Research article

Possible counter-intuitive impact of local vaccine mandates for vaccine-preventable infectious diseases

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Abstract: We modeled the impact of local vaccine mandates on the spread of vaccine-preventable infectious diseases, which in the absence of vaccines will mainly affect children. Examples of such diseases are measles, rubella, mumps, and pertussis. To model the spread of the pathogen, we used a stochastic SIR (susceptible, infectious, recovered) model with two levels of mixing in a closed population, often referred to as the household model. In this model, individuals make local contacts within a specific small subgroup of the population (e.g., within a household or a school class), while they also make global contacts with random people in the population at a much lower rate than the rate of local contacts. We considered what would happen if schools were given freedom to impose vaccine mandates on all of their pupils, except for the pupils that were exempt from vaccination because of medical reasons. We investigated first how such a mandate affected the probability of an outbreak of a disease. Furthermore, we focused on the probability that a pupil that was medically exempt from vaccination, would get infected during an outbreak. We showed that if the population vaccine coverage was close to the herd-immunity level, then both probabilities may increase if local vaccine mandates were implemented. This was caused by unvaccinated pupils possibly being moved to schools without mandates.

Keywords: stochastic SIR epidemic; household model; vaccine preventable infectious diseases; vaccine mandates; branching processes

1. Introduction

1.1. Background

Measles is a viral disease that yearly kills over a hundred thousand people worldwide [1]. It is one of the most contagious diseases known, with a basic reproduction number R_0 estimated to be between 12 and 18 [2] (see Section 1.2 and [3] for a definition of R_0). Because measles is extremely contagious and immunity after infection is typically life-long, in the absence of vaccination, most people will be infected before reaching adulthood, hence measles is often called a childhood disease.

In some countries, measles and other vaccine-preventable infectious diseases, such as rubella, mumps, and pertussis, are still endemic. That is, they are always present and cause regular peaks of prevalence. In many other countries, measles is largely absent because of successful vaccination campaigns [1, 4], which caused *herd-immunity* in the population [3, p69]. We focus on those countries. In several of the countries in which there have not been considerable outbreaks of measles for decades, the vaccine coverage has been declining, and there is fear that the "pool of susceptibles" will grow big enough to lose herd-immunity and allow for new waves of infection. Governments and public health authorities try to tackle this with campaigns to promote vaccine uptake and sometimes by legislation, which makes some vaccines compulsory for children, e.g., in Italy [5], or considering giving schools/daycare centers the freedom to use a vaccine mandate, as was considered in the Netherlands [6]. The ethical debate on whether childhood vaccination should be mandatory or not is a complex and contested topic. See, e.g., [7] for a review of the ethical literature.

In this paper, we consider the case in which vaccination is not mandatory for everybody and the decision of requiring the vaccine is left to the schools, as was proposed in the Netherlands. In this situation, children who cannot get a vaccine for medical reasons can freely choose where to attend school, whether it has a mandate or not. We refer to those children as *medically exempt* children. So, in this situation, unvaccinated children that are not medically exempt all go to schools without a mandate, and in those schools the fraction of unvaccinated children may be higher than if vaccine mandates are not allowed, which (as we show) may lead to higher risk of outbreaks of the vaccine-preventable diseases.

Skepticism and opposition toward vaccination represent a serious matter, to the point that the World Health Organization (WHO) has listed *vaccine hesitancy* as one of the ten threats to global health in 2019 [8]. Fear of possible side effects, religious convictions, and spread of misinformation are some of the causes of decline in vaccination coverage [9, 10]. It is reasonable to argue that, if given the choice, parents who are reluctant toward vaccines are more likely to enroll their children in schools that do not require vaccination. On the other hand, it is likely that medically exempt children go to schools that require vaccination, in order to obtain a safer environment. We are interested in the question whether and under which conditions local vaccine mandates will lead to increased risk of infection for unvaccinated children that are medically exempt.

1.2. Model

In this paper, we use a mathematical model to analyze the spread of measles among elementary school-aged children, where we take into account that most spread of the pathogen takes place within classes. Although we take measles as the pathogen of interest, the analysis applies equally to other highly infectious vaccine preventable diseases. We use a stochastic SIR (susceptible, infectious, recovered) epidemic model in which the relevant population is closed and partitioned in school classes. So, the total number N of children is fixed: We do not consider enrollments nor graduations during the time of study, which correspond to births and deaths, respectively, in the usual SIR model terminology, and we assume that the time scale of an outbreak is such that we may ignore children moving to different classes. We define n_c as the number of children per class, which we, for convenience of exposition, consider fixed, and m_c as the number of classes. So, $N = n_c m_c$. In Section 4.1, we show that the mathematical analysis is easily expanded to allow for variation in the class sizes. We remark that, strictly speaking, we analyze our model for a sequence of populations, where $N \to \infty$.

In SIR models, each individual can be *susceptible* to the disease, *infectious* or *recovered* and immune. A contact trough which the virus is spread is called an *infectious contact*. An infectious individual remains so for a random length of time that is called the *infectious period*, during which they are able to infect other individuals in the population. In our analysis we only focus on the probability that an introduction of a virus may lead to a large outbreak and on the probability that a given child gets infected. We do not consider the dynamics of such an outbreak in real time. Because of this, possible exposed/latent periods between a child getting infected and becoming infectious have no impact on our quantities of interest [11].

In many epidemic models, it is assumed that the population is homogeneous, which means that the individuals mix uniformly. However, in real life this is rarely the case. Here, we introduce heterogeneity by allowing individuals to have local (within class) and global contacts (between classes) with different probabilities. We assume that, conditioned on an individual being infected, the events that whether or not infectious contacts are made with different individuals are independent. So, we implicitly assume that the infectious period is not random [12]. Obviously, the infectious period for measles is random. However, the variance of time between the start of the infectious period and the onset of rash (which is close to the period that a child stays socially active and is likely to infect other children) is very small [13]. This makes that we can simplify the analysis and assume that contacts between individuals occur independently [12]. In our model, an infectious individual makes an infectious contact with each classmate with probability p_L, which for the moment we assume to be equal to 1, and with other individuals in the population with probability p_G. This model is also referred to as the *household model*, see e.g., [14] or [11, Section 6.3], since people are divided into small groups (such as workplaces, households, or classrooms) where transmission of the virus is more frequent. The global transmission probability is typically much smaller than the local probability, and in our case we assume that p_G is asymptotically inversely proportional with the population size. That is, if we consider a sequence of epidemic models indexed by the population size N, then there exists a constant $\lambda_G \in (0, \infty)$ such that $\lim_{N \to \infty} N p_G = \lambda_G$. This assumption implies that in the model, the rate of global contacts made by each child is asymptotically independent of the total population size [3, page 4].

The basic reproduction number R_0 is the expected number of infections caused by a "typical" infected individual infected during the initial stages of an epidemic in a population that is mostly susceptible [3] (so, R_0 refers to a situation in which there is no vaccination.) In homogeneously mixing populations, defining R_0 is straightforward. Although there is some issue with deciding what a typical infected individual is, it is also possible to define R_0 and other reproduction numbers for household models [15, 16]. In Section 2, we deduce the model parameter λ_G from estimates of R_0 from literature.

For measles, the basic reproduction number R_0 is estimated to be between 12 and 18 [2, 13], which makes the virus one of the most contagious viruses in the world. Assuming $p_L = 1$ means that if one of the unvaccinated children in a class gets the disease, then they will infect all of their unvaccinated classmates. Setting $p_L < 1$ would complicate the exposition considerably, while the qualitative behavior of the model stays similar, as long as p_L is large enough (say, larger than 4 divided by the class size, when the probability that the entire class gets infected is roughly 98%); see Section 4.2.

