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Explaining vaccination decisions: A system dynamics model of the interaction between epidemiological and behavioural factors

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ABSTRACT

The WHO goal of eradicating measles is delayed by widespread scepticism of parents against the recommended MMR vaccination. In this context, a model of the prevalence of measles that incorporates behavioural aspects is desirable. Parental decisions can be influenced by epidemiological and behavioural factors. The former include vaccination coverage and its impact on the prevalence of the disease. The latter include perceptions of the risk to be infected, which affects vaccination decisions, as well as government campaigns to affect vaccination behaviour, vaccination scares or changes in disease control policies. We develop a model that incorporates both kinds of effects. In particular, we illustrate how incorporating parental response to a change in the prevalence of the disease impacts the outcome of governmental policies aiming to increase the vaccination coverage. While calibrated to measles, this model is also applicable to other childhood diseases, such as pertussis or diphtheria. Different scenarios illustrate the long-term consequences of the interaction between health policies (in particular, vaccination campaigns) or the agenda of social institutions (e.g., drawing attention to specific events to create vaccination scares) and parental reactions. Periodic ups and downs of the disease's prevalence, characteristic of epidemiological feedback, are the consequence of the interaction between parental behaviour and events such as vaccination campaigns or vaccination scares. International and national health authorities, pursuing the fight against measles, may be helped by the potential of the model to provide understanding in the way different predictors of vaccination behaviour interact.

1. Introduction

The eradication of measles is one of the most prominent aims of global health policies. The major device to reach this aim is vaccination. A vaccine has been available since 1971, which is usually applied in combination with vaccines against mumps and rubella and referred to as MMR vaccination. Plans for eradication of measles have been delayed in many countries in the last two decades by a growing scepticism of, and hesitancy towards, MMR vaccination, or vaccination in general among parents. One of the origins of the scepticism was the claim of a British research group that the MMR vaccine could cause autism [1]. The claim was heavily criticized, and the study was officially retracted in 2010 [2] but some of the scepticism seems to endure.

International and national health authorities, if they want to retain the aim of eradicating measles, are faced with the task of overcoming parents' hesitancy or resistance towards MMR vaccination. That task will be much helped if the predictors of parental decision-making on having their children vaccinated are known. This article aims at contributing to broaden our understanding of these predictors and their consequences on vaccination rates and prevalence of measles.

Research has well supported that people decide in favour of vaccination the more efficacious they think the vaccine is, the less they expect side effects, the more they fear the severity of the disease and the higher they rate the likelihood of catching it [3,4]. These influences of perceptions and appraisals on individual vaccination decisions have to be put in the context defined by influences exerted by political institutions, social groups or systems such as research or mass media of communication. And the exciting question is: how do these forces interact in an epidemiological context? This paper seeks to provide insight into this question by incorporating the influence of these factors in an epidemiological model.

The epidemiological part of the model includes logical relationships that are based on stable mathematical relationships between the likelihood of health care events (getting a disease, showing side effects,

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vaccination coverage). The behavioural part focusses on the perception of risk and its impact on vaccination behaviour. The success of vaccination programmes reduces the prevalence of the disease and thus changes people's perception of the necessity of vaccination, which will increasingly appear to be negligible. As more people are vaccinated, the (perceived) prevalence of vaccine side effects will grow, influenced by the emergence of anti-vaccine movements [5,6]. These processes can be assumed to foster vaccination hesitancy. One can say that the basis of vaccination is damaged or destroyed by the success of vaccination programmes. Vaccination scepticism in this approach does not emerge as an individual belief but as a logical consequence of a lower prevalence. Singular activities, such as the fielding of campaigns run by public health institutions, research results, or periods of heightened media attention lead to behavioural reactions. The claim that the MMR vaccination causes autism is a perfect example: originating in science, it was popularized by the mass media and continues to affect vaccination decisions to date [7,8]. Such input can be added to the models of epidemiologically relevant behaviours.

In this paper we develop a stylised model of a childhood disease which explicitly integrates the epidemic/medical and the behavioural parts. By "stylised" we mean that our objective is not to build a medically detailed model, enabling exact epidemiological predictions, but rather to develop the simplest possible model that captures the essential epidemiological dynamics. This model allows us to analyse the long-term consequences of health policies (e.g., communication campaigns aimed at increasing the fraction of parents positively predisposed towards vaccination) and the attention paid to specific events by social institutions (e.g., vaccination scares). While the model is calibrated for measles, the insights apply to other contagious childhood diseases such as pertussis or diphtheria.

The remainder of this paper is structured as follows: after reviewing the literature on the situational factors of the vaccination decision and on the use of models to study vaccination decisions, we provide a model description. Next we present our simulation results and provide a sensitivity analysis of our main behavioural assumption. We conclude with a general discussion and suggestions for further work.

2. Literature review

2.1. Situational factors of vaccination decisions

Epidemiological factors relate to medical conditions such as the number of infections or vaccination rates. Behavioural factors include parental decisions, policy making by healthcare authorities, and the public communication about these. Some attention has been paid to factors such as the organization of vaccination programmes, the issue of whether measles vaccination should become mandatory or the efficacy of surveillance systems [9]. Considering objective elements, the environment is similar for people living similar lives. What differentiates people living in a similar environment is the awareness of these elements. For instance, a person living in a community that experiences a measles outbreak might or might not be aware of it [10]. Only if the person is aware of it can she react to it. Awareness brings back the issue to the individual level, but most research on epidemiological factors remains at the aggregate level and seeks to make connections between indicators of aggregate behaviour such as vaccination rates or the prevalence of infections.

Research into behavioural factors has focussed on five elements: disease outbreaks or epidemics, immunization campaigns by healthcare institutions, waves of media coverage of vaccination-preventable diseases, vaccine adverse events such as severe side-effects of vaccination, which can go as far as people dying, and adverse campaigning. Deaths in temporal proximity to vaccinations are seldom, and what is known of them does not provide cause for concern [9,11,12]. Outbreaks, campaigns and media coverage should be instrumental to the pro-vaccination side as they can be assumed to strengthen the resolve to get immunised or have one's children immunised, while vaccine adverse events and campaigning would be considered to play into the hands of vaccination sceptics.

A recent compilation of influence factors on vaccination status and intention to have children vaccinated [13] highlights that parents most frequently name vaccination safety concerns, especially the risk of side effects, as barriers to having children vaccinated. Other beliefs that make parents shun vaccination relate to an allegedly too high number of vaccinations babies receive and fears that vaccinations might do harm to a child's immune system. Still other beliefs have to do with the necessity of vaccination, which is questioned either by the assumption of low susceptibility of their children to a disease or by doubts as to the effectiveness of vaccines. Trust in one's family doctor strengthens the resolve to have one's children vaccinated, while distrust in government or pharmaceutical companies weakens it. Motivators have been researched less than barriers. Physician reminders appear to be the most important motivator.

A somewhat similar study by [4], covering Northern, Western and Southern Europe over the period 2000–2014, provides a systematic qualitative review of 45 published studies focussing specifically on MMR vaccination. A total of 26 of these were additionally studied with meta-analysis methodology. Results show that, by and large, the likelihood of not being properly immunised with the MMR vaccination was high when parents held wrong beliefs about vaccination, had gaps in their vaccination knowledge, perceived vaccination negatively, held unfavourable attitudes towards vaccination, had had less formal schooling, were less well off, or were affiliated with an ethnic minority in their respective countries. A child's older age, a high number of children and living arrangements other than traditional marriage also went along with low inclination to get vaccinated. The role of one's general practitioner as a source of information and a reminder of some possibly missed vaccination is stressed here as elsewhere.

In summary, evidence overwhelmingly points to a positive effect of an increased perceived threat by the disease (most often caused by an outbreak [10,14,15]; with deviating results [16,17] as well as of official pro-vaccination campaigns on vaccination behaviour or other indicators that signal support for vaccination [18,19]. The effects of temporary media attention to a disease (including its prevention) is less clear and appears to be dependent on the context: if media attention is triggered by an outbreak or a campaign, its effect tends to be positive [20,21], but it can also be negative in other situations [22].

Vaccination-adverse events include vaccination side effects, the publication of scientific evidence either of harm done by vaccination or of failure to reach its aims [9], and the withdrawal of a vaccine by government agencies or pharmaceutical companies. Vaccination-adverse events appear to have a clear negative impact on vaccination behaviour, especially if government or industry retract or suspend vaccines [9,23–25]. For people, interest groups, companies or media outlets who think they recognize a risk too high to continue present policies, the only ways to promote change is by litigation or campaigning. Compared to the other types of predictive factors, this has received less attention, but a potential to affect vaccination behaviour negatively by adverse campaigning has been shown [26]. Low coverage rate for childhood vaccinations has been associated with a number of attitudinal and perceptual variables that are linked with parents' vaccination hesitancy [3,27].

Various reviews of reviews [13,28], as well as the review in [29] find no convincing evidence in favour of any particular intervention. This might have to do with the large variety of approaches, methods, types of vaccination, types of interventions, settings and addressed populations in the published research.

A very recent and extremely comprehensive review [30] expresses reservations about the validity of many studies due, among others, to potential biases and inappropriate choices of outcome measures. They point out that the success of specific interventions depends on the specific barrier to vaccination. For instance, comparing lack of knowledge

to vaccine hesitancy, face-to-face interventions appear to be more effective to overcome the former.

