



Evolutionary Vaccination Games with Premature Vaccines to Combat Ongoing Deadly Pandemic

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Abstract. We consider a vaccination game that results with the introduction of premature and possibly scarce vaccines introduced in a desperate bid to combat the otherwise ravaging deadly pandemic. The response of unsure agents amid many uncertainties makes this game completely different from the previous studies. We construct a framework that combines SIS epidemic model with a variety of dynamic behavioral vaccination responses and demographic aspects. The response of each agent is influenced by the vaccination hesitancy and urgency, which arise due to their personal belief about efficacy and side-effects of the vaccine, disease characteristics, and relevant reported information (e.g., side-effects, disease statistics etc.). Based on such aspects, we identify the responses that are stable against static mutations. By analysing the attractors of the resulting ODEs, we observe interesting patterns in the limiting state of the system under evolutionary stable (ES) strategies, as a function of various defining parameters. There are responses for which the disease is eradicated completely (at limiting state), but none are stable against mutations. Also, vaccination abundance results in higher infected fractions at ES limiting state, irrespective of the disease death rate.

Keywords: Vaccination games · ESS · Epidemic · Stochastic approximation · ODEs

1 Introduction

The impact of pandemic in today's world is unquestionable and so is the need to analyse various related aspects. There have been many disease outbreaks in the past and the most recent Covid-19 pandemic is still going on. Vaccination is known to be of great help; however, the effectiveness depends on the responses of the population ([1] and references therein). The vaccination process gets more challenging when vaccines have to be introduced prematurely, without much

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information about their efficacy or side effects, to combat the ravaging on-going pandemic. The scarcity of vaccines makes it all the more challenging.

There is a vast literature developed during the current pandemic that majorly focuses on exhaustive experiments. Recently authors in [2] discuss the importance of game theoretic and social network models for better understanding the pandemic. Our paper exactly aims to achieve this purpose. We aim to develop a mathematical framework that mimics the ongoing pandemic as closely as possible. *We consider vaccination insufficiency, hesitancy, impact of individual vaccination responses, possibility of excess deaths, lack of information (e.g., possible end of the disease, vaccine details) etc. Our model brings together the well known epidemic SIS model [3,4], evolutionary game theoretic framework [11], dynamic behavioural patterns of the individuals and demographic aspects.*

Majority of the literature on vaccination games assumes some knowledge which aids in vaccination plans; either they consider seasonal variations, or the time duration and or the time of occurrence of the disease is known etc. For example, in [5,6] authors consider a replicator dynamics-based vaccination game; each round occurs in two phases, vaccination and the disease phase. Our paper deals with agents, that continually operate under two contrasting fears, vaccination fear and the deadly pandemic fear. They choose between the two fears, by estimating the perceived cost of the two aspects. In [7] authors consider perceived vaccination costs as in our model and study the ‘wait and see’ equilibrium. Here the agents choose one among 53 weeks to get vaccinated. In contrast, *we do not have any such information*, and the aim is to eradicate the deadly disease.

In particular, we consider a population facing an epidemic with limited availability of vaccines. Any individual can either be infected, susceptible or vaccinated. *It is evident nowadays (with respect to Covid-19) that recovery does not always result in immunization. By drawing parallels, the recovered individuals become susceptible again.* We believe the individuals in this current pandemic are divided in opinion due to vaccination hesitancy and vaccination urgency. A vaccination urgency can be observed depending upon the reported information about the sufferings and deaths due to disease, lack of hospital services etc. On the other hand, the vaccination hesitancy could be due to individuals’ belief in the efficacy and the emerging fears due to the reported side effects.

To capture above factors, we model a variety of possible vaccination responses. We first consider the individuals who exhibit the *follow-the-crowd (FC)* behavior, i.e., their confidence (and hence inclination) for vaccination increases as more individuals get vaccinated. In other variant, the interest of an individual in vaccination reduces as vaccinated proportion further grows. Basically such individuals attempt to enjoy the resultant benefits of not choosing vaccination and still being prevented from the disease; we refer them as *free-riding (FR) agents*. Lastly, we consider individuals who make more informed decisions based also on the infected proportion; these agents exhibit increased urge towards vaccination as the infected population grows. We name such agents as *vigilant agents*.

In the era of pre-mature vaccine introduction and minimal information, the agents depend heavily on the perceived cost of the two contrasting (vaccination/infection cost) factors. The perceived cost of infection can be large leading to a vaccination urgency, when there is vaccine scarcity. In case there is abundance, one may perceive a smaller risk of infection and procrastinate the vaccination till the next available opportunity. *Our results interestingly indicate a larger infected proportion at ES equilibrium as vaccine availability improves.*

We intend to investigate the resultant of the vaccination response of the population and the nature of the disease. Basically, we want to understand if the disease can be overpowered by given type of vaccination participants. Our analysis is layered: firstly, *we use stochastic approximation techniques to derive time-asymptotic proportions and identify all possible equilibrium states, for any given vaccination response and disease characteristics.* Secondly, we derive the vaccination responses that are *stable against mutations*. We slightly modify the definition of classical evolutionary stable strategy, which we refer as *evolutionary stable strategy against static mutations (ESS-AS)*¹. We study the equilibrium/limiting states reached by the system under ES vaccination responses.

Under various ES (evolutionary stable) vaccination responses, the dynamic behaviour at the beginning could be different, but *after reaching equilibrium, individuals either vaccinate with probability one or zero.* Some interesting patterns are observed in ES limiting proportions as a function of important defining parameters. For example, the ES limit infected proportions are concave functions of birth rate. *With increased excess deaths, we have smaller infected proportions at ES limiting state.* Further, *there are many vaccination responses which eradicate the disease completely at equilibrium; but none of them are evolutionary stable, unless the disease can be eradicated without vaccination.* At last, we corroborate our theoretical results by performing exhaustive Monte-Carlo simulations.

2 Problem Description and Background

We consider a population facing an epidemic, where at time t , the state of the system is $(N(t), S(t), V(t), I(t))$. These respectively represent total, susceptible, vaccinated and infected population. Observe $N(t) = S(t) + V(t) + I(t)$.

At any time t , any susceptible individual can contact anyone among the infected population according to exponential distribution with parameter $\lambda/N(t)$, $\lambda > 0$ (as is usually done in epidemic models, e.g., [8]). In particular, a contact between a susceptible and an infected individual may result in spread of the disease, which is captured by λ . Any infected individual may recover after an exponentially distributed time with parameter r to become susceptible again. A susceptible individual can think of vaccination after exponentially distributed time with parameter ν . At that epoch, the final decision (to get vaccinated) depends on the information available to the individual. We refer to the probability of a typical individual getting vaccinated as q ; more details will follow

¹ Agents use dynamic policies, while mutants use static variants (refer Sect. 2).

below. *It is important to observe here that ν will also be governed by vaccination availability; the individual decision rate is upper bounded by availability rate.* Further, there can be a birth after exponentially distributed time with parameter $bN(t)$. A death is possible in any compartment after exponentially distributed time with parameter $dI(t)$ (or $dS(t)$ or $dV(t)$), and excess death among infected population with parameter $d_e I(t)$. Furthermore, we assume $b > d + d_e$.

One of the objectives is to analyse the (time) asymptotic proportions of the infected, vaccinated and susceptible population depending upon the disease characteristics and vaccination responses. To this end, we consider the following fractions:

$$\theta(t) := \frac{I(t)}{N(t)}, \quad \psi(t) := \frac{V(t)}{N(t)}, \quad \text{and} \quad \phi(t) := \frac{S(t)}{N(t)}. \quad (1)$$

2.1 Responses Towards Vaccination

In reality, the response towards available vaccines depends upon the cost of vaccination, the severity of infection and related recovery and death rates. However, with lack of information, the vaccination decision of any susceptible depends heavily on the available data, $(\theta(t), \psi(t))$, the fractions of infected and vaccinated population at that time. Further, it could depend on the willingness of the individuals towards vaccination. The final probability of getting vaccinated at the decision/availability epoch is captured by probability $q = q(\theta, \psi, \beta)$; this probability is also influenced by the parameter β which is a characteristic of the given population. Now, we proceed to describe different possible behaviours exhibited by agents/individuals in the population:

Follow the Crowd (FC) Agents: These type of agents make their decision to get vaccinated by considering only the vaccinated proportion of the population; they usually ignore other statistics. They believe vaccination in general is good, but are hesitant because of the possible side effects. The hesitation reduces as the proportion of vaccinated population increases, and thus accentuating the likelihood of vaccination for any individual. In this case, the probability that any individual will decide to vaccinate is given by $q := \beta\psi(t)$.

Free-Riding (FR) Agents: These agents exhibit a more evolved behaviour than FC individuals, in particular free-riding behaviour. In order to avoid any risks related to vaccination or paying for its cost, they observe the crowd behaviour more closely. Their willingness towards vaccination also improves with $\psi(t)$, however they tend to become free-riders beyond a limit. In other words, as $\psi(t)$ further grows, their tendency to vaccinate starts to diminish. We capture such a behaviour by modelling the probability as $q := \beta\psi(t)(1 - \psi(t))$.