Because $p_L = 1$, a child and their non-vaccinated classmates together can be seen as a "super individual", of which the susceptibility and infectivity is proportional to the number of unvaccinated children in the class. We assume that the latent period of measles is relatively long, so that we can safely assume that all secondary infections in a class are directly caused by the initially infected child

in the class, and not by another secondary infected in the population. Lastly, we define a time unit as the fixed infectious period [11, p4].

To analyze the impact of vaccination (or the lack thereof), we assume that the vaccine against measles is perfect, i.e., once vaccinated, a person can neither get infected nor infect other people. So, vaccinated people are effectively omitted from the model. A fraction v of the population is vaccinated; we call $N_u(v) = (1-v)N$ the number of unvaccinated children, which are initially the susceptibles in our model. Another way to introduce vaccines is to say that each child is independently vaccinated with probability v. The two assumptions lead to slightly different models, but since they are asymptotically equivalent, we use one or the other depending on what is more convenient in the situation. For example, when it is useful to think about N_u as a fixed number, we will use the first assumption; if we want the number of initially susceptible children in different classes to be independent, then the second model is better.

In order to protect children at a school, the school may (if allowed by law) institute a local vaccine mandate, which requires all pupils that are not medically exempt to be vaccinated. In our model, we denote the fraction of classes with such a mandate by π. We require $\pi \leq v$ for reasons of consistency; see Section 2.

2. Analysis

To evaluate the global infection rate λ_G for given R_0, we consider a situation with no vaccines nor mandates, i.e., both v and π are set to zero. Recall that classes are all of fixed size n_c. We distinguish between children that are infected through a global contact and children that are infected within a class through a local contact. The first group of children are the *primary cases* or *type-one individuals*, which are the first individuals of their classes to get infected; each primary case will then infect all the $n_c - 1$ other (unvaccinated) children in their class, which we refer to as *secondary cases* or *type-two individuals*. We assume that both types make global contacts (and cause primary cases) at rate λ_G, but only primary cases cause secondary cases. For this household model, it is known that the epidemic threshold parameter R_0 is the dominant eigenvalue of the *next generation matrix*

$$A = \begin{bmatrix} \lambda_G & n_c - 1 \\ \lambda_G & 0 \end{bmatrix} \tag{2.1}$$

where element $A_{i,j}$ is given by the mean number of type-j individuals that a type-i individual infects during their infectious period ([14, 17]). So, R_0 solves

$$-(\lambda_G - R_0)R_0 - \lambda_G(n_c - 1) = 0.$$

So,

$$\lambda_G = \frac{R_0^2}{R_0 + n_c - 1} \tag{2.2}$$

If $R_0 = 15$ and $n_c = 25$, this leads to $\lambda_G \approx 5.8$.

Note that in [17], the dimension of the next generation matrix is $n_H \times n_H$, where n_H is the maximal household size. In our case, it is enough to consider a 2×2 matrix because we only need to divide individuals into *primary* and *secondary* cases. In Section 4.2, when dealing with $p_L < 1$, we need higher dimensional matrices.

The dominant eigenvalue of the matrix A is sometimes called the individual reproduction number and is denoted by R_I [16]. Assuming $p_L = 1$, the reproduction numbers R_0 and R_I are the same because all secondary cases are infected by primary ones only.

As mentioned before, there are in total m_c classes, each with n_c students. So, $N = m_c n_c$. The number of classes without mandate is $m_c(1 - \pi)$, so the maximum possible number of unvaccinated children is $m_c n_c(1 - \pi) = N(1 - \pi)$. Therefore, the number of unvaccinated children $N(1 - v)$ has to be less or equal than $N(1 - \pi)$. So, for the model to be consistent, we require $\pi \le v$.

In a class without a vaccine mandate, the distribution of the number X of unvaccinated children is $Bin(n_c, \frac{1-v}{1-\pi})$ and we write $\pi_k = P(X = k)$. In what follows, we use

$$u := \frac{1 - v}{1 - \pi}.$$

The expected number of unvaccinated children per class is given by $\mu_c = n_c u = n_c \frac{1-v}{1-\pi}$. We observe that the probability that a child, chosen uniformly at random among the unvaccinated children in the population, belongs to a class with k unvaccinated children is given by the size-biased distribution determined through

$$\bar{\pi}_k = \frac{k\pi_k}{E[X]} = \frac{k\binom{n_c}{k}(u)^k(1 - u)^{n_c-k}}{n_c u} = \binom{n_c - 1}{k - 1}u^{k-1}(1 - u)^{n_c-1-k+1}. \tag{2.3}$$

We let $\bar{X}$ be a random variable with probability masses defined through the $\bar{\pi}_k$ and we notice that $\bar{X}$ is distributed as $Y + 1$, where $Y = Y(v)$ is a random variable with a binomial distribution with parameters $n_c - 1$ and u.

We are interested in how the reproduction number $R_{0,e} = R_{0,e}(v, \pi)$ (where the e stands for effective) changes once the vaccine is introduced in the population and depends on v and π. To analyze this, we compute the dominant eigenvalue of the next generation matrix

$$A' = A'(v, \pi) = \begin{bmatrix} \lambda_G(1 - v) & (n_c - 1)\frac{(1-v)}{(1-\pi)} \\ \lambda_G(1 - v) & 0 \end{bmatrix},$$

where $\lambda_G(1 - v)$ is the infection rate of global contacts between the unvaccinated. The results are depicted in Figure 1.

Using λ_G from (2.2), we also compute $R_{*,e} = R_{*,e}(v, \pi)$, which is the *household reproduction number* after vaccination for the household model [14, 16] and represents the mean number of clumps (or in our case, classes) that are infected by a typical infectious clump/class. It is defined to be $R_{*,e} = \lambda_{G,v}\mu$, where $\lambda_{G,v}$ is the expected number of global infections caused by a typical infected individual, and $\mu = \mu(v)$ is the mean number of infected children in an infected class. $\lambda_{G,v}$ is given by $\lambda_G(1 - v)$, while

$$\mu = \mathbb{E}[\bar{X}] = \sum_{k=1}^{n_c} k\bar{\pi}_k = 1 + (n_c - 1)u.$$

The latter follows from the size biased distribution in (2.3). Substituting this in the expression for $R_{*,e}$, we obtain

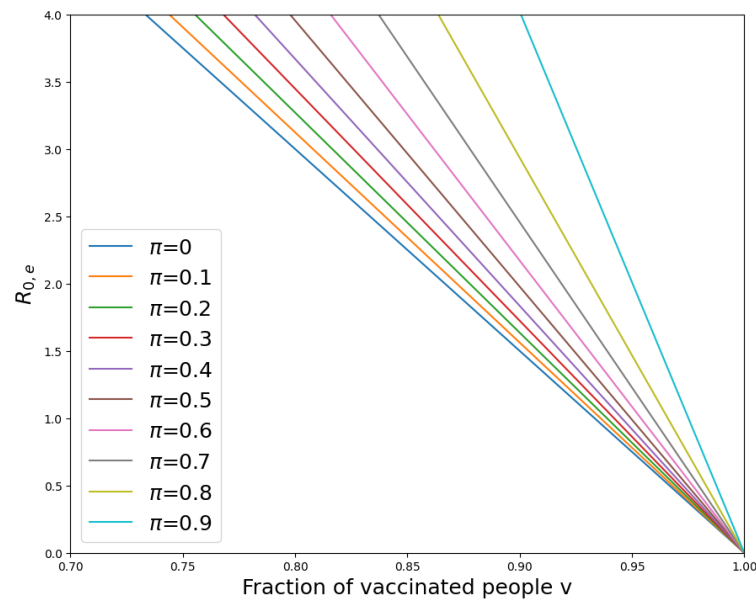$$R_{*,e} = [1 + (n_c - 1)u](1 - v)\lambda_G. \tag{2.4}$$

Figure 1. $R_{0,e}$ as a function of v, if both vaccines and mandates are introduced.