Another systematic review [25] identifies several positive influences on child vaccination: not believing in side effects, positive parent attitudes, positive recommendations, and the absence of practical barriers to having children vaccinated. Knowledge, social influences and trust in one's doctor worked in the same direction. The authors also conclude that the evidence of a link between vaccination and perceived susceptibility to an illness is stronger than the link between vaccination and perceived severity.

2.2. Modelling epidemic control

There is a very broad literature on modelling work relating to epidemic control and vaccination, which has been summarised in a large number of review articles over the last decade [31–33]. The journal *Risk Analysis* has devoted two special issues, introduced by [34,35] to models aimed at managing the risks of measles and rubella.

Most work in this area focusses on evaluating the cost effectiveness of different vaccination policies [32,36–38] and references in these articles). Other authors focus on operational aspects of vaccination programmes, for instance on vial size [39], storage facilities [40], or the need to stockpile vaccines [41,42]. Another strand of literature focusses on disaster management, see for instance [33] and the references therein.

While policy models cannot be expected to include all the medical details, they should capture the essential elements that enable drawing reliable conclusions [43]. provide useful guidelines in this respect. They argue among others that models should be open (i.e., including birth and death processes) and dynamic (infection probabilities vary over time as the prevalence of the disease evolves). Only dynamic models can correctly capture the consequences of a change in vaccination policy, such as the non-monotonic impact of an increase in vaccination on the number of cases and the changing distribution of the age at which the disease is caught. The need to use a dynamic modelling framework to capture the herd immunity effect has been emphasised by many authors [36,38,44,45]. In his recent overview of policy models regarding vaccination programmes for measles and rubella, [46] considers only dynamic transmission models.

Most of this literature focusses on comparing vaccination policies for different diseases in various parts of the world, dealing for instance with measles in Taiwan [47], influenza in Washington D.C. [48], cervical cancer in Kenya [49] and tuberculosis in South Africa [50]. But these articles, and most others, have in common the implicit assumption that the proposed vaccination policy can be implemented, i.e., if the policy requires an 80% vaccination rate, this rate is actually achieved; no attention is given to the possibility that people may refuse vaccination. The same implicit assumption is found in the majority of the more theoretical economics models dealing with vaccination policies, see for instance Refs. [51,52].

The need to pay attention to individual behaviour and decision making has been pointed out by several authors, one of the earliest being [53], who provides a graphical illustration of the dynamics resulting from the interaction between vaccine coverage, disease incidence and vaccine adverse events. [6] argues the need to incorporate economic incentives in mathematical epidemiological models, as a lower perceived risk is likely to lead to more risky behaviour and less support for preventive measures. They conclude (p. 716) that "it is impossible to eradicate a vaccine-preventable disease through voluntary vaccination if people act in their own self-interest". In a similar vein, [34] discusses how, on the one hand, a low incidence of measles leading to a decrease in the perceived danger of this disease and, on the other hand, a worry about the safety of the MMR vaccine, combine to reduce willingness to vaccinate, increasing both the cost and the time required to achieve immunisation objectives. [54] formalises these ideas in a theoretical economics paper: they argue that neither private markets nor public support programmes can succeed in eradicating a disease as increasing vaccination levels will always ultimately result in some individuals finding it in their interest not to be vaccinated.

[55] explicitly considers individual decision making in an agent-based simulation model developed to study the impact of social distancing and vaccination on the transmission of influenza. The model is calibrated using a controlled experiment performed on a university campus, enabling the comparison of the outcome of the behavioural pattern of the general student population to that of a student population having received information from a health care expert.

[56] argues that simulation has become a well-established research approach, complementary to theoretical and experimental work. System dynamics (SD) based simulations are particularly useful to address vaccination policies, which are characterised by dynamic complexity rather than combinatorial or detail complexity [57], as this methodology enables incorporating feedback and delay structures, as well as behavioural aspects [56,58]. [59] provide examples of successful use of SD policy models to address public health problems. More recently, [49] has analysed vaccination policies for cervical cancer in Kenya. While the authors refer to the challenges of being able to reach the right population and possible vaccination resistance, these elements remain exogenous to their models. Our work is a first step towards making these elements endogenous.

It is worth emphasising that the objective of SD models is not to yield precise numerical forecasts, but to gain understanding of the possible outcomes of envisaged decision policies, given various behavioural assumptions. The focus is on the dynamics of the transition period resulting from, e.g., policy changes.

3. Model description and testing

We combine a highly stylised representation of an epidemiological model with a more detailed model of behavioural factors of vaccination decisions. The epidemiological part contains elements such as the number of susceptible people, infections per week and immunization rates. The behavioural aspects include the perception of the risks of disease and vaccination, and their impact on vaccination behaviour.

To illustrate the impact of behavioural elements on the outcome of vaccination policies, we have chosen to focus on two factors among the many reviewed in the previous section: (i) a long term behavioural aspect (observing less cases results in a tendency to vaccinate less) and (ii) a shorter term aspect (a scare leads to a temporary change in behaviours). This is a first step towards including behavioural aspects in models aimed at supporting vaccination policy decisions: we aim to build a stylised policy model characterised by a low level of detail complexity, but a high level of behavioural complexity, that can provide insights into the long-term consequences of today's vaccination decisions. In particular, our model captures the transitional effects and the long-term consequences of variations in the coverage level resulting from changing health policies and parental attitudes, the latter's evolution being modelled endogenously.

To calibrate the key model parameters, we focus on measles, a disease close to being eradicated in many countries (including most of the Americas), but remaining endemic in other parts of the world (e.g., India, Italy) [60]. The simulation model is developed using the VENSIM PLE 8.0 software.

3.1. Model description

Fig. 1 shows a simplified view of the model structure, focussing on the main feedback loops. A full equation listing is provided in Appendix. For reasons of clarity this diagram shows neither age-classes, nor deaths, except those resulting from the disease. The rectangles represent accumulations of people in the different possible states (e.g., Susceptible, Infected, etc.). The double arrows represent people moving between states (e.g., people who become infected ("Infections") move from the

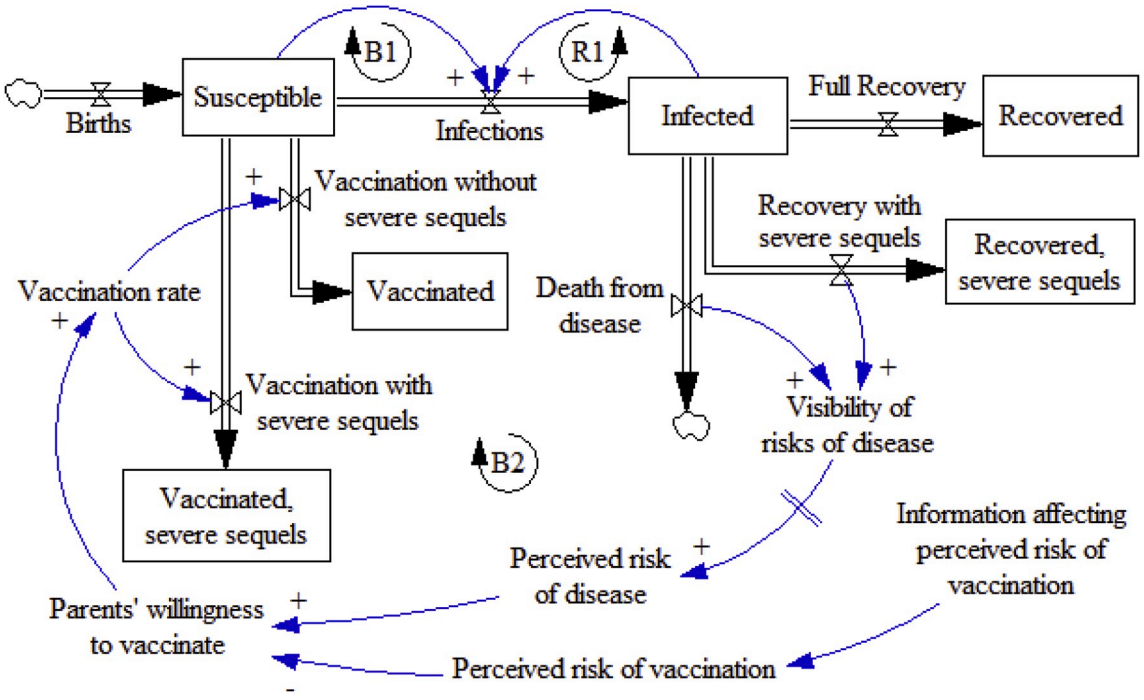


Fig. 1. *Model overview.*

Susceptible state to the Infected state. The single arrows represent causal links. Two parallel lines on an arrow (//) indicate the existence of a delay. For instance, there is a delay between a change in the Visibility of risks of disease and the Perceived risk of disease. A "+" ("-") sign at the head of an arrow indicates that an increase in the first element will cause an increase (decrease) in the second element, other things remaining constant. For instance, an increase in the number of Susceptibles leads to more Infections, while an increase in the Perceived risks of vaccination lowers Parents' willingness to vaccinate.