Vigilant Agents: Many a times individuals are more vigilant, and might consider vaccination only if the infected proportion is above a certain threshold, in which case we set $q = \beta\psi(t)\mathbb{1}_{\{\theta(t) > \Gamma\}}$ (Γ is an appropriate constant). This dependency could also be continuous, we then model the probability as $q = \beta\theta(t)\psi(t)$. We refer to them as Vigilant Follow-the-Crowd (VFC) agents.

Vaccination Policies: We refer the vaccination responses of the agents as vaccination policy, which we represent by $\pi(\beta)$. When the vaccination response policy is $\pi(\beta)$, the agents get vaccinated with probability $q_{\pi(\beta)}(\theta, \psi)$ at the vaccination decision epoch, based on the system state (θ, ψ) at that epoch. For ease of notation, we avoid π and β while representing q .

As already explained, we consider the following vaccination policies: a) when $\pi(\beta) = F_C(\beta)$, i.e., with follow-the-crowd policy, the agents choose to vaccinate with probability $q(\theta, \psi) = \min\{1, \tilde{q}(\theta, \psi)\}$, $\tilde{q}(\theta, \psi) = \beta\psi$ at vaccination decision epoch; b) when $\pi(\beta) = F_R(\beta)$, the free-riding policy, $\tilde{q}(\theta, \psi) = \beta\psi(1 - \psi)$; and (c) the policy $\pi(\beta) = V_{FC}^1(\beta)$ represents vigilant (w.r.t. θ) follow the crowd (w.r.t. ψ) policy and $\tilde{q}(\theta, \psi) = \beta\theta\psi$. We define $\Pi := \{F_C, F_R, V_{FC}^1\}$ to be the set of all these policies. We discuss the fourth type of policy V_{FC}^2 with $\tilde{q}(\theta, \psi) = \beta\theta\mathbb{1}_{\{\theta > \Gamma\}}$ separately, as the system responds very differently to these agents. Other behaviour patterns are for future study.

2.2 Evolutionary Behaviour

The aim of this study is three fold: (i) to study the dynamics and understand the equilibrium states (settling points) of the disease depending upon the agents' behavior and the availability of the vaccine, (ii) to compare and understand the differences in the equilibrium states depending on agents' response towards vaccine, and (iii) to investigate if these equilibrium states are stable against mutations, using the well-known concepts of evolutionary game theory. Say a mutant population of small size invades such a system in equilibrium. We are interested to investigate if the agents using the original (vaccination response) are still better than the mutants. Basically we use the concept of evolutionary stable strategy (ESS), which in generality is defined as below (e.g., [11]):

By definition, for $\pi(\beta)$ to be an ESS, it should satisfy two conditions, i) $\pi(\beta) \in \arg \min_{\pi \in \tilde{\Pi}} u(\pi; \pi(\beta))$, where $\tilde{\Pi}$ is the set of the policies, $u(\pi, \pi')$ is the utility/cost of the (can be mutant) user that adapts policy π , while the rest of the population uses policy π' ; and ii) it should be stable against mutations, i.e. there exist an $\bar{\epsilon}$, such that for all $\epsilon \leq \bar{\epsilon}(\pi)$,

$$u(\pi, \pi_\epsilon(\beta, \pi)) > u(\pi(\beta), \pi_\epsilon(\beta, \pi)) \text{ for any } \pi \neq \pi(\beta),$$

where $\pi_\epsilon(\beta, \pi) := \epsilon\pi + (1 - \epsilon)\pi(\beta)$, represents the policy in which ϵ fraction of agents (mutants) use strategy π and the other fraction uses $\pi(\beta)$.

In the current paper, we restrict our definition of ESS to cater for mutants that use static policies, Π^D . Under any static policy $\pi \equiv q$, the agent gets vaccinated with constant probability q at any decision epoch, irrespective of the system state. We now define the *Evolutionary Stable Strategies, stable Against Static mutations* and the exact definition is given below.

Definition 1. [ESS-AS] A policy $\pi(\beta)$ is said to be ESS-AS, i) if $\{q_{\pi(\beta)}^*\} = \mathcal{B}(\pi(\beta))$, where the static-best response set

$$\mathcal{B}(\pi(\beta)) := \arg \min_{q \in [0,1]} u(q, \pi(\beta)),$$

and $q_{\pi(\beta)}^* = q(\theta^*, \psi^*)$ is the probability with which the agents get vaccinated after the system reaches equilibrium under strategy $\pi(\beta)$; and ii) there exists an $\bar{\epsilon}$ such that $\{q_{\pi(\beta)}^*\} = \mathcal{B}(\pi_\epsilon(\beta, q))$, for any $\epsilon \leq \bar{\epsilon}(q)$ and any q .

Anticipated Utility/Cost of a User: Once the population settles to an equilibrium (call it $\hat{\theta}, \hat{\psi}$) under a certain policy $\pi(\hat{\beta})$, the users (assume to) incur a cost that depends upon various factors. To be more specific, any user estimates² its overall cost of vaccination considering the pros and cons as below to make a judgement about vaccination.

The cost of infection (as perceived by the user) can be summarized by $p_I(\hat{\theta})(c_{I_1} + c_{I_2}d_e\hat{\theta})$, where $p_I(\hat{\theta})$ equals the probability that the user gets infected before its next decision epoch (which depends upon the fraction of infected population $\hat{\theta}$ and availability/decision rate ν), c_{I_1} is the cost of infection without death (accounts for the sufferings due to disease, can depend on r), while $c_{I_2}d_e\hat{\theta}$ is the perceived chance of death after infection. Observe here that $d_e\hat{\theta}$ is the fraction of excess deaths among infected population which aids in this perception.

On the contrary, the cost of vaccination is summarized by $c_{v_1} + \min\{\bar{c}_{v_2}, \frac{c_{v_2}}{\hat{\psi}}\}$, where c_{v_1} is the actual cost of vaccine. Depending upon the fraction $\hat{\psi}$ of the population vaccinated and their experiences, the users anticipate additional cost of vaccination (caused due to side-effects) as captured by the second term $\frac{c_{v_2}}{\hat{\psi}}$. Inherently we assume here that the side effects are not significant, and hence in a system with a bigger vaccinated fraction, the vaccination hesitancy is lesser. Here \bar{c}_{v_2} accounts for maximum hesitancy. In all, the expected anticipated cost of vaccination by a user in a system at equilibrium (reached under $\pi(\hat{\beta})$) equals: the probability of vaccination (say q) times the anticipated cost of vaccination, plus $(1 - q)$ times the anticipated cost of infection. Thus we define:

Definition 2. [User utility at equilibrium]

When the population is using policy $\pi(\hat{\beta})$ and an agent attempts to get itself vaccinated with probability q , then, the user utility function is given by:

$$\begin{aligned} u(q; \pi(\hat{\beta})) &:= q \left(c_{v_1} + \min \left\{ \bar{c}_{v_2}, \frac{c_{v_2}}{\hat{\psi}} \right\} \right) + (1 - q)p_I(\hat{\theta})(c_{I_1} + c_{I_2}d_e\hat{\theta}) \\ &= qh(\pi(\hat{\beta})) + p_I(\hat{\theta})(c_{I_1} + c_{I_2}d_e\hat{\theta}), \text{ where} \\ h(\pi(\hat{\beta})) &= h(\hat{\theta}, \hat{\psi}) := c_{v_1} + \min \left\{ \bar{c}_{v_2}, \frac{c_{v_2}}{\hat{\psi}} \right\} - p_I(\hat{\theta})(c_{I_1} + c_{I_2}d_e\hat{\theta}). \end{aligned} \quad (2)$$

In the next section, we begin with ODE approximation of the system, which facilitates in deriving the limiting behaviour of the system. Once the limiting behaviour is understood, we proceed towards evolutionary stable strategies.

² We assume mutants are more rational, estimate various rates using reported data.

3 Dynamics and ODE Approximation

Our aim in this section is to understand the limiting behaviour of the given system. The system is transient with $b > d + d_e$; it is evident that the population would not settle to a stable distribution (it would explode as time progresses with high probability). However the fraction of people in various compartments (given by (1)) can possibly reach some equilibrium and we look out for this equilibrium or limiting proportions (as is usually considered in literature [9]).

To study the limit proportions, it is sufficient to analyse the process at transition epochs. Let τ_k be the k^{th} transition epoch, and infected population immediately after τ_k equals $I_k := I(\tau_k^+) = \lim_{t \downarrow \tau_k} I(t)$; similarly, define N_k, S_k and V_k . Observe here that $T_{k+1} := \tau_{k+1} - \tau_k$ is exponentially distributed with a parameter that depends upon previous system state (N_k, S_k, I_k, V_k) .

