p}(y_{1:n}|\theta^{(0)})$ and sample $x_{0:n}^*$ from $\hat{p}(x_{0:n}|y_{1:n}, \theta^{(0)})$.
 - 3: **for** $i = 1 \dots N^\theta$ **do**
 - 4: Sample θ^* from $q(\cdot|\theta^{(i)})$
 - 5: Using SMC algorithm, compute $L(\theta^*) = \hat{p}(y_{1:n}|\theta^*)$ and sample $x_{0:n}^*$ from $\hat{p}(x_{0:n}|y_{1:n}, \theta^*)$
 - 6: Accept θ^* (et $x_{0:n}^*$) with probability $1 \wedge \frac{L(\theta^{(i)})q(\theta^*)}{L(\theta^*)q(\theta^{(i)})}$
 - 7: If accepted, $\theta^{(i+1)} = \theta^*$ and $x_{0:n}^{(i+1)} = x_{0:n}^*$. Otherwise $\theta^{(i+1)} = \theta^{(i)}$ and $x_{0:n}^{(i+1)} = x_{0:n}^{(i)}$.
 - 8: **end for**
-

362 SMC⁵⁰ is a filtering method that enables to recover the latent variables and estimate the
363 likelihood for a given set of parameters. The data is treated sequentially, by adding one more
364 data point at each iteration. The initial distribution of the state variables is approximated
365 by a sample a particles, and from one iteration to the next, all the particles are projected
366 according to the dynamic given by the model. The particles receive a weight according to
367 the quality of their prediction regarding the observations. Before the next iteration, all the
368 particles are resampled using these weights, in order to eliminate low weight particles and
369 concentrate the computational effort in high probability regions. Model likelihood is also
370 computed sequentially at each iteration^{41;51}.

Algorithm 2 SMC (*Sequential Monte Carlo*, as implemented in SSM⁴¹)

In a model with n observations and J particles.

L is the model likelihood $p(y_{1:T}|\theta)$. $W_k^{(j)}$ is the weight and $x_k^{(j)}$ is the state associated to particle j at iteration k .

- 1: Set $L = 1$, $W_0^{(j)} = 1/J$.
 - 2: Sample $(x_0^{(j)})_{j=1:J}$ from $p(x|\theta_0)$.
 - 3: **for** $k = 0 : n - 1$ **do**
 - 4: **for** $j = 0 : J$ **do**
 - 5: Sample $(x_{k+1}^{(j)})_{j=1:J}$ from $p(x_{k+1}|x_k, \theta)$
 - 6: Set $\alpha^{(j)} = h(y_{k+1}, x_{k+1}^{(j)}, \theta)$
 - 7: **end for**
 - 8: Set $W_{k+1}^{(j)} = \frac{\alpha^{(j)}}{\sum_{l=1}^J \alpha^{(l)}}$ and $L = L \frac{1}{J} \sum_j \alpha^{(j)}$
 - 9: Resample $(x_{0:k+1}^{(j)})_{j=1:J}$ from $W_{k+1}^{(j)}$
 - 10: **end for**
-

371 A gaussian kernel $q(\cdot|\theta^{(i)})$ is used in the PMCMC algorithm, with mean $\theta^{(i)}$ and fixed
372 variance Σ^q (random walk Metropolis Hastings).

373 **Initialization**

374 PMCMC algorithm is very sensitive to initialization of both the parameter values $\theta^{(0)}$ and
375 the covariance matrix Σ^q . Several steps of initialization are therefore used.

376

377 Firstly, parameter values are initialized by maximum likelihood through simplex algorithm
378 on a deterministic version of the model. We apply the simplex algorithm to a set of 1000
379 points sampled in the prior distributions and we select the parameter set with the highest
380 likelihood.

381

382 Secondly, in order to initialise the covariance matrix, an adaptative MCMC (Metropolis
383 Hastings) framework is used^{41;52}. It uses the empirical covariance of the chain $\Sigma^{(i)}$, and
384 aims to calibrate the acceptance rate of the algorithm to an optimal value. The transition
385 kernel is also mixed (with a probability $\alpha = 0.05$) with another gaussian using the identity
386 matrix to improve mixing properties.

387

$$q^A(\cdot|x^{(i)}) = \alpha N(x^{(i)}, \lambda \frac{2.38^2}{d} Id) + (1 - \alpha) N(x^{(i)}, \frac{2.38^2}{d} \Sigma^{(i)})$$

388 The parameter λ is approximated by successive iterations using the empirical acceptance
389 rate of the chain.

$$\lambda_{i+1} = \lambda_i a^i (AccRate_i - 0.234)$$

390

391

392 The adaptative PMCMC algorithm itself may have poor mixing properties without initialization.
393 A first estimation of the covariance matrix is computed using KMCMC algorithm.⁴¹
394 In the KMCMC algorithm, the model is simulated with stochastic differential equations
395 (intermediate between deterministic and Poisson with stochastic rates frameworks) and the
396 SMC part of the adaptative PMCMC is replaced by the extended Kalman filter. When
397 convergence is reached with KMCMC, then, adaptative PMCMC is used.

398

399 The PMCMC algorithm is finally applied on the output of the adaptative PMCMC, using
400 50,000 iterations and 10,000 particles. Calculations are performed with SSM software⁴¹
401 and R version 3.2.2.

402 **R_0 calculation**

403 R_0 is calculated using the Next Generation Matrix approach⁹ (NGM).

404

405 **R_0 calculation in Pandey model**

$$F = \begin{pmatrix} 0 & 0 & 0 & \beta_H \\ 0 & 0 & 0 & 0 \\ 0 & \beta_V & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \quad V = \begin{pmatrix} -\sigma & 0 & 0 & 0 \\ \sigma & -\gamma & 0 & 0 \\ 0 & 0 & -(\mu + \tau) & 0 \\ 0 & 0 & \tau & -\mu \end{pmatrix}$$

406 Then we have,

$$V^{-1} = \begin{pmatrix} -1/\sigma & 0 & 0 & 0 \\ -1/\gamma & -1/\gamma & 0 & 0 \\ 0 & 0 & -1/(\mu + \tau) & 0 \\ 0 & 0 & -\tau/[\mu(\tau + \mu)] & -1/\mu \end{pmatrix}$$

407 and

$$FV^{-1} = \begin{pmatrix} 0 & 0 & -\beta_H\tau/[\mu(\tau + \mu)] & -\beta_H/\mu \\ 0 & 0 & 0 & 0 \\ -\beta_V/\gamma & -\beta_V/\gamma & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

408 We calculate the eigen values α of $-FV^{-1}$:

$$\begin{vmatrix} -\alpha & 0 & \beta_H\tau/[\mu(\tau + \mu)] & \beta_H/\mu \\ 0 & -\alpha & 0 & 0 \\ \beta_V/\gamma & \beta_V/\gamma & -\alpha & 0 \\ 0 & 0 & 0 & -\alpha \end{vmatrix} = \alpha^2 \left(\alpha^2 - \frac{\beta_H\beta_V\tau}{\gamma\mu(\tau + \mu)} \right) = 0$$

409 Then $\alpha = 0$ or $\alpha = \pm \sqrt{\frac{\beta_H\beta_V\tau}{\gamma\mu(\tau + \mu)}}$ and the highest eigen value is $R_0 = \sqrt{\frac{\beta_H\beta_V\tau}{\gamma\mu(\tau + \mu)}}$.

410

411 This formula defines R_0 as "the number of secondary cases per generation"⁵³: i.e R_0 can be
 412 written as the geometric mean $R_0 = \sqrt{R_0^v R_0^h}$, where R_0^v is the number of infected mosquitoes
 413 after the introduction of one infected human in a naive population, and R_0^h is the number of
 414 infected humans after the introduction of one infected mosquito in a naive population. With
 415 this definition, herd immunity is reached when $(1 - R_0^{-2})$ of the population is vaccinated⁵³.