The letters B and R surrounded by an arrow indicate respectively balancing and reinforcing loops. The loops B1 and R1 are the classical epidemiology loops. On the one hand, more Susceptibles leads to more Infections, which reduces the number of Susceptibles, thus yielding a balancing loop. On the other hand, more Infected leads to further Infections, and thus even more Infected, yielding a reinforcing loop. In the initial stage of an outbreak the reinforcing loop dominates, while in the latter part the balancing one dominates, resulting in the well-known S-shaped curve for the cumulative number of affected people.

Loop B2 represents behavioural aspects. It captures the idea that as the rate of Infections decreases, fewer people suffer from severe sequels or die. Thus the Visibility of the risks of the disease decreases, leading to a lower Perceived risk of disease. This in turn lowers Parent's willingness to vaccinate, leading to a lower Vaccination rate and, in the longer term, to more Susceptibles, and thus more Infections. The model thus captures the important behavioural element that an increase in the vaccination rate negatively affects parents' willingness to vaccinate through a decreased visibility of the risks of the disease. Note that we do not include an explicit link between the number of cases of measles and the vaccination rate. As adverse effects (severe sequels or death) are proportional to the number of infections, and this proportion is assumed to remain constant over the simulation period, this link is captured implicitly in our model. The system includes many delays, but only a few have been highlighted in the sketch. For instance, whenever perceptions are involved, there is a delay between the event that will result in changed perceptions, and the actual change in perceptions.

We choose not to model a direct endogenous impact of adverse effects from vaccination on parents' willing to vaccinate for the following reasons. First, such events are extremely rare, less than 1 in a million (see e.g. [61,62]), and hard to quantify. Second, and more importantly, parents' reaction to such events results not so much from the event per se, as from the actions of health authorities and media coverage of such events. The presence of, for instance, anti-vaccination movements, can have more impact than actual events [5]. We have therefore chosen to model this element exogenously using the variable Information affecting perceived risk of vaccination, which allows us to analyse how a vaccination scare affects the system behaviour.

Our stylised model is based on the following simplifying assumptions.

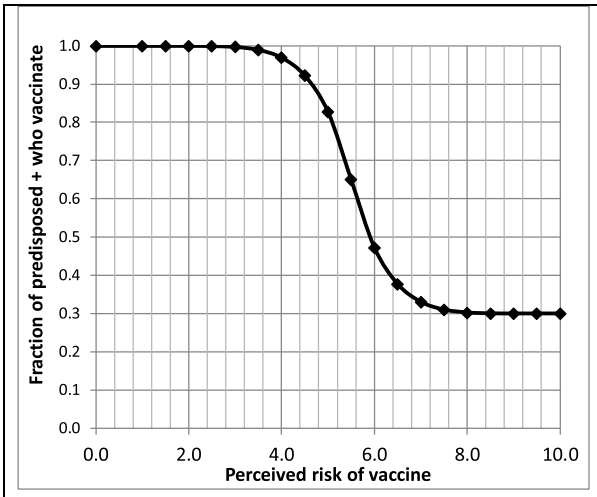
- There are two age-classes: pre-school children and all other individuals, referred to as adults. There is perfect mixing, but pre-school children have a lower contact rate than adults.
- Vaccinated individuals have received two doses of the MMR vaccine and are immunised; all other individuals remain susceptible until they are infected; individuals who recover are immunised.
- Susceptible children are vaccinated (second dose of MMR) when entering school; only children are vaccinated.
- We only distinguish between vaccination without sequels and vaccination with severe sequels.
- Infected individuals recover fully, recover with severe sequels, or die from the disease.
- The model is deterministic, focussing on the long-term underlying trends which lead to major outbreaks, ignoring random short term fluctuations.

Next we describe in detail the modelling of the behavioural aspects leading to the vaccination decision. The visibility of risks of the disease is measured as a weighted average of the number of cases with sequels or resulting in death. We arbitrarily assign a weight of 1 to a case of severe sequels and a weight of 10 to a death. As the occurrence of both severe sequels and deaths are proportional to the total number of cases (which is endogenous), the choice of this parameter does not influence the system behaviour. We then take an exponentially smoothed average of this value to capture people's memory of adverse events. Exponential smoothing, also referred to as adaptive expectations, is the most commonly used method to capture how people update their perceptions

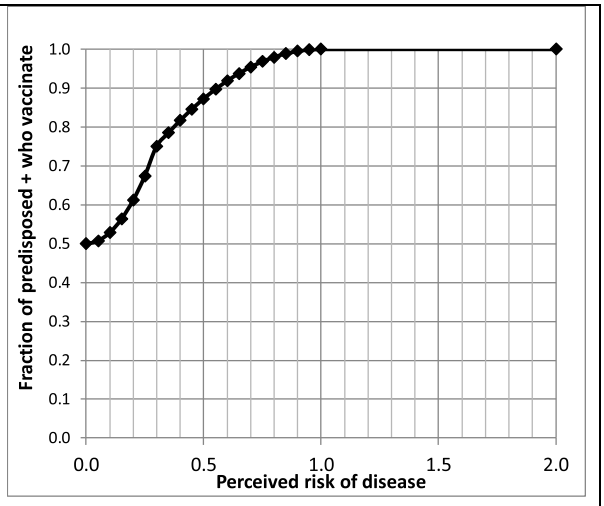
based on new information [58]. The resulting value is normalised to equal one in the steady state at the start of each scenario. This normalised value is referred to as the Perceived risk of disease. The perceived risk of vaccination is modelled exogenously; it is assumed to result, e.g., from media-coverage of a vaccination scare, which could result from an adverse event or from misinformation.

Parents are divided into two subgroups, depending on whether they are positively or negatively predisposed towards vaccination. Parents who are positively predisposed will vaccinate their children, unless they either perceive an increased risk of vaccination (compared to the initial conditions of the simulation), or a decreased risk of disease. So their behaviour will be affected by a vaccination scare and by a lower prevalence of the disease, as the latter leads to fewer adverse disease outcomes. Parents who are negatively predisposed will not vaccinate their children, unless they perceive a significantly increased risk of disease. Their behaviour will be affected by outbreaks of the disease leading to a larger number of adverse outcomes. We do not consider the impact of a reduction in the perceived risk of vaccination, as the risk documented in the medical literature, is already extremely low.

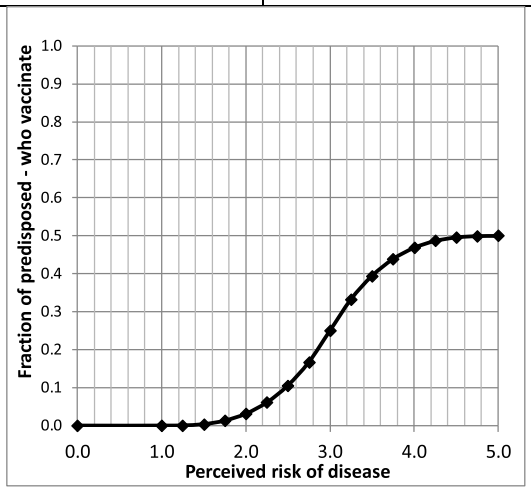
Next we motivate our behavioural hypotheses, which are encompassed in the three graphs shown in Fig. 2. Several studies report data on the impact of reports of adverse events of vaccination on vaccination rates. Examples include [24,63] (both Hepatitis B in Viet Nam), [64] (Measles in the UK), [65] (Pertussis in the UK), and [5] (Pertussis in a dozen developed countries). The reported impacts vary widely. For instance [24] reports that the fraction hesitant or refusing to vaccinate increased six fold, while [65] reports a vaccination rate decreasing from 80% to 30%. [5] mentions the extreme case of a decrease in the vaccination rate against pertussis from 90% in 1974 to 12% in 1979, observed in Sweden after years of criticism of this vaccination in the medical profession and ensuing policy changes. In our model we hypothesize that the parental reaction to a scare depends on the severity of the scare, ranging from little or no reaction to a minor scare, to a very strong reaction to a major scare. To calibrate these reactions, we use as upper



(a) Impact of a vaccination scare on the fraction of positively predisposed parents who vaccinate



(b) Impact of the perceived risk of disease on the fraction of positively predisposed parents who vaccinate



(c) Impact of the perceived risk of disease on the fraction of negatively predisposed parents who vaccinate

Fig. 2. *Impact of perceived risk of disease and vaccination on willingness to vaccinate.*

bound that 70% of positively predisposed parents would refuse vaccination, a high but not extreme value compared to the numbers reported in the literature mentioned above.

Data on the impact of a perceived increase of the risk of a disease is scarce. Two exceptions are [10,66] who both report on parental reactions to measles outbreaks, i.e., situations where the risk of the disease suddenly increases. Based on the figures reported in these papers, we make the rough estimate that in case of a major outbreak, up to 50% of negatively predisposed parents would accept vaccination. Similarly, we assume that as the disease gets close to being eradicated, half the positively predisposed parents will stop vaccinating. In other words: we assume that 50% of this group will continue to vaccinate as long as the vaccine is available and the disease is not considered to be eradicated.

