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Efficacy versus abundancy: Comparing vaccination schemes
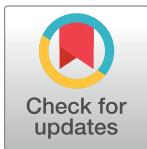
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Abstract

We introduce a novel compartmental model accounting for the effects of vaccine efficacy, deployment rates and timing of initiation of deployment. We simulate different scenarios and initial conditions, and we find that higher abundancy and rate of deployment of low efficacy vaccines lowers the cumulative number of deaths in comparison to slower deployment of high efficacy vaccines. We also forecast that, at the same daily deployment rate, the earlier introduction of vaccination schemes with lower efficacy would also lower the number of deaths with respect to a delayed introduction of high efficacy vaccines, which can however, still achieve lower numbers of infections and better herd immunity.

1 Introduction

Humanity has been struggling with viral pandemics and infectious diseases that caused great catastrophes throughout the entire history of mankind. Since the Athenian, Antonine and Justinian Plagues to the Black Death, Spanish Flu, Cholera, and Smallpox up until the HIV pandemic, SARS, Swine flu, Ebola outbreak and, most recently, the Coronavirus infection, pandemics have been a major source of disease, death, economic crises and political turmoils [1–3]. Nevertheless, those outbreaks also lead to major discoveries and advancements in sciences and public health, especially in medicine, pharmaceuticals, vaccines and development of public policies [4–6].

The Coronavirus disease (COVID-19) associated to the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) continues as a main cause of hospitalization and death and as a main public health risk since the first case was registered in Wuhan, China in December 2019 [7]. It was declared as a global pandemic on March 11, 2020 [8] after spreading from China into several Asian and European countries and consequently into tens of countries across the world. By December 22, 2021, the world has suffered about 278 million registered infections and 5.4 million deaths associated to COVID-19 in 224 different countries, territories and entities [9]. Health systems were put under enormous pressure, and the consequent mitigation measures and associated closures and lockdowns taken across the world for lengthy periods of time contributed to a global economic slowdown and recession in several countries [10, 11], in

addition to a heavy negative impact on education, employment, tourism and social activities [12–14] exacerbating existing political discontent and stirring political unrest [15].

Similarly to the other respiratory pathogens, airborne transmission of SARS-CoV-2 occurs by inhaling droplets loaded with the virus emitted by infectious people. Infection can also occur through mucous membranes like the eyes, nose or mouth [16].

Many variants of the virus emerged with an increased risk to global public health. The World Health Organization (WHO) characterized variants of interest (VOI) and variants of concern (VOC) to track and monitor [17]. The main variants of concern have stronger capabilities of transmissibility and health risks, and they are labeled by the WHO as the Alpha, Beta, Gamma, Delta and Omicron variants which were first documented in the United Kingdom, South Africa, Brazil and India, respectively [18]. The recent emergence of variants creates a major cause of concern since they can lead to an epidemic rebound especially with the possibility of vaccine resisting and the emergence of deadlier and/or more transmissible future variants. Increased viral transmission means higher probability of emergence of variants as well [19].

Several types of vaccines were developed and approved by global (WHO) or by national health agencies, using different techniques like the viral vector vaccines (Oxford/AstraZeneca, Sputnik V/Gamaleya, Janssen), genetic vaccines (Pfizer/BioNTech, Moderna), inactivated vaccines (Sinovac, Sinopharm, Bharat) and protein vaccines (Novavax, Sanofi) [20]. By December 22, 2021, a total of about 8.85 billion doses of those vaccines were administered with main concentration in China, USA, India and Europe [21]. Other vaccines, inhaled aerosol dry vaccines and antiviral drugs are under extensive research and development [22–24]. Antibody therapies are under development as well and they offer an effective treatment for the most seriously ill patients, but they remain expensive and in short supply [25].

A global roll-out of vaccines is needed to guarantee a swift elimination of the infection, and for this end vaccines need to be available, affordable, and accessible at a global scale. In high-income countries, large stocks were produced and purchased, combined with active logistic and public health resources, but in low-income countries, vaccination is still slow mainly due to insufficiency of vaccines. The WHO has called for more equity and stronger support for its COVAX initiative in order to supply wider vaccine access for poor countries, but statistics show that the gap remains deep as many rich countries have already bought multiple times of doses needed per person [26]. A similar gap also arises on the national level as health illiteracy, religious beliefs and sometimes political partisanship are slowing down vaccinations and creating irregular immunity rates among different regions or groups [27]. As such, the global efforts to stop the spread remains far from successful, and the priority should be focused to optimize the use, deployment and timing of available vaccines, whether those recognized by the WHO or by national health agencies across the globe in order to prevent further deaths as well as emergence of new variants.

The spread patterns of infectious diseases have been and are being actively studied and simulated using several mathematical and computational models in the interdisciplinary fields of health economics, physical biology and medical physics, applied mathematics and epidemiology. A wide variety of techniques were employed ranging from compartmental models, Agent-Based models (ABM), spatio-temporal analysis to data-driven analysis and artificial intelligence [28, 29]. The first compartmental model was the famous Kermack–McKendrick Susceptible-Infectious-Removed (SIR) model which divides the total population under study into three compartments where agents would move from one compartment to another upon infection or removal through recovery or death [30]. Numerous variations of this model were introduced to account for characteristics and dynamics of different diseases as well as in simulating similar interactions and spreads in the social and behavioral sciences [31–33]. The

research on COVID-19 spread extensively employed SIR models that were improved to account for other effects like exposure, travel, quarantine, vaccination, public measures and many other alterations to best describe the underlying features of the contagion [34–38], as well as general and country specific ABMs, spatio-temporal and data driven studies [39–47].

However, with the mass introduction of various globally and nationally recognized vaccines with varying efficacies, and with the varying degrees of availability and logistic capabilities across the globe, an important question arises for decision makers: which vaccine to choose to deploy in a certain country from the available options in that country, given their efficacies, their availability timeline and expected deployment rate of each. This is a major concern for public health officials aiming at minimizing the number of deaths and/or the number of active infections, that was not thoroughly discussed and analyzed in the literature. This paper studies the trade-off between vaccine efficacy and abundance and then between efficacy and time of availability, and the corresponding expected outcomes for deaths and infections.