416 R_0 calculation in Laneri model

417 Following the analogy with Pandey model, we compute the spectral radius of the NGM for
 418 the Laneri model.

$$F = \begin{pmatrix} 0 & 0 & 0 & 1 \\ 0 & 0 & 0 & 0 \\ 0 & \beta\tau & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \quad V = \begin{pmatrix} -\sigma & 0 & 0 & 0 \\ \sigma & -\gamma & 0 & 0 \\ 0 & 0 & -\tau & 0 \\ 0 & 0 & \tau & -\tau \end{pmatrix}$$

419 Then we have,

$$V^{-1} = \begin{pmatrix} -1/\sigma & 0 & 0 & 0 \\ -1/\gamma & -1/\gamma & 0 & 0 \\ 0 & 0 & -1/\tau & 0 \\ 0 & 0 & -1/\tau & -1/\tau \end{pmatrix}$$

420 and

$$FV^{-1} = \begin{pmatrix} 0 & 0 & -1/\tau & -1/\tau \\ 0 & 0 & 0 & 0 \\ -\beta\tau/\gamma & -\beta\tau/\gamma & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

421 We calculate the eigen values α of $-FV^{-1}$:

$$\begin{vmatrix} -\alpha & 0 & 1/\tau & 1/\tau \\ 0 & -\alpha & 0 & 0 \\ \beta\tau/\gamma & \beta\tau/\gamma & -\alpha & 0 \\ 0 & 0 & 0 & -\alpha \end{vmatrix} = \alpha^2 \left(\alpha^2 - \frac{\beta\tau}{\gamma\tau} \right) = 0$$

422 Then $\alpha = 0$ or $\alpha = \pm\sqrt{\frac{\beta}{\gamma}}$ and the highest eigen value is $\alpha_R = \sqrt{\frac{\beta}{\gamma}}$.

423

424 Because λ and κ can be seen as parameters rather than state variables, the interpretation of
 425 the spectral radius as the R_0 of the model is not straightforward. Therefore, we computed
 426 the R_0 of the model through simulations, by counting the number of secondary infections
 427 after the introduction of a single infected individual in a naive population. Since Laneri
 428 model is considered here as a vector model, the number of infected humans after the
 429 introduction of a single infected is considered as R_0^2 . We simulated 1000 deterministic

430 trajectories, using parameter values sampled in the posterior distributions for all parameters
431 except initial conditions. With this method, the confidence intervals for number of infected
432 humans (R_0^2) are similar to the ones of α_R^2 estimated by the model. As a consequence,
433 R_0 was approximated by the spectral radius of the NGM in our results with our stochastic
434 framework (cf. Table 5).

435

436 As a robustness check, the same method was applied to Pandey model : the confidence
437 intervals for the number of secondary cases in simulations are very similar to the ones of
438 R_0^2 (cf. Table 5).

	Pandey model	Laneri model
Yap	3.1 (2.5-4.3)	2.2 (1.9-2.6)
Moorea	2.6 (2.2-3.3)	1.8 (1.6-2.0)
Tahiti	2.5 (2.0-3.3)	1.6 (1.5-1.7)
New Caledonia	2.0 (1.8-2.2)	1.6 (1.5-1.7)

Table 5 – Square root of the number of secondary cases after the introduction of a single infected individual in a naive population. Median and 95% credible intervals of 1000 deterministic simulations using parameters from the posterior distribution.

439 Sensitivity analysis

440 In order to analyse the influence of parameter values on the model's outputs, a sensitivity
441 analysis was performed, using LHS/PRCC technique⁵⁴, on Tahiti example. Similar results
442 were obtained for the other settings. Three criteria were retained as outputs for the analysis:
443 the seroprevalence at the last time point, the intensity of the peak of the outbreak and the
444 date of the peak. We used uniform distributions for all parameters, which are listed in
445 Tables 6 and 7. For model parameters, we used the same range as for the prior distribution.
446 For initial conditions, the observation rate r and the fraction involved in the epidemic ρ ,
447 we used the 95% confidence interval obtained by PMCMC, in order to avoid unrealistic
448 scenarios.

449

Parameters		Seroprevalence		Peak intensity		Peak date	
		PRCC	p-value	PRCC	p-value	PRCC	p-value
Model parameters							
R_0^2	Uniform[0,20]	0.88	<0.001	0.91	<0.001	-0.53	<0.001
β_V	Uniform[0.1,10]	-0.69	<0.001	-0.77	<0.001	0.29	<0.001
γ^{-1}	Uniform[4,7]	-0.25	<0.001	0.09	0.003	0.20	<0.001
σ^{-1}	Uniform[2,7]	-0.03	0.324	-0.12	<0.001	0.18	<0.001
τ^{-1}	Uniform[4,20]	0.00	0.921	-0.05	0.102	0.08	0.012
μ^{-1}	Uniform[4,30]	-0.61	<0.001	-0.75	<0.001	0.51	<0.001
Initial conditions							
Hi_0	Uniform[$2 \cdot 10^{-5}$,0.011]	-0.03	0.403	-0.04	0.204	0.03	0.417
Vi_0	Uniform[10^{-4} ,0.034]	0.06	0.044	-0.03	0.290	-0.24	<0.001
Fraction involved and observation model							
ρ	Uniform[0.46,0.54]	0.50	<0.001	0.14	<0.001	-0.03	0.307
r	Uniform[0.048,0.072]	0.02	0.578	0.03	0.383	0.04	0.250

Table 6 – Sensitivity analysis in Pandey model. Tahiti island. 1000 parameter sets were sampled with latin hypercube sampling (LHS), using "lhs" R package⁵⁵. On each parameter set, the model was simulated deterministically. PRCC were computed using the "sensitivity" R package.⁵⁶ P-values were calculated using the Student distribution approximation provided by Blower and Dowlatabadi.⁵⁴

450 For all criteria, the key parameters in both models are transmission parameters (R_0 and β_V).
 451 High values for R_0 are positively correlated with a large seroprevalence and a high and early
 452 peak. On the contrary, high values for the parameters introducing a delay in the model, the
 453 incubation periods in human (σ^{-1}) and in mosquito (τ^{-1}), are associated with a lower and
 454 later peak, and have no significant effect on seroprevalence. Moreover, the simulations are
 455 clearly sensitive to the other model parameters, in particular the mosquito lifespan (μ^{-1}) in
 456 Pandey model.
 457 Concerning other parameters, the initial conditions have a noticeable effect on the date
 458 of the peak only. As expected, the fraction involved in the epidemic (ρ) influences the
 459 magnitude of the outbreak, by calibrating the proportion of people than can be infected, but
 460 it has no significant effect on the timing of the peak.

461

Parameters	Distribution	Seroprevalence		Peak intensity		Peak date	
		PRCC	p-value	PRCC	p-value	PRCC	p-value
Model parameters							
R_0^2	Uniform[0,20]	0.62	<0.001	0.93	<0.001	-0.50	<0.001
γ^{-1}	Uniform[4,7]	0.01	0.731	0.62	<0.001	0.15	<0.001
σ^{-1}	Uniform[2,7]	-0.03	0.373	-0.54	<0.001	0.21	<0.001
τ^{-1}	Uniform[4,20]	-0.03	0.323	-0.70	<0.001	0.47	<0.001
Initial conditions							
Hi_0	Uniform[10^{-5} ,0.015]	0.05	0.135	0.02	0.622	-0.32	<0.001
L_0	Uniform[$2 \cdot 10^{-5}$,0.004]	0.05	0.133	0.00	0.930	-0.16	<0.001
Fraction involved and observation model							
ρ	Uniform[0.49,0.59]	0.80	<0.001	0.34	<0.001	0.02	0.558
r	Uniform[0.048,0.068]	-0.01	0.727	0.01	0.738	-0.02	0.635

Table 7 – Sensitivity analysis in Laneri model. Tahiti island. 1000 parameter sets were sampled with latin hypercube sampling (LHS), using "lhs" R package⁵⁵. On each parameter set, the model was simulated deterministically. PRCC were computed using the "sensitivity" R package.⁵⁶ P-values were calculated using the Student distribution approximation provided by Blower and Dowlatabadi.⁵⁴

462 Complementary results

463 These complementary results include PMCMC results for both models in the four settings:
 464 the epidemic trajectories regarding the human compartments for infected and recovered
 465 individuals (Figures 4-5), the detailed posterior distributions for all parameters (Figures
 466 6-13) and correlation plots for all models (Figures 14-21).

