# On the vaccination threshold for Covid-19 in French Polynesia

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#### Abstract

High immunization rates are often sought to contain epidemics with target values typically 70% or greater. Our objective is to independently assess this value in the context of the 2020 Covid-19 pandemic in French Polynesia. To this extent, we develop a graph-based epidemic model tailored to this pandemic and compute the vaccination threshold required to prevent exponential spread of the communicable disease. Our results indicate that herd immunity increases drastically when a threshold percentage of vaccinated individuals is reached. Experimental data using our idealized model indicates that the threshold value is approximately 45%. We conclude that vaccination is much more effective at preventing pandemics than usually predicted.

## 1 Introduction

It is well-known that highly communicable diseases such as measles can only be thwarted when a very large proportion of the population is immune, and vaccination is an effective way to artificially boost public immunity. In this paper, we seek to compute the *herd immunity threshold* (HIT), that is, the proportion of individuals who must be immune in order to ensure that reintroducing the disease in an otherwise healthy population only leads to contained, non-exponential spread.

This threshold is often confused with the *final cumulative incidence rate* (FCIR) which is the eventual proportion of recovered individuals in a naturally spreading pandemic. For simple compartmental models such as SIR, those values are in fact equal, and we have

$$HIT = FCIR = 1 - 1/R_0$$

where  $R_0$  denotes the basic reproductive number of the disease. For Covid-19, current estimates (Billah & Khan, 2020; Kucharski et al., 2020) give  $R_0 \in [2.4, 3.4]$ . Considering a worst-case scenario of  $R_0 \approx 3.4$ , government officials thus seek an immunization rate of  $1 - 1/3.4 \approx 70\%$  to contain further epidemics.

Over the past year it has been widely argued that the herd immunity threshold for Covid-19 ought in fact to be smaller (Britton et al., 2020; Gomes et al., 2022). We investigate this claim by developing a graph-based epidemic model. Such models provide finer-grained methods for simulating the spread of a communicable disease through a population with a heterogeneous social graph. We calibrate our model on public data specific to the 2020 Covid-19 pandemic in French Polynesia.

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We then use this model to compute the effectiveness of vaccination as measured by the resulting FCIR when reintroducing the disease in a partly immune population. Our computations show that vaccination sharply increases in effectiveness when a threshold proportion of about 45% immune individuals is reached. While considerations not taken into account by our idealized model (such as variants or antibody decay) surely affect this threshold value, we argue that the overall effect stands.

## 2 Epidemic models

### 2.1 The SIR model

The SIR model (Kermack & McKendrick, 1927) aims to predict the spread of an infectious disease; to this extent, it partitions the population in compartments: *susceptible individuals* (S), *infectious individuals* (I), *recovered individuals* (R). Flow patterns between compartments are generally described by ordinary differential equations such as:

$$\frac{\partial S}{\partial t} = -\frac{\beta I S}{N}, \qquad \frac{\partial I}{\partial t} = \frac{\beta I S}{N} - \gamma I, \qquad \frac{\partial R}{\partial t} = \gamma I$$

where N is the total population,  $\beta$  is the probability of contagion per individual per unit of time, and  $\gamma$  is the inverse of the duration of contagion.

This model and its many variants implicitly assume perfect and uniform interaction between the populations of each compartment, as if each individual was in contact with every other in a homogeneous way. This is equivalent to assuming the social graph to be complete. This profound assumption on spreading patterns makes such models very simple and thus easy to work with but exhibits suboptimal correlation with observed data.

#### 2.2 Graph-based models

To simulate the spread of an epidemic while taking into account the complexity of social interactions, we rely on graph-based models, also known as network-based models.

A graph consists of a set of vertices V and a set of edges  $E \subset V^2$ . In the social graph, vertices represent individuals and edges correspond to significant social interactions. In this context we restrict the study to graphs which are non-directed, simple, and connected. Since the social graph cannot be rigorously defined or even computed, we use randomly generated graphs with specific properties: vertices are laid out on a two-dimensional lattice; for each vertex, a degree is chosen randomly according to a Poisson distribution; as many vertices are then randomly chosen from neighboring lattice points and connected to it.

Figure 1 shows the first four steps of a simulation of an epidemic along the edges of a social graph. Our model computes such simulations by tracking the state of each vertex: susceptible, incubating, contagious or recovered. Initially, the entire population is assumed susceptible, and we randomly select a given number to be incubating. After a period of incubation, they become contagious and are then able to pass on the disease to their neighbors in the social graph. Those vertices eventually become recovered and thus immune.

We refer the reader to (Kiss et al., 2017) for an overview of graph-based models and we note that such models have already produced important results concerning the Covid-19 pandemic for specific geographical areas (Chang et al., 2021; Pizzuti et al., 2020).