The household reproduction number $R_{*,e}$, as well as most other reproduction numbers defined in literature, including $R_{0,e}$, is useful because an epidemic goes extinct with probability 1 if and only if $R_{*,e} \leq 1$ [16]. The minimal value of v for which $R_{*,e} \leq 1$ is *the critical vaccination coverage v_{cr}*.

$$R_{*,e} > 1 \iff \frac{(1-v)^2}{(1-\pi)}\lambda_G(n_c - 1) > 1 - (1-v)\lambda_G.$$

Since n_c represents the number of children in a class, the left-hand side is always nonnegative. So, if the expected number of contacts with unvaccinated children (i.e., $(1-v)\lambda_G$) is greater than 1, i.e., $1 - v > \frac{1}{\lambda_G}$, then the epidemic is above threshold. Assume instead that both terms are positive; then this inequality gives us an upper bound for the fraction $1 - \pi$ of schools without mandate:

$$1 - \pi < \frac{(1-v)^2\lambda_G(n_c - 1)}{1 - (1-v)\lambda_G}.$$

So, if the number of schools without mandate is small enough and the unvaccinated children are concentrated in those schools, the value of $R_{*,e}$ is above 1 (see Figure 2).

2.1. Branching process approximation

2.1.1. The branching process

Assume that the epidemic starts with one randomly chosen unvaccinated child in the population and say that the class of the child has ℓ non-vaccinated children. Then, the probability that a class with k unvaccinated children gets infected by the class of the initially infected child is given by the following expression:

$$\frac{\pi_k[1 - (1 - p_G)^{k\ell}]}{\sum_{k'=0}^{n_c} \pi_{k'}[1 - (1 - p_G)^{k'\ell}]}. \tag{2.5}$$
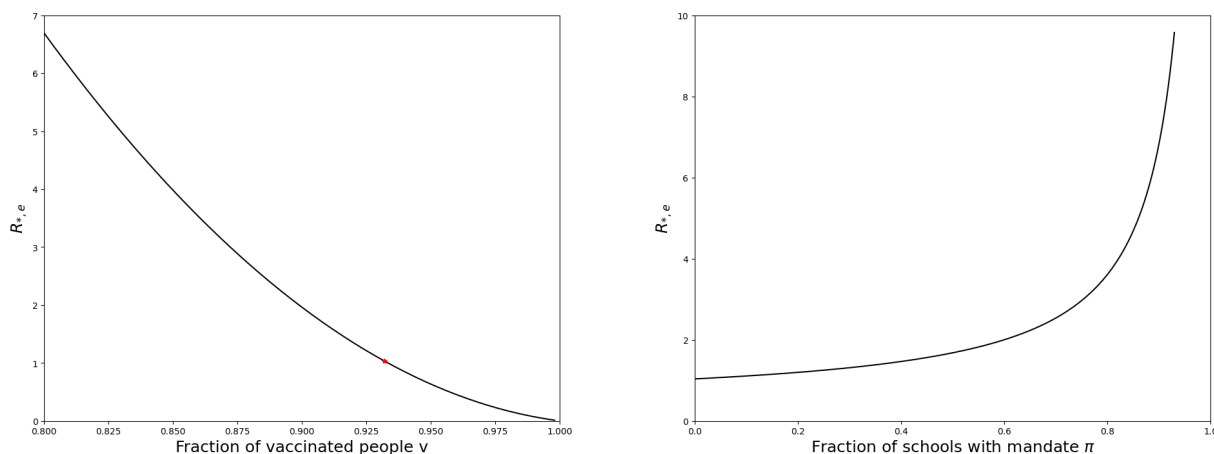
Figure 2. Left: plot of $R_{*,e}$ as a function of $v \in [0.8, 1]$ when $R_0 = 15$, $n_c = 25$, and $\pi = 0$. The red asterisk denotes when $R_{*,e} = 1$ and we call v_{cr} the correspondent vaccination coverage. Right: plot of $R_{*,e}$ as a function of π when $v = v_{cr}$, again for $R_0 = 15$ and $n_c = 25$.

When $p_G \ll 1$ (as we assume), the expression $1 - (1 - p_G)^{k\ell}$ is well approximated by $k\ell p_G$, and we obtain that (2.5) is approximated by

$$\frac{\pi_k p_G k\ell}{\sum_{k'=0}^{n_c} \pi_{k'} p_G k'\ell} = \frac{\pi_k k}{\sum_{k'=0}^{n_c} \pi_{k'} k'} = \bar{\pi}_k.$$

If the number of classes is large, also the population size N and the number of unvaccinated children N_u are large. So, we can use a branching process approximation to describe the initial stages of the epidemic: In those early stages, infectious individuals contact only individuals that – with large probability – are still susceptible, so the number of new infections caused by households are asymptotically independent and global contacts lead to a tree-structured epidemic generated graph, which has the law of a multitype branching process, in which individuals correspond to classes in the epidemic process. A class of size k (i.e., a class with k unvaccinated children) corresponds to a type-k individual of the Galton-Watson approximating tree. Since the infection rate for global contacts between unvaccinated is $\lambda_G(1 - v)$, the offspring of a type-k individual is $\sim Poiss(\lambda_G(1 - v)k)$ [18].

More formally, by a birthday problem approach [21, p.24], we can argue that, with high probability, the first k global contacts, $k = o(\sqrt{N_u})$, are all with different households. Furthermore, it is well-known (e.g., [11, Chapter 3]) that if we denote the number of infected children in an SIR epidemic by $|\mathcal{E}_{N_u}|$ and the size of the approximating branching process (i.e., a Galton-Watson process with offspring distribution described above) by $|\mathcal{B}|$, then as $N_u \to \infty$,

$$\mathbb{P}(|\mathcal{E}_{N_u}| > \log N_u) \to \mathbb{P}(|\mathcal{B}| = \infty).$$

If $|\mathcal{E}_{N_u}| > \log N_u$, we talk about a major outbreak of the epidemic.