467

468 **Infected and recovered**

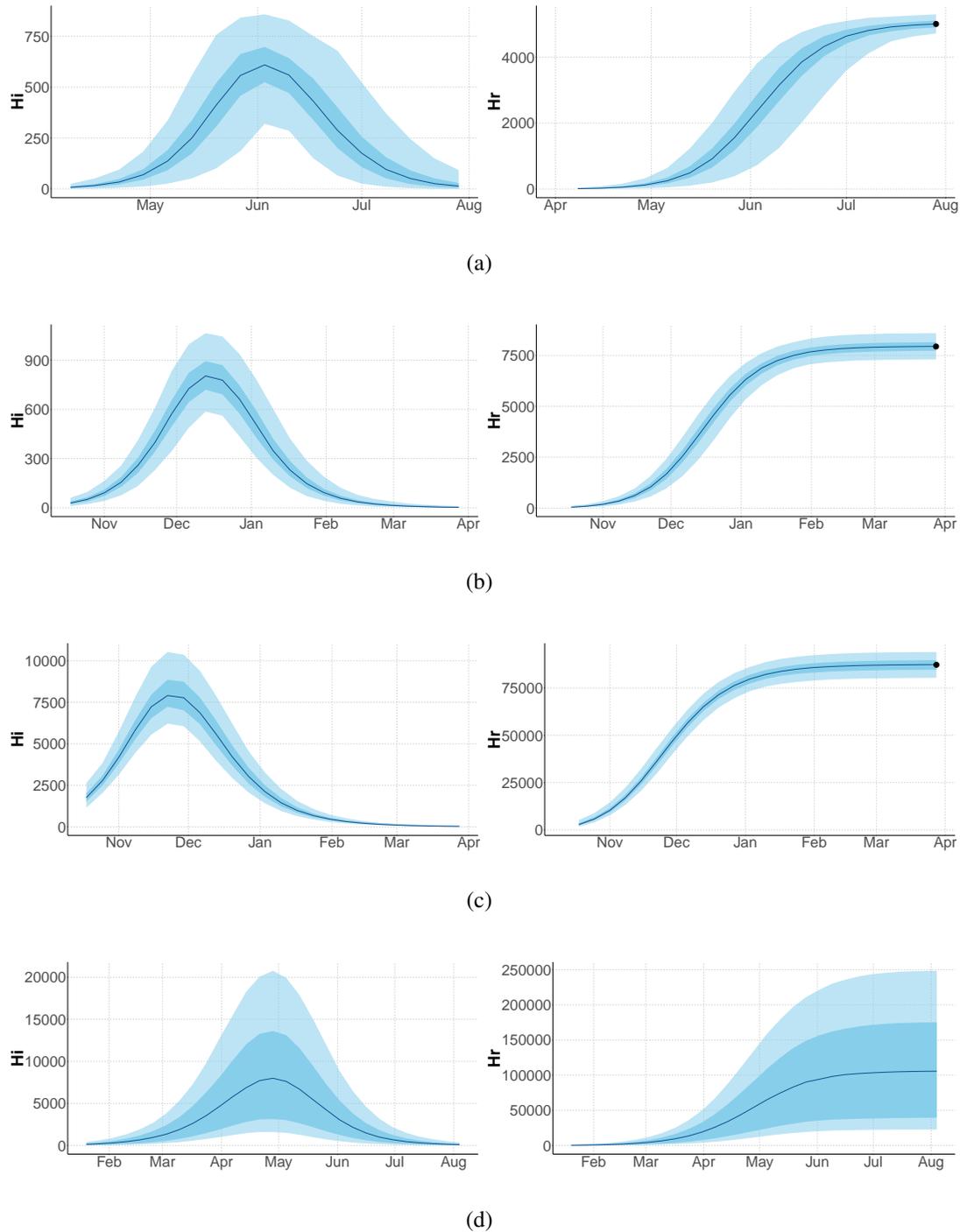


Figure 4 – Infected and recovered humans evolution during the outbreak with Pandey model. Simulations from the posterior density: posterior median (solid line), 95% and 50% credible intervals (shaded blue areas) and observed seroprevalence (black dots). First column: Infected humans (H_I). Second column: Recovered humans (H_R). a) Yap. b) Moorea. c) Tahiti. d) New Caledonia.

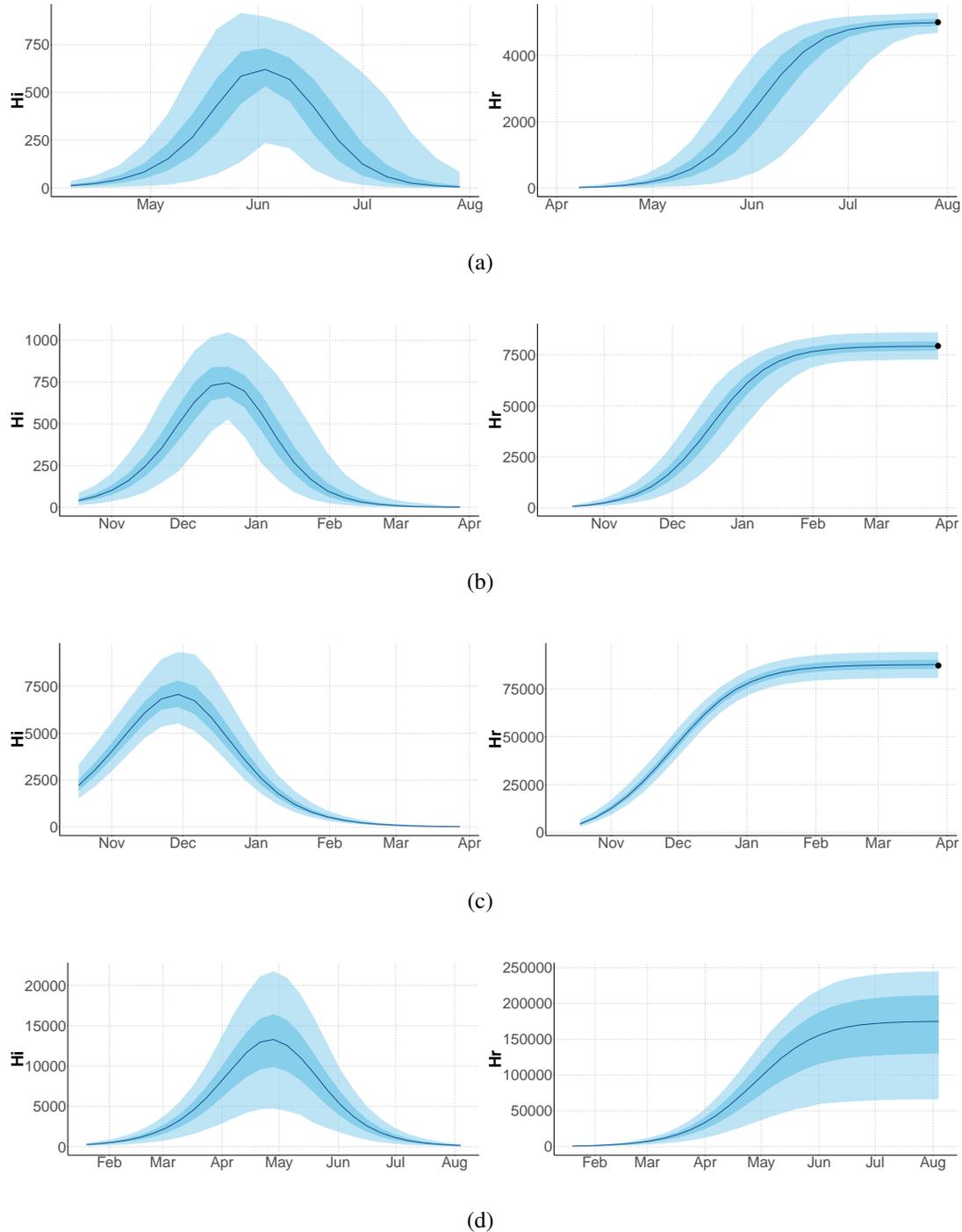


Figure 5 – Infected and recovered humans evolution during the outbreak with Laneri model. Simulations from the posterior density: posterior median (solid line), 95% and 50% credible intervals (shaded blue areas) and observed seroprevalence (black dots). First column: Infected humans (H_I). Second column: Recovered humans (H_R). a) Yap. b) Moorea. c) Tahiti. d) New Caledonia.