The paper is organized as follows: section 2 presents the theoretical model, section 3 puts forward the results and discussions about the efficacy, deployment and timing rates while section 4 concludes the paper.

2 Theoretical model

We introduce a novel compartmental model to account for the effect of vaccines, taking into account different vaccination deployment rates (v), efficacies (e) under several scenarios of infection spread rates represented by the reproductive number (R_t), starting with different initial conditions in relation to different numbers of infections, deaths and immune populations in different countries. The $SS_V II_V RD$ model consists of 6 compartments: the susceptible unvaccinated population S, the susceptible vaccinated population S_V who might become infected during the assumed effective immunity period depending on the efficacy of the vaccine, the infectious unvaccinated population I, the infectious vaccinated population I_V, the recovered population R who are either fully protected by the vaccine during its effective protection time range or who have already recovered from the infection and still possess immunity, and finally the dead D. Each compartment is normalized with respect to the total population so that $N = S + S_V + I + I_V + R + D = 1$.

The 6 compartments are interlinked (Fig 1), and their dynamics can be modeled by a system of coupled linear ordinary differential equations described below.

$$\begin{cases} \dfrac{dS}{dt} & = -\beta(I + I_V)S - evS + \dfrac{evS}{T_{VI}} + \dfrac{\gamma_R I + \gamma_{RV} I_V}{T_{II}} \\[2mm] \dfrac{dS_V}{dt} & = (1 - e)vS - \beta'(I + I_V)S_V \\[2mm] \dfrac{dI}{dt} & = \beta(I + I_V)S - (\gamma_D + \gamma_R)I \\[2mm] \dfrac{dI_V}{dt} & = \beta'(I + I_V)S_V - (\gamma_{DV} + \gamma_{RV})I_V \\[2mm] \dfrac{dR}{dt} & = evS + \gamma_R I + \gamma_{RV} I_V - \dfrac{evS}{T_{VI}} - \dfrac{\gamma_R I + \gamma_{RV} I_V}{T_{II}} \\[2mm] \dfrac{dD}{dt} & = \gamma_D I + \gamma_{DV} I_V \end{cases} \qquad (1)$$

The parameter β is the incidence rate between susceptible (S) and all infectious people ($I + I_V$) and it can be determined using the reproductive number of the infectious spread R_t and
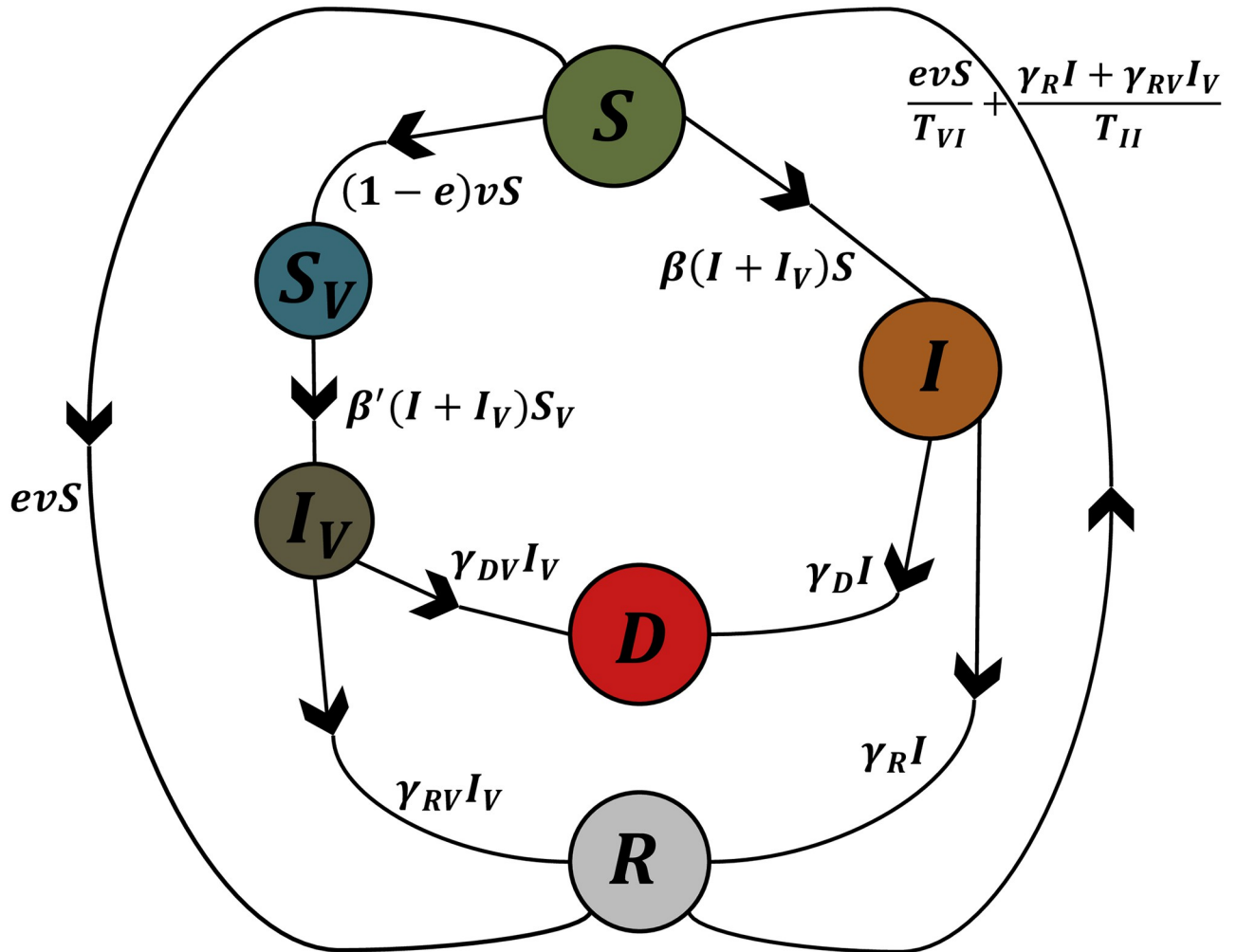
Fig 1. A schematic diagram of the $SS_V II_V RD$ model showing the six compartments of the model (susceptible, vaccinated susceptible, infectious, vaccinated infectious, recovered and dead) together with their transfer dynamics and the coefficients relating transfers among different compartments.

the average rate of recovery of unvaccinated people γ_R. In this sense, $\beta = \gamma_R R_t$. The rate of recovery of vaccinated people is given by γ_{RV} and the incidence rate between vaccinated susceptible people and infectious people is given by $\beta' = \gamma_{RV} R_t$. The rate of death of unvaccinated people is γ_D and that of vaccinated ones is γ_{DV}. Finally, T_{VI} represents the average time duration of acquired vaccine immunity and T_{II} represents the average duration of acquired post infection immunity. The change in the total population is $\frac{dN}{dt} = 0$, assuming that N remains constant during that period, neglecting other changes due to natural growth, immigration, etc. . .