We assume an S-shaped form to capture these three behavioural hypotheses, as shown in Fig. 2. This shape captures the idea that a small change in perceived risk will have a very limited impact on people's behaviour. As the change becomes more noticeable, an increasing number of parents will modify their behaviour, until a level is reached where little or no further reaction is observed (i.e., some parents will always vaccinate, others never, whatever the perceived risks). The curvature is least pronounced for the impact of the perceived risk of the disease on positively predisposed parents (power of 2), and steepest for the impact of a vaccine scare (power of 6). For the impact of a decrease in prevalence on positively predisposed parents (Fig. 2(b)), we assume an asymmetric shape, which captures the hypothesis that it will take a significant (long-term) drop in prevalence for a large share of this population group to stop vaccinating their children. Fig. 2(c) implies that negatively predisposed parents will seriously consider vaccination if the perceived risk of disease is multiplied by about 3.

The graphs shown in Fig. 2 are for the base case, which assumes that at the start of the simulation 50% of the parents are positively predisposed, implying an initial 50% vaccination rate. This scenario aims to capture a situation where the vaccination coverage is well below that required to achieve herd immunity. We also consider scenarios with a very low level of prevalence (corresponding to a situation with a very low initial fraction of negatively predisposed parents); in these instances, Fig. 2(c) is rescaled by a factor of 10 to take into account that these parents are particularly hard to convince and will only react to a major outbreak. We have performed extensive sensitivity analysis concerning these behavioural assumptions; the main observations are summarised in the results section. Table 1 summarises how the different model parameters were estimated and provides the main data sources.

3.2. Model testing

Following [58], we use the term model testing rather than model validation and verification. The following discussion provides a summary of the tests we have performed; these are the classical tests used to evaluate whether a system dynamics model is suitable for its intended purpose [58]. The boundaries of our model were chosen to enable us to study specific behaviours. For instance, the perceived risk of disease is modelled endogenously, as it results from the (also endogenous) degree of prevalence, while the perceived risk of vaccination is modelled exogenously, as it is largely influenced by media reports and anti-vaccine movements not necessarily linked to true adverse events. Although the model was tested for much longer simulation runs, most results are reported for a time-horizon of 80 years, which correspond to the hypothesised expected life-time; this enables observing the consequences of vaccination decisions over the life-time of the individuals concerned.

The model has been checked for dimensional consistency. Parameters are based on available literature whenever possible and have real-world counterparts. We have performed extensive extreme condition tests and sensitivity analysis. For instance, we have simulated the model starting with a non-vaccinated population, with and without behavioural feedback, and for different speeds of vaccination introduction.

Table 1
Parameter values.

Parameter		Value	Motivation and data sources
Vaccination age		4 years	This is the approximate age at which children start school in European countries.
Life expectancy		80 years	This is a representative number for most European countries.
Infectious period		1.25 weeks	Medical and governmental sources (e.g. [67,68] indicate an infectious period of about 9 days. We approximate this by 1.25 weeks (the time-unit of the model is weeks, with 1 year being approximated as 52 weeks).
Reduction in life-expectancy due to severe sequels of disease		0.75	This parameter depends on how one defines "severe sequels", i.e., what complications are included. The few information sources we identified, e.g., [69] cannot be adapted to our needs, as they calculate DALYs for a population, not for an individual with severe sequels. We have therefore chosen to focus on one of the major causes of severe sequels: subacute sclerosing panencephalitis (SSPE). This sequel is developed mainly by very young victims of measles, 6–15 years after the disease, and leads to death within 1–3 years [70]. This yields a life-expectancy of 20 years which, combined with our assumption of a life expectancy at birth of 80 years, implies a 75% reduction of life expectancy. For lack of better information, we assume the same reduction for adults.
Reduction in life-expectancy due to severe sequels of vaccination		0.75	We have not been able to find any data on this parameter. Given the extremely low occurrence of this event, and that panics due to adverse vaccination events are modelled exogenously, this parameter does not affect the simulation results. We use the same value as for the impact of severe sequels of the disease on life-expectancy.
Basic reproduction number (R0)		13.5	Different sources provide very different values. The basic reproduction number is context dependent. For instance, the value is higher in a crowded city than in a rural area as people interact much more frequently. In a review of 137 studies, [46] identifies values ranging from 4 to 20, with most studies using a value between 9 and 18. The basic reproduction numbers for the two population groups (children and adults) have been selected to obtain a value of 13.5 for R0, the midpoint of this range, a reasonable estimate for a European country.
Relative contact rate of pre-school children compared to adults		0.5	Contact rates vary significantly across age-groups. See for instance [71] for a detailed matrix of relative contact rates by age-group. In our stylised model we assume homogeneous mixing of the population, but do account for the fewer contacts pre-school children have compared to school-aged children and adults. We assume that pre-school children have on average half as many contacts as the remaining population.
Probability of severe sequels from vaccination		1 per million	All official information sources and the medical literature concur that such events are extremely rare. Based on [62] we assume a value of one per million.
Probability of consequences of disease			The numbers below are based on data from [72]. The figures for the Adults are a weighted average of the numbers given for the different age groups (age 5 and higher).
Children	Severe Sequels	0.066%	This figure combines the 0.2% probability of post infectious encephalomyelitis, and the 33% probability of having severe sequels from this complication (Table 2 of [72].
	Death	0.30%	Table 2 of [72]

(continued on next page)

Table 1 (*continued*)

Parameter		Value	Motivation and data sources
Adults	Severe Sequels	0.25%	Same approach as for children, using data from [72]
	Death	0.53%	Weighted average based on Table 2 of [72]
Weighting Severe sequels and deaths		10	When evaluating the visibility of risks associated with the disease, the weight given to a death equals 10 times the weight given to a case of severe sequels.
Time to perceive changes in the risk of disease			
Positively predisposed parents		25 years	We assume that the positively predisposed parents' perception of the risk of the disease evolves very slowly: it takes a long time (one generation) with little or no cases with sequels or deaths before a significant share of them will consider not vaccinating their children. The model assumes that expectations are formed using exponential smoothing, i.e., a weighted average of past values, with the most recently observed value receiving the most weight, and the older values exponentially decaying weights. This is the most common way to represent perceptions in SD models [58].
Negatively predisposed parents		5 years	We assume that in case of an increase of the prevalence of the disease negatively predisposed parents' perception of risk will evolve over a time-span of 5 years.

Table 2
Overview of scenarios.

(a) Scenarios concerning vaccination campaigns aimed at positively predisposed parents

Scenario number	1	2	3	4	5	6	7	8
Activation of behavioural loops	No	Yes	No	Yes	Yes	Yes	Yes	Yes
Event, beginning after five years	Vaccination campaign aimed at increasing share of positively predisposed parents						Sensitivity* Low	High
Share of positively predisposed parents/ vaccination rate at the start (%)	50	50	50	50	50	50	50	50
Target share of positively predisposed parents (%)	95	95	95	95	75	75/ 95	95	95
Horizon to achieve target (years)	1	1	20	20	5	5/ 20	20	20
Shown in Figure, Column	3 left	3 left	3 right	3 right	4	4	5	5

(b) Scenarios mimicking small and large vaccination scares in population with a 50% or 95% vaccination rate

Scenario number	9	10	11	12
Activation of behavioural loops	Yes	Yes	Yes	Yes
Event, beginning after five years	Vaccination scare causing a sudden drop in vaccination rate			
Share of positively predisposed parents/vaccination rate at the start (%)	50	50	95	95
Size of scare: vaccination rate drops within a month to (%)	40	15	90	80
Vaccination rate returns to initial level after (years)	1	1 ½	1	1 ½
Shown in Figure	6	6	7	7

*with respect to the reactivity of positively predisposed parents to changes in prevalence.

Results were as expected. Similarly, changing the values of the various parameters within reasonable ranges does not affect the qualitative results. Sensitivity analyses concerning the key behavioural assumption are discussed in the results section.

The results presented in the paper are obtained using the Euler method with a time-step of 0.125 weeks. We have validated the use of this method (size of integration error) by varying the time-step and by using the Classical Runge–Kutta method (RK4) with variable time-step, all of which yield the same results, to within numerical error. A full equation listing is included in appendix to ensure replicability.

4. Simulation results

This section presents a number of simulations in order to demonstrate how including behavioural aspects affects the impact of different policies on the evolution of the disease. Table 2 provides an overview of the selected scenarios. All scenarios start in steady state, and policy changes (e.g., a communication campaign) or events (e.g., a vaccination scare) occur after five years. Unless specified otherwise, we consider an initial situation where half the parents are positively predisposed to vaccination. They all vaccinate their children, while none of the negatively predisposed parents do. In other words, in the initial situation predisposition is assumed to fully translate into behaviour.

4.1. Scenarios 1–4: vaccination campaigns aimed at increasing positive predispositions

Our first set of scenarios (1–4) compares the consequences of increasing the fraction of positively predisposed parents, for instance through an information campaign, when behavioural feedback is either ignored (as is the case in most previous research, i.e., we disconnect the behavioural loops in our model) or accounted for. Disconnecting the behavioural loops implies that all positively predisposed parents vaccinate, while negatively predisposed parents don't, whatever the evolution of the prevalence of the disease. Including the behavioural loops implies that, as the campaign successfully reduces the number of cases, some of the positively predisposed parents will stop vaccinating their child. In these scenarios we exogenously increase the fraction of positively predisposed parents from 50% to 95% over respectively a very short period (1 year, Scenarios 1–2, Fig. 3, left column) and a more reasonable period (20 years, Scenarios 3–4, Fig. 3, right column). While the 1-year scenario seems rather unrealistic, it can be interpreted as an approximation for a governmental policy imposing mandatory vaccination at school-entry, as for instance recently decided in Italy [73].