469 **Posterior distributions**

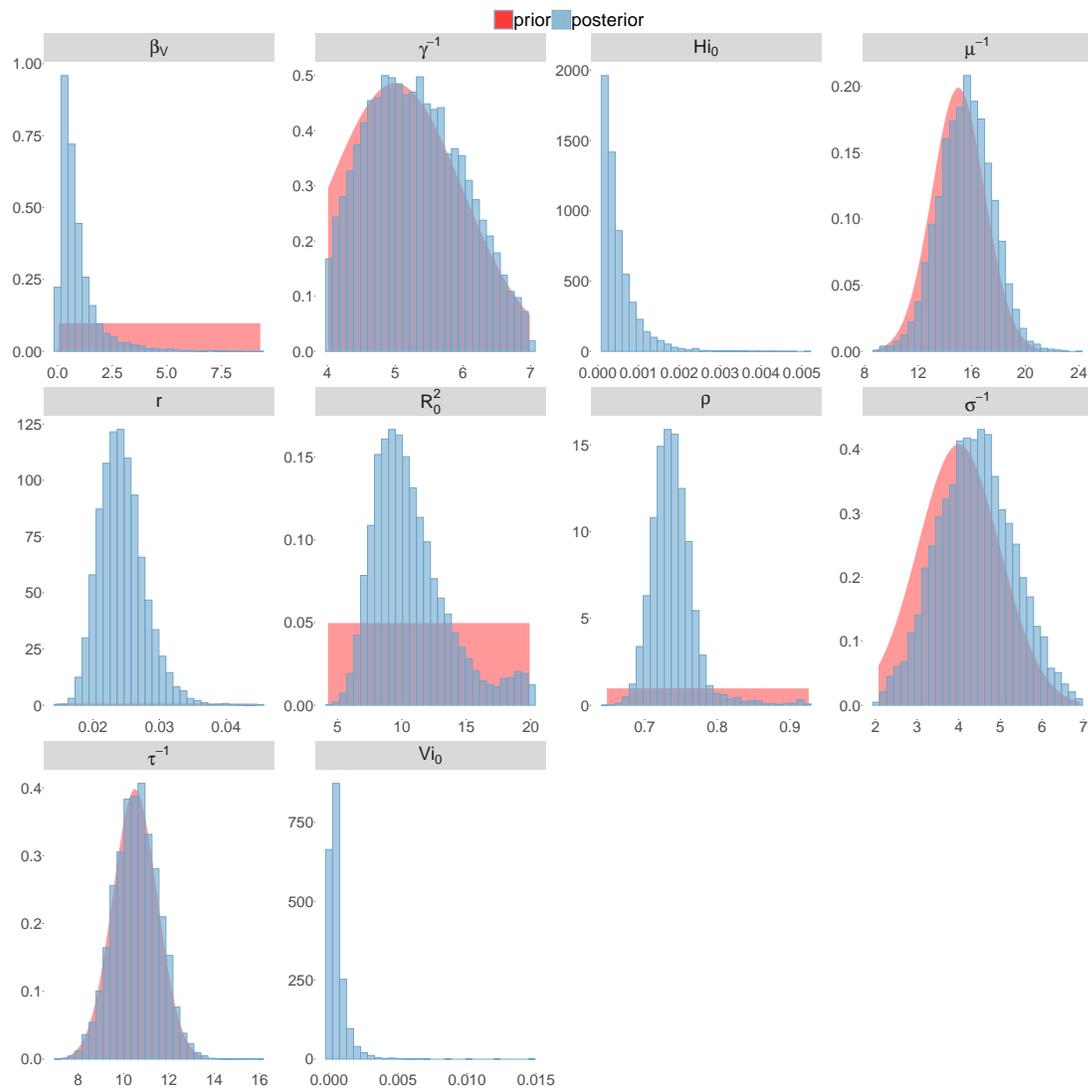


Figure 6 – Posterior distributions. Pandey model, Yap island.

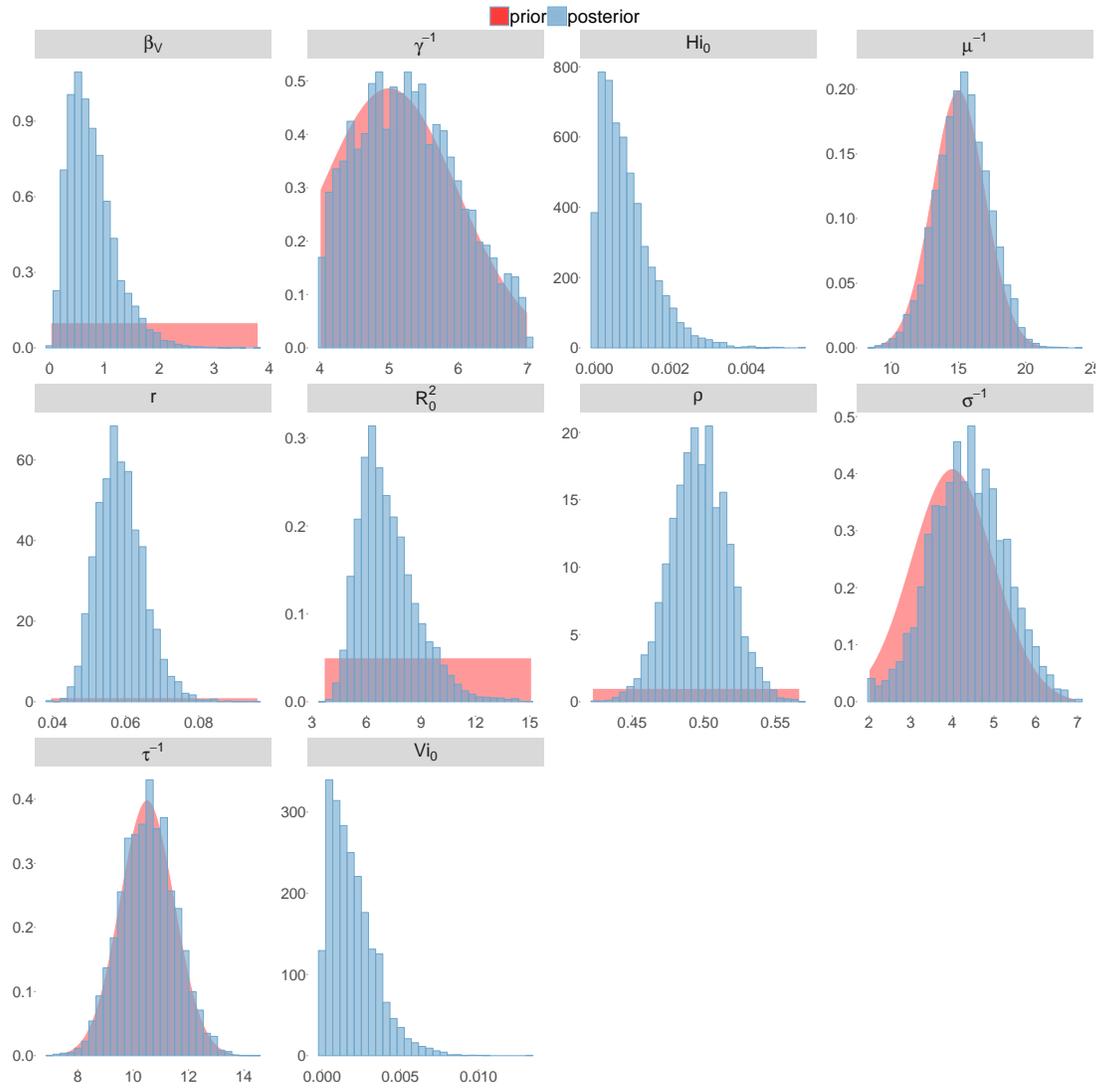


Figure 7 – Posterior distributions. Pandey model, Moorea island.