The transition dynamics between different compartments can be summarized according to the following:

- The susceptible S may become infected I upon contact with the infectious (vaccinated or non-vaccinated) at rate β. They may also become recovered R upon receiving a vaccine with efficacy e given at a daily rate v. In addition, recovered people R would become susceptible again some time after recovery. Mainly, recovered people due to infection would become

susceptible again in an infection immunity period T_{II} and recovered people due to vaccination would become susceptible again in a vaccine immunity period T_{VI}.

- The vaccinated susceptible population S_V are the susceptibles S who got vaccinated but are still susceptible to infection, and may become infectious I_V upon a meeting rate β' with other infectious people.

- The infectious compartment I is populated by unvaccinated susceptibles S encountering other infectious agents at a rate β, while exit from this compartment is attributed to deaths at rate γ_D into compartment D and recoveries at rate γ_R into compartment R.

- The vaccinated infectious compartment I_V includes vaccinated susceptibles S_V catching the disease at a rate β' and diminished by people dying at rate γ_{DV} into D and people recovering a rate γ_{RV} into R.

- The recovered population R is formed by effectively vaccinated susceptible people coming from S and people surviving the infection at rates γ_R and γ_{RV} from infectious and vaccinated infectious populations I and I_V. Simultaneously, recovered people would eventually lose their acquired immunity on average periods of T_{II} after infection and T_{VI} after vaccination, thus would exit the recovered compartment R back to the susceptibles S.

- Finally, the dead D increase at death rates γ_D and γ_{DV} among infectious and vaccinated infectious populations I and I_V. In this model, we only consider the deaths caused by COVID-19, disregarding net population changes (natural deaths as well as natural births). Consequently, the dead compartment gets populated from the infected and vaccinated infected populations.

The vaccination deployment rate v depends on the available supply of the vaccine as well as the logistical capability and the popular demand at a given time [48]. In this paper it is assumed to be equivalent to the daily vaccination percentage of the susceptible population, with different scenarios representing slow, moderate and fast deployment rates. The vaccine efficacy varies among different employed vaccines as well as in relation to new emerging variants in addition to the possibility of supplying a single dose of a double dosed vaccine under short supply. The model accounts for a range of scenarios with low, moderate and high efficacy corresponding to the former cases. We also inspect those scenarios under different reproductive rates R_t which depend on different values of mitigation measures related to social distancing, protective masks, sanitation, and other factors that alter the rate of infection spread. We assume different scenarios of low, moderate, high and alternating (where R_t varies in a periodic pattern between high and low extrema) reproductive rates.

The numerical values of these parameters are taken in relation to available data and research. The efficacy values vary between $0.5 \leq e \leq 0.95$ depending on the available vaccines [49–55]. The rate of recovery for vaccinated people $\gamma_R = \frac{1}{14}$ in relation of an average of 14 days needed for recovery [56], while the death rate amounts to around 2% [9] of the infected which leads to $\gamma_D = \frac{\gamma_R}{50}$. We also assume that those who are infected after vaccination would need a similar time of recovery, despite the fact that their symptoms would be much reduced [57, 58], thus $\gamma_{RV} = \gamma_R$ but their death rate, as studies reveal, would be considerably lower by 70–85% [58, 59], which is modeled through $\gamma_{DV} = \frac{\gamma_D}{5}$. We take $T_{VI} = 90$ days and $T_{II} = 360$ days to represent expected periods of acquired immunity after recovery and after vaccination respectively [60]. The deployment rate v is assigned hypothetical values varying between 0.1% of the susceptible population per day for slow vaccination rollout and 1.5% for the highest pace of vaccination rollout.

The reproduction rate R_t is assigned values of 0.7, 1.1 and 1.5 corresponding to low, medium and high reproduction rates, in addition to a fourth scenario where R_t alternates sinusoidally between 0.4 and 2 according to the relation $R_t = 0.8\cos\left(\frac{t}{15}\right) + 1.2$ where t represents the time in days, with a period of around 94 days to simulate the effect of the consecutive waves of the spread of the infection, as observed empirically [61, 62]. The periodicity of the function $\cos(at + b)$ is mathematically defined to be $\frac{2\pi}{a}$. The parameter $\frac{1}{15}$ in our model simulates a periodicity of 30π which approximately corresponds to a 94 day period. Its dimension is time given in the unit of days. Thus, this forecast simulates the recurrence of waves of high then low infection rates on an average period of 94 days. The growth rate of the active cases of COVID-19 infections is studied in the United Kingdom, South Africa and Brazil [63, 64]. These articles present the progression of the registered values of R_t since the start of the spread until recently. In both countries, R_t assumed values that varied between 0.5 up to 3 depending on the mitigation measures and closures. The registered data constitutes a motivation to simulate the possible scenarios of infection spreads in presence of vaccines for representative values of R_t corresponding to low, middle and high rates of spread.

We simulate this model under different combinations of efficacy e, deployment rate v and reproductive number R_t to analyze the corresponding cumulative numbers of infected, recovered and dead populations.

3 Results and discussions

3.1 Efficacy vs abundance

We simulate the theoretical model introduced in (1) to determine relative numbers of active cases, infected vaccinated cases, immune population and total cumulative deaths under nine different combinations of efficacies (e) of available vaccines and their deployment rates (v) given by: $e = 92\%$, $v = 0.5\%$, $e = 92\%$, $v = 0.3\%$, $e = 92\%$, $v = 0.1\%$, $e = 72\%$, $v = 0.7\%$, $e = 72\%$, $v = 0.5\%$, $e = 72\%$, $v = 0.3\%$, $e = 55\%$, $v = 1.5\%$, $e = 55\%$, $v = 1\%$ and $e = 55\%$, $v = 0.7\%$ in order to compare the levels of infection, death and immunity between higher efficacy vaccines at lower abundance and lower efficacy vaccines with more abundance or deployment rate, together with middle values between them.

