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Abstract

We develop a graph-based epidemic model tailored to the 2020 Covid-19 pandemic in French Polynesia. Our computations indicate that herd immunity increases drastically when a threshold percentage of vaccinated individuals is reached. Our idealized model yield a threshold value of about 45%.

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

$$\text{HIT} \; = \; \text{FCIR} \; = \; 1 - 1/R_0$$

where R_0 denotes the basic reproductive number of the disease. For coronavirus disease 2019 (Covid-19), current estimates [13, 1] 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 [8, 3]. 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.

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.

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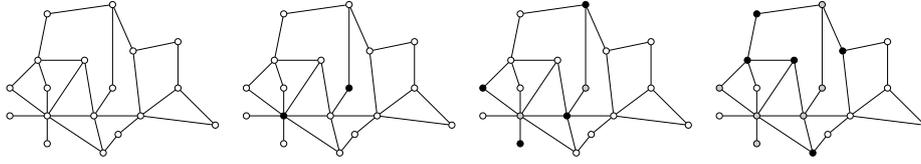
Figure 1: Simulating the propagation of the disease along the social graph.

2 Epidemic models

2.1 The SIR model

The SIR model [11] 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, β is the probability of contagion per individual per unit of time, and γ 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 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 [12] 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 [16, 5].
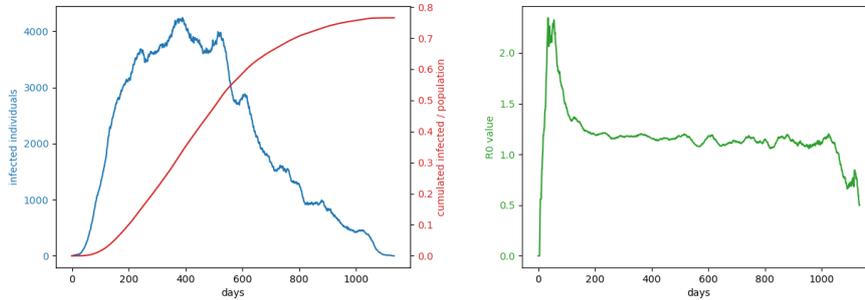
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Figure 2: Sample run of our graph-based model.

3 Impact of vaccination on FCIR

3.1 Calibrating our model

3.1.1 Graph-generating procedure

Our graphs are generated as described above to best model 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 [15] as well as to account for virus-specific phenomenon such as overdispersion [6].

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 interactions.

3.1.2 Disease-related parameters

Our model relies on state-of-the-art range estimates for Covid-19 incubation period [14] and contagion period [9]. 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 [18, 17] including: higher humidity and thus less communicability via droplets [2]; and higher temperatures and thus a lower frequency of indoor social activities [4].

3.1.3 Sample runs

We compute ten thousand sample runs of our model on an 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. As expected the resulting FCIR lies in the range $[75\%, 85\%]$ which is widely accepted for Covid-19 [20, 10].
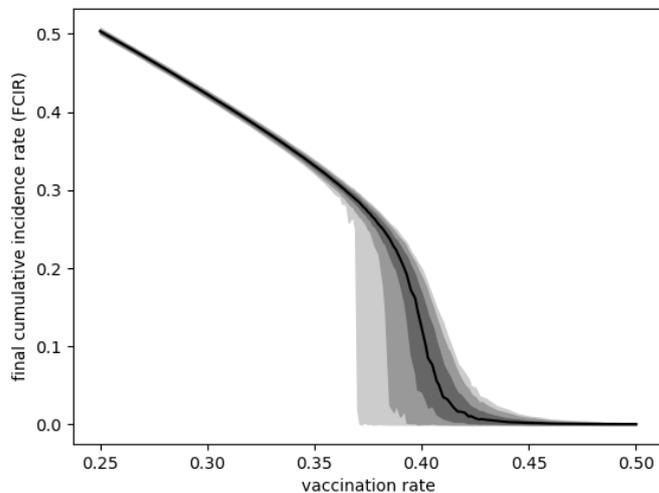
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Figure 3: FCIR for an epidemic in a partly-vaccinated population. For each vaccination rate in 0.1% increment, FCIR has been computed for a thousand randomly chosen graphs; the black line shows the median value, and darker to lighter gray areas display probability ranges $[25\%, 75\%]$, $[10\%, 90\%]$, and $[5\%, 95\%]$.

3.2   Experimental data

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. In order 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.

Our computations reveal 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 [7] and vaccine effectiveness [19].

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.

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4   Conclusion

We conclude that, as a public health strategy, vaccination is much more effective at preventing Covid-19 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. While 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 the generally accepted 70% target.

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