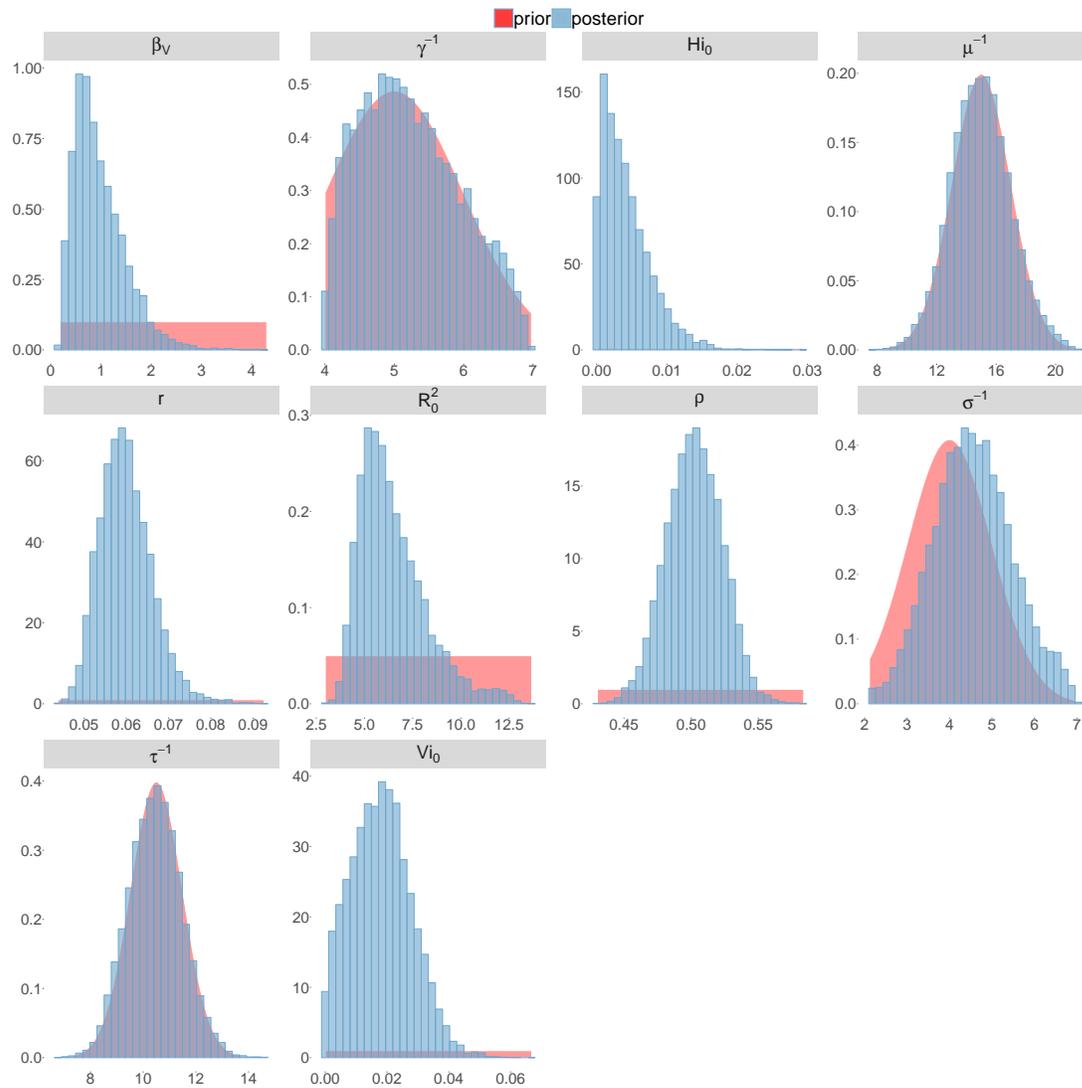


Figure 8 – Posterior distributions. Pandey model, Tahiti island.

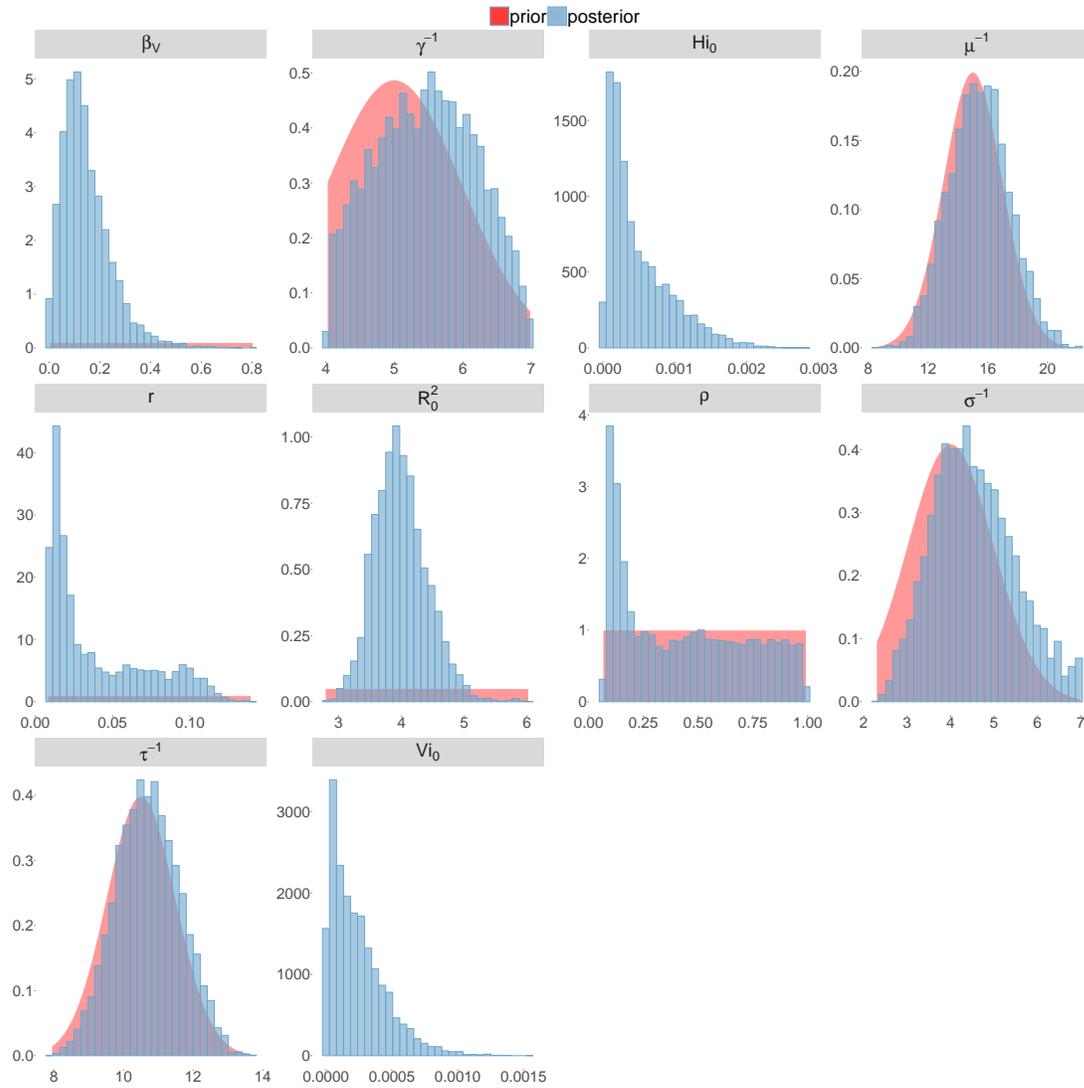


Figure 9 – Posterior distributions. Pandey model, New Caledonia.