Case study. We simulate in this paper the actual data of infections, deaths and vaccination in two countries with high vaccination rate (United Kingdom) and low vaccination rate (South Africa), according to data publically available in [9]. The initial conditions corresponding to the United Kingdom are: 73.1% vaccinated population 2.0% currently infected and 0.23% dead, while those of South Africa are:29.2% vaccinated population 0.02% currently infected and 0.16% dead. In this sense, the results of this forecast are not country specific but are of global significance. They can be applied to any other country with similar conditions. We repeat this simulation for different levels of infection spread modeled through low, medium, high and alternating reproductive rates R_t defined before. The results are displayed in Figs 2–5, respectively.

Fig 2 shows that when the reproductive rate is low ($R_t = 0.7$), the number of active cases would fall down quickly under all vaccination schemes, for various efficacies and deployment rate, both in countries that are in the middle or early stages of the spread (upper and lower figures respectively). The number of infected vaccinated people is the highest for the low efficacy vaccine. Herd immunity would be achieved optimally under fast deployment of the high efficacy vaccine and the middle efficacy vaccine, while the lowest immunity is achieved under the slowest deployment of the high efficacy vaccine. This shows that the deployment rate is an essential factor in combination with efficacy for achieving immunity. The figure also shows that the number of deaths is most reduced under the two fastest deployment schemes of the
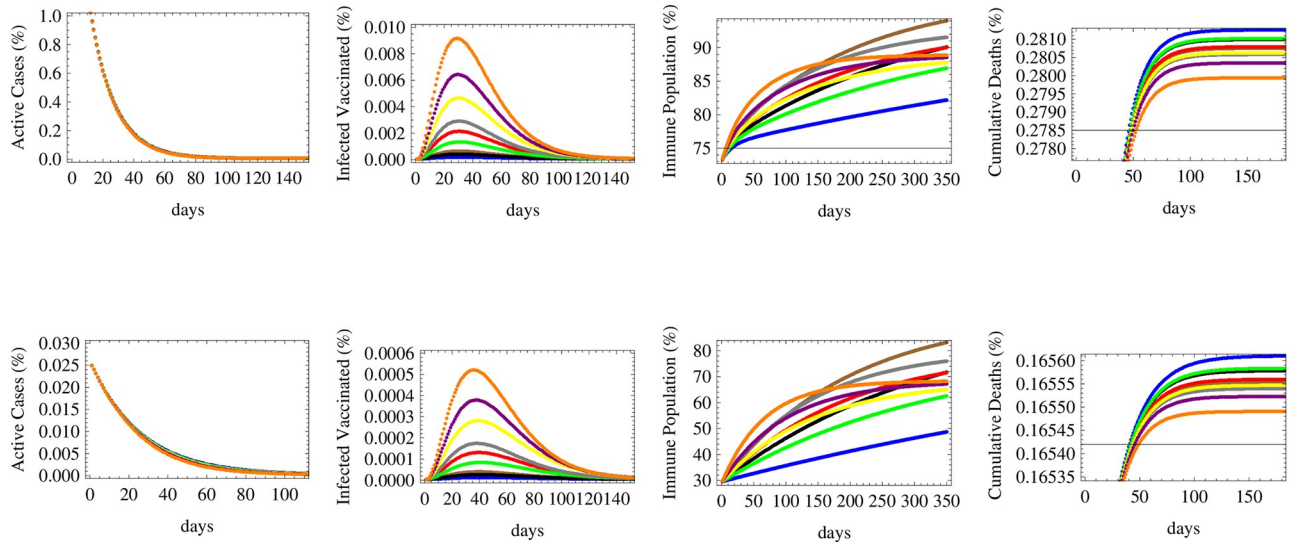
Fig 2. The relative numbers of active cases, infected vaccinated cases, immune population and total cumulative deaths are shown for a low reproductive rate $R = 0.7$ under initial conditions of: 73.1% vaccinated population 2.0% currently infected and 0.23% dead (upper row, United Kingdom) and 29.2% vaccinated population 0.02% currently infected and 0.16% dead (lower row, South Africa) for nine different vaccination scenarios. The forecast corresponds to vaccine efficacy (e) and daily deployment rate (v) of: $e = 92\%$, $v = 0.5\%$ (in brown), $e = 92\%$, $v = 0.3\%$ (in black), $e = 92\%$, $v = 0.1\%$ (in blue), $e = 72\%$, $v = 0.7\%$ (in gray), $e = 72\%$, $v = 0.5\%$ (in red), $e = 72\%$, $v = 0.3\%$ (in green), $e = 55\%$, $v = 1.5\%$ (in orange), $e = 55\%$, $v = 1\%$ (in purple) and $e = 55\%$, $v = 0.7\%$ (in yellow).

