Vaccination, asymptomatics and public health information in COVID-19

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Abstract. The dynamics of the COVID-19 pandemic is greatly influenced by vaccine quality, as well as by vaccination rates and the behaviour of infected individuals, both of which reflect public health policies. We develop a model for the dynamics of relevant cohorts within a fixed population, taking extreme care to model the reduced social contact of infected individuals in a rigorous self-consistent manner. The basic reproduction number R_0 is then derived in terms of the parameters of the model. Analysis of R_0 reveals two interesting possibilities, both of which are plausible based on known characteristics of COVID-19. Firstly, if the population in general moderates social contact, while infected individuals who display clinical symptoms tend not to isolate, then increased vaccination can drive the epidemic towards a disease-free equilibrium (DFE). However, if the reverse is true, then increased vaccination can destabilise the DFE and yield an endemic state. This surprising result is due to the fact that the vaccines are leaky, and can lead to an increase in asymptomatic individuals who unknowingly spread the disease. Therefore, this work shows that public policy regarding the monitoring and release of health data should be combined judiciously with modeling-informed vaccination policy to control COVID-19.

Keywords: vaccination, asymptomatics, nonlinear incidence rate

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1. Introduction

The COVID-19 (SARS-CoV-2) pandemic, which caused wide-ranging suffering and unprecedented disruption, and which now seems to be abating mainly due to the reduced virulence of the prevalent strains of the virus, has given rise to a number of interesting challenges to modellers. Some of these have to do with understanding (nonlinear) infection rates in the context of public health policies. Of particular interest is understanding how levels of awareness amongst the general population impact the dynamics of the pandemic.

The literature on SARS-CoV-2 modelling is immense and so we can only be very selective in our remarks and reference and by necessity omit much important work. The understanding that the susceptible population is heterogeneous, thus making ODE models of the type considered here not completely satisfactory is discussed in [10]. Vaccination strategies and quality of protection are considered in [15, 20, 24, 25]. Particular role of asymptomatics in epidemics dynamics are the main topic in [1, 6, 9, 11, 22]. Leakiness of vaccines is analysed in [17]. Other mathematical aspects of related models are treated in [14].

In our previous study [12], performed before the availability of vaccination, we considered the role that public information plays in the evolution of the pandemic. Using just two cohorts, comprising susceptible individuals and infected individuals who display clinical symptoms of the disease, respectively, we showed how the level of new infections can reach a stationary value, yielding a linear growth in cases as observed in various datasets. Adding a third cohort, representing asymptomatics who are capable of spreading the disease but who themselves show no clinical symptoms, did not change the conclusions of our model.

In the work presented here, we now consider how public information can be included in models with an additional cohort of vaccinated people. Our contribution here is purely methodological and focuses on the ideas needed to model contact rates realistically; this particular focus sets our work apart from the available literature.

The cohorts in our model are illustrated in Figure 1. We consider a fixed population that can be divided into susceptibles $S(t)$, vaccinated $V(t)$, infected $I(t)$, and asymptomatics $A(t)$. We also have a cohort of those recovered from recent infection $R(t)$, who have some protection from re-infection. The size of these cohorts can vary over time t, with people moving between cohorts, as illustrated in Figure 1, at rates which themselves can be nonlinear. Of particular interest to us is the fact that the vaccinated cohort $V(t)$ will mingle quite freely with the rest of the population, as will asymptomatics $A(t)$ since, by definition, they do not realise that they are spreading the infection. However, the infected cohort $I(t)$ will, on average, reduce their contact with the rest of the population, and the level of restraint will reflect public health policy regulations and all other available information. As we discuss below, formulating this effect in a

rigorously self-consistent manner is possible if due care is taken to describe contact rates.