2.1.2. The probability of avoiding infection for a medically exempt child

If the first infectious child belongs to a class of size k (i.e., a class with k unvaccinated children), then the probability that the process dies out is equal to the probability that the infectious offspring

of each student in the first infectious class dies out. This leads to the following expressions for $\bar{q}$, the extinction probability of the approximating branching process for an epidemic initiated by a uniformly chosen unvaccinated individual from the population.

$$
\begin{aligned}
\bar{q} &= \sum_{k=1}^{n_c} \mathbb{P}(\bar{X} = k) \sum_{\ell=0}^{\infty} \frac{[\lambda_G(1-v)k]^{\ell}}{\ell!} e^{-\lambda_G(1-v)k} \bar{q}^l \\
&= \sum_{k=1}^{n_c} \mathbb{P}(\bar{X} = k) e^{-\lambda_G(1-v)k(1-\bar{q})} \\
&= \sum_{j=0}^{n_c-1} \mathbb{P}(\bar{X} - 1 = j) e^{-\lambda_G(1-v)(j+1)(1-\bar{q})} \\
&= \sum_{j=0}^{n_c-1} \binom{n_c-1}{j} u^j (1-u)^{n_c-1-j} e^{-\lambda_G(1-v)(j+1)(1-\bar{q})} \\
&= \left(1 - u + u e^{-\lambda_G(1-v)(1-\bar{q})}\right)^{n_c-1} e^{-\lambda_G(1-v)(1-\bar{q})}
\end{aligned}
\tag{2.6}
$$

which means that $\bar{q}$ is a solution of the fixed point equation

$$
\bar{q} = \left(1 - u + u e^{-\lambda_G(1-v)(1-\bar{q})}\right)^{n_c-1} e^{-\lambda_G(1-v)(1-\bar{q})}.
$$

From the theory of branching processes [19], we know that $\bar{q}$ is the smallest solution of this equation, which is strictly less than 1 if and only if $R_{*,e} > 1$.

By assumption, a medically exempt child is enrolled in a school that has a vaccine mandate. This means that they belong to a class of effective size 1. By the epidemic generated graph representation of an epidemic [18], we know that p_{me}, the probability that a medically exempt child avoids being infected if a large outbreak occurs, is equal to the probability that a large outbreak occurs if the outbreak starts with a single infected child in a class of effective size 1. This result can also be obtained by using susceptibility sets [20] and see that the so-called forward and backward branching process have the same offspring distribution. Using arguments as in (2.6), we obtain that

$$
p_{me} = p_{me}(v, \pi) = e^{-\lambda_G(1-v)(1-\bar{q})}.
\tag{2.7}
$$

If there is no vaccine mandate, a medically exempt child is just like any other unvaccinated child and the probability of avoiding infection is $\bar{q}$ for the case that $\pi = 0$. We denote this probability by $p_{wm} = p_{wm}(v)$, where *wm* stands for "without mandate".

3. Results

Considering the entire population, we may expect that mandates make the global situation worse, because unvaccinated children have, on average, more other unvaccinated children in their class, and children in the same class contact each other at a higher rate than other pairs of children. However, in this situation, a medically exempt child can only be infected by global contacts. In the case without mandate, even if the global situation is more controlled, such a child could be enrolled in a class with other unvaccinated children, and the probability p_{me} for a given medically exempt child to avoid
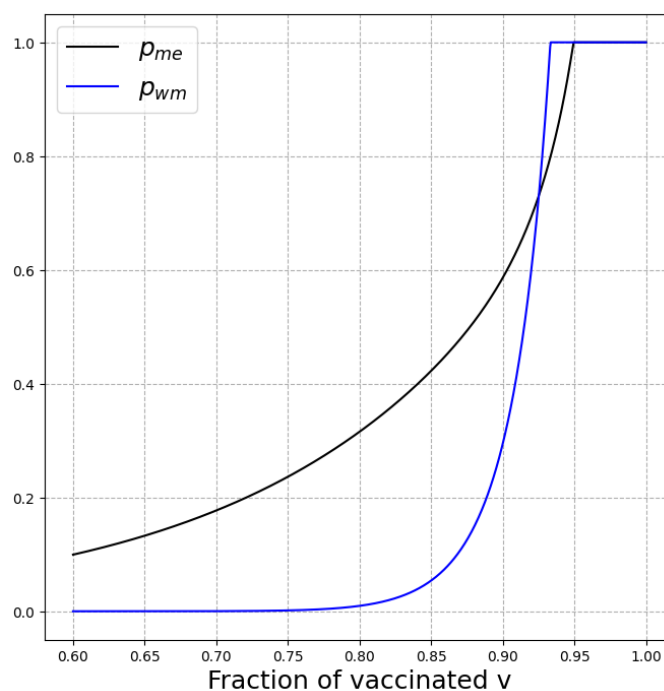
Figure 3. Plot of the probability p_{me} that a medically exempt child avoids infection as a function of v with π set to 0.5, and of the probability p_{wm} that a random unvaccinated child avoids infection in the case with no mandates ($\pi = 0$). We see that the two lines cross at $v_s \approx 0.925$. The minimal value of v for which $p_{wm} = 1$ is at $v \approx 0.933$, while the minimal value of v for which $p_{me} = 1$ is at $v \approx 0.95$.

infection in a situation with vaccine mandates may be larger than that probability in a situation without mandates p_{wm}. In this section, we analyze the model to justify this intuition.

In Figure 3 we consider the case where $\pi = 1/2$ and v is varying. Here, p_{me} is higher than p_{wm} up to $v \leq v_s \approx 93\%$, and we have the opposite inequality for $v > v_s$, where the s stands for switching level. The graph tells us that if v is low, a medically exempt child is profiting from vaccine mandates at schools, since they will for sure be enrolled in a class with no other unvaccinated children. However, when the vaccine percentage is sufficiently high, the probability of having only one unvaccinated child per class is already high and mandates worsen the global situation for everyone. Note that p_{wm} reaches 1 at a lower v value than p_{me}. This confirms the intuition that without mandates, a lower vaccine coverage is sufficient to guarantee herd-immunity.

In Figure 4, we again consider p_{me}, the probability that a medically exempt individual avoids infection, but now as a function of π, when $v = 0.93$ and $v = 0.90$. For $v = 0.93$, we can see that, for low values of π (lower than approximately 0.20), the medically exempt are more protected in the model with mandates. But if π increases, then the mandates are actually disadvantageous to the medically exempt children, even if they are the only unvaccinated children in their class. If $v = 0.90$, already without any vaccine mandates, the global spread of the disease is so easy that the global spread does not increase much by introducing vaccine mandates, while the relative protection of having no other unvaccinated children in the class is considerable and vaccine mandates are always beneficial for medically exempt children.
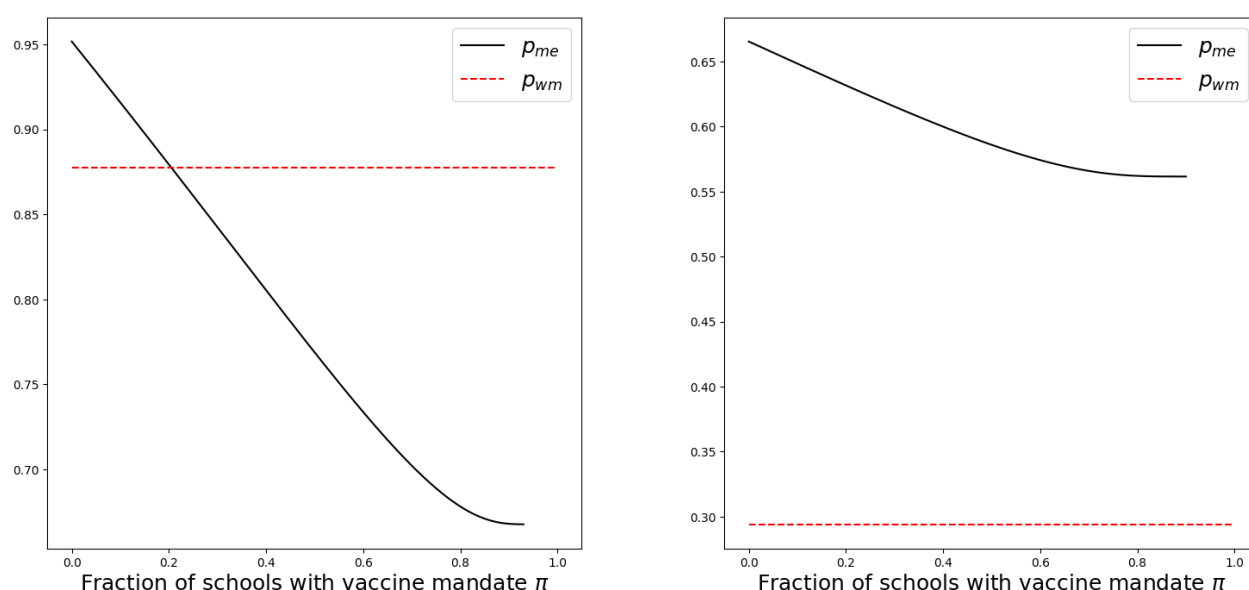
Figure 4. Plot of p_{me}, the probability that a medically exempt child avoids infection as a function of π, when $v = 0.93$ (left) and when $v = 0.90$ (right). We compare it with the probability p_{wm} that a random unvaccinated child avoids infection in the case with no mandates ($\pi = 0$).