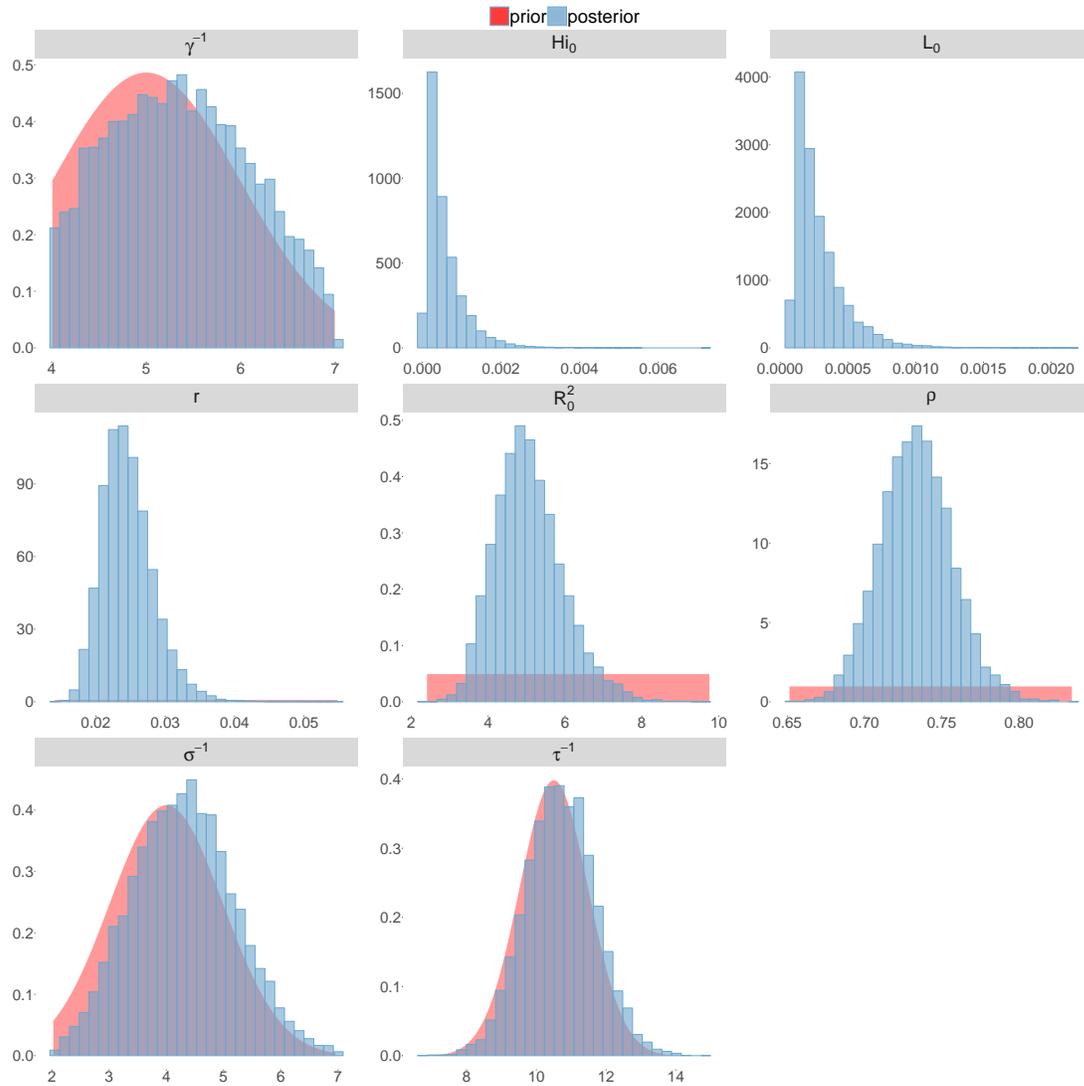


Figure 10 – Posterior distributions. Laneri model, Yap island.

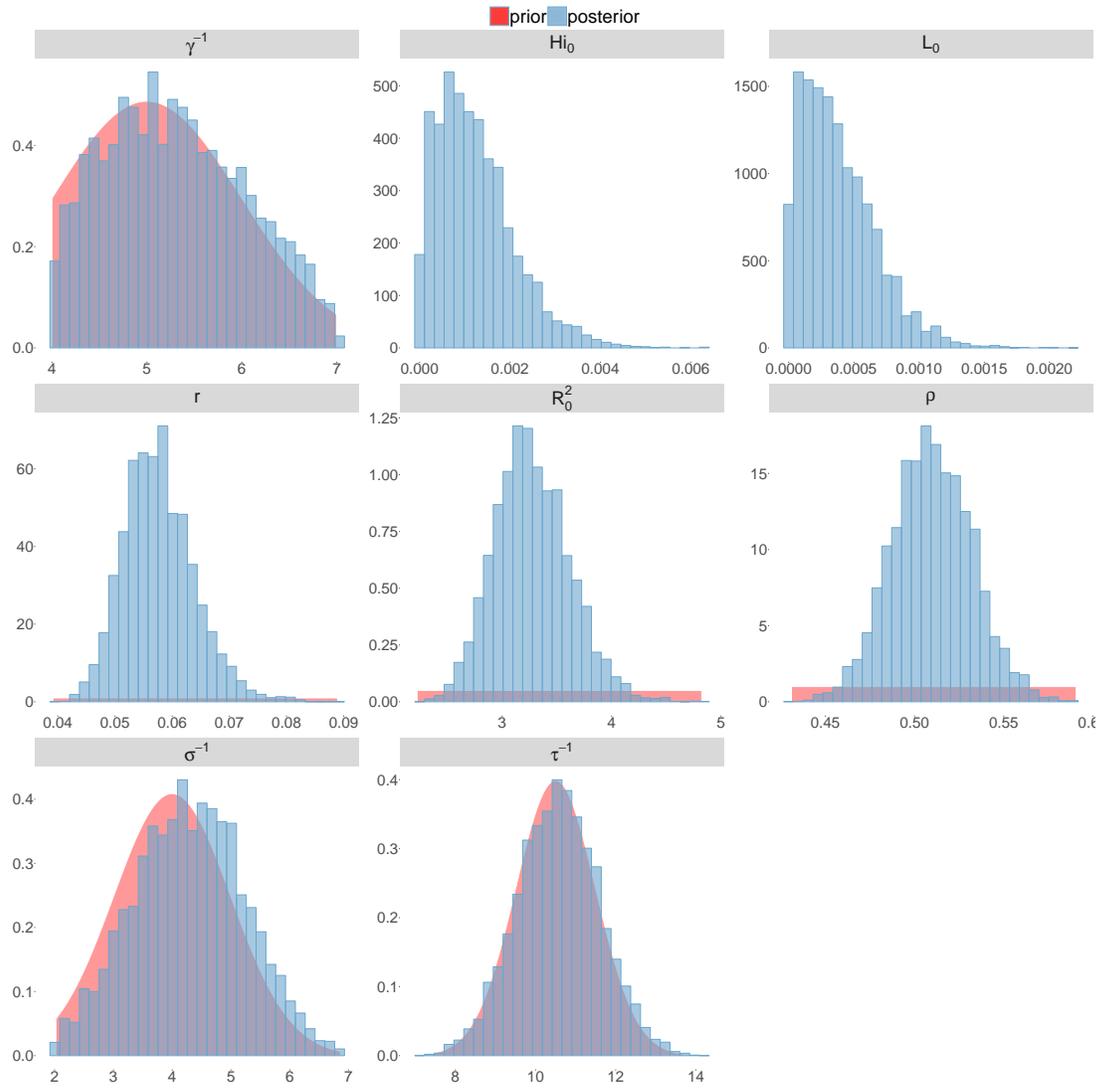


Figure 11 – Posterior distributions. Laneri model, Moorea island.