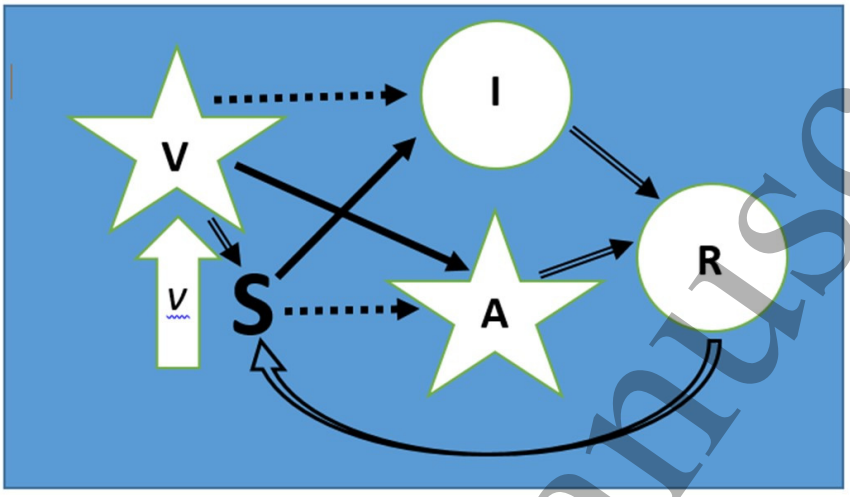

Figure 1. Illustration of the cohorts and interactions used in this work.

Members of the vaccinated cohort have some protection from the disease that wanes over time. Nevertheless, the COVID vaccines are known to be leaky, so that vaccinated individuals can become infected with the disease and move to either $A(t)$ or $I(t)$. We therefore note that there is now the interesting possibility of increased vaccination rates leading to more asymptomatics, which themselves can increase $I(t)$, thus yielding higher levels of infection overall in the population. In fact, we will show that this is indeed the case under reasonable estimates of various transmission rates.

Before we proceed, we should reassure the reader that what we propose is not mere fitting of data to our model, which unavoidably contains many parameters as described below. Rather, we will show that it is the relative size of combinations of parameters that is important, and that these can readily be identified from known properties of the disease, leading to clear understanding of the behaviour of our model.

2. The model

Though clearly the human population is highly heterogeneous with respect to its communicability [10], and its resistance to and tolerance of viral infection, and while the evolution of the virus is a crucially important aspect of the pandemic, we operate in the simpler context of lumped (ODE) models, on a timescale that allows us also to neglect demographics; see [18] for a modern overview and standard notation which we follow. Thus we assume that the total population N_0 is fixed, and people can only move from one cohort to another; belonging to a cohort determines at what rate movement to another

cohort can take place. For clarity, we will write down the ODEs for S, I, A, V, R, i.e. for the expected numbers of people in the susceptible, infected, asymptomatic, vaccinated and recovered cohorts, but in our analysis we will always take $R = N_0 - S - I - A - V$. We have

$$\begin{aligned} S' &= -\alpha_S L_S(I,A)S - \nu S + \beta R + \gamma V, \\ V' &= \nu S - \alpha_V L_V(I,A)V - \gamma V, \\ I' &= \delta_S \alpha_S L_S(I,A)S + \delta_V \alpha_V L_V(I,A)V - \rho_I I, \\ A' &= (1-\delta_S)\alpha_S L_S(I,A)S + (1-\delta_V)\alpha_V L_V(I,A)V - \rho_A A, \\ R' &= \rho_I I + \rho_A A - \beta R. \end{aligned} \tag{1}$$

Here ν is the rate of vaccination, $L_S(I,A)$ and $L_V(I,A)$ are the environmental pathogen levels [7], encountered by the S and the V cohorts, respectively; we will discuss these below in detail. α_S and α_V measure the transmissibility of the pathogen to susceptibles and the vaccinated, respectively. Thus higher α_S, for example, means lower resistance to infection. δ_S and δ_V, that take values in $[0,1]$, measure the lack of tolerance: e.g. for a wholly tolerant susceptible host, in which the virus can only cause asymptomatic disease, $\delta_S = 0$. ρ_A, ρ_I are recovery rates for the asymptomatic and the infected cohorts (i.e. the rate at which they stop producing the virus); β measures the rate of waning of natural immunity, and γ measures the rate of waning of vaccination-provided immunity.

We assume that the vaccine is effective, which means that $\alpha_V \leq \alpha_S$ and that it increases tolerance, by which we mean that $\delta_V \leq \delta_S$. It is also logical to assume that $\rho_I \leq \rho_A$. The relation between β and γ, a matter of some heated controversy, does not concern us here. As $\alpha_V \neq 0$, we are dealing with a leaky vaccine.