Up to now, we have assumed that the introduction of mandates in a society will not change the vaccine percentage. This is not realistic, since some parents might vaccinate their children in order to let them go to the school of their preference. We call those parents "converted". We may think of v as the sum of two components: v_i is the initial proportion of vaccinated individuals and v_c is the additional portion of "converted". Note that $v_c \leq 1 - v_i$. The graph in Figure 5 shows a plot of $R_{*,e}$ as a function of v_c, when $\pi = 0.5$, $v_i = 0.9$, and $n_c = 25$. As expected, $R_{*,e}$ is strictly decreasing in v_c: If more people get "converted" and, therefore, more children get vaccinated, the overall public health situation improves.

4. Extensions of the basic model

In this section, we present some extensions to the basic model. In Section 4.1, we look at the case in which the number of children per class is random. In Section 4.2, we study the case of nontrivial local infection probabilities, with $p_L < 1$. Finally, in Section 4.3, we analyze the spread of other infectious diseases with basic reproduction number R_0 lower than the one of measles.

4.1. Random class sizes

Our analysis is easily extended to random class sizes, in which the number of children in a class is distributed as a random variable N_c. Call $p_k = \mathbb{P}(N_c = k)$; then the probability that a newly infected class is of size k is given by the size-biased version $\bar{N}_c$ of N_c and we define $\bar{p}_k = \mathbb{P}(\bar{N}_c = k)$. To evaluate the expected number of type-2 individuals that a type-1 individual infects (which is element $(1,2)$ of
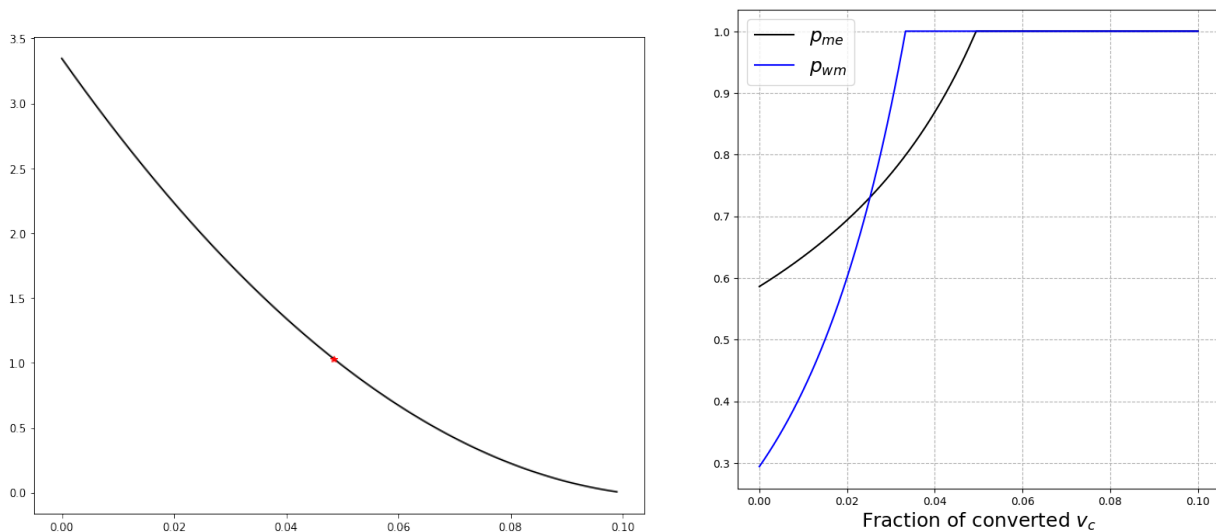
Figure 5. Left: $R_{*,e}$ as a function of v_c when $\pi = 0.5$, $v_i = 0.9$, and $n_c = 25$. The red asterisk is the intersection point with $y = 1$. (Right: Plot of p_{me} and p_{wm} as a function of v_c when $\pi = 0.5$, $v_i = 0.9$, and $n_c = 25$. Note that this figure may be deduced from Figure 3 with v restricted to $[0.9, 1]$. This is because we fix the initial fraction of vaccinated v_i and then define $v = v_i + v_c$ and compute p_{me} and p_{wm} with such combined v.)

matrix A in (2.1)), we compute

$$\mathbb{E}[\bar{N}_c - 1] = \sum_{k=1}^{\infty} (k-1)\bar{p}_k = \sum_{k=1}^{\infty} \frac{k(k-1)p_k}{\mathbb{E}[N_c]} = \frac{\mathbb{E}[N_c(N_c - 1)]}{\mathbb{E}[N_c]}.$$

For example, if $N_c \sim Poiss(m)$, say, with $m = 25$, then $\mathbb{E}[N_c] = m$ and, with the same procedure as in Section 2, we obtain $\lambda_G \approx 5.6$.

We denote the random number of unvaccinated per class after both vaccines and mandates are introduced in the model again by X. Then, X conditioned on $N_c = k$ is $Bin(k, u)$ distributed and

$$\mathbb{P}(X = l) = \sum_k \mathbb{P}(X = l|N_c = k)\mathbb{P}(N_c = k)$$

while

$$\mathbb{P}(\bar{X} = l) = \frac{l\mathbb{P}(X = l)}{\mathbb{E}[X]} = \sum_k \mathbb{P}(\bar{X} = l|N_c = k)\mathbb{P}(N_c = k). \tag{4.1}$$

We observe that

$$\mathbb{P}(\bar{X} = l|N_c = k) = \frac{l\mathbb{P}(X = l|N_c = k)}{\mathbb{E}[X|N_c = k]}$$

which implies, after some computations, that $\bar{X}$ conditioned on $N_c = k$ is distributed as $1 + Bin(k-1, u)$, and then allows us to compute the unconditioned distribution of $\bar{X}$ using (4.1).

4.2. Nontrivial local infection probabilities

Throughout all our analysis above, we have considered the case in which the local infection probability $p_L = 1$, which simplifies the model as explained in Section 1.2. Here, we want to study nontrivial cases with $p_L < 1$ that correspond to a different contact pattern.