In the 1-year scenario the number of weekly infections drops to virtually zero within five years of the start of the campaign, but the disease resurfaces about five years later. In the absence of behavioural feedback, the vaccination rate stays at 95%; future outbreaks are therefore limited in size, and distant in time: the disease is slowly dying out, as expected with this vaccination rate. With vaccination only occuring at school-entry, it is not a surprise that it takes so long for the disease to disappear. Faster elimination would require catch-up vaccination of the adult population.

When the reaction of positively predisposed parents is incorporated in the model, the situation looks quite different (recall loop B2 in Fig. 1): as an increasing share of positively predisposed parents stop vaccinating, the disease soon resurfaces, leading to strong epidemic cycles. Although the vaccination rate converges towards 78%, significantly higher than the initial rate of 50%, certain epidemic cycles peak above the initial number of weekly cases. Over time, the cycles become less pronounced, but the disease remains endemic.

This scenario also illustrates a behavioural aspect not included in the model, but occasionally referred to in the literature: the "it does not work" phenomenon. A (very) fast introduction initially seems to lead to eradication, but the disease comes back with huge surges, which can be perceived as "worse than before" by part of the population. During those peaks parents may get doubts about the effectiveness of the vaccine and be less inclined to vaccinate, i.e., the opposite of the effect hypothesised in the model (we assume that in the case of a resurgence, parents tend to vaccinate more).

With a more gradual approach (Fig. 3, right column), in the very long

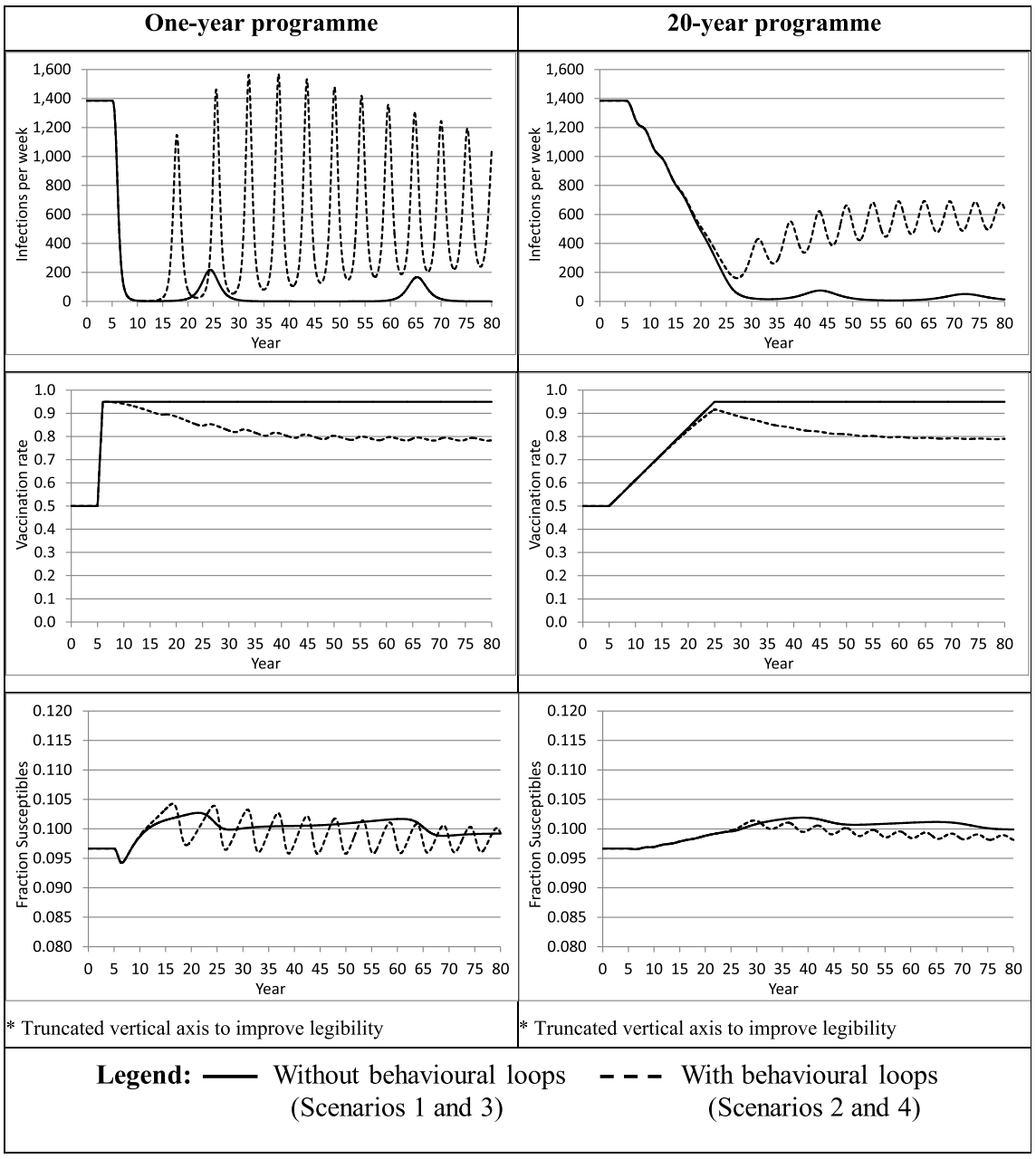


Fig. 3. Effect of an increase in the fraction of positively predisposed parents from 50% to 95%, with and without behavioural loops, over a 1-year (left) or a 20-year period (right).

term the outcome is the same, but the dynamics do differ. Without behavioural feedback it takes longer for the disease to disappear; the future outbreaks have a lower magnitude but last longer. With behavioural feedback the objective of a 95% vaccination rate is not reached: as the disease gradually seems to be eliminated, some positively predisposed parents stop vaccinating; the disease picks up, but the magnitude of the outbreaks is much smaller. It is worth noting that in all four scenarios the fraction of susceptible people stabilises slightly above the initial level as the decrease in the number of people immunised by catching the disease exceeds the increase in the number of vaccinated individuals.

4.2. Scenarios 5–6: effects of introducing a second campaign to increase positive predispositions

While international bodies such as the WHO do set long-term goals, national governments tend to have a sequence of incremental short-term objectives. We therefore consider a sequence of two government campaigns aimed at increasing the fraction of positively predisposed parents, in the presence of the behavioural feedback loops. We initially consider a 5-year campaign, starting in year 5, aimed at increasing the fraction of positively predisposed parents from 50% to 75% (Fig. 4, Scenario 5). This campaign is initially successful, with the cases per week being approximately halved. The 75% vaccination rate objective for children entering school is briefly achieved, but soon starts to erode as the lower prevalence reduces the perceived risk of the disease. Additionally, this vaccination rate is insufficient to eliminate the disease; we observe regular spikes in the number of cases, with an increasing trend resulting from a gradual erosion of the vaccination rate.

Observing this, the government launches a second communication campaign (Scenario 6), starting in year 25, aimed at increasing the fraction of positively predisposed parents to 95%. This boosts the

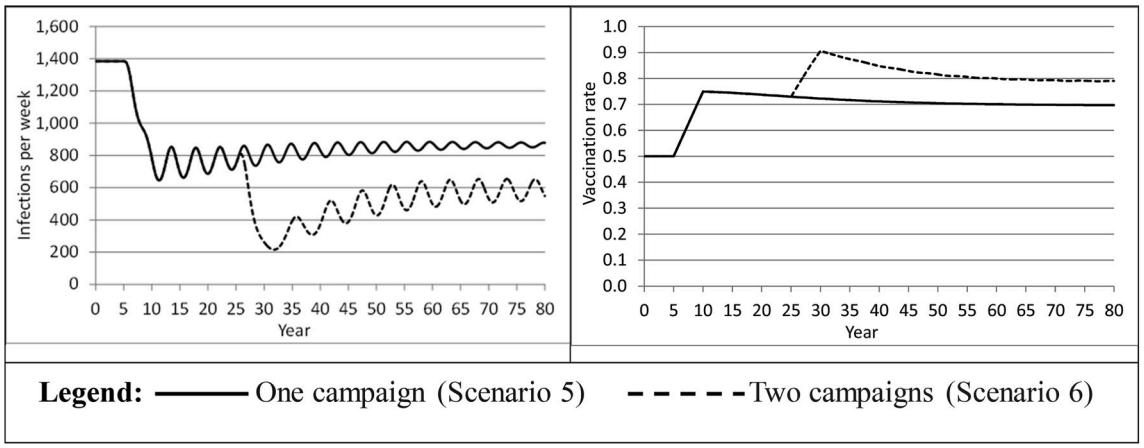


Fig. 4. Policy example: a five-year communication campaign aiming to increase the fraction of positively predisposed parents to 75%, followed by a second campaign 15 years later with a 95% target.

vaccination rate, resulting in a further reduction in the number of cases. But again, the behavioural loop kicks in: the vaccination rate tapers off and epidemic cycles occur, again with an increasing trend, until the vaccination rate stabilises and the cycles gradually dampen.