We assume here that the rate of vaccination ν is constant, set by public health policy (as, for example, in [3, 4]). In future work, it will be interesting to explore the effects of making the rate dependent, through public health measures and change in behavioural patterns, on the size of the infected cohort I. For example, we could assume that

$$\nu(I) = \nu_0 + \frac{K_1 I}{I + K_2} \tag{2}$$

for some positive constants K_1, K_2. ν turns out to be of particular interest in the discussion of the basic reproduction number R_0 below.

We could have subdivided the infected cohort into two, depending on the origin of the person showing clinical symptoms, i.e. S or V; each of these sub-cohorts would have its own infectivity and recovery rate, but for simplicity we only encode the difference between the vaccinated and the un-vaccinated susceptible cohort via the parameters α_S, α_V and δ_S, δ_V. We also do not consider latency or vaccination of the recovered cohort.

The remaining issue is to specify $L_S(I,A)$ and $L_V(I,A)$, which is far from obvious and we proceed to show our thinking in detail.

We assume that the A cohort is behaviourally identical to the S cohort, but these two cohorts, and the V and the I cohorts, can be distinguished by their behaviour.

We assume that the public health information that people receive is in terms of the magnitude of I; equivalently, this information is (or can easily be obtained) in terms of $i := I/N_0$. We associate with each cohort a **contact propensity per capita** and assume that all these propensities are functions of i; we will denote them by $c_j(i)$, $j \in \{S, I, A, V\}$. It stands to reason that these propensities are decreasing functions of i. We remind the reader that we take the S and the A cohort to be behaviourally the same, and different from the V and the I cohorts. Let us deal first with the S, A and V cohorts. Our assumptions imply that

$$c_S(i) = c_A(i) \neq c_V(i).$$

For simplicity we choose

$$c_j(i) = \frac{C_j}{1 + \kappa_j i}, \quad j \in \{S, A, V\} \tag{3}$$

for some positive constants C_j and κ_j. Of course other choices of dependence on i are possible. Human psychology suggests that

$$\kappa_V < \kappa_S = \kappa_A.$$

The simplest assumption that is already sufficiently interesting is taking $\kappa_V = 0$ and $\kappa_S = \kappa_A$. Concerning C_j, it is logical to assume that $C_A = C_S = C_V =: C_0$.

Note that for all cohorts $j \in \{S, A, V\}$,

$$c_j(0) = C_0,$$

as it should be.

Considering the I cohort, we assume that the contact propensity satisfies

$$c_I(i) = \frac{C_0}{1 + \kappa_I i},$$

with $\kappa_I = \kappa_S = \kappa_A$, but with the additional clinically plausible stipulation that only a fraction $\xi \in (0, 1)$ of this cohort that exhibits clinical symptoms participates in social interaction. Clearly, ξ is a function of the virus strain in the population which impacts on hospitalisation, and of the psychological pressure that influences rates of self-isolation. We call ξ the **participation ratio**.

We will now discuss in detail the rate of loss of the susceptibles due to contact with the infected cohort.

Assuming random mixing, the proportion of people from the I cohort a susceptible individual, given participation ratio ξ, will meet under normal circumstances is $c_S(i)\xi I/N_0$. However, out of these, the proportion that will turn up is $c_I(i)/c_I(0)$. Hence the contact rate with infectives per capita for susceptibles is $c_S(i)\xi I c_I(i)/(N_0 C_0)$. It is easily checked that starting with an individual from I and computing the contact rate with susceptibles, we obtain the same contact rate of the two populations, so that the model is internally self-consistent.

The logic of the analysis of the S/A , V/A and V/I contacts is similar.

We need to specify the probability of a contact resulting in an infection. We denote by L_I the production rate of virus by an infective and by L_A the production rate by an asymptomatic. It is commonly believed that $L_I > L_A$.

Putting all this together, we have that

$$L_S(I,A) = c_S(i)\frac{\xi L_I c_I(i) I + L_A c_A(i) A}{N_0 C_0}$$

and

$$L_V(I,A) = c_V(i)\frac{\xi L_I c_I(i) I + L_A c_A(i) A}{N_0 C_0}.$$

Note that a priori we do not know whether $\xi L_I > L_A$.