Assume that an infectious individual infects their classmates with probability $p_L = 1/2$ per classmate during their infectious period. Setting both v and π to zero, we compute the global infection rate λ_G as in Section 2. We consider a class of size 25 in which one student gets infected from a global contact. The probability that a given one of their classmates avoids infection within two steps is $\frac{1}{2}\left(1 - \frac{1}{4}\right)^{23}$ and the probability that at least one of the classmates avoids infection is at least $24 \times \frac{1}{2}\left(1 - \frac{1}{4}\right)^{23} \approx 0$. This means that all the pupils of that class will have contracted the virus within two generations of infection with very high probability. A primary case infects, on average, $(n_c - 1)/2$ classmates, which will, in turn, infect as many remaining susceptibles. The next generation matrix (2.1) then becomes

$$A = \begin{bmatrix} \lambda_G & \frac{n_c-1}{2} & 0 \\ \lambda_G & 0 & 1 \\ \lambda_G & 0 & 0 \end{bmatrix}$$

and R_0 solves

$$R_0^2(\lambda_G - R_0) + R_0\lambda_G\frac{n_c - 1}{2} + \lambda_G\frac{n_c - 1}{2} = 0.$$

If $R_0 = 15$ and $n_c = 25$, then

$$\lambda_G = \frac{R_0^3}{R_0^2 + \frac{n_c-1}{2} + R_0\frac{n_c-1}{2}} \approx 8.1.$$

Recall that if $p_L = 1$, then $\lambda_G \approx 5.8$.

Now, introduce both vaccines and mandates in the model. We consider a class with $n + 1$ unvaccinated children, one of which is infected through a global contact. The epidemic that starts spreading via local contacts in the class is described by – to use the notation of Andersson and Britton [11, Chapter 2] – the *standard SIR epidemic model* $E_{n,1}(\lambda_L, 1)$, where λ_L indicates the local infectious rate. To compute the value of λ_L, we link it to p_L by observing that an infectious individual makes contacts with a given classmate at the time steps of a Poisson(λ_L/n) process; therefore, the probability $1 - p_L$ of having no contacts with a given individual is given by $e^{-\lambda_L/n}$.

We indicate by P_j^n the probability that j of the n initial susceptibles get infected during the (local) epidemic. The distribution of these probabilities is obtained by solving a lower triangular linear system given in [11, Chapter 2, Theorem 2.2]. Then, in this case, the probability of extinction $\bar{q}$ in (2.6) will be

$$\bar{q} = \sum_{k=1}^{n_c} \mathbb{P}(\bar{X} = k) \sum_{j=0}^{k-1} P_j^{k-1} \sum_{l=0}^{\infty} \frac{[\lambda_G(1 - v)(j + 1)]^l}{l!} e^{-\lambda_G(1-v)(j+1)} \bar{q}^l$$

$$= \sum_{k=1}^{n_c} \mathbb{P}(\bar{X} = k) \sum_{j=0}^{k-1} P_j^{k-1} e^{-\lambda_G(1-v)(j+1)(1-\bar{q})}. \tag{4.2}$$

We observe that, keeping p_L fixed and increasing the number of susceptibles (of the local epidemic) n, the probability P_n^n that everyone gets infected gets closer and closer to 1, as seen in Figure 6.

In Figure 7 we see, on the left, a plot of the probabilities p_{me} and p_{wm}, that a medically exempt child avoids infection in the case with and without class mandates, respectively, when $\pi = 1/2$, as functions of v. On the right, we can see a comparison between the cases $p_L = 1$ and $p_L = 1/2$. We observe that even if in the latter case the local contact probability is halved, the probability p_{me} that a medically exempt child avoids infection is higher in the first case. This is caused by the dependence on the value
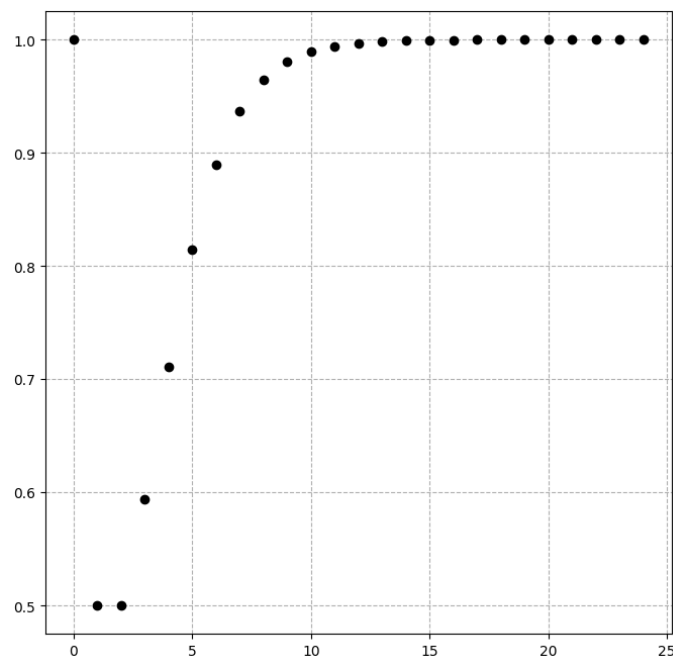
Figure 6. Value of P_n^n plotted as a function of n.

of the global infection rate λ_G, which we remember increases from 5.8 in the first case to 8.1 in the second: If R_0 stays the same but p_L decreases, then p_G increases, which means that the medically exempts are more exposed to the virus. For the probability p_{wm} that a random unvaccinated avoids infection in the case without mandates, we conversely see almost no difference in the two cases.

Similarly, we can analyze the cases $p_L = 1/3$ and $p_L = 1/5$. For the same reason as above, we can assume each individual in a class to be infected within a maximum of two generations of infectives. If one child is infected by a global contact, it will infect, on average, $(n_c - 1)/3$ and $(n_c - 1)/5$ classmates, respectively, which will, in turn, infect the remaining $2(n_c - 1)/3$ and $4(n_c - 1)/5$ children. The next generation matrices become

$$A_{1/3} = \begin{bmatrix} \lambda_G & \frac{n_c-1}{3} & 0 \\ \lambda_G & 0 & 2 \\ \lambda_G & 0 & 0 \end{bmatrix} \qquad A_{1/5} = \begin{bmatrix} \lambda_G & \frac{n_c-1}{5} & 0 \\ \lambda_G & 0 & 4 \\ \lambda_G & 0 & 0 \end{bmatrix}$$

which lead to $\lambda_G \approx 9.3$ and 10.7, respectively. Then, using the same expression for $\bar{q}$ found in (4.2), we obtain similar graphs for p_{me} and p_{wm}, as seen in Figure 8.