Transitions: Our aim is to derive the (time) asymptotic fractions of (1). Towards this, we define the same fractions at transition epochs,

$$\theta_k := \frac{I_k}{N_k}, \psi_k := \frac{V_k}{N_k}, \text{ and } \phi_k := \frac{S_k}{N_k}.$$

Observe that $\theta_k + \psi_k + \phi_k = 1$. To facilitate our analysis, we also define a slightly different fraction, $\eta_k := N_k/k$ for $k > 1$ and $\eta_0 := N(0)$, $\eta_1 := N(1)$. As described in previous section, the size of the infected population evolves between two transition epochs according to:

$$I_{k+1} = I_k + G_{I,k+1}, \text{ with } G_{I,k+1} := \mathbb{I}_{k+1} - \mathbb{R}_{k+1} - \mathbb{D}_{I,k+1}, \quad (3)$$

where \mathbb{I}_{k+1} is the indicator that the current epoch is due to a new infection, \mathbb{R}_{k+1} is the indicator of a recovery and $\mathbb{D}_{I,k+1}$ is the indicator that the current epoch is due to a death among the infected population. Let $\mathcal{F}_k := \sigma(I_j, S_j, V_j, N_j, j \leq k)$ represent the sigma algebra generated by the history until the observation epoch k and let $E_k[\cdot]$ represent the corresponding conditional expectation. By conditioning on \mathcal{F}_k , using the memory-less property of exponential random variables,

$$\begin{aligned} E_k[\mathbb{I}_{k+1}] &= \frac{\frac{\lambda I_k S_k}{N_k}}{N_k b + N_k d + I_k d_e + \frac{\lambda I_k S_k}{N_k} + S_k \nu + I_k r} = \frac{\lambda \theta_k \phi_k}{\varrho_k}, \text{ with,} \\ \varrho_k &:= b + d + d_e \theta_k + \lambda \theta_k \phi_k + \nu \phi_k + r \theta_k, \\ E_k[\mathbb{R}_{k+1}] &= \frac{r \theta_k}{\varrho_k}, \text{ and, } E_k[\mathbb{D}_{I,k+1}] = \frac{\theta_k (d + d_e)}{\varrho_k}. \end{aligned} \quad (4)$$

In similar lines the remaining types of the population evolve according to the following, where $\mathbb{V}_{k+1}, \mathbb{B}_{k+1}, \mathbb{D}_{V,k+1}$ and $\mathbb{D}_{S,k+1}$ are respectively the indicators of vaccination, birth and corresponding deaths,

$$V_{k+1} = V_k + G_{V,k+1}, \quad G_{V,k+1} := \mathbb{V}_{k+1} - \mathbb{D}_{V,k+1}, \quad (5)$$

$$N_{k+1} = N_k + G_{N,k+1}, \quad G_{N,k+1} := \mathbb{B}_{k+1} - \mathbb{D}_{I,k+1} - \mathbb{D}_{V,k+1} - \mathbb{D}_{S,k+1}, \text{ and,}$$

$$S_{k+1} = N_{k+1} - I_{k+1} - V_{k+1}.$$

As before,

$$\begin{aligned} E_k[\mathbb{V}_{k+1}] &= \frac{\nu q(\theta_k, \psi_k) \phi_k}{\varrho_k}, \quad E_k[\mathbb{D}_{V,k+1}] = \frac{\psi_k d}{\varrho_k}, \\ E_k[\mathbb{D}_{S,k+1}] &= \frac{\phi_k d}{\varrho_k}, \quad \text{and, } E_k[\mathbb{B}_{k+1}] = \frac{b}{\varrho_k}. \end{aligned} \quad (6)$$

Let $\Upsilon_k := [\theta_k, \psi_k, \eta_k]^T$. The evolution of Υ_k can be studied by a three dimensional system, described in following paragraphs. To facilitate tractable mathematical analysis we consider a slightly modified system that freezes once η_k reaches below a fixed small constant $\delta > 0$. The rationale and the justification behind this modification is two fold: a) once the population reaches below a significantly small threshold, it is very unlikely that it explodes and the limit proportions in such paths are no more interesting; b) the initial population $N(0)$ is usually large, let $\delta = 2/(N(0) - 1)$ and then with $b > d + d_e$, it is easy to verify that the probability, $P(\eta_k < \delta \text{ for some } k | N(0)) \rightarrow 0$ as $N(0) \rightarrow \infty$. From (3),

$$\begin{aligned} \frac{I_{k+1}}{N_{k+1}} &= \frac{I_k}{N_k} + \frac{I_{k+1}}{N_{k+1}} - \frac{I_k}{N_k} = \frac{I_k}{N_k} + \frac{1}{k+1} \frac{k+1}{N_{k+1}} \left[I_{k+1} - \frac{I_k N_{k+1}}{N_k} \right], \\ &= \frac{I_k}{N_k} + \frac{1}{k+1} \frac{k+1}{N_{k+1}} \left[G_{I,k+1} - \frac{N_{k+1} - N_k}{N_k} I_k \right], \quad \text{and thus including } \mathbb{1}_{\{\eta_k > \delta\}}, \\ \theta_{k+1} &= \theta_k + \epsilon_k \frac{\mathbb{1}_{\{\eta_k > \delta\}}}{\eta_{k+1}} [G_{I,k+1} - (N_{k+1} - N_k) \theta_k], \quad \epsilon_k := \frac{1}{k+1}. \end{aligned} \quad (7)$$

We included the indicator $\mathbb{1}_{\{\eta_k > \delta\}}$, as none of the population types change (nor there is any evolution) once the population gets almost extinct. Similarly, from equation (5),

$$\begin{aligned} \psi_{k+1} &= \psi_k + \epsilon_k \frac{\mathbb{1}_{\{\eta_k > \delta\}}}{\eta_{k+1}} [G_{V,k+1} - (N_{k+1} - N_k) \psi_k], \\ \eta_{k+1} &= \eta_k + \mathbb{1}_{\{\eta_k > \delta\}} \epsilon_k [G_{N,k+1} - \eta_k]. \end{aligned} \quad (8)$$

We analyse this system using the results and techniques of [10]. In particular, we prove equicontinuity in extended sense for our non-smooth functions (e.g., $q(\theta, \psi)$ may only be measurable), and then use [10, Chapter 5, Theorem 2.2]. Define $L_{k+1} := [L_{k+1}^\theta, L_{k+1}^\psi, L_{k+1}^\eta]^T$, with

$$\begin{aligned} L_{k+1}^\theta &= \frac{\mathbb{1}_{\{\eta_k > \delta\}}}{\eta_{k+1}} [G_{I,k+1} - (N_{k+1} - N_k) \theta_k], \\ L_{k+1}^\psi &= \frac{\mathbb{1}_{\{\eta_k > \delta\}}}{\eta_{k+1}} [G_{V,k+1} - (N_{k+1} - N_k) \psi_k], \quad \text{and, } L_{k+1}^\eta = \mathbb{1}_{\{\eta_k > \delta\}} (G_{N,k+1} - \eta_k). \end{aligned} \quad (9)$$

Thus (7)–(8) can be rewritten as, $\Upsilon_{k+1} = \Upsilon_k + \epsilon_k L_{k+1}$. Conditioning as in (4):

$$\begin{aligned} E_k[L_{k+1}^\theta] &= \frac{\theta_k \mathbb{1}_{\{\eta_k > \delta\}}}{\eta_k \varrho_k} [\phi_k \lambda - r - d_e - (b - d_e \theta_k)] + \alpha_k^\theta \\ &=: g^\theta(\Upsilon_k) + \alpha_k^\theta, \quad \text{where, } \alpha_k^\theta = E_k \left[L_{k+1}^\theta - \frac{\eta_{k+1}}{\eta_k} L_{k+1}^\theta \right]. \end{aligned} \quad (10)$$

In exactly similar lines, we define $g(\Upsilon_k) = [g^\theta(\Upsilon_k), g^\psi(\Upsilon_k), g^\eta(\Upsilon_k)]^T$ (details just below) and α_k^ψ such that $E_k[L_{k+1}^\psi] = g^\psi(\Upsilon_k) + \alpha_k^\psi$ and $E_k[L_{k+1}^\eta] = g^\eta(\Upsilon_k)$. Our claim is that the error terms would converge to zero (shown by Lemma 3 in Appendix) and ODE $\dot{\Upsilon} = g(\Upsilon)$ approximates the system dynamics, where,

$$\begin{aligned} g^\theta(\Upsilon) &= \frac{\theta \mathbb{1}_{\{\eta > \delta\}}}{\eta \varrho} [\phi \lambda - r - d_e - (b - d_e \theta)], \quad \phi = 1 - \theta - \psi \\ g^\psi(\Upsilon) &= \frac{\mathbb{1}_{\{\eta > \delta\}}}{\eta \varrho} [q(\theta, \psi) \phi \nu - (b - d_e \theta) \psi], \quad \text{and,} \\ g^\eta(\Upsilon) &= \mathbb{1}_{\{\eta > \delta\}} \left(\frac{b - d - d_e \theta}{\varrho} - \eta \right), \quad \varrho = b + d + d_e \theta + \lambda \theta \phi + \nu \phi + r \theta. \end{aligned} \quad (11)$$

We now state our first main result (with proof in Appendix A).

A. Let the set A be locally asymptotically stable in the sense of Liapunov for the ODE (11). Assume that $\{\Upsilon_n\}$ visits a compact set, S_A , in the domain of attraction, D_A , of A infinitely often (i.o.) with probability $\rho > 0$.