Figure 1: Simulating the propagation of the disease along the social graph.

# 3 Calibrating social-graph parameters

For our model, we selected graphs generated as described above to best approximate the social graph of French Polynesia. The latest count reports 275,916 inhabitants and we thus generate graphs containing a total of 250,000 vertices each. The degree of vertices is randomly distributed in  $[4, \infty)$  according to a Poisson distribution with mean-value 12. This allows us to model a wide range of individuals with both small and large social circles (Meyerowitz et al., 2021) as well as to account for virus-specific phenomenon such as overdispersion (Endo et al., 2020).

We further ensure that our graphs are connected with a diameter of about 30 which is significantly higher than the degree of separation but more realistic given that Covid-19 mainly spreads through close relationships.

#### 3.1 Calibrating disease-related parameters

Our model relies on state-of-the-art range estimates for the Covid-19 incubation period (Lauer et al., 2020) and contagion period (X. He et al., 2020). Since there is no public data on the basic reproductive number  $R_0$  in French Polynesia, we conservatively use the upper bound 3.4 of the worldwide estimated range. Our algorithm's parameters are further chosen such that this value matches the initial observed spike of  $R_0$  where the epidemic grows exponentially.

However we note that the actual value of  $R_0$  should be slightly smaller in French Polynesia due to multiple factors pertaining to tropical climate (Prather et al., 2020; Raines et al., 2021) including: higher humidity and thus less communicability via droplets (Božič & Kanduč, 2021); and higher temperatures and thus a lower frequency of indoor social activities (Bulfone et al., 2021).

# 4 Results

#### 4.0.1 Sample runs

The procedures described above have been implemented in Python; the code is freely available online at:

### https://gaati.org/oyono/pandemic-code/

Using that code, we compute ten thousand sample runs of our model on a healthy population and verify that the output matches the expected values. See Figure 2 for one such run which is typical of what would be expected of a naturally evolving pandemic without any protective measures such as lockdowns or vaccination.



Figure 2: Sample run of our graph-based model.

### 4.1 Simulating the impact of vaccination on FCIR

We now consider a healthy population of which a given percentage has been vaccinated and thus considered immune. The disease is then introduced, and we compute the final cumulative incidence rate (FCIR) of the epidemic. To determine the herd immunity threshold (HIT), that is, the threshold vaccination rate which prevents the reintroduced disease from spreading exponentially, we compute, for each percentage of vaccinated individuals in 0.1% increments, a thousand sample runs of our model over randomly chosen graphs. Figure 3 shows our results.

#### 4.2 Discussion

For sample runs of our model simulating a naturally evolving pandemic on a healthy population, the resulting FCIR lies in the range [75%, 85%] which is widely accepted for Covid-19 (S. He et al., 2020; Wangping et al., 2020). This confirms the relevance of our model.

We now turn to the simulation of an epidemic in a population of which a given percentage has been vaccinated and thus considered immune. Our computation of FCIR as a function of the vaccination rate is displayed in Figure 3. It reveals a sharp increase in vaccination effectiveness around a threshold rate of about 40%. For vaccination rates below this threshold, the level of protection as measured by FCIR varies roughly linearly with the vaccination rate, as predicted by homogeneous models such as SIR. For vaccination rates above this threshold, the level of protection quickly reaches its maximum: exponential spread of the disease is not observed at vaccination rates of 45% and above.

We stress actual threshold values may differ since our model reflects an idealized version of the 2020 Covid-19 pandemic and, as such, does not account for several factors including multiple circulating variants of the virus (Fowlkes et al., 2021) and vaccine effectiveness (Rosenberg et al., 2021).

Nevertheless, we argue that the general behavior stands, namely that vaccination sharply increases in effectiveness around a threshold value. Said HIT value is necessarily smaller than the FCIR for a naturally evolving pandemic. Further research remains necessary to confidently assess the HIT value.



Figure 3: FCIR for an epidemic in a partly-vaccinated population. For each vaccination rate in 0.1% increment, FCIR has been computed for a thousand randomly chosen graphs; the black line shows the median value, and darker to lighter gray areas display probability ranges [25%, 75%], [10%, 90%], and [5%, 95%].

# 5 Conclusion

We conclude that, as a public health strategy, vaccination is much more effective at preventing pandemics than predicted by homogeneous models. While its effectiveness initially grows linearly in the proportion of immune individuals, it sharply increases when a threshold immunity rate is reached. Although our model is unsuited to determine actual values, it indicates that, at least in the context of French Polynesia, the actual HIT is likely closer to 50% than to 70%.

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