Finally, in Figure 9 we can see a comparison between the plots for p_{me} (on the left) and p_{wm} (on the right) for the different p_L values we have previously considered. What we observe is that, while p_{me} tends to decrease when p_L decreases, p_{wm} has the opposite behavior. When mandates are not present and unvaccinated are uniformly spread in the classrooms, having a lower p_L value slightly increases the probability p_{wm} of avoiding infection, as the clusters of infectives forming during the spread of the virus are smaller. When mandates are present, the probability p_{me} that a medically exempt child avoids infection decreases with p_L, because the global infection rate λ_G reaches higher values.
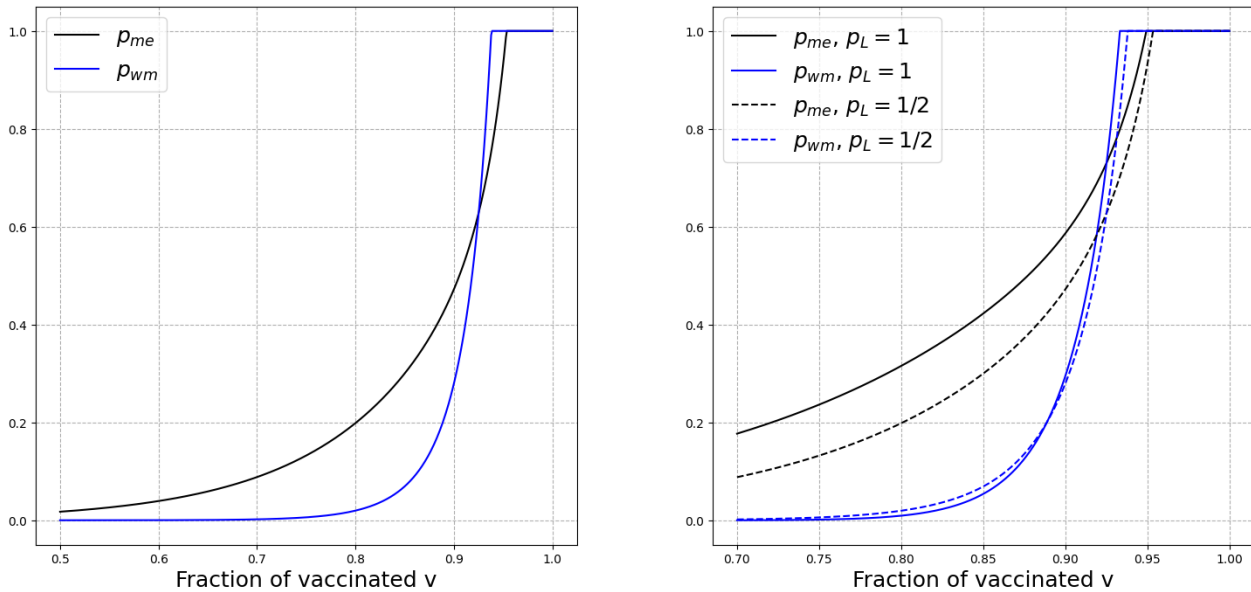
Figure 7. Left: Plot of the probability p_{me} that a medically exempt child avoids infection as a function of v with π set to 0.5, and of the probability p_{wm} that a random unvaccinated child avoids infection in the case with no mandates ($\pi = 0$), when the local probability p_L is set to 1/2. The two lines cross at $v_s \approx 0.925$, as in Figure 3. (Right: Contrast between the cases $p_L = 1$ (see Figure 3) and $p_L = 1/2$.)
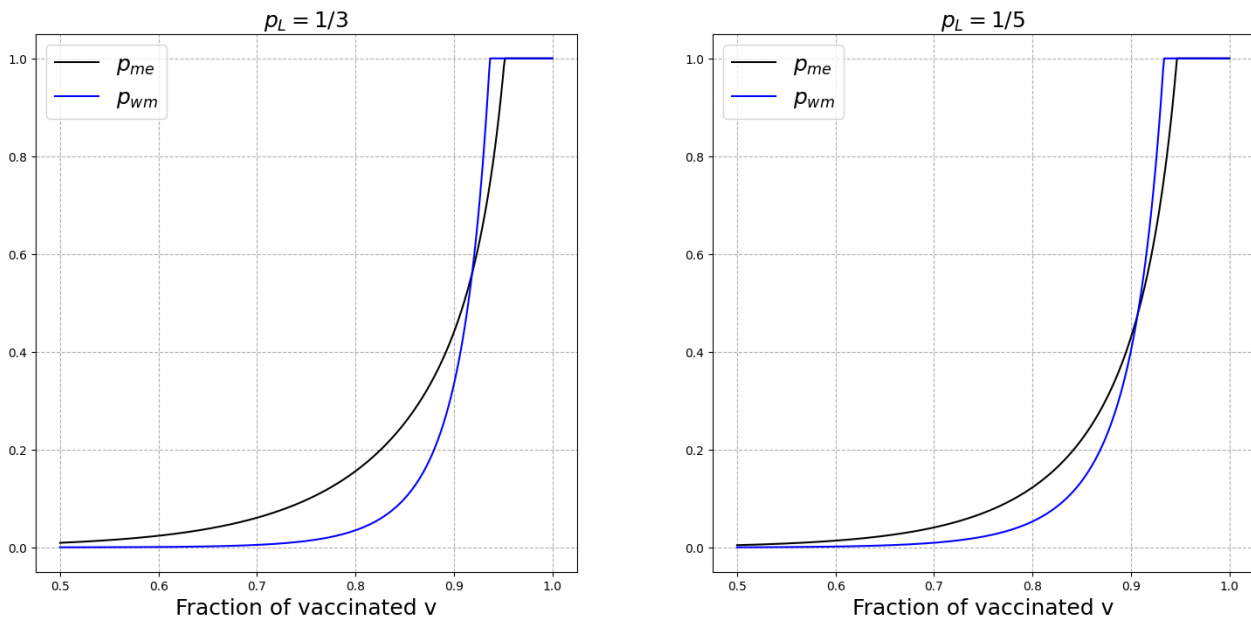

Figure 8. Plot of the probability p_{me} that a medically exempt child avoids infection as a function of v with π set to 0.5, and of the probability p_{wm} that a random unvaccinated child avoids infection in the case with no mandates ($\pi = 0$), when the local probability p_L is set to 1/3 (left) and 1/5 (right).
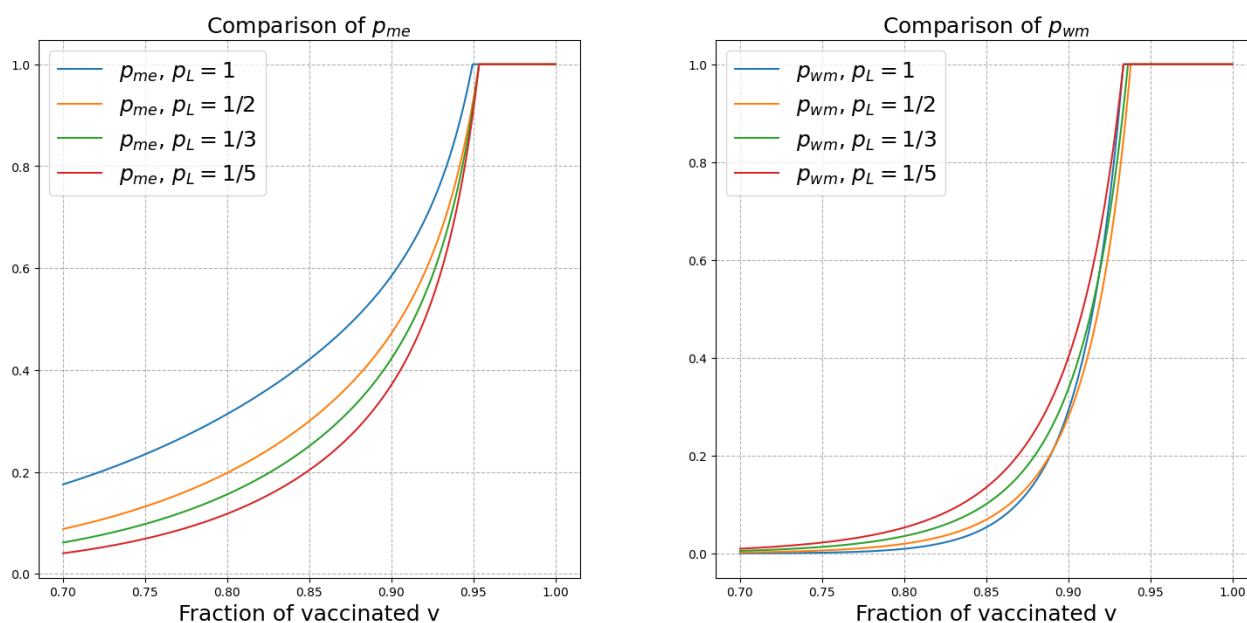
Figure 9. Comparison of the plots of the probability p_{me} that a medically exempt child avoids infection as a function of v when $\pi = 0.5$ (left), and of the probability p_{wm} that a random unvaccinated child avoids infection in the case of no vaccine mandates (right), for the different p_L values considered above.