Finally, we divide the equations in (1) by N_0, set $s := S/N_0$, $a := A/N_0$, $r := R/N_0$ and $v := V/N_0$ and obtain

$$L_S(i,a) = \frac{c_S(i)}{C_0}(\xi L_I c_I(i) i + L_A c_A(i) a) \text{ and } L_V(i,a) = \frac{c_V(i)}{C_0}(\xi L_I c_I(i) i + L_A c_A(i) a),$$

so the final form of the equations is

$$\begin{aligned}
s' &= -\alpha_S L_S(i,a) s - \nu s + \beta r + \gamma v,\\
v' &= \nu(i) s - \alpha_V L_V(i,a) v - \gamma v,\\
i' &= \delta_S \alpha_S L_S(i,a) s + \delta_V \alpha_V L_V(i,a) v - \rho_I i,\\
a' &= (1-\delta_S)\alpha_S L_S(i,a) s + (1-\delta_V)\alpha_V L_V(i,a) v - \rho_A a,\\
r' &= \rho_I i + \rho_A a - \beta r.
\end{aligned} \tag{4}$$

Observe that unlike the situation with Marek's disease [2, 16], to which SARS-CoV-2 has many epidemiological similarities, people have a good notion in which cohort they are, unless they are asymptomatic carriers, who believe that they are in the susceptible cohort.

3. Computation of R_0

The disease-free equilibrium (DFE) for (4) is

$$s = s_0 := \frac{\gamma}{\gamma + \nu}, \quad v = v_0 := \frac{\nu}{\gamma + \nu}, \tag{5}$$

with the rest of the cohorts being zero.

Using the next generation matrix technique [26], with the relevant matrices all being 2×2, we obtain the following expression for the basic reproduction number R_0:

$$R_0 = \frac{C_0}{\gamma + \nu} \left[\xi L_I \frac{\gamma \alpha_S \delta_S + \nu \alpha_V \delta_V}{\rho_I} + L_A \frac{\gamma \alpha_S (1 - \delta_S) + \nu \alpha_V (1 - \delta_V)}{\rho_A} \right]. \tag{6}$$

It is worthwhile to understand the dependence of R_0 given by (6) has on ν, the rate of vaccination at the DFE. Differentiating (6) with respect to ν shows that it is monotone decreasing in ν, if the following inequality is satisfied:

$$L_A \rho_I \left[\alpha_V (1 - \delta_V) - \alpha_S (1 - \delta_S) \right] < \rho_A \xi L_I \left[\alpha_S \delta_S - \alpha_V \delta_V \right], \tag{7}$$

and monotone increasing if the opposite inequality holds.

We observe that (7) automatically holds if

$$\lambda := \frac{\alpha_V (1 - \delta_V)}{\alpha_S (1 - \delta_S)} \leq 1. \tag{8}$$

The value of λ is clearly strain- and vaccine-dependent.

Let us assume now that $\lambda > 1$. If the participation ratio ξ is 1, so that no people in cohort I are self-isolating or hospitalised, then the biologically reasonable assumptions $L_I > L_A$, $\rho_A > \rho_I$, $\delta_s > \delta_V$ and $\alpha_S > \alpha_V$ imply that that inequality (7) is satisfied. This is true because

$$L_A \rho_I \left[\alpha_V (1 - \delta_V) - \alpha_S (1 - \delta_S) \right] < L_A \rho_I \left[\alpha_S \delta_S - \alpha_V \delta_V \right] < L_I \rho_A \left[\alpha_S \delta_S - \alpha_V \delta_V \right].$$

On the other hand, it is also clear that if the participation ratio is close enough to zero, that is, if most infectives are ill enough or self-isolate effectively and $\lambda > 1$, the opposite inequality to (7) holds. Hence we can define

$$\xi_{crit} = \frac{L_A \rho_I \left[\alpha_V (1 - \delta_V) - \alpha_S (1 - \delta_S) \right]}{L_I \rho_A \left[\alpha_S \delta_S - \alpha_V \delta_V \right]} \tag{9}$$

such that, under biologically plausible assumptions, if $\xi < \xi_{crit}$, R_0 is a monotone increasing function of ν and if $\xi > \xi_{crit}$, it is monotone decreasing. This observation has epidemiological significance.