Theorem 1. *Under assumption A., i) the sequence converges, $\Upsilon_n \rightarrow A$ as $n \rightarrow \infty$ with probability at least ρ ; and ii) for every $T > 0$, almost surely there exists a sub-sequence (k_m) such that: $(t_k := \sum_{i=1}^k \epsilon_i)$*

$$\sup_{k: t_k \in [t_{k_m}, t_{k_m} + T]} d(\Upsilon_k, \Upsilon_*(t_k - t_{k_m})) \rightarrow 0, \quad \text{as } m \rightarrow \infty, \quad \text{where,}$$

$\Upsilon_*(\cdot)$ is the solution of ODE (11) with initial condition $\Upsilon_*(0) = \lim_{k_m} \Upsilon_{k_m}$. ■

Using above Theorem, one can derive the limiting state of the system using that of the ODE (in non-extinction sample paths, i.e., when $\eta_k > \delta$ for all k). Further, for any finite time window, there exists a sub-sequence along which the disease dynamics are approximated by the solution of the ODE. The ODE should initiate at the limit of the system along such sub-sequence.

4 Limit Proportions and ODE Attractors

So far, we have proved that the embedded process of the system can be approximated by the solutions of the ODEs (11) (see Theorem (1)). We will now analyse the ODEs and look for equilibrium states for a given vaccination policy $\pi(\hat{\beta})$. The following notations are used throughout: we represent the parameter by $\hat{\beta}$ and the corresponding equilibrium states by $(\hat{\theta}, \hat{\psi})$. Let $\hat{q} := q(\hat{\theta}, \hat{\psi})$ and $\hat{\varrho} := \varrho(\hat{\theta}, \hat{\psi})$. In the next section, we identify the evolutionary stable (ES) equilibrium states (θ^*, ψ^*) , among these equilibrium states, and the corresponding vaccination policies $\pi(\beta^*)$.

We now identify the attractors of the ODEs (11) that are locally asymptotically stable in the sense of Lyapunov (referred to as attractors), which is a requirement of the assumption A. However, we are yet to identify the domains

of attraction, which will be attempted in future. Not all infectious diseases lead to deaths. One can either have: (i) *non-deadly disease*, where only natural deaths occur, $d_e = 0$, or (ii) *deadly disease* where in addition, we have excess deaths due to disease, $d_e > 0$. We begin with the non-deadly case and FC agents.

The equilibrium states for FC agents ($\hat{\beta} \geq 0$) are (proof in Appendix B):

Theorem 2. [FC agents] Define $\rho := \lambda/(r + b + d_e)$, $\mu := b/\nu$. When $d_e = 0$ and $\tilde{q}(\theta, \psi) = \hat{\beta}\psi$, at the attractor we have $\hat{\eta} = (b-d)/\hat{e}$. The remaining details of the attractors are in Table 1. The interior attractors (when $(\hat{\theta}, \hat{\psi}) \in (0, 1) \times (0, 1)$) are the zeros of the right hand side (RHS) of ODE (11). ■

Table 1. Attractors for FC agents, $(\theta_E, \psi_E) := \left(1 - \frac{1}{\rho} - \frac{1}{\mu\rho}, \frac{1}{\mu\rho}\right)$

Nature	Parameters		$(\hat{\theta}, \hat{\psi})$
Endemic, $\rho > 1$	$\hat{\beta} < \mu\rho$		$\left(1 - \frac{1}{\rho}, 0\right)$
	$\hat{\beta} > \mu\rho$, $\tilde{q}(0, 1 - \frac{\mu}{\hat{\beta}}) < 1$ implies $\hat{\beta} < \mu + 1^*$		$\left(0, 1 - \frac{\mu}{\hat{\beta}}\right)$
	$\hat{\beta} > \mu\rho$, $\tilde{q}(0, 1 - \frac{\mu}{\hat{\beta}}) > 1$ implies $\hat{\beta} > \mu + 1$	$\mu\rho < \mu + 1$	$\left(0, \frac{1}{\mu+1}\right)$
		$\mu\rho > \mu + 1$	(θ_E, ψ_E)
SE, $\rho < 1$	$\mu > \hat{\beta}$		$(0, 0)$

In all the tables of this section, the * entries are also valid when $\rho < 1$. When $\hat{\beta} = \mu\rho$, the ODE (and hence the system) is not stable; such notions are well understood in the literature and we avoid such marginal cases. We now consider the FR agents (proof again in Appendix B):

Theorem 3. [FR agents] When $\tilde{q}(\theta, \psi) = \hat{\beta}\psi(1 - \psi)$ and $d_e = 0$, then the attractors for ODE (11) are $(\hat{\theta}, \hat{\psi}, (b-d)/\hat{e})$, which are provided in Table 2. The interior attractors are the zeros of the RHS of ODE (11). ■

Table 2. Attractors for FR agents, $(\theta_E, \psi_E) := \left(1 - \frac{1}{\rho} - \frac{1}{\mu\rho}, \frac{1}{\mu\rho}\right)$

Nature	Parameters		$(\hat{\theta}, \hat{\psi})$
Endemic, $\rho > 1$	$\hat{\beta} < \mu\rho$		$\left(1 - \frac{1}{\rho}, 0\right)$
	$\hat{\beta} > \mu\rho$, $\tilde{q}(\hat{\theta}, \hat{\psi}) < 1$	$\hat{\beta} > \rho^2\mu^*$	$\left(0, 1 - \sqrt{\frac{\mu}{\hat{\beta}}}\right)$
		$\hat{\beta} < \rho^2\mu$	$\left(\frac{\mu\rho}{\hat{\beta}} - \frac{1}{\rho}, 1 - \frac{\mu\rho}{\hat{\beta}}\right)$
	$\hat{\beta} > \rho^2\mu$, $\tilde{q}\left(0, 1 - \sqrt{\frac{\mu}{\hat{\beta}}}\right) > 1$	$\mu + 1 > \mu\rho$	$\left(0, \frac{1}{\mu+1}\right)$
		$\mu + 1 < \mu\rho$	(θ_E, ψ_E)
SE, $\rho < 1$	$\mu\rho < \hat{\beta} < \rho^2\mu$, $\tilde{q}\left(\frac{\mu\rho}{\hat{\beta}} - \frac{1}{\rho}, 1 - \frac{\mu\rho}{\hat{\beta}}\right) > 1$ $\implies \mu\rho > (\mu + 1)$		(θ_E, ψ_E)
	$\mu > \hat{\beta}$		$(0, 0)$

As seen from the two theorems, we have two types of attractors: (i) interior attractors (for e.g., third row in Tables 1 and 2) in which $(\hat{\theta}, \hat{\psi}) \in (0, 1) \times (0, 1)$, and (ii) boundary attractors, where at least one of the components is 0. In the latter case, either the disease is eradicated with the help of vaccination ($\hat{\theta} = 0$, $\hat{\psi} > 0$) or the disease gets cured without the help of vaccination ($\hat{\theta} = \hat{\psi} = 0$) or no one gets vaccinated ($\hat{\psi} = 0$). In the last case, the fraction of infected population reaches maximum possible level for the given system, which we refer to as non-vaccinated disease fraction (NVDF), ($\hat{\theta}^N = 1 - 1/\rho$) (first row in Tables 1 and 2). Further, $\hat{\eta}$ is always in $(0, 1)$. Furthermore, important characteristics of the attractors depend upon quantitative parameters describing the nature and the spread of the disease, and the vaccination responses:

- *Endemic disease*: The disease is not self-controllable with $\rho > 1$ and the eventual impact of the disease is governed by the attitude of agents towards vaccination (all rows other than the last in Tables 1 and 2).
- *Self-eradicating (SE) Disease*: The disease is not highly infectious ($\rho < 1$) and can be eradicated without exogenous aid (vaccine).

The above characterisation interestingly draws parallels from queuing theory. One can view λ as the arrival of infection and $r + b + d_e$ as its departure. Then $\rho = \lambda/(r+b+d_e)$, resembles the load factor. It is well known that queuing systems are stable when $\rho < 1$, similarly in our case, the disease gets self-eradicating with $\rho < 1$. The attractors for VFC1 agents (proof in Appendix B):

Theorem 4. [VFC1 agents] When $\hat{\beta} \leq 2\mu\rho^2$ or when $\hat{q} = 1$ with $\tilde{q}(\hat{\theta}, \hat{\psi}) \neq 1$, the attractors for ODE (11) are $(\hat{\theta}, \hat{\psi}, {}^{(b-d)/\hat{\theta}})$, and are provided in Table 3. The interior attractors are the zeroes of the RHS of ODE (11). ■

Table 3. Attractors for VFC1 agents: Disease never gets eradicated with $\rho > 1$

Nature	Parameters	$(\hat{\theta}, \hat{\psi})$
Endemic, $\rho > 1$	$\hat{\beta} < \mu \left(\frac{\rho^2}{\rho-1} \right)$	$\left(1 - \frac{1}{\rho}, 0 \right)$
	$\hat{\beta} > \mu \left(\frac{\rho^2}{\rho-1} \right), \quad \tilde{q}(\hat{\theta}, \hat{\psi}) < 1$	$\left(\frac{\mu\rho}{\hat{\beta}}, 1 - \frac{1}{\rho} - \frac{\mu\rho}{\hat{\beta}} \right)$
	$\tilde{q} \left(\frac{\mu\rho}{\hat{\beta}}, 1 - \frac{1}{\rho} - \frac{\mu\rho}{\hat{\beta}} \right) > 1 \implies \rho\mu > \mu + 1$ and $\hat{\beta} > \frac{(\rho\mu)^2}{\rho\mu - \mu - 1}$	(θ_E, ψ_E)
SE, $\rho < 1$		$(0, 0)$

Key observations and comparisons of the various equilibrium states:

- When the disease is self-eradicating, agents need not get vaccinated to eradicate the disease. For all the type of agents, $\hat{\theta} = 0$ (and so is $\hat{\psi} = 0$) for all $\hat{\beta} < \mu$.