4.3. Less infectious diseases

We are now interested in considering the case of a less infectious disease than measles, such as mumps or rubella; as an example, we take the basic reproduction number $R_0 = 6$.

Comparing Figures 3 and 10, we see that in the first case, the functions describing p_{me} and p_{wm} increase much faster than in the case $R_0 = 6$. This means that with a less infectious virus, the difference between the cases with and without mandates is more pronounced. In fact, as shown in Figure 10, a medically exempt child escaping infection is significantly more probable in the case with vaccine mandates, up to the switching point $v \approx 0.82$. However, there is a wider range of vaccine coverage v, for which medically exempt children are worse off with than without vaccine mandates.

In Figure 11, we see how p_{me} and p_{wm} vary as functions of π when the value of v is fixed and close to the herd immunity level; the behavior is the same as the one observed in Figure 4.

5. Discussion

In this paper, we study how vaccine mandates might affect the spread of an infectious disease in elementary schools, and especially how the probability of being infected changes for medically exempt children. This effect may be counterintuitive and local mandates might worsen the risks for medically exempt children. This work was inspired by a law that was proposed by the Dutch House of Representatives and was later rejected by the Senate [6]. In this measure, childcare owners could freely decide whether or not joining the public vaccination program was compulsory for children to enroll at the daycare.
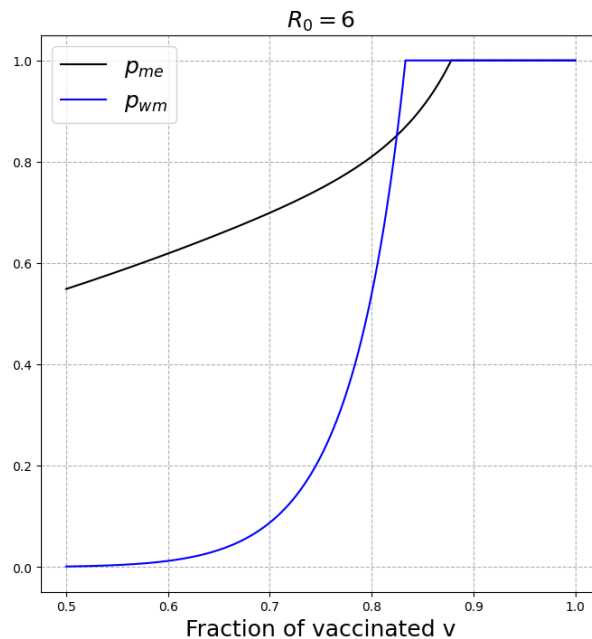
Figure 10. Plot of the probability p_{me} that a medically exempt child avoids infection as a function of v with π set to 0.5, and of the probability p_{wm} that a random unvaccinated child avoids infection in the case with no mandates ($\pi = 0$). We see that the two lines cross at $v_s \approx 0.82$. The minimal value of v for which $p_{wm} = 1$ is at $v \approx 0.83$, while the minimal value of v for which $p_{me} = 1$ is at $v \approx 0.88$. To compare it with the case $R_0 = 15$, see Figure 3.
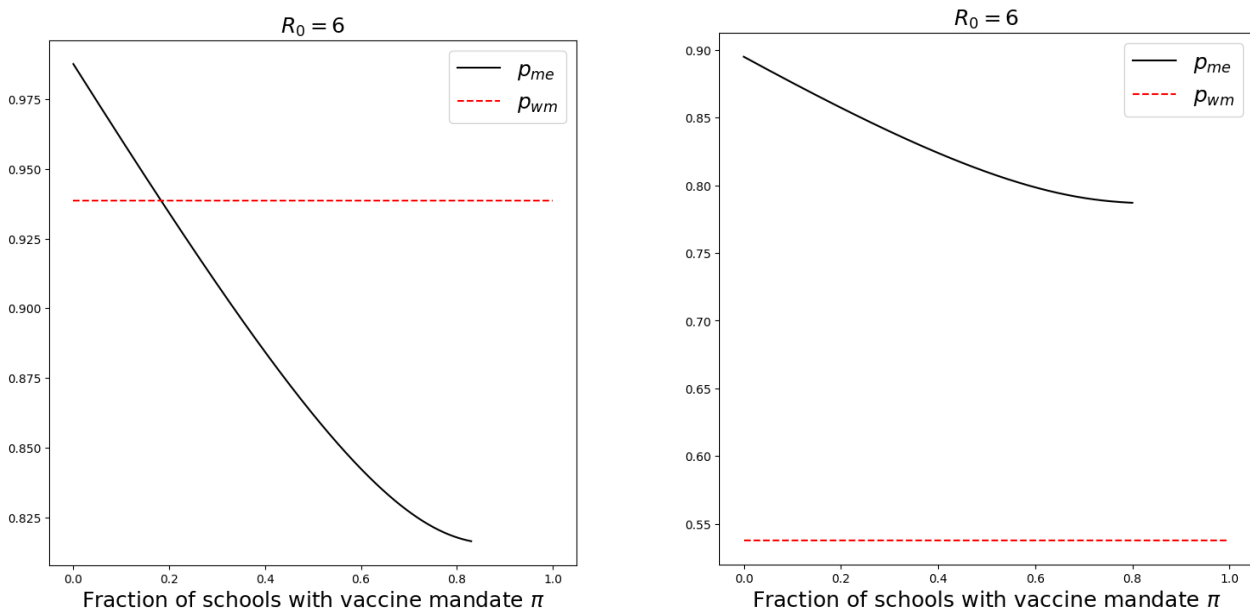

Figure 11. Plot of p_{me}, the probability that a medically exempt child avoids infection as a function of π, when $v = 0.83$ (left) and when $v = 0.80$ (right). We compare it with the probability p_{wm} that a random unvaccinated child avoids infection in the case with no mandates ($\pi = 0$). See Figure 4 for comparison with the case $R_0 = 15$.

In our study, we do not distinguish between child-daycare and elementary schools, and we only allow the disease to spread within schools, while the population structure and contact patterns are actually much more complex and the infection could take place in households, sport centers, and so on. Moreover, we do not consider multiple waves of infection nor the possibility of becoming susceptible after being recovered, which indeed is not that relevant for measles, but might be for other diseases.

Despite the limitations due to the model hypotheses, the analysis describes the qualitative different behaviors of the two situations (with and without vaccine mandates) and can be important for further ethical and political decisions made on vaccines.

The analysis can easily be extended to diseases with lower reproduction numbers than measles; see Section 4.3. It depends on the relative spread within and between households whether this will lead to less or more pronounced possible undesirable effects of vaccine mandates.

Use of AI tools declaration

The authors declare they have not used Artificial Intelligence (AI) tools in the creation of this article.

Conflict of interest

The authors declare there is no conflict of interest.

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