4.3. Scenarios 7–8: sensitivity analysis

Next we consider the sensitivity of our results to one of our key behavioural assumptions: how do positively predisposed parents react to a significant, lasting decrease in the prevalence of the disease? As mentioned in the model description, there is no hard data on this, and it will not come as a surprise that our results are sensitive to this assumption. We have tested an extensive range of shapes, and report here some of the more extreme results to illustrate what type of behaviours can occur. In particular, in the most extreme case where we assume that this parent group is highly reactive, and that all stop

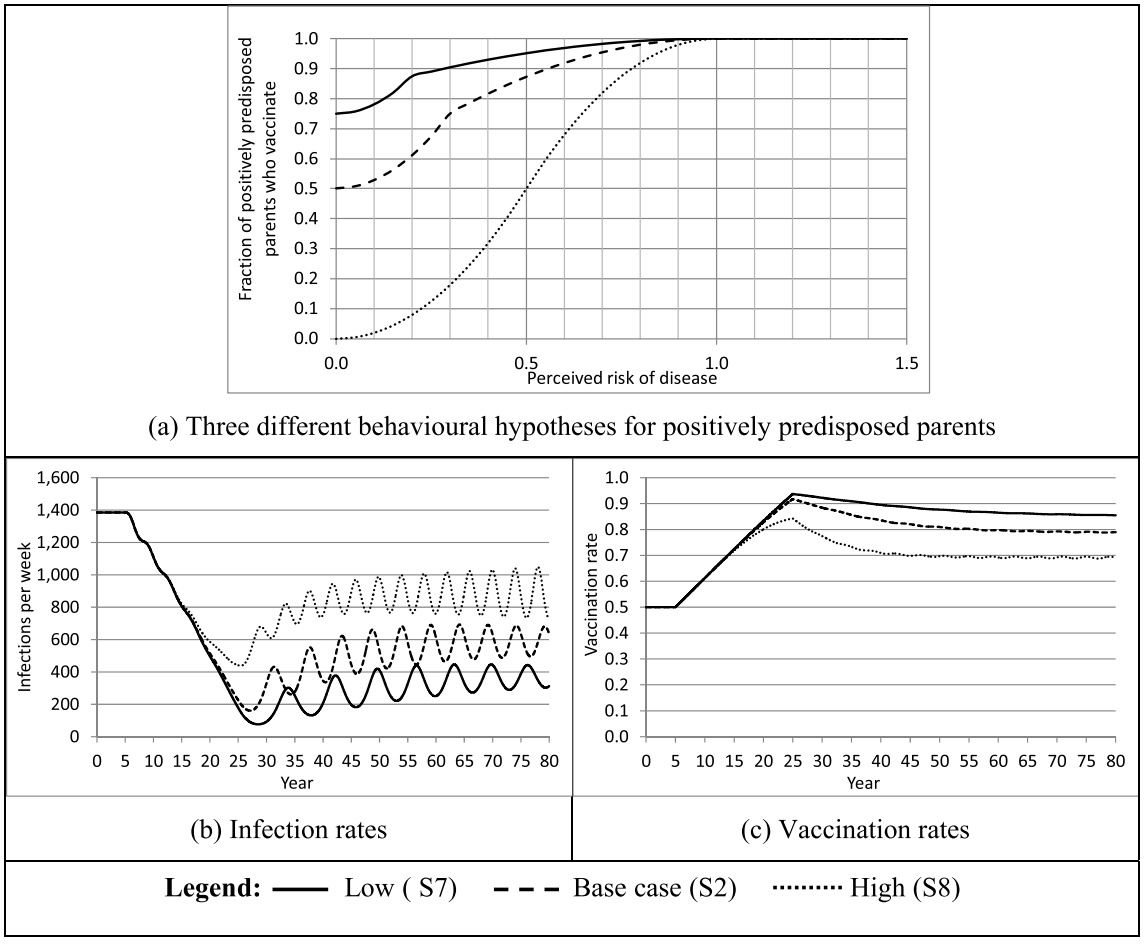


Fig. 5. Sensitivity analysis regarding the impact of the gradual disappearance of the disease on the attitude towards vaccination of positively predisposed parents when targeting an increase in the fraction of positively predisposed parents from 50% to 95%.

vaccinating when the perceived risk of the disease becomes negligible, we observe a limit cycle for both the vaccination rate and the weekly number of infections. Recall that this perception is formed over a 25-year period, so it would take one generation with little or no cases for the perceived risk to tend to zero.

We again consider the scenario of a campaign aimed at increasing the fraction of positively predisposed parents from 50% to 95% over 20 years under three different behavioural assumptions. In the low reactivity scenario, we assume that 75% of positively predisposed parents continue to vaccinate until the disease is formally eradicated (scenario 7). In the high reactivity scenario we assume that if the perceived risk decreases by 50%, half the positively predisposed parents stop vaccinating and that if the perceived risk tends to zero, all stop vaccinating (scenario 8). Fig. 5(a) shows these two extreme cases, as well as the hypothesis used in the previous scenarios. The results (Fig. 5(b) and (c)) are as expected: the more reactive the parents, the lower the final vaccination rate, and the higher the fluctuations. While the first two cases converge towards equilibrium, the case with highly reactive parents converges towards a limit cycle, i.e. sustained fluctuations.

4.4. Scenarios 9–10: minor and major vaccination scares

Scenarios 9 and 10 (Fig. 6) consider the impact of a minor and a major vaccination scare, resulting in a cohort of children having a lower vaccination rate. The minor scare is calibrated to cause the vaccination rate to drop from 50% to 40% (i.e., 20% of positively predisposed parents stop vaccinating) and to return to normal one year after the scare. The major scare is calibrated for the vaccination rate to drop from 50% to 15% (i.e., an extreme case where 70% of positively predisposed parents stop vaccinating), returning to normal after 18 months. While significant, a decrease of this magnitude is not unrealistic; more drastic reactions have been documented in the literature (see e.g., [5]).

A scare leads to a short-term change in parental behaviour: the vaccination rate quickly returns to normal. But, unless catch-up vaccination is organised, the impact on the system lasts for many years (Fig. 6, left panel). This is not a surprise: given a life expectancy of 80 years, a less vaccinated cohort will affect the evolution of the disease for several generations. This phenomenon is visible in Fig. 6 (right panel), which shows the evolution of the number of susceptible adults.

4.5. Scenarios 11–12: effects of scares when coverage is high

Finally, in scenarios 11 and 12 (Fig. 7) we consider an initial situation where the vaccination coverage is 95%, i.e., close to the eradication level. For this scenario, the graphical function capturing the reaction of negatively predisposed parents to the risk of disease (leading to a willingness to vaccinate) needs to be recalibrated. As an example, going from one infection every 10 years to 1 infection a year in a total population of about 10 million will not have much of an impact, while going from 10 to 100 cases a year would draw much more attention. For the scenarios below, the parents' reaction function has been rescaled by a factor of 10, i.e., it takes a 50-fold increase in the number of cases rather than a 5-fold increase to convince half the negatively predisposed parents to vaccinate.

The small scare scenario considers a short-term drop of the vaccination rate from 95% to 90%. While this results in an increase in the number of cases, it takes about 10 years before one can talk about a noticeable outbreak (the number of cases per week increases from 2.6 to 5.6 per week (Fig. 7)). This depletes the remaining (small) pool of susceptible people, leading to the disease being close to elimination. Not surprisingly, we observe the same pattern, but in a much stronger form, when considering a larger scare: the drop in vaccination rate from 95% to just under 80% causes an outbreak peaking at just over 25 people per week about 10 years after the event. This graph nicely illustrates the delay between the event (a scare) and the outbreak (which starts instantly but peaks quite a few years later).

Note that contrary to the impression given by the graph, the disease is not eradicated. The very low prevalence rate leads to an increasingly large number of parents deciding not to vaccinate their children. Unless government intervenes, the gradual accumulation of susceptible people will lead to large outbreaks in the future (not shown).

5. Discussion and conclusion

Against the background of plans for the eradication of measles and their delay over the last two decades due to growing scepticism of and hesitancy towards vaccinations, in particular the MMR vaccination recommended for small children, this paper introduced a stylised model of a childhood disease which explicitly integrates the behavioural influences on aspects of parental decision making. In particular, we have illustrated how incorporating parental response to a change in the prevalence of the disease impacts the outcome of governmental policies aiming to increase the vaccination coverage. The success of vaccination programmes reduces the prevalence of the disease and thus people's perception of the necessity of vaccination. At the same time, anti-vaccine movements and vaccination scares foster vaccination hesitancy. While calibrated to measles, the insights generated are applicable to other childhood diseases (e.g., pertussis or diphtheria).