- With endemic disease, *it is possible to eradicate the disease only if the agents get vaccinated aggressively*. The FC agents with $\hat{\beta} > \rho\mu$ and FR agents with a bigger $\hat{\beta} > \rho^2\mu$ can completely eradicate the disease; this is possible only when $\mu\rho < \mu + 1$. However interestingly, *these parameters can't drive the system to an equilibrium that is stable against mutations, as will be seen in the next section*.
- With a lot more aggressive FR/FC agents, the system reaches disease free state ($\hat{\theta} = 0$ for all bigger $\hat{\beta}$), however with bigger vaccinated fractions ($\leq 1/(\mu+1)$).
- Interestingly *such an eradicating equilibrium state is not observed with vigilant agents* (Table 3). This is probably analogous to the well known fact that the rational agents often pay high price of anarchy.
- For certain behavioural parameters, system reaches an equilibrium state at which vaccinated and infected population co-exist. Interestingly for all three types of agents some of the co-existing equilibrium are exactly the same (e.g., (θ_E, ψ_E) in Tables and left plot of Fig. 1). In fact, such equilibrium are stable against mutations, as will be seen in the next section.

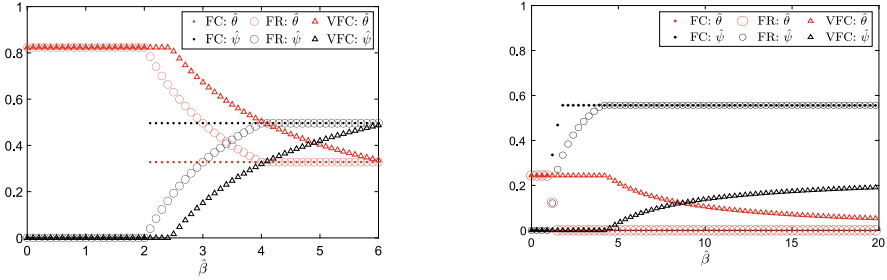


Fig. 1. Attractors for FC, FR, VFC1 agents versus β

A numerical example is presented in Fig. 1 that depicts many of the above observations. The parameters in respective plots are ($r = 1.188, \nu = 0.904, \lambda = 8.549$) and ($r = 1.0002, \nu = 0.404, \lambda = 1.749$), with $b = 0.322$. From the left plot, it can be seen that for all agents, $(\hat{\theta}, \hat{\psi})$ equals NVDF for smaller values of $\hat{\beta}$. As $\hat{\beta}$ increases, proportions for FC agents directly reach (θ_E, ψ_E) . However, with FR and VFC1 agents, the proportions gradually shift from another interior attractor to finally settle at (θ_E, ψ_E) . Further, for the right plot ($\mu\rho < \mu + 1$) the disease is eradicated with FC (when $\hat{\beta} > \rho\mu$), FR (when $\hat{\beta} > \rho^2\mu$), i.e., $(\hat{\theta}, \hat{\psi})$ settles to $(0, 1/(\mu+1))$, which is not a possibility in VFC1 agents. For the latter type, the proportions traverse through an array of co-existence equilibrium, and would approach $(0, 1 - 1/\rho)$ as $\hat{\beta}$ increases (see row 2 in Table 3).

Table 4. Attractors for FC agents (deadly disease)

Nature	Parameters		$(\hat{\theta}, \hat{\psi})$
Endemic, $\rho > 1$	$\hat{\beta} > \rho\mu^*$		$\left(0, 1 - \frac{\mu}{\beta}\right)$
	$\hat{\beta} < \rho\mu$	$\rho_e\mu_e > 1$ or $\hat{\beta}\nu < b - d_e$	$\left(1 - \frac{1}{\rho_e}, 0\right)$
		$\rho_e\mu_e < 1, \hat{\beta}\nu > b - d_e$	$\left(\theta^*, 1 - \theta^* \left(1 - \frac{d_e}{\lambda}\right) - \frac{1}{\rho}\right),$ $\theta^* = \frac{\beta\nu \left(\frac{\mu}{\beta} - \frac{1}{\rho_e}\right)}{d_e \left(1 - \frac{\hat{\beta}\nu}{\lambda}\right)}$
SE, $\rho < 1$	$\hat{\beta} < \mu$		$(0, 0)$

Deadly Disease: We now consider the deadly disease scenario ($d_e > 0$) with FC and FR agents. Let $\rho_e := \frac{\lambda - d_e}{r + b}, \mu_e := \frac{b - d_e}{\beta\nu - d_e}$. We conjecture the attractors with $\hat{\beta} \leq 1$ in Tables 4, 5 respectively, with $A := d_e\hat{\beta}\nu$, $B := -(r + b + d_e)\hat{\beta}\nu + d_e\lambda$, and $C := b\lambda + d_e(r + d_e)$. Further, the candidate attractors with $\bar{q}(\hat{\theta}, \hat{\beta}) > 1$ are provided in the next section, which is of interest to ESS-AS. We expect that the proofs can be extended analogously and will be a part of future work, along with identifying and proving the attractors for other cases.

Table 5. Attractors for FR agents (deadly disease)

Nature	Parameters		$(\hat{\theta}, \hat{\psi})$
Endemic, $\rho > 1$	$\hat{\beta} > \rho\mu$	$\hat{\beta} > \rho^2\mu^*$	$\left(0, 1 - \sqrt{\frac{\mu}{\beta}}\right)$
		$\hat{\beta} < \rho^2\mu$	$\left(1 - \frac{1}{\rho_e} - \frac{\lambda\hat{\psi}}{\lambda - d_e}, \hat{\psi}\right),$ $\hat{\psi} = 1 - \frac{-B - \sqrt{B^2 - 4AC}}{2A}$
	$\hat{\beta} < \rho\mu$	$\rho_e\mu_e > 1$ or $\hat{\beta}\nu < b - d_e$	$\left(1 - \frac{1}{\rho_e}, 0\right)$
		$\rho_e\mu_e < 1, \hat{\beta}\nu > b - d_e$	$\left(1 - \frac{1}{\rho_e} - \frac{\lambda\hat{\psi}}{\lambda - d_e}, \hat{\psi}\right),$ $\hat{\psi} = 1 - \frac{-B - \sqrt{B^2 - 4AC}}{2A}$
SE, $\rho < 1$	$\hat{\beta} < \mu$		$(0, 0)$

5 Evolutionary Stable Vaccination Responses

Previously, for a given user behaviour, we showed that the system reaches an equilibrium state, and identified the corresponding equilibrium states in terms of limit proportions. If such a system is invaded by mutants that use a different vaccination response, the system can get perturbed, and there is a possibility that the system drifts away. We now identify those equilibrium states, which are evolutionary stable against static mutations. Using standard tools of evolutionary game theory, we will show that the mutants do not benefit from deviating

under certain subset of policies. That is, we identify the ESS-AS policies defined at the end of Sect. 2.

We begin our analysis with $d_e = 0$, the case with no excess deaths (due to disease). From Tables 1, 2 and 3, with $\rho > 1$, for all small values of $\hat{\beta}$ (including $\hat{\beta} = 0$), irrespective of the type of the policy, the equilibrium state remains the same (at NVDF), $(\hat{\theta}, \hat{\psi}) = (1 - 1/\rho, 0)$. Thus, the value of $h(\cdot)$ in user utility function (2) for all such small $\hat{\beta}$ equals the same value and is given by:

$$h_m := h(\pi(0)) = h\left(1 - \frac{1}{\rho}, 0\right) = c_{v1} + \bar{c}_{v2} - p_I(\theta) c_{I1}, \text{ with, } p_I(\theta) := \frac{\lambda\theta}{\lambda\theta + \nu}. \quad (12)$$

In the above, $p_I(\cdot)$ is the probability that the individual gets infected before the next vaccination epoch. The quantity h_m is instrumental in deriving the following result with $\rho > 1$. When $\rho < 1$, the equilibrium state for all the policies (and all $\hat{\beta}$) is $(0, 0)$ leading to the following (proof in [12]):

Lemma 1. *If $\rho < 1$, or if $\rho > 1$ with $h_m > 0$, then $\pi(0)$ is an ESS-AS, for any $\pi \in \Pi$.* ■

When the disease is self-eradicating ($\rho < 1$), the system converges to $(0, 0)$, an infection free state on it's own without the aid of vaccination. Thus we have the above ESS-AS. When $\rho > 1$, if the inconvenience caused by the disease captured by $-h_m$ is not compelling enough (as $-h_m < 0$), the ES equilibrium state again results at $\hat{\beta} = 0$. In other words, policy to never vaccinate is evolutionary stable in both the cases. Observe this is a static policy irrespective of agent behaviour (i.e., for any $\pi \in \Pi$), as with $\hat{\beta} = 0$ the agents never get vaccinated irrespective of the system state.