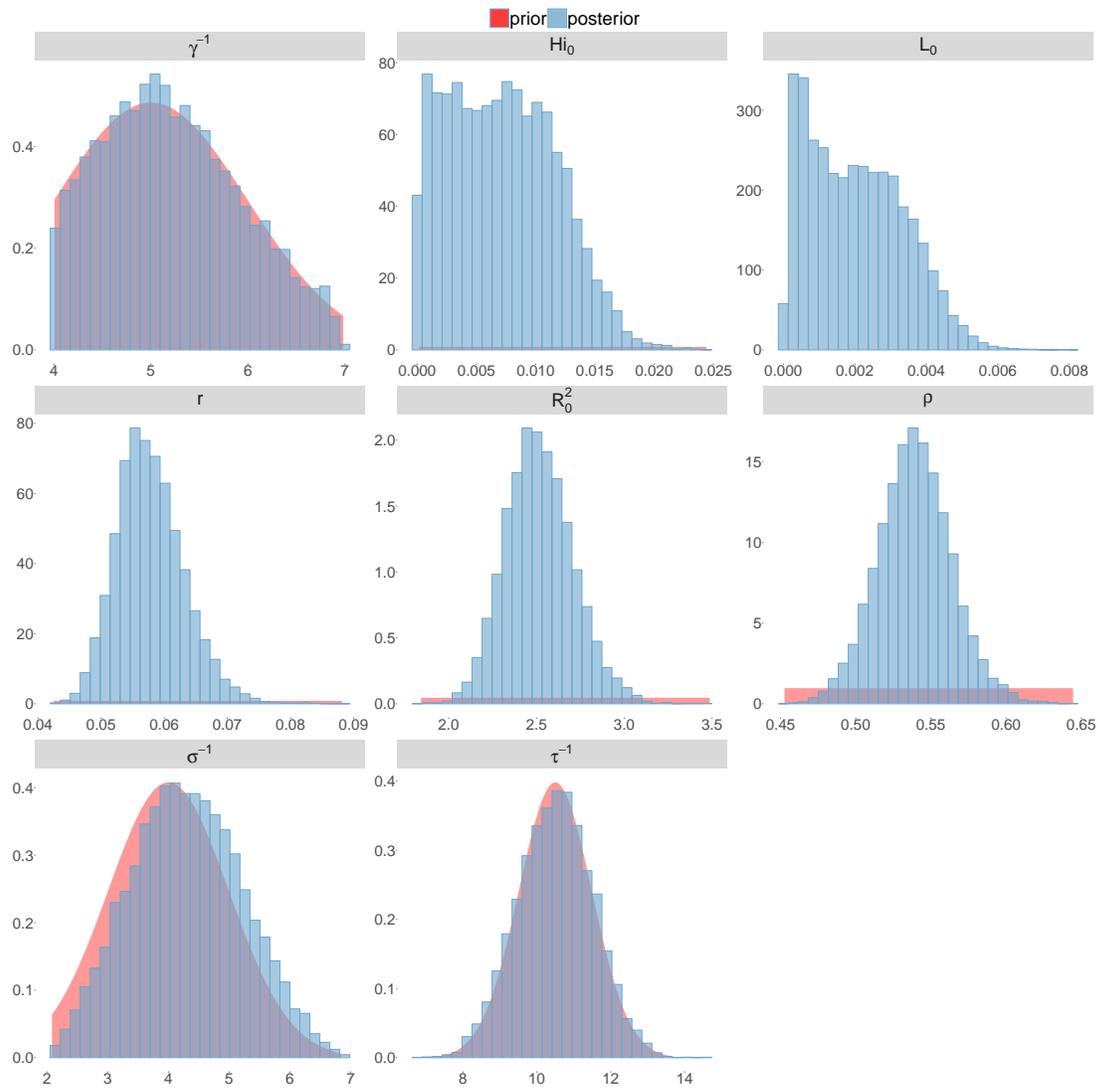


Figure 12 – Posterior distributions. Laneri model, Tahiti island.

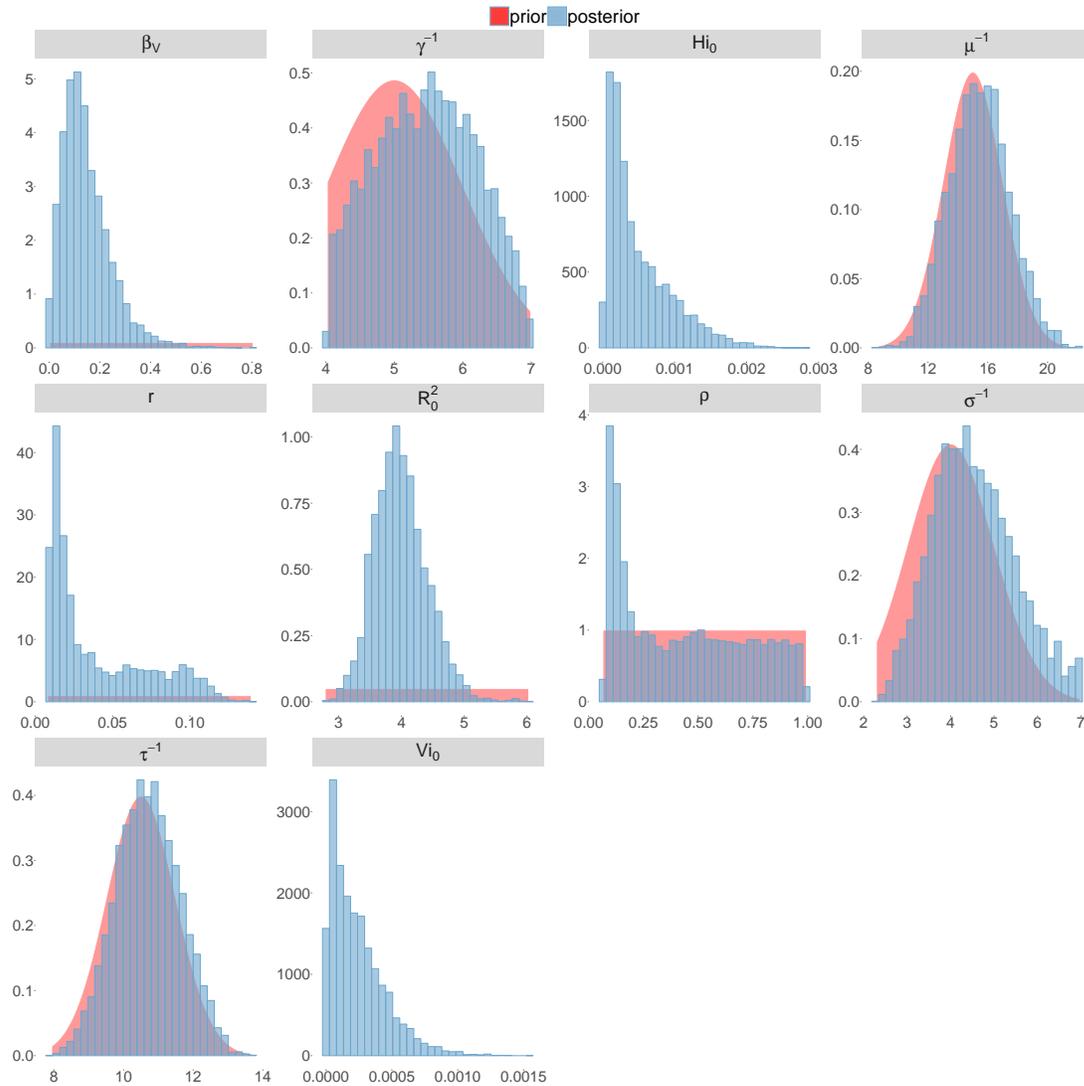


Figure 13 – Posterior distributions. Laneri model, New Caledonia.

470 Correlation between estimated parameters

471 The marginal posterior densities (Figures 6-13) do not indicate the correlation between
472 parameters, i.e when the observed value of one parameter is highly influenced by the value
473 of another one. In some cases, this phenomenon reveals identifiability issues : the model
474 manages to estimate only a pair of parameters but cannot identify each one separately. In
475 our case, the observation rate and the fraction of the population involved in the epidemic are
476 strongly negatively correlated when no information is provided on seroprevalence (Figures
477 9 and 13).