Set

$$R_0^0 := R_0|_{\nu=0} = \frac{C_0\alpha_S}{\rho_A\rho_I}\left[\xi L_I\rho_A\delta_S + L_A\rho_I(1-\delta_S)\right] \tag{10}$$

and

$$R_0^\infty := \lim_{\nu\to\infty} R_0 = \frac{C_0\alpha_V}{\rho_A\rho_I}\left[\xi L_I\rho_A\delta_V + L_A\rho_I(1-\delta_V)\right]. \tag{11}$$

Thus we have proved

Proposition 1 *Assume that λ, defined in (8), satisfies $\lambda > 1$. Then* (a) *If $R_0^0 > 1 > R_0^\infty$ and $\xi > \xi_{crit}$, one can decrease R_0 below 1 by increasing the disease-free vaccination rate ν;* (b) *If $R_0^0 < 1 < R_0^\infty$ and $\xi < \xi_{crit}$, one can destabilise the DFE by increasing the disease-free vaccination rate ν.*

4. Numerical Examples

Here we provide some purely illustrative numerical results for the set of equations (4), with solutions computed using the Euler method with step size 0.01, where our time unit is one day. The solutions are computed up to $t = 5000$ to ensure convergence to steady state. We investigate the impact of vaccination rate $\nu \in [0.003, 0.03]$, which represents a time between approximately one month and one year to vaccinate the whole population. The values of parameters we use are displayed in Table 1.

We remark that many important parameters are not available from the voluminous data collected during the SARS-CoV-2 epidemic. Of particular interest to us are β and γ, the rates, respectively of the waning of natural immunity and of the vaccine-conferred one, as well as L_I and L_A, the rates of virus release by, respectively, the infective and asymptomatic individuals.

Table 1. Parameter values used in the numerical examples.

Parameter	Value
α_S, α_V	0.1, 0.1
β, γ	0.01, 0.01
δ_S, δ_V	0.8, 0.1
ρ_I, ρ_A	0.1, 0.1
L_I, L_A	0.4, 0.2
κ_j	1

From (9), using these parameter values, we have $\xi_{crit} = 0.5$. With this is mind, we first display solutions for $\xi = 0.8$ using $C_0 = 4$. Clearly, (8) does not hold. With such a high value of participation ratio (i.e. high level of social participation by members of

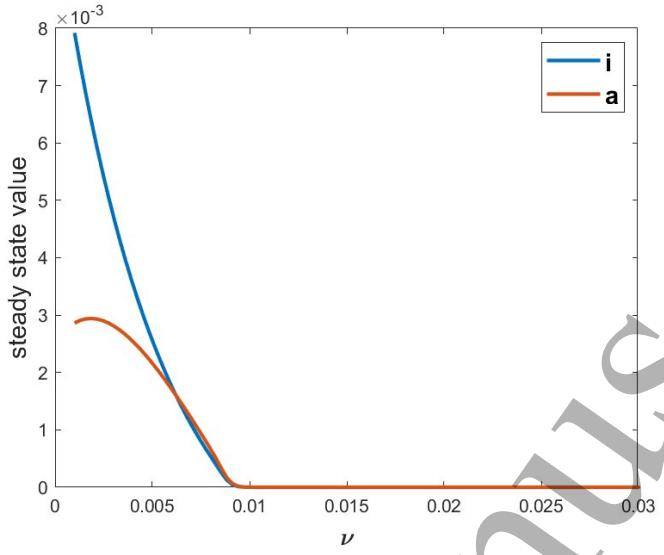

Figure 2. Numerical results for equations (4) showing steady state values of the i and a cohorts as functions of vaccination rate ν in the case $R_0^0 > 1 > R_0^\infty$.

the infected cohort), coupled with modest general contact propensity, equations (10) and (11) yield $R_0^0 = 1.184$ and $R_0^\infty = 0.800$. Thus R_0 monotonically decreases with ν, becoming less than one at $\nu = 0.009$.

In Figure 2 we show the steady state values of the infected cohort i and the asymptomatic cohort a as a function of ν. It is clear that below $\nu = 0.009$ the virus is endemic in the population, but declines with increasing vaccination rate until a disease-free equilibrium (DFE) becomes stable.