From tables of the previous section, there exists $\bar{\beta}(\pi)$ such that the equilibrium state remains at NVDF for all $\hat{\beta} < \bar{\beta}$ (including $\hat{\beta} = 0$) and for all $\pi \in \Pi$. For such $\hat{\beta}$, we have $h(\hat{\theta}, \hat{\psi}) < 0$ if $h_m < 0$. Thus from the user utility function (2) and ESS-AS definition, the static best response set $\mathcal{B}(\pi(\hat{\beta})) = \{1\}$ for all $\pi \in \Pi$ and all $\hat{\beta} < \bar{\beta}$. Thus with $\rho > 1$ and $h_m < 0$, any policy $\pi(\hat{\beta})$ such that $\hat{\beta} < \bar{\beta}$ is not an ESS-AS. This leads to the following (proof available in [12]):

Theorem 5. [Vaccinating-ESS-AS] *When $\rho > 1$ and $h_m < 0$, there exists an ESS-AS among a $\pi \in \Pi$ if and only if the following two conditions hold:*

- (i) *there exist a $\beta^* > 0$ such that $q(\mathcal{R}^*) = 1$ and $\tilde{q}(\mathcal{R}^*) \neq 1$ under policy $\pi(\beta^*)$,*
- (ii) *the equilibrium state is $(\theta^*, \psi^*) = (\theta_E, \psi_E)$, with $\mu\rho > \mu + 1$ and the corresponding user utility component, $h(\pi(\beta^*)) = h(\theta_E, \psi_E) < 0$.* ■

Remarks: Thus when $\rho > 1$ and $h_m < 0$, there is no ESS-AS for any $\pi \in \Pi$ if $h(\theta_E, \psi_E) \geq 0$; observe that (θ_E, ψ_E) is equilibrium state with $\hat{q} = 1$ and hence from ODE (11), does not depend upon π . On the contrary, if $h(\theta_E, \psi_E) < 0$, $\pi(\beta^*)$ is ESS-AS for any $\pi \in \Pi$, with $\beta^* > \mu\rho$, $\beta^* > (\mu\rho)^2/(\mu\rho-1)$, and $\beta^* > (\mu\rho)^2/(\mu\rho-1-\mu)$ respectively for *FC*, *FR* and *VFC* policies (using Tables 1, 2 and 3).

Thus interestingly, evolutionary stable behaviour is either possible in all, or in none. However, the three types of dynamic agents require different set of parameters to arrive at ES equilibrium. *An ES equilibrium with vaccination is possible only when $\mu\rho > \mu + 1$* and interestingly, the infected and vaccinated fractions at this equilibrium are indifferent of the agent's behaviour. *In conclusion the initial dynamics could be different under the three different agent behaviours, however, the limiting proportions corresponding to any ESS-AS are the same.*

Numerical Examples: We study the variations in vaccinating ESS (θ_E, ψ_E) , along with others, with respect to different parameters. In these examples, we set the costs of vaccination and infection as $c_{v1} = 2.88, c_{v2} = 0.65, \bar{c}_{v2} = 1.91, c_{I1} = 4.32/r$. Other parameters are in the respective figures; black curves are for $d_e = 0$. In Fig. 2, we plot the ESS-AS for different values of birth-rate. Initially, $\rho > 1$, and vaccinating ESS-AS exists for all $b \leq 0.54$; here $h(\theta_E, \psi_E) < 0$ as given by Theorem 5. As seen from the plot, θ_E is decreasing and approaches zero at $b \approx 0.54$. Beyond this point there is no ESS because $\mu\rho$ reduces below $\mu + 1$. With further increase in b , non-vaccinating ESS emerges as ρ becomes less than one. Interestingly, a much larger fraction of people get vaccinated at ESS for smaller birth-rates. This probably could be because of higher infection rate per birth. In fact from the definition of θ_E , the infected fractions at ES equilibrium are concave functions of birth rate. When infection rate per birth is sufficiently high, *it appears people pro-actively vaccinate themselves, and bring infected fraction (at ES equilibrium) lower than those at smaller ratios of infection rate per birth.*

In Fig. 3 we plot ESS-AS for different values of ν . For all $\nu \leq 0.31$, the vaccinating ESS-AS exists. Beyond this, there is no ESS because $\mu\rho$ reduces below $\mu + 1$. As ν further increases, h_m becomes³ positive, leading to NVDF as ESS. One would expect a smaller infected proportion at ES equilibrium with increased availability rate, however we observe the converse; this is because the users' perception about infection cost changes with abundance of vaccines.

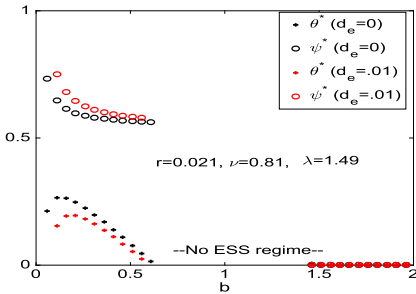


Fig. 2. ESS versus birth-rate (Color figure online)

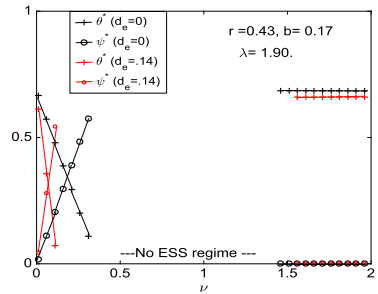


Fig. 3. ESS versus vaccine availability (Color figure online)

³ This is because the chances of infection before the next vaccination epoch decrease with increase in the availability rate ν .

With Excess Deaths ($d_{e>0}$): The analysis will follow in exactly similar lines as above. In this case, we have identified the equilibrium points of the ODE (11) but are yet to prove that they are indeed attractors. We are hoping that proof of attractors will go through similar to case with $d_e = 0$, but we have omitted it due to lack of time and space. Once we prove that the equilibrium points are attractors, the analysis of ESS would be similar to the previous case. When $\rho < 1$ or if $\rho > 1$ and $h_m^{d_e} := h(\pi(0)) = h\left(1 - \frac{1}{\rho_e}, 0\right) > 0$ (see (2), Table 4 and 5), then $\pi(0)$ is an ESS-AS, as in Lemma 1. Now, we are only left with case when $\rho > 1$ and $h_m^{d_e} < 0$ and one can proceed as in Theorem 5. In this case the only candidate for ESS-AS is $\pi(\hat{\beta})$ with $\hat{\beta}$ such that $q = 1$ and $\tilde{q} \neq 1$ and $h(\hat{\theta}, \hat{\psi}) < 0$. So, we will only compute the equilibrium points for $\hat{\beta}$ such that $q = 1$ and $\tilde{q} \neq 1$. From ODE (11), such an equilibrium point is given by ($B := \lambda b + d_e(r + d_e - \lambda - \nu)$):

$$\theta_E^{d_e} = 1 - \frac{1}{\rho_e} - \frac{\lambda \psi_E^{d_e}}{\lambda - d_e} \text{ and } \psi_E^{d_e} = \frac{-B + \sqrt{B^2 + 4\lambda d_e \nu(r + b)}}{2\lambda d_e}.$$

One can approximate this root for small d_e (by neglecting second order term $\lambda d_e \approx 0$), the corresponding ES equilibrium state $(\theta_E^{d_e}, \psi_E^{d_e})$ (again from (11)):

$$\psi_E^{d_e} \approx \frac{(r + b)\nu}{\lambda b + d_e(r + d_e - \lambda - \nu)}, \text{ that is,}$$

$$(\theta_E^{d_e}, \psi_E^{d_e}) \approx \left(1 - \frac{1}{\rho_e} - \frac{o^{d_e}}{\mu \rho_e}, \frac{o^{d_e}}{\mu \rho_e} \frac{\lambda - d_e}{\lambda}\right) \text{ with } o^{d_e} := \frac{1}{1 + \frac{d_e(r + d_e - \lambda - \nu)}{\mu \lambda \nu}}.$$

As before, there is no ESS if $\mu + o^{d_e} \geq \mu \rho_e$ (for larger d_e , when $\theta_E^{d_e} < 0$). From Figs. 2, 3 (red curves), the ES equilibrium state in deadly case has higher vaccinated fraction and lower infected fraction as compared to the corresponding non-deadly case (all parameters same, except for d_e). More interestingly the variations with respect to the other parameters remain the same as before.

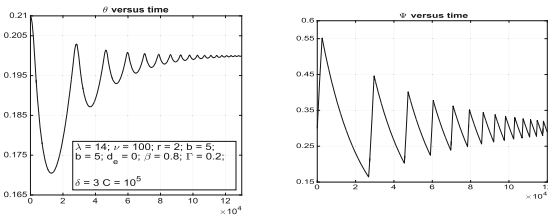


Fig. 4. VFC2 agents: Limit behaviour

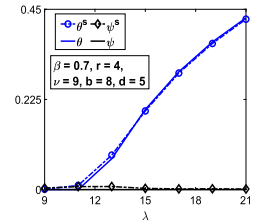


Fig. 5. FC agents, against λ

6 Numerical Experiments

We performed Monte-Carlo simulations to reinforce our ODE approximation theory. We plotted attractors of the ODE (11) represented by (θ, ψ) , and the

corresponding infected and vaccinated fractions (θ^s, ψ^s) obtained via simulations for different values of λ , ν and β . Our Monte-Carlo simulation based dynamics mimic the model described in Sect. 3. In all these examples we set $N(0) = 40000$. The remaining parameters are described in the respective figures. We have several plots in Figs. 5, 6, 7 and 8, which illustrate that the ODE attractors well approximate the system limits, for different sets of parameters.