The model goes beyond these epidemiological considerations by incorporating a focus on parental behaviour, perceptions and policies. The long-term consequences of the unfounded claim that the MMR vaccine causes autism illustrate the importance of accounting for these

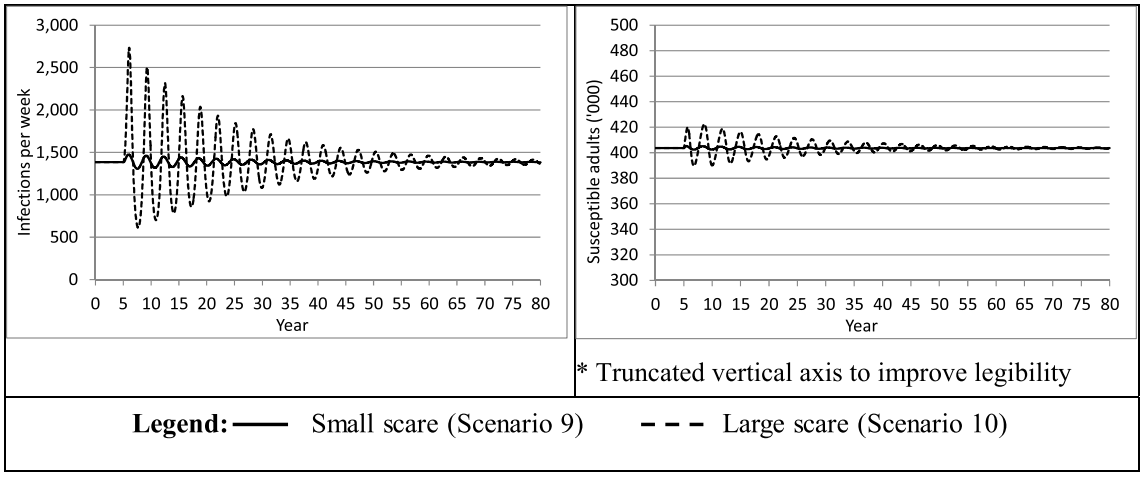


Fig. 6. Consequences of a small and large scare in a population with a 50% vaccination rate.

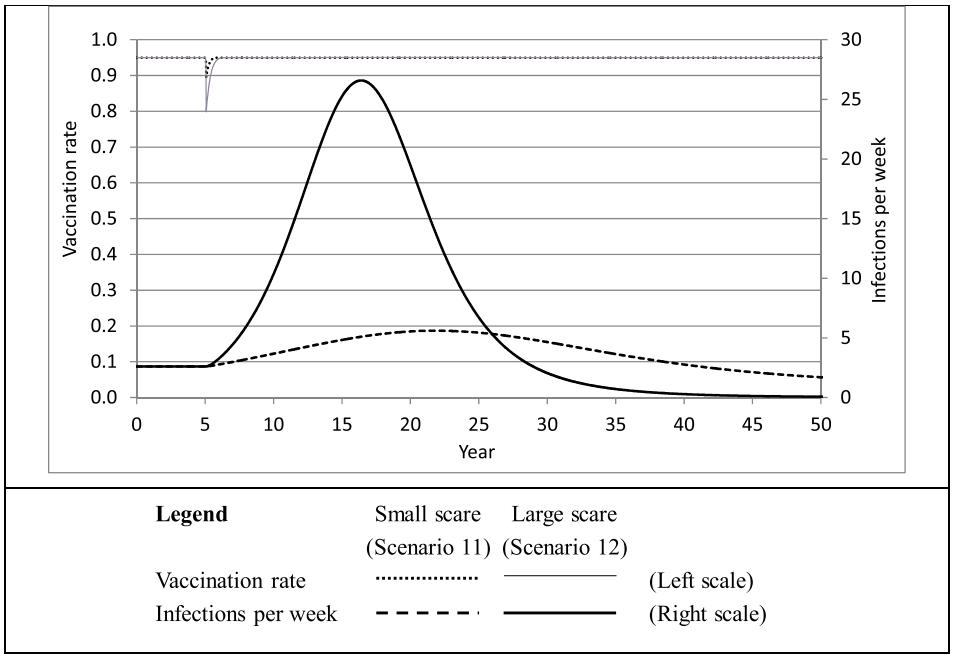


Fig. 7. Consequences of a small and a large scare in a population with a 95% vaccination rate: Infection rates (left axis) and infections per week (right axis).

issues. Including such elements in epidemiological models enables us to learn more about how these elements interact. That is what this article attempted to achieve, aiming for a better assessment of how the epidemiological effects and feedbacks interact with situational and perceptual input produced by outside institutions that vie for public attention. Our simulations illustrate the importance of incorporating the behavioural feedback loops in the model: for a given governmental policy, simulating without these feedbacks may indicate that the policy is successful in eliminating the disease, while this is far from being the case in the more realistic scenario where the behavioural loops are activated.

This model illustrates the long-term consequences of the interaction between health policies such as vaccination campaigns and the agenda of social institutions that might draw attention to specific events instrumental to their aims and thus create vaccination scares. The modelled consequences, no matter whether the vaccination rate, the number of infections or the number of susceptible persons are considered, partly endure for decades, highlighting the fact that health policy measures or public health discourse are not only matters of the day, but might have repercussions in the lives of our children and grandchildren. Therefore, our objective is to take this work further. At each step, the trade-off is between increasing model complexity and achieving more insights. For instance, from a policy point of view, including catch-up vaccination is without doubt one of the more important aspects, but this implies a significant increase in model complexity (a doubling of the number of stocks and flows).

Despite the deterministic nature of the model, the periodic ups and downs of the disease's prevalence, characteristic for epidemiological feedback, are observed, resulting not from random events, but as a consequence of behavioural reactions to vaccination campaigns or vaccination scares. This provides an indication that our model, despite being highly stylised, succeeds in capturing the essential epidemiological dynamics while incorporating selected behavioural factors, a first step towards more realistic models providing an acceptable representation of both epidemiological and behavioural factors. Faced with the task of overcoming parents' hesitancy or resistance towards the MMR vaccination, international and national health authorities, pursuing the fight against measles, may be helped by the potential of the model to describe the way different predictors of vaccination behaviour interact.

The cyclical behaviour in the number of new infections may have implications for the evaluation of vaccination programs, especially innovative programs. The time at which a programme starts, and the data to perform the evaluation are collected, may coincide with a period of high or low, increasing or decreasing stages of a cycle. A lack of awareness of this by researchers and policymakers could lead to an inaccurate evaluation of the programme, and also affect the attention paid to vaccination in the course of the program or in the wake of the publication of the evaluation results.[1]

While the governmental health policies tend to have limited durations, rarely exceeding 5 years, international bodies such as the WHO tend to set much longer-term goals, which are more appropriate given the long time-delays between actions and their consequences in the context of vaccination and epidemics. Policymakers can be advised to take a long-term perspective in both policy planning and supervision of infections. Infections should be monitored closely, even after interventions have stopped or policies abandoned, as the consequences endure well beyond.

The epidemiological part of this stylised model is purposely highly simplified: this is a conscious trade-off, as our focus is on the behavioural aspects of the parental vaccination decision. In particular, we only consider two age-classes: pre-schoolers and people over 4 years old. While this significantly simplifies the modelling, it results in certain limitations, such as the inability to simulate the impact of catch-up vaccination aimed at certain age-classes. Other forces that may turn out to be influential, but were not included in the present model, are a separate consideration of positive and negative information, which are likely to affect perceived risk differently, and influence factors affecting parents' willingness to vaccinate that are of a more general nature such as their own experience with infection in their youth or their general health beliefs.

In this paper the various scenarios have mostly focussed on the vaccination decision of positively predisposed parents. In further work we will consider in more detail the reaction of negatively predisposed parents. We also envisage refining the parental decision process, in particular by addressing the question of how long-term changes in the

[1] We thank a reviewer for pointing out the potential consequences of changes in vaccination policies coinciding with specific times of an epidemic cycle.

prevalence of a disease affect parents' reference points (i.e., what they perceive as a normal level of prevalence), and the resulting impact on their reaction to outbreaks.

Declaration of competing interest

None.

Appendix. Equation listing with comments in curly brackets

Notation

Fr: Fraction

Population groups: A: Adults, C: Children

TTP: Time to perceive {delay parameter of the exponential smoothing functions used to model perceptions, e.g., of the risk of vaccination}

InVal: Initial value of the population groups, dependent on the vaccination rate at the start of the simulation run. {The base case model is calibrated for a population of about 10 million people.}

+ (−) refers to positively (negatively) predisposed parents

Status: I: Infected, Im: Immune, V: Vaccinated, Su: Suceptible, Vsq: Severe sequels from vaccination, Sq: Severe sequels from disease, D: Death from disease, R: Recovered from disease

Indicator variables {These take on a value of 1 or 0 and are used to turn certain model parts on or off.}

I Behavioural loops	(Dmnl)
I Media coverage vaccination scare	(Dmnl)

Initial values of the population stocks and value of the parameter "Instantaneous visibility of risk of disease" for the different scenarios

	Initial vaccination rate		
	0%	50%	95%
Adults Infected	1,991.04	1,137.91	2.1266
Adults Recovered	6.35139e006	3.65397e006	6902
Adults Sequels	2,502.14	1,434	2.6923
Adults Susceptible	338,589	403,682	487,449
Adults Vaccinated	0	4.0035e006	9.36928e006
Adults Vaccinated with Sequels	0	1.00062	2.3451
Children Infected	1,026.59	593.663	1.1339
Children Recovered	85,100	49,212.8	93.9917
Children Sequels	56.3715	32.6	0.0623
Children Susceptible	349,156	421,214	519,808
Instantaneous visibility of risk of disease	113.56	65.08	0.1222