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low efficacy vaccine, followed by the fastest deployment rate of that of middle efficacy. The latter result shows that to reduce deaths, the most essential measure is to ensure the the availability and the fast deployment of the vaccine, while reaching herd immunity and decreasing infections (thus lowering the pressure on the health sector and decreasing the possibility of appearance of new mutations) depends relatively more on its efficacy.
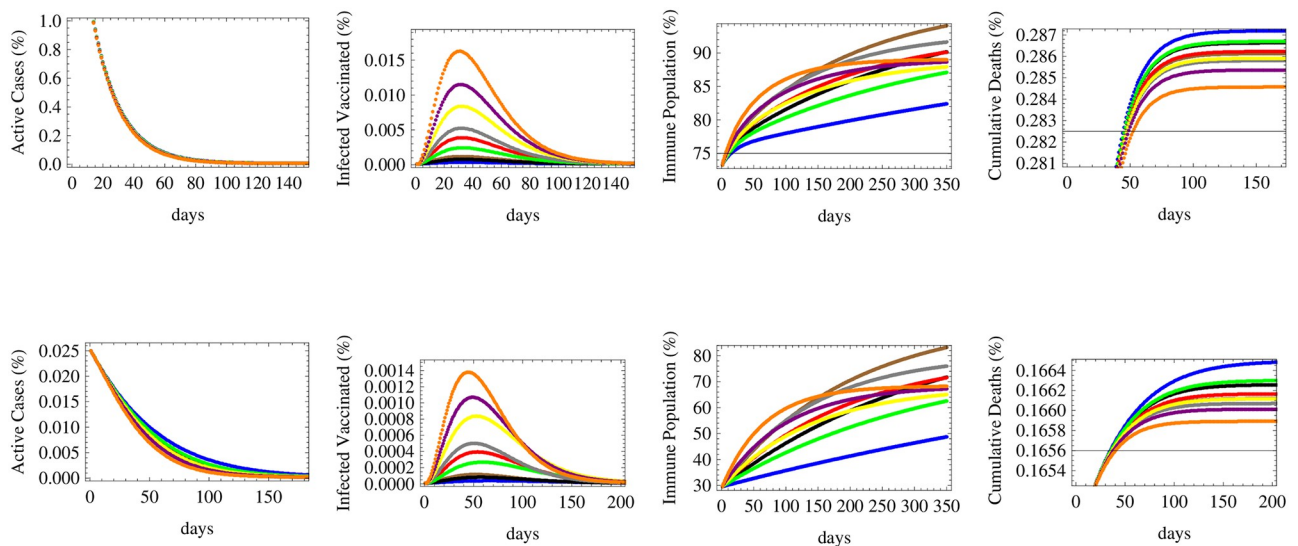

Fig 3. The figure shows the relative numbers of active cases, infected vaccinated cases, immune population and total cumulative deaths for a medium reproductive rate $R = 1.1$ with the same two initial conditions and nine vaccination scenarios of Fig 2.

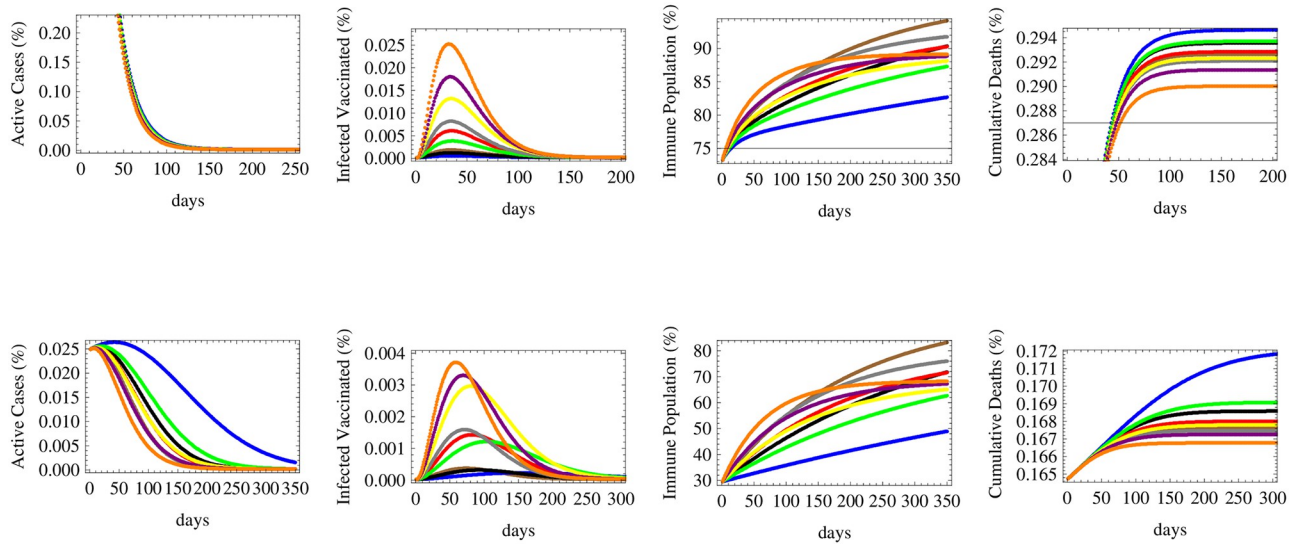https://doi.org/10.1371/journal.pone.0267840.g003

Fig 4. The figure shows the relative numbers of active cases, infected vaccinated cases, immune population and total cumulative deaths for a high reproductive rate _R_ = 1.5 with the same two initial conditions and nine vaccination scenarios of Fig 2.

In the case of a medium reproductive rate (R_t = 1.1) corresponding to a slow increase in the number of infections in absence of vaccine, Fig 3 shows that the number of active cases would decrease in countries that are in their middle stages of infection, while they would keep on increasing for a limited interval of time in countries that are in their early stages of spread. The duration needed for the active cases to start decreasing is shortest for the vaccine with fastest deployment rate (despite its low efficacy), while that with the slowest deployment rate (with high efficacy) needs more time to decrease the number of active cases. The highest number of
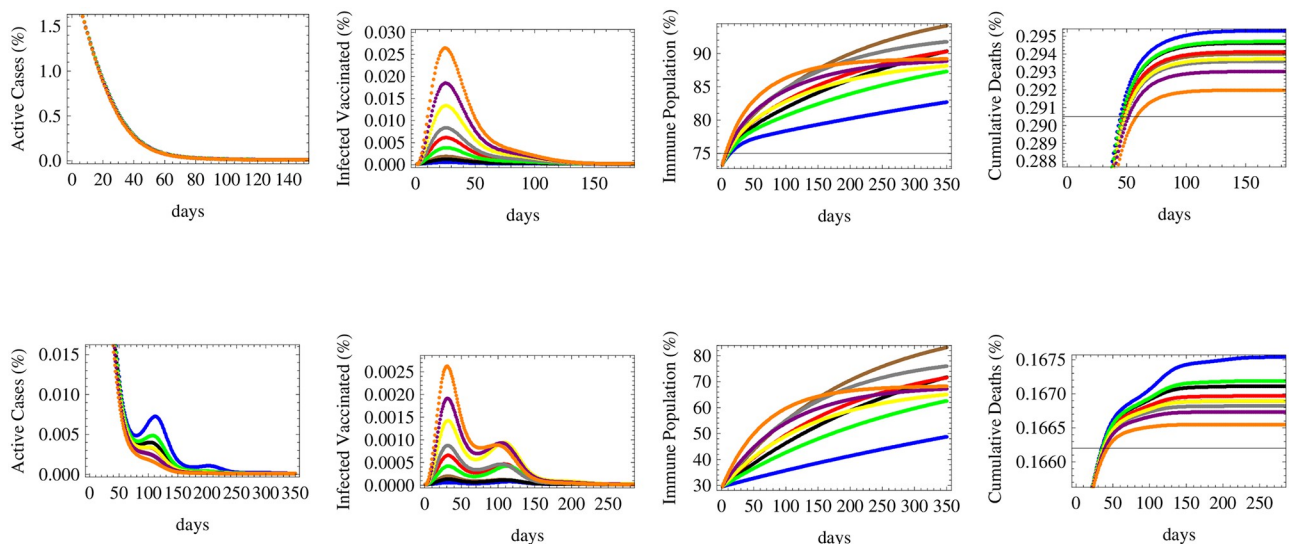