VFC2 Agents: These agents attempt to vaccinate themselves only when the disease is above a certain threshold Γ , basically $\tilde{q} = \beta\psi\mathbb{1}_{\{\theta > \Gamma\}}$. As one may anticipate, the behaviour of such agents is drastically different from the other type of agents. Theorem 1 is applicable even for these agents (approximation in finite windows will be required here). However with a close glance at the ODE, one can identify that the ODE does not have a limit point or attractor, but rather would have a limiting set. From the RHS of the ODE (11), one can observe that the ψ derivative fluctuates between positive and negative values, and hence ψ goes through increase-decrease phases if at-all $\theta(t)$ reaches near Γ . This indeed happens, the fact is supported by a numerical example of Fig. 4. Thus interestingly with such a vaccine response behaviour, the individuals begin to vaccinate the moment the infection is above Γ , which leads to a reduced infection, and when it reaches below Γ , individuals stop vaccinating themselves. This continues forever, and one can observe such behaviour even in real world.

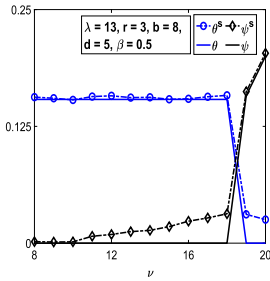


Fig. 6. FC agents vs ν

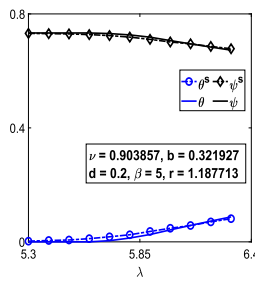


Fig. 7. FR agents vs λ

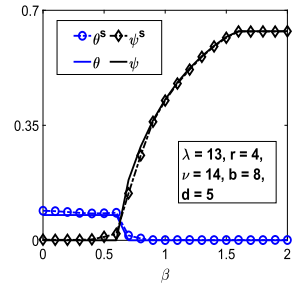


Fig. 8. FC agents vs β

7 Conclusions

With the ongoing pandemic in mind, we consider a scenario where the vaccines are being prematurely introduced. Further, due to lack of information about the side-effects and efficacy of the vaccine, individuals exhibit vaccination hesitancy. This chaos is further amplified sometimes due to reported disease statistics, unavailability of the vaccine, leading to vaccination urgency. We developed an epidemic SIS model to capture such aspects, where the system changes due to births, deaths, infections and recoveries, while influenced by the dynamic vaccination decisions of the population. As observed in reality, a variety of behavioral

patterns are considered, in particular follow-the-crowd, free-riding and vigilant agents. Using stochastic approximation techniques, we derived the time asymptotic proportions of the infected and vaccinated population for a given vaccination response. Additionally, we considered the conflict of stability for dynamic policies against static mutations, identified the strategies which are stable against static mutations and studied the corresponding equilibrium states.

Interestingly, the agents exhibit different behaviors and lead to different equilibrium states, however, at ESS-AS, all of the agents reach same limit state, where they choose vaccination either with probability 1 or 0, only based on system parameters. Also, by analysing the corresponding ODEs, we obtained many responses under which disease can be eradicated completely, but none of those are stable against mutations. Ironically, this is a resultant of the rationality exhibited by agents, which prevents them from reaching the disease-free state.

We observed certain surprising patterns at evolutionary stable equilibrium:

- (a) no one gets vaccinated with abundant vaccines; scarcity makes them rush;
- (b) the limit infected proportions are concave functions of birth rates.

Lastly, the excess deaths did not change the patterns of ES equilibrium states versus parameters, however, the limit vaccination fractions are much higher. So, in all it appears individuals rush for vaccine and we have smaller infected fractions at ES equilibrium, when there is a significant scare (of either deaths, or of scarcity of vaccines, or high infection rates etc.). Ironically, the disease can be better curbed with excess deaths.

Appendix A: Stochastic approximation related proofs

Lemma 2. *Let $\delta = 2/(N(0) - 1)$. Then for any k , $\eta_k \geq \bar{\delta} := \frac{N(0)-3}{(N(0)-1)^2}$ a.s. And thus,*

$$E \left[\frac{1}{\eta_k^2} \right] \leq \frac{1}{\bar{\delta}^2} \text{ and } E_k \left[\left| \left(\frac{1}{\eta_{k+1}} - \frac{1}{\eta_k} \right) \right| \right] \leq \epsilon_k \frac{\bar{\delta} + 1}{\bar{\delta}^2} \text{ a.s., for any } k.$$

Proof is provided in [12]. ■

Lemma 3. *The term $\alpha_k^m \rightarrow 0$ a.s., and, $\sum_k \epsilon_k |\alpha_k^m| < \infty$ a.s. for $m = \theta, \psi$.*

Proof: We will provide the proof for α_k^ψ and proof goes through in exactly similar line for α_k^θ . From equation (9), as in (10),

$$\alpha_k^\psi = E_k \left[L_{k+1}^\psi - \frac{\eta_{k+1}}{\eta_k} L_{k+1}^\psi \right], \text{ where } L_{k+1}^\psi = \frac{\mathbb{1}_{\{\eta_k > \delta\}}}{\eta_{k+1}} [G_{V,k+1} - (N_{k+1} - N_k)\psi_k].$$

By Lemma 2 and because $|G_{V,k+1} - (N_{k+1} - N_k)\psi_k| \leq 2$ a.s., we have:

$$|\alpha_k^\psi| \leq 2E_k \left[\left| \frac{1}{\eta_{k+1}} - \frac{1}{\eta_k} \right| \right] \leq 2\epsilon_k \frac{\bar{\delta} + 1}{\bar{\delta}^2} \text{ a.s.} \quad (13)$$

Thus we have:

$$\sum_{k=1}^{\infty} \epsilon_k |\alpha_k^\psi| \leq 2 \frac{\bar{\delta} + 1}{\bar{\delta}^2} \sum_{k=1}^{\infty} \epsilon_k^2 < \infty \quad a.s. \quad \blacksquare$$

Lemma 4. $\sup_k E|L_k^m|^2 < \infty$ for $m = \theta, \psi, \eta$.

Proof: The result follows by Lemma 2 and (9) (for an appropriate C):

$$|L_k^\theta|^2 \leq \frac{4}{\eta_k^2} \quad a.s., \text{ and } |L_k^\psi|^2 \leq \frac{4}{\eta_k^2} \quad a.s., \text{ and } |L_k^\eta|^2 \leq C < \infty, \quad a.s. \quad \blacksquare$$

Proof of Theorem 1: As in [10], we will show that the following sequence of piece-wise constant functions that start with Υ_k are equicontinuous in extended sense. Then the result follows from [10, Chapter 5, Theorem 2.2]. Define $(\Upsilon^k(t))_k := (\theta^k(t), \psi^k(t), \eta^k(t))_k$ where,

$$\theta^k(t) = \theta_k + \sum_{i=k}^{m(t_k+t)-1} \epsilon_k L_{k+1}^\theta, \quad \psi^k(t) = \psi_k + \sum_{i=k}^{m(t_k+t)-1} \epsilon_k L_{k+1}^\psi, \quad \eta^k(t)_k = \eta_k + \sum_{i=k}^{m(t_k+t)-1} \epsilon_k L_{k+1}^\eta,$$

where $m(t) := \max\{k : t_k \leq t\}$. This proof is exactly similar to that provided in the proof of [10, Chapter 5, Theorem 2.1] for the case with continuous g , except for the fact that $g(\cdot)$ in our case is not continuous. We will only provide differences in the proof steps towards $(\theta^k(t))_k$ sequence, and it can be proved analogously for others. Towards this we define $M_k^\theta = \sum_{i=0}^{k-1} \epsilon_i \delta M_i^\theta$ with $\delta M_k^\theta := L_{k+1}^\theta - g^\theta(\Upsilon_k) - \alpha_k^\theta$ as in [10] and show the required uniform continuity properties in view of Lemmas 3–4. Observe that $\eta_k \leq 1 + N(0)/k$, $\theta_k \leq 1$ for any k . Now the uniform continuity of integral terms like the following is achieved because our $g(\cdot)$ are bounded:

$$\left| \int_s^t g^\theta(\Upsilon^k(z)) dz \right| \leq \int_s^t |g^\theta(\Upsilon^k(z))| dz \leq \int_s^t \frac{\lambda + r + b + 2d_e}{\delta(d+b)} dz \leq \bar{m}(t-s) \leq \bar{m}\delta_1.$$

Such arguments lead to the required equicontinuity (details are in [12]). \blacksquare

Appendix B: ODE Attractors Related Proofs

Proof of Theorem 2: Let $\Upsilon := (\theta, \psi, \eta)$. Let $\hat{\Upsilon}$ represent the corresponding attractors from Table 1. Here $q(\theta, \psi) = \min\{\tilde{q}(\theta, \psi), 1\}$ with $\tilde{q}(\theta, \psi) = \beta\psi$.