Parameters and intermediate variables based on parameters

Birthrate = 2,500	(People/ Week)
Life expectancy = 80 * 52	(Weeks)
Childhood = 4 * 52	(Weeks)
A life expectancy = Life expectancy - Childhood	(Weeks)
Perfect mixing hypothesis = 0.5{This hypothesis implies that on average, events occur halfway through the period individuals spent in a given state. For instance, Childhood lasts 4 years, so the remaining childhood period after infection (next equation) lasts 2 years}	(Dmnl)
Remaining childhood = Childhood * Perfect mixing hypothesis	(Weeks)
Reduction in life expectancy due to severe sequels = 0.75	(Dmnl)
Reduction in Life expectancy due to vaccination sequels = 0.75	(Dmnl)
Infection period = 1.25	(Weeks)
A Fr D = 0.0053; A Fr Sq = 0.0025; A Fr R = 1 - A Fr D - A Fr Sq	(Dmnl)
C Fr D = 0.003; C Fr Sq = 0.00066; C Fr R = 1 - C Fr D - C Fr Sq	(Dmnl)
Probability of Sequels of vaccination = 1/1e+006	(Dmnl)
A Infectivity factor = 11.115 {Parameter calibrated to obtain the desired value of the basic reproduction number R0}	(Dmnl/Week)
C fraction Infectivity factor = 0.5	(Dmnl)
C Infectivity factor = A Infectivity factor * C fraction Infectivity factor	(Dmnl/Week)
"TTP Risk of disease +" = 52 * 25	(Weeks)
"TTP Risk of disease -" = 52 * 5	(Weeks)
Weight Disease A Deaths = 10	(Dmnl)
Weight Disease A Sequels = 1	(Dmnl)
Weight Disease C Deaths = 10	(Dmnl)
Weight Disease C Sequels = 1	(Dmnl)
"Reference fr predisposed +" = {Fraction of parents positively predisposed, this variable equals 0.5 at time zero in the base case, and evolves over time, depending on the different communication scenarios}	(Dmnl)
"Reference fr predisposed -" = 1 - "Reference fr predisposed +"	(Dmnl)

Population groups (People)

{These are the state variables of the model, referred to as stocks. Their initial value (InVal) depends on the hypothesised vaccination rate at the start of the simulation. Their values evolve according to the population movements (in- and out-flows). For instance, Children Susceptible equals InVal at the start of the simulation, is increased each week by the Birthrate, and is decreased by the number of children being infected, or reaching the age of 4; the latter can have no vaccination, be vaccinated or be vaccinated with sequels.}

Children Susceptible
= INTEG(Birthrate - C infections - No vaccination – Vaccination - Vaccination with Sequels, InVal)
Children Infected = INTEG(C infections - C Full Recovery - C I Deaths - C Sequels, InVal)
Children Recovered = INTEG(C Full Recovery – C R to Adult, InVal)
Children Sequels = INTEG(C Sequels - C Sq to Adult, InVal)
Adults Vaccinated = INTEG(Vaccination - A V Deaths InVal)
Adults Vaccinated with Sequels = INTEG(Vaccination with Sequels - A VSq Deaths, InVal)
Adults Susceptible = INTEG(No vaccination - A infections - A Su Deaths, InVal)
Adults Infected = INTEG(A infections - A Full Recovery - A I Deaths - A Sequels, InVal)
Adults Recovered = INTEG(A Full Recovery + C R to Adult - A R Deaths, InVal)
Adults Sequels = INTEG(A Sequels + C Sq to Adult - A Sq Deaths, InVal)

Endogeneous population flows (People/Week)

Vaccination
= Children Susceptible * Vaccination rate * (1 - Probability of Sequels of vaccination)/Childhood
Vaccination with Sequels
= Children Susceptible * Vaccination rate * Probability of Sequels of vaccination/Childhood
No vaccination = Children Susceptible * (1 - Vaccination rate)/Childhood
C infections = Children Susceptible * C Weekly infection probability
C Full Recovery = Children Infected * C Fr R/Infection period
C Sequels = Children Infected * C Fr Sq/Infection period
C I Deaths = Children Infected * C Fr D/Infection period
C R to Adult = Children Recovered/Remaining childhood
C Sq to Adult = Children Sequels/Remaining childhood
A infections = Adults Susceptible * A Weekly infection probability
A Full Recovery = Adults Infected * A Fr R/Infection period
A Sequels = Adults Infected * A Fr Sq/Infection period
A I Deaths = Adults Infected * A Fr D/Infection period
A R Deaths = Adults Recovered/A Im life expectancy
A Sq Deaths = Adults Sequels/A Sq life expectancy
A Su Deaths = Adults Susceptible/A life expectancy
A V Deaths = Adults Vaccinated/A life expectancy
A VSq Deaths = Adults Vaccinated with Sequels/A VSq Life expectancy

Behavioural factors (Dmnl unless specified otherwise)

Size of scare = {Parameter used to simulate different sizes of scare, calibrated to obtain desired initial reduction in vaccination rate}
Perceived risk of vaccination = (1 + Size of scare * I Media coverage vaccination scare) *I Behavioural loops + 1 * (1 - I Behavioural loops)
"Impact of risk of vaccination on fr predisposed +" = {Function of (Perceived risk of vaccination). See Fig. 2(b) for the functional form.}
Instantaneous visibility of risks of disease = C I Deaths * Weight Disease C Deaths + C Sequels * Weight Disease C Sequels + A I Deaths * Weight Disease A Deaths + A Sequels * Weight Disease A Sequels (People/Week)
"Visibility of risks of disease +" = SMOOTHI (Instantaneous visibility of risks of disease, "TTP Risk of disease +") (People/Week) {SMOOTHI denotes the build-in exponential smoothing function}
Reference negative consequences of disease = (People/Week)
{Value of "Instantaneous visibility of risk of disease" at the start of each simulation}
"Perceived risk of disease +" = "Visibility of risks of disease +"/Reference negative consequences of disease * I Behavioural loops + 1 * (1 - I Behavioural loops)
"Impact of risk of disease on fr predisposed +" = {Function of ("Perceived risk of disease +"). See Fig. 2(a) for functional form.}
"Fr predisposed + and +" = "Reference fr predisposed +" * "Impact of risk of disease on fr predisposed +" * "Impact of risk of vaccination on fr predisposed +"
"Fr predisposed + but -" = "Reference fr predisposed +" - "Fr predisposed + and +"
"Visibility of risks of disease -" = SMOOTHI (Instantaneous visibility of risks of disease, "TTP Risk of disease -") (People/Week)
"Perceived risk of disease -" = "Visibility of risks of disease -"/Reference negative consequences of disease * I Behavioural loops + 1 * (1 - I Behavioural loops)
Fr negatively predisposed who will vaccinate = {Function of ("Perceived risk of disease -"). See Fig. 2(c) for functional form.}
"Impact of risk of disease on Fr predisposed -" = (1 - Fr negatively predisposed who will vaccinate)
"Fr predisposed - and -"= "Reference fr predisposed -" * "Impact of risk of disease on fr predisposed -"
"Fr predisposed - but +" = "Reference fr predisposed -" - "Fr predisposed - and -"
Vaccination rate = "Fr predisposed + and +" + "Fr predisposed - but +"

Other intermediate variables

A VSq Life expectancy = A life expectancy * (1 - Reduction in Life expectancy due to Vaccination sequels)	(Weeks)
A Im life expectancy = (A life expectancy * Perfect mixing hypothesis * A Full Recovery + C R to Adult * A life expectancy)/(A Full Recovery + C R to Adult)	(Weeks)
A Sq life expectancy = (1 - Reduction in life expectancy due to severe sequels) * (A life expectancy * Perfect mixing hypothesis * A Sequels + A life expectancy * C Sq to Adult)/(A Sequels + C Sq to Adult)	(Weeks)
Infections = A infections + C infections	(People/Week)
IDeaths = A I Deaths + C I Deaths	(People/Week)
Deaths = A R Deaths + A Sq Deaths + A Su Deaths + A V Deaths + I Deaths	(People/Week)
Adults = Adults Susceptible + Adults Vaccinated + Adults Infected + Adults Recovered + Adults Sequels	(People)
Children = Children Susceptible + Children Infected + Children Recovered + Children Sequels	(People)
Population Susceptible = Adults Susceptible + Children Susceptiblerowhead	(People)
Population Immune = Adults Sequels + Children Sequels + Adults Recovered + Children Recovered + Adults Vaccinated + Adults Vaccinated with Sequels	(People)
Population Infected = Adults Infected + Children Infected	(People)
Population Total = Population Immune + Population Infected + Population Susceptible	(People)
Fr Immune = Population Immune/Population Total	(Dmnl)
Fr Inf = Population Infected/Population Total	(Dmnl)
Fr R = (Adults Recovered + Children Recovered)/Population Total	(Dmnl)
Fr Se = (Adults Sequels + Children Sequels)/Population Total	(Dmnl)
Fr Su = Population Susceptible/Population Total	(Dmnl)
Fr V = Adults Vaccinated/Population Total	(Dmnl)
A Weekly infection probability = Fr Inf * A Infectivity factor	(Dmnl/Week)
C Weekly infection probability = Fr Inf * C Infectivity factor	(Dmnl/Week)
Basic reproduction number R0 = (A Infectivity factor * Adults + C Infectivity factor * Children)/Population Total * Infection period	(Dmnl)
R = Basic reproduction number R0 * Fr Su	(Dmnl)

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