Fig 5. The figure shows the relative numbers of active cases, infected vaccinated cases, immune population and total cumulative deaths for an alternating reproductive rate, varying sinusoidally between _R_ = 0.4 and _R_ = 2, with the same two initial conditions and nine vaccination scenarios of Fig 2.

infected vaccinated people would be obtained through the rapidly deployed low efficacy for countries in their middle stages of spread, while for early stage countries, each scenario of deployment of the low efficacy vaccine would lead to maximal infections under different times. In a similar manner to what happens for low reproductive rate, immunity will be maximally obtained under the deployment of high efficacy vaccines at high rates, while the least deaths are achieved under the fastest deployment rates of vaccines.

For a country under a fast spread scenario simulated by $R_t = 1.5$, Fig 4 shows that the number of active cases would fall in a middle spread stage country, though it takes more time than that with lower R_t. However, in early spread countries, the number of active cases will rise and peak after a period of time before falling down after a relatively long time (around 100–200 days) of vaccination. The slowest vaccination schemes of the low and middle efficacy vaccines would reach the highest peaks and need more time for controlling the spread while the fastest vaccination schemes would achieve it the quickest. The number of infected vaccinated people would be the largest in the case of the low efficacy vaccine deployed rapidly in a middle spread stage country, while for a country at early spread stages, the two slowest deployments of the low efficacy vaccine lead to the highest infections among the vaccinated population. Immunity will be maximally obtained under the deployment of high efficacy vaccines at high rates, while the least deaths are achieved under the fastest deployment rates of vaccines, like what was shown before for low and medium spread rates, but with a much higher magnitude of deaths in all scenarios due to a higher reproductive rate.

In the more realistic case of an alternating reproductive number varying between $0.4 \leq R_t \leq 2$ and corresponding to consecutive patterns of low and high spread waves, we realize that in a country at a middle spread stage, all vaccination strategies would lead to bringing down the number of active cases with a small peak arising after the return of the next wave, while in a country at early stages of spread, the slow deployment of vaccines, whether with high or low efficacies, would cause the re-emergence of several peaks of infections and high number of active cases. Only the fast deployment of vaccines of various efficacies would lead to curb the number of infections just after the first peak of spread. The number of infected vaccinated people would be the highest for the case of fast deployment of low efficacy vaccines in countries with middle stages of spread while stronger peaks of infections among the vaccinated people would occur under slower vaccination scenarios of low efficacy vaccine during the second wave in countries with early spread stages. The highest herd immunity levels would be attained under the scenarios of fastest deployments of high and medium efficacy vaccines in both categories of countries under early or middle stages of spread after 3–4 months, despite the early lead and the sharp rise in the immune population during the first few weeks of fast deployment of low efficacy vaccines. The cumulative number of deaths would rise in jumps corresponding to successive waves of spread especially in early stage countries, but in both cases, the fastest vaccination schemes of low efficacy vaccines would ultimately lead to the minimal number of deaths while the slowest pattern of high efficacy vaccine deployment would result in the highest cumulative rate of deaths. Those simulations are displayed in Fig 5.

3.2 Efficacy vs time of availability

An important aspect of vaccination strategies that we consider in this paper is the question of vaccine efficacy versus time of availability or start of deployment. In this simulation, we forecast the relative number of active cases, infected vaccinated people, the immune population and the cumulative number of deaths under low, medium and high reproductive rates $R = 0.7$, 1.1 and 1.5 respectively in Figs 6–8. Four cases simulating the effect of efficacy and the time of initiation of the vaccination process were considered: a vaccine of low efficacy $e = 52\%$ to start
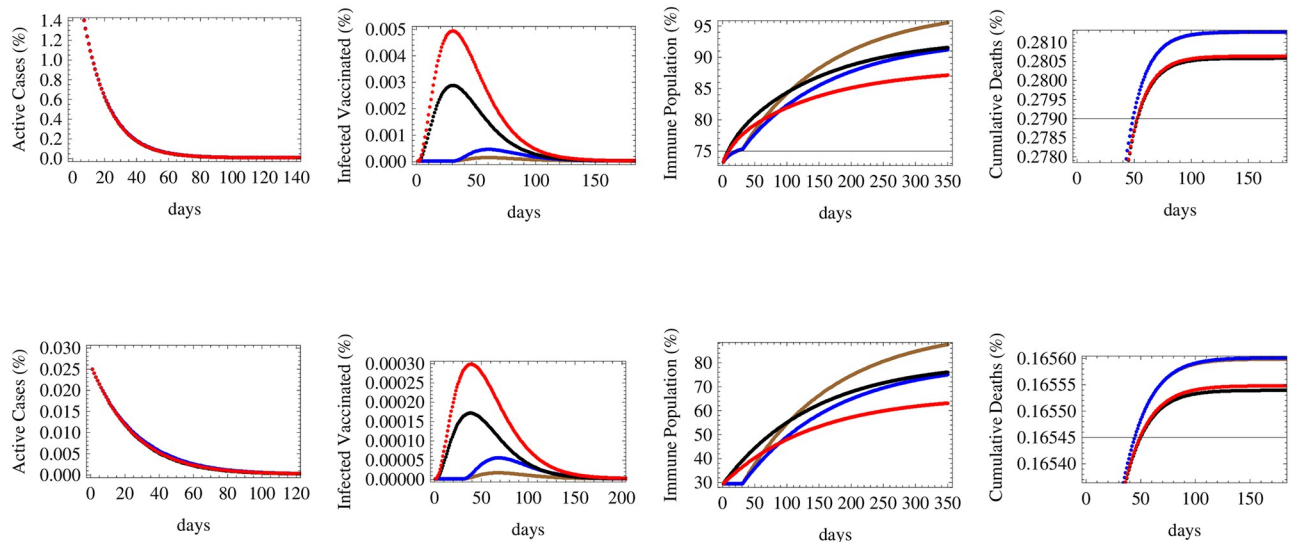
Fig 6. The figure shows the expected relative numbers of active cases, infected vaccinated cases, immune population and cumulative deaths at a low reproductive rate $R = 0.7$ in relation to four cases simulating the effect of efficacy and the time of initiation of the vaccination process: A vaccine of efficacy $e = 52\%$ to start deployment immediately (red), a vaccine of efficacy $e = 72\%$ to start deployment immediately (black), a vaccine of efficacy $e = 72\%$ to start deployment after 30 days (blue) and a vaccine of efficacy $e = 92\%$ to start deployment after 30 days (brown), all being deployed at an equal rate of 0.7% of the susceptible population per day. The upper row corresponds to initial conditions of 40.6% vaccinated population 1.8% currently infected and 0.18% dead while the lower row corresponds to 3.1% vaccinated population 0.46% currently infected and 0.11% dead.