We first consider the case where $\tilde{q}(\hat{\theta}, \hat{\psi}) < 1$. Further, note that one can re-write ODEs, $\dot{\Upsilon} = g(\Upsilon)$, as below:

$$\dot{\theta} = \frac{\mathbb{1}_{\{\eta > \delta\}} A \theta}{\eta \varrho}, \quad \dot{\psi} = \frac{\mathbb{1}_{\{\eta > \delta\}} B \psi}{\eta \varrho}, \quad \text{and } \dot{\eta} = \mathbb{1}_{\{\eta > \delta\}} C, \quad \text{where}$$

$A = A(\Upsilon) := (1 - \theta - \psi)\lambda - r - b$, $B = B(\Upsilon) := (1 - \theta - \psi)\beta\nu - b$ and $C = C(\Upsilon) := (b-d)/\varrho - \eta$. To this end, we define the following Lyapunov function based on the regimes of parameters:

$$V(\Upsilon) := \begin{cases} \left(\hat{A}(\theta) \right)^2 + \psi \left(\hat{B}(\psi) \right)^2 + C(\eta)(\hat{\eta} - \eta), & \text{if } \beta < \rho\mu, \rho > 1, \\ \theta \left(\hat{A}(\theta) \right)^2 + \left(\hat{B}(\psi) \right)^2 + C(\eta)(\hat{\eta} - \eta), & \text{if } \beta > \rho\mu, \text{ and } \rho > 1 \\ \theta \left(\hat{A}(\theta) \right)^2 + \psi \left(\hat{B}(\psi) \right)^2 + C(\eta)(\hat{\eta} - \eta), & \text{if } \rho < 1, \end{cases}$$

where $\hat{A}(\theta) := A(\theta, \hat{\psi}, \hat{\eta})$, $\hat{B}(\psi) := B(\hat{\theta}, \psi, \hat{\eta})$. We complete this proof using the above functions and the details are in [12]. ■

Lemma 5. *Let $\hat{\theta}, \hat{\psi} > 0$. If $\tilde{q}(\hat{\theta}, \hat{\psi}) \neq 1$, there exists a Lyapunov function such that $(\hat{\theta}, \hat{\psi}, \hat{\eta})$ is locally asymptotically stable attractor for ODE (11) in the sense of Lyapunov.*

Proof: We use similar notations as in previous proof. Let us first consider the case where $\tilde{q}(\hat{\theta}, \hat{\psi}) < 1$, i.e., $q(\hat{\theta}, \hat{\psi}) = \tilde{q}(\hat{\theta}, \hat{\psi})$. Then, one can choose a neighborhood (further smaller, if required) such that $\hat{q} - \delta < q(\theta, \psi) < \hat{q} + \delta$, and $q(\theta, \psi) = \tilde{q}(\theta, \psi)$ for some $\delta > 0$. Define the following Lyapunov function (for some $w_1, w_2 > 0$, which would be chosen appropriately later):

$$V(\Upsilon) := w_1(\hat{\theta} - \theta)\hat{A}(\theta) + w_2(\hat{\psi} - \psi)\hat{B}(\psi) + C(\hat{\eta} - \eta), \text{ where} \quad (14)$$

$\hat{A}(\theta) := 1 - \theta - \hat{\psi} - \frac{1}{\rho}$, and $\hat{B}(\psi) := \hat{q}(1 - \hat{\theta} - \psi) - \mu\psi$ (recall $\hat{q} := q(\hat{\theta}, \hat{\psi})$). Call $\tilde{\theta} := \hat{\theta} - \theta$ and $\tilde{\psi} := \hat{\psi} - \psi$. The derivative of $V(\Upsilon(t))$ with respect to time is:

$$\begin{aligned} \dot{V} &= \langle \nabla V, g(\Upsilon) \rangle \\ &= - \left(\hat{A}(\theta) + \tilde{\theta} \right) \frac{A\theta\lambda w_1}{\eta\varrho} - \left(\hat{B}(\theta) + \tilde{\psi}(\hat{q} + \mu) \right) \frac{B\nu w_2}{\eta\varrho} - (C(\eta) + \hat{\eta} - \eta)C. \end{aligned} \quad (15)$$

One can prove that the last component, i.e., $-(C + \hat{\eta} - \eta)C$ is strictly negative in an appropriate neighborhood of $\hat{\Upsilon}$ as in proof of Theorem 2. Now, we proceed to prove that other terms in \dot{V} (see (15)) are also strictly negative in a neighborhood of $\hat{\Upsilon}$.

Consider the term⁴ $\left(\hat{A}(\theta) + \tilde{\theta} \right) A$, call it A_1 :

$$A_1 = 2\tilde{\theta}A = 2 \left(\tilde{\theta}^2 + \tilde{\theta}\tilde{\psi} \right) = 2 \left(\left(\tilde{\theta}c_1 + \frac{1}{2c_1}\tilde{\psi} \right)^2 + (1 - c_1^2)\tilde{\theta}^2 - \frac{1}{4c_1^2}\tilde{\psi}^2 \right), \quad (16)$$

where c_1 will be chosen appropriately in later part of proof. Similarly the term corresponding to B is (details in [12]),

⁴ Observe that $\hat{A}(\theta)A = (\hat{A}(\hat{\theta}) + \tilde{\theta})A$, and $\hat{A}(\hat{\theta}) = 0$.

$$B_1 = 2(\hat{q} + \mu) \left[\tilde{\psi}^2 \left(\mu + p_1(\mathcal{Y}) + \hat{q}(1 - c_2^2) \right) - \frac{1}{4c_2^2\hat{q}} (\hat{q} - p_2(\mathcal{Y}))^2 \tilde{\theta}^2 \right] \\ + 2(\hat{q} + \mu)\hat{q} \left(c_2\tilde{\psi} + \frac{1}{2c_2} \left(1 - \frac{p_2(\mathcal{Y})}{\hat{q}} \right) \tilde{\theta} \right)^2. \quad (17)$$

Thus, we get: $\dot{V} < -A_1 \frac{\theta\lambda w_1}{\eta\varrho} - B_1 \frac{\nu w_2}{\eta\varrho}$. Now, for \dot{V} to be negative, we need (using terms, in (16) and (17), corresponding to $\tilde{\theta}^2, \tilde{\psi}^2$):

$$\nu(\hat{q} + \mu) \leq \frac{4c_2^2\hat{q}}{w_2(\hat{q} - p_2(\mathcal{Y}))^2} \lambda w_1(1 - c_1^2)\theta, \text{ and} \\ \frac{1}{2c_1^2} \theta \lambda w_1 \leq 2\nu w_2(\hat{q} + \mu) \left(\mu + \hat{q}(1 - c_2^2) + p_1(\mathcal{Y}) \right).$$

By appropriately choosing the constants (for various agents), we complete the proof (details in [12]). ■

Appendix C: ESS Related Proofs

Lemma 6. *Let $\rho > 1$. Assume $\tilde{q}(\mathcal{Y}) \neq 1$ where $q(\mathcal{Y}) = \min\{\tilde{q}(\mathcal{Y}), 1\}$. Consider a policy $\pi(\hat{\beta})$ where $\pi \in \Pi$ and $\hat{\mathcal{Y}}$ is the attractor of the corresponding ODE (11). Let $\hat{\mathcal{Y}}_\epsilon$ be attractor corresponding to ϵ -mutant of this policy, $\pi_\epsilon(\hat{\beta}, p)$ for some $p \in [0, 1]$. Then, i) there exists an $\bar{\epsilon}(p) > 0$ such that the attractor is unique and is a continuous function of ϵ for all $\epsilon \leq \bar{\epsilon}$ with $\hat{\mathcal{Y}}_0 = \hat{\mathcal{Y}}$. ii) Further $\bar{\epsilon}$ could be chosen such that the sign of $h(\hat{\mathcal{Y}}_\epsilon)$ remains the same as that of $h(\hat{\mathcal{Y}})$ for all $\epsilon \leq \bar{\epsilon}$, when the latter is not zero. ■*

Proof: We begin with an interior attractor. Such an attractor is a zero of a function like the following (e.g., for VFC1 it equals, see (11)):

$$\phi\lambda - r - b, \quad \min\{1, \psi\theta\hat{\beta}\} \phi\nu - b\psi, \quad \text{and} \quad \frac{b-d}{\varrho} - \eta. \quad (18)$$

Under mutation policy, $\pi_\epsilon(\hat{\beta}, p)$, the function modifies to the following:

$$\phi\lambda - r - b, \quad \left((1 - \epsilon) \min\{1, \hat{\beta}\psi\theta\} + \epsilon p \right) \phi\nu - b\psi, \quad \text{and} \quad \frac{b-d}{\varrho} - \eta. \quad (19)$$

By directly computing the zero of this function, it is clear that we again have unique zero and these are continuous⁵ in ϵ (in some $\bar{\epsilon}$ -neighbourhood) and that they coincide with $\hat{\mathcal{Y}}$ at $\epsilon = 0$. Further using Lyapunov function as defined in the corresponding proofs (with obvious modifications) one can show that these zeros are also attractors in the neighborhood. The remaining part of the proof is completed in [12]. The last result follows by continuity of h function (2). ■

⁵ When $\tilde{q}(\mathcal{Y}) > 1$, the zeros are $(\epsilon p + (1 - \epsilon))/(\mu\rho)$, otherwise they are the zeros of a quadratic equation with varying parameters, we have real zeros in this regime.

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