In Figure 3 we display the solutions for $\xi = 0.3$ and $C_0 = 6$. Here we have $R_0^0 = 0.816$ and $R_0^\infty = 1.200$. Thus R_0 monotonically increases with ν, becoming greater than one at $\nu := 0.009$. Figure 3 shows that at $\nu = 0.009$ the DFE is destabilised by increased vaccination rate to make the virus endemic in the population. This counter-intuitive result comes from the increase in the asymptomatic population caused by the leaky vaccine itself.

We note finally that we find no evidence of backwards bifurcation such as occurs in [4] in our numerics. In a sense, this is not surprising, as very often [8, 13] backward bifurcation requires that $\beta \gg \gamma$, that is, the rate of waning of natural immunity is much faster than the rate of waning of vaccine-conferred immunity and, in the absence of epidemiological evidence, we have taken these parameters to be equal. In future work we will use the techniques advocated in [19] to formulate conditions for backward bifurcation in our model (4). Note that Nadim and Chattopadhyay [21] obtain backward bifurcation in a model of SARS-CoV-2 that takes lockdowns explicitly into account, which we do not do.

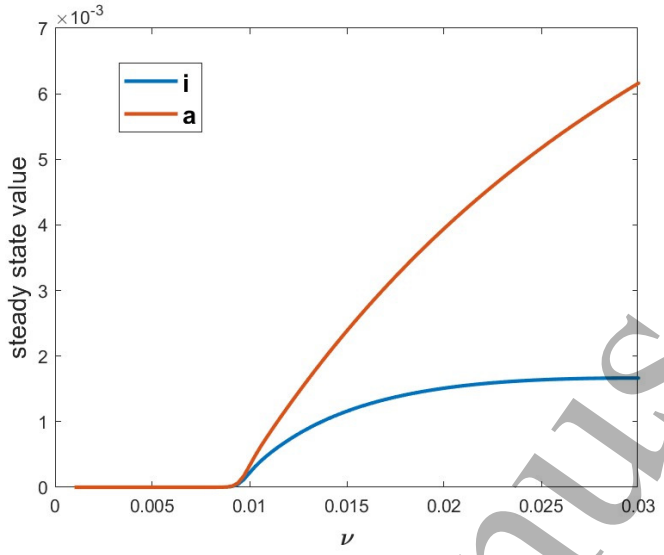

Figure 3. Numerical results for equations (4) showing steady state values of the i and a cohorts as functions of vaccination rate ν, in the case $R_0^0 < 1 < R_0^\infty$.

5. Summary and Conclusions

We have carefully developed a set of ODEs for the dynamics of the COVID-19 epidemic in the spirit of [5], having analysed in detail the influence of both vaccination rate and public health information in the disease transmission rates. In (8) we have also introduced in our opinion an interesting descriptor of the interaction between strain and vaccine properties, λ.

The basic reproduction number R_0 has been derived for this model, and reveals interesting behaviour:

Firstly, we see that it is possible for increased vaccination rates to drive the infection from an endemic state to a disease-free-equilibrium (DFE), as might reasonably be expected. However, this is not the only possibility, and indeed it relies upon general social constraint throughout the population.

Secondly, it is possible for vaccination to actually destabilise a DFE. If the disease is well controlled by the disciplined self-isolation of infected individuals who display clinical symptoms of the disease, high levels of vaccination give rise to high proportions of asymptomatics who, by the very nature being symptom-free, mix within society and cause endemic levels of the disease.

Thirdly, even with high vaccination rates, if infected people do not self-isolate, the disease remains endemic, albeit at a level that is lowered by increased vaccination (in this case, $R_0^0 > R_0^\infty > 1$).

Thus we see that public health information and policy have vital roles to play in the

outcome of a vaccination program, and care must be taken to develop a coherent public health package when tackling a pandemic like COVID-19, which takes into account the properties of pathogen strains and vaccine quality.

Finally, we consider the parameters β, γ, L_I and L_A to be of crucial importance in the analysis of SARS-CoV-2 and related, asymptomatics-influenced epidemics and would encourage epidemiologists to devise methods for their estimation.

Data Availability Statement. This paper does not involve any dataset.

Conflict of interest. The authors declare no conflict of interest.

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