deployment immediately (red), a vaccine of medium efficacy $e = 72\%$ to start deployment immediately (black), a medium efficacy vaccine with $e = 72\%$ to start deployment after 30 days (blue) and a vaccine of high efficacy $e = 92\%$ to start deployment after 30 days (brown), all being deployed at an equal rate of the susceptible population per day. As in previous simulations, the upper row corresponds to countries at middle stages of spread with initial conditions of 40.6% vaccinated population 1.8% currently infected and 0.18% dead while the lower row corresponds to countries at early stages with 3.1% vaccinated population 0.46% currently infected and 0.11% dead.

Under the circumstances of low reproductive rate $R_t = 0.7$ depicted in Fig 6, it is clear that the number of active cases will fall significantly under the four efficacy-timing schemes in both early and middle spread stage categories. The number of infected vaccinated people in both categories would occur under the scenario of immediate deployment of low efficacy vaccines. Herd immunity would be maximally attained through the adoption of high efficacy vaccines being deployed with a one month period of delay while it would be the lowest using low efficacy vaccines deployed immediately. On the level of cumulative deaths, the least number of deaths is realized under the scenario of immediate deployment of medium then low efficacy vaccines, while the delay would raise the number of deaths even while using high efficacy vaccines.

Fig 7 simulates same efficacy-timing scenarios under medium reproductive rate $R_t = 1.1$. We notice that in countries in their middle stage of infection spread, the number of active cases would fall quickly under all scenarios, while in early stage countries, the late deployment scenarios would cause a rise and a peak in infections before falling down significantly, whereas the quick deployment of low or medium efficacy vaccines would lower the active cases faster during the first few months. We also notice that in about 100 days, the number of active cases
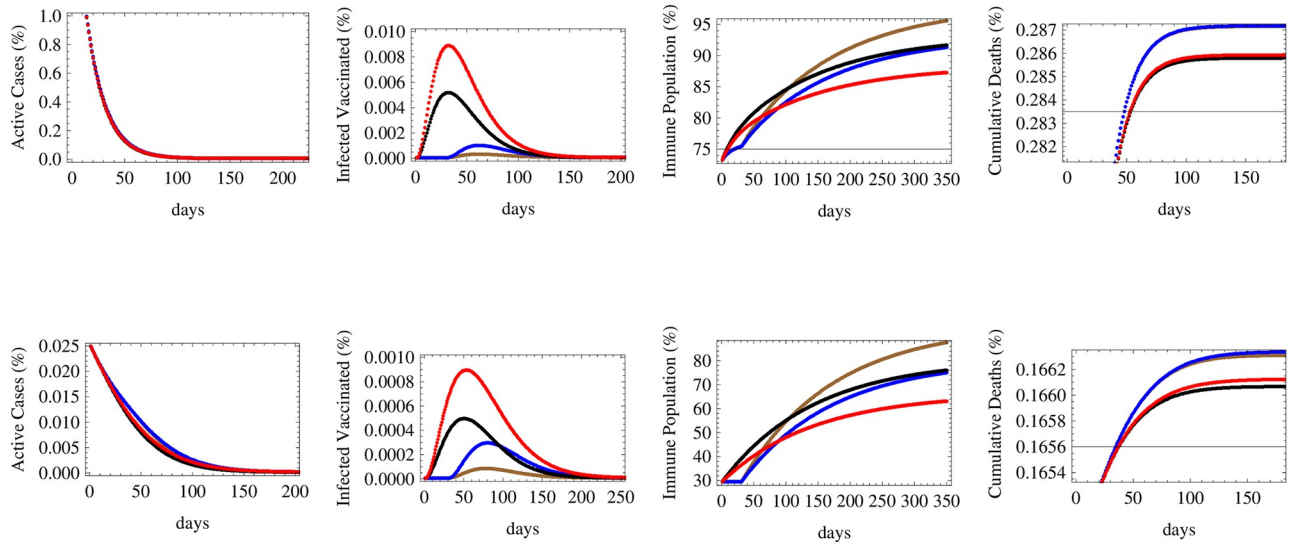
Fig 7. The figure shows the expected relative numbers of active cases, infected vaccinated cases, immune population and cumulative deaths corresponding to a medium reproductive rate *R* = 1.1 with same initial conditions, efficacies and time of initiation as those detailed in Fig 6.