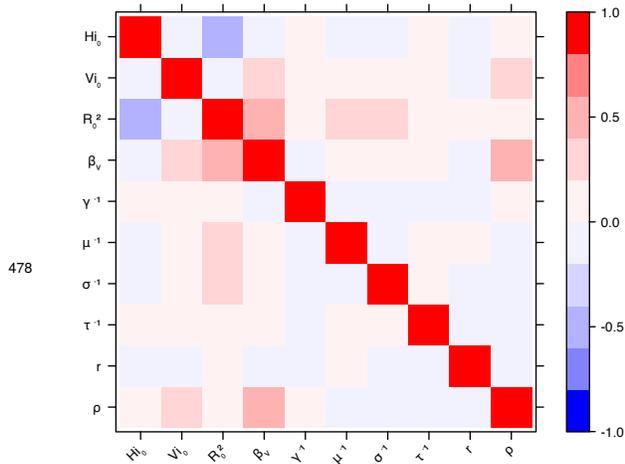


Figure 14 – Correlation plot of MCMC output. Pandey model, Yap island.

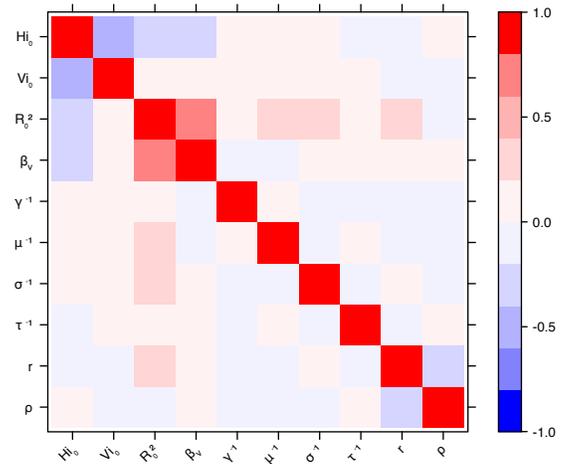


Figure 15 – Correlation plot of MCMC output. Pandey model, Moorea island.

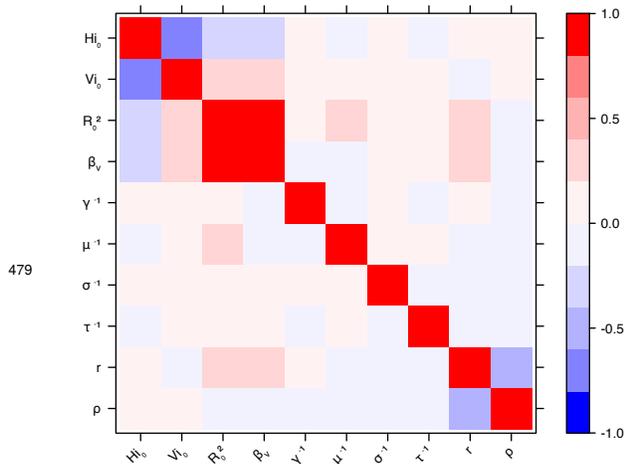


Figure 16 – Correlation plot of MCMC output. Pandey model, Tahiti island.

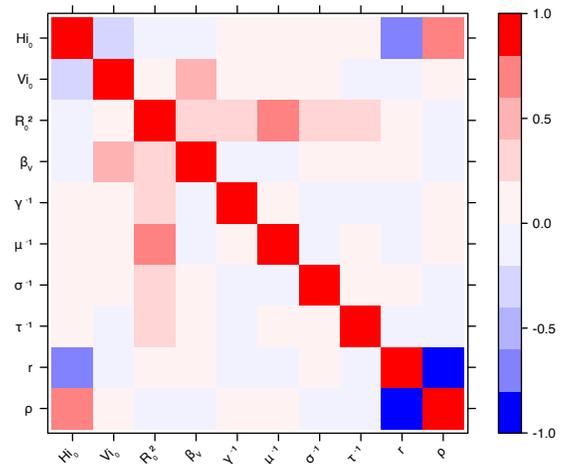


Figure 17 – Correlation plot of MCMC output. Pandey model, New Caledonia.

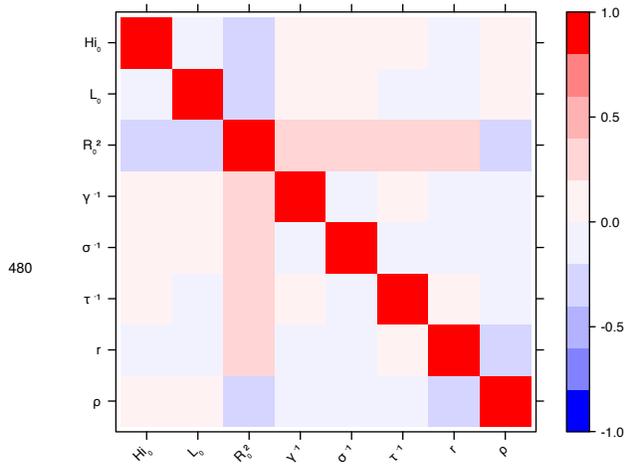


Figure 18 – Correlation plot of MCMC output. Laneri model, Yap island.

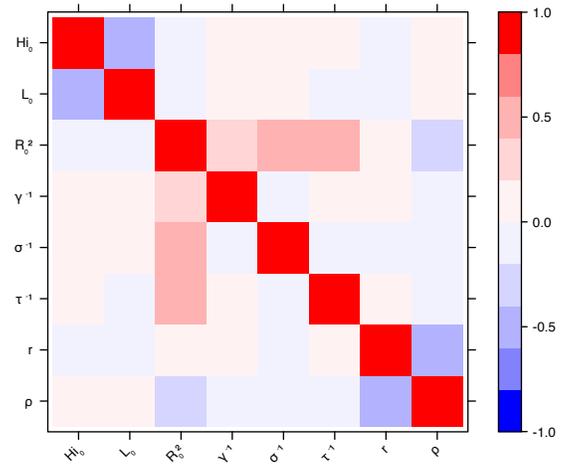


Figure 19 – Correlation plot of MCMC output. Laneri model, Moorea island.

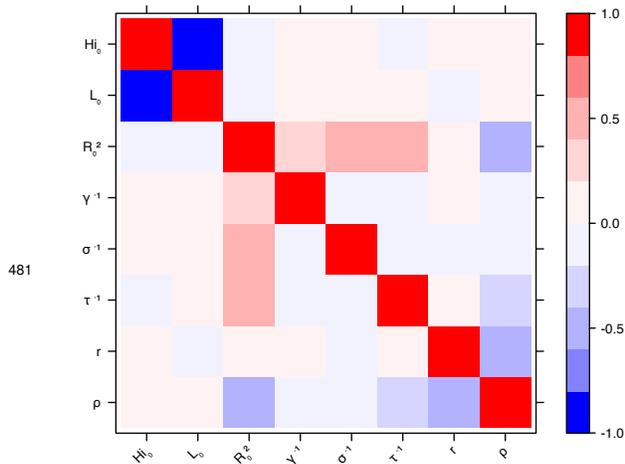


Figure 20 – Correlation plot of MCMC output. Laneri model, Tahiti island.

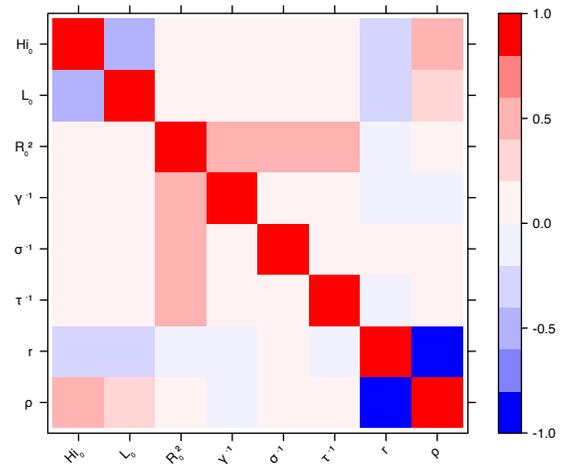


Figure 21 – Correlation plot of MCMC output. Laneri model, New Caledonia.

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493 Declaration of interests

494 The authors have declared that no competing interests exist.

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