due to a delayed high efficacy vaccine will catch up and fall below the expected active cases under an immediately deployed low efficacy vaccine. The number of infected vaccinated people is the highest for the lowest efficacy vaccine and the lowest for the highest efficacy vaccine in both country categories. Similarly, for both categories, herd immunity is maximally attained using the delayed high efficacy vaccine rather than the immediate low efficacy one which provides the lowest percentile of immune population. However, regarding deaths, the lowest numbers of deaths are attributed to the immediate deployment of medium then low efficacy
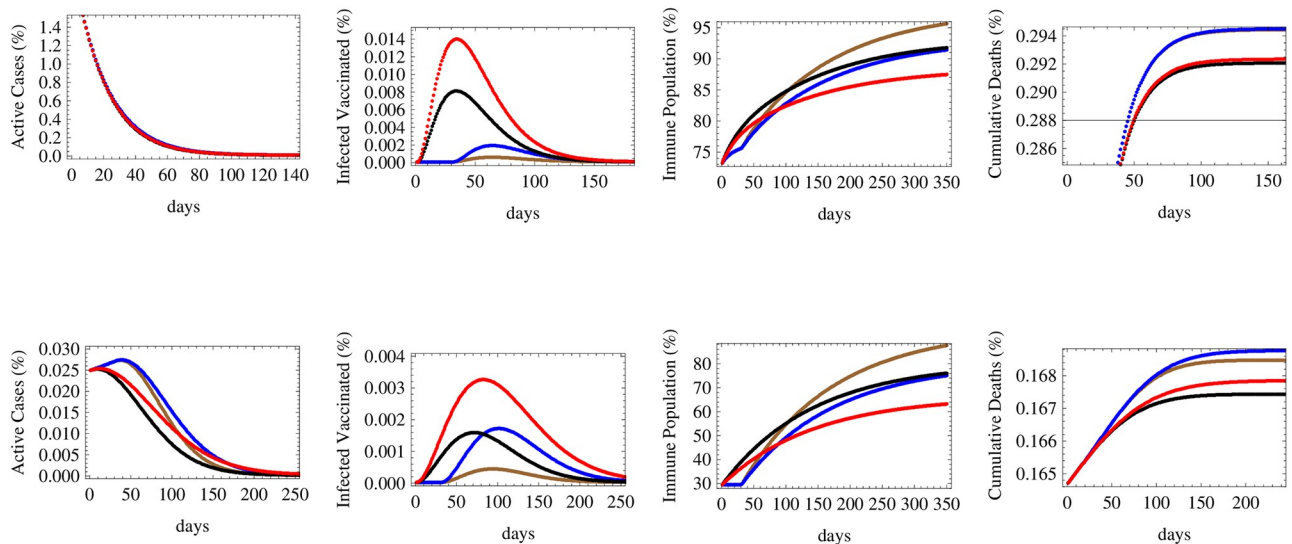

Fig 8. The figure shows the expected relative numbers of active cases, infected vaccinated cases, immune population and cumulative deaths corresponding to a high reproductive rate *R* = 1.5 with same initial conditions, efficacies and time of initiation as those detailed in Fig 6.

vaccines, while a delayed deployment will cause more deaths even while using high and medium efficacy vaccines.

Under the scenario of high reproductive number simulated by $R_t = 1.5$, the number of active cases would fall slowly in countries that are in their middle stages of vaccination and spread, while it would rise and peak under all vaccination scenarios in early stage countries. The highest peaks are attributed to delayed vaccinations even though infections would fall rapidly once vaccination starts, and eventually the corresponding number of active cases would fall below that of low efficacy vaccine being deployed immediately. Immediate deployment also helps to flatten the curve on infections, thus reducing the expected peak number of cases. The maximum number of infected vaccinated people would correspond low efficacy vaccine deployed immediately while the lowest corresponds to the high efficacy vaccine deployed late, for both country categories. Similarly, herd immunity is maximally achieved by the high efficacy vaccine despite being introduced late while the lowest level of immunity is caused by the low efficacy vaccine despite early introduction. The immediate deployment of medium efficacy vaccine minimizes the number of deaths, followed by the immediate low efficacy vaccine, while late deployment raises the number of deaths for both country categories, but with higher relative differences among the death outcomes for middle stage countries.

3.3 Limitations

This study takes into account all parameters related to vaccine efficacy and deployment rates under several infectious rates and initial conditions. However, there is a limiting factor related to the maximal proportion of the population who are willing or at least who would eventually take the vaccine. Here, we took this into account indirectly by using a daily deployment rate v which is proportional to the susceptibles S, and not to the total population N. In this sense, in the initial phases of vaccination, the number of people taking the vaccine would be the highest, but as time progresses, the number of susceptibles decreases, hence the daily number of vaccinated people decreases. It is natural to assume that, as when a country reaches a high level of vaccination, less people will be willing to get the vaccine. If vaccination rates were only connected to abundance or logistic infrastructure, they would have been linked to the total population N. It is also important to mention that the reproduction number R_t can differ for different population groups. Its value is dominated by the group in which most transmission occurs. In this sense, it is not a homogeneous parameter that applies on a nationwide or social scale but depends on demographic, socio-economic and other factors.

Due to various reasons ranging from religious and political beliefs, into non-scientific and anti-vaxxer fears, there might be a sizable sector of the society who would refuse to get vaccinated [27]. Vaccine hesitancy is not directly simulated in the model, but it is indirectly represented through relating the daily deployment rate to the number of susceptibles hence it decreases as the number of vaccinated people increases. In our scenario, immunity of this portion of the population would still be achieved through infection rather than vaccination.

4 Conclusion

In this paper we introduced a general novel compartmental model accounting for the vaccinated population, infected vaccinated population, active infections, and deaths with various vaccine efficacies and vaccination deployment rates.

We simulated different scenarios and initial conditions, and we showed that abundance and higher rate of deployment of low efficacy vaccines would lower the cumulative number of deaths in comparison to slower deployment of high efficacy vaccines. However, the high

efficacy vaccines can better lower the number of active cases and achieve faster and higher herd immunity.

We also discovered that at the same daily deployment rate, the earlier introduction of vaccines with lower efficacy would also lower the number of deaths with respect to a delayed introduction of high efficacy vaccines, which can, however, lower the number of infections and attain higher levels of herd immunity.

Author Contributions

Conceptualization: Omar El Deeb, Maya Jalloul.

Formal analysis: Omar El Deeb, Maya Jalloul.

Methodology: Omar El Deeb, Maya Jalloul.

Writing – original draft: Omar El Deeb.

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