The importance of sustained compliance with physical distancing during COVID-19 vaccination rollout

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The importance of sustained compliance with physical distancing during COVID-19 vaccination rollout

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Abstract

Mass vaccination campaigns against SARS-CoV-2 are under way in many countries with the hope that increasing vaccination coverage will enable reducing current physical distancing measures. Compliance with these measures is waning, while more transmissible virus variants such as B.1.1.7 have emerged. Using SARS-CoV-2 transmission model we investigated the impact of the feedback between compliance, the incidence of infection, and vaccination coverage on the success of a vaccination programme in the population where waning of compliance depends on vaccine coverage. Our results suggest that the combination of fast waning compliance, slow vaccination rates, and more transmissible variants may result in a higher cumulative number of infections than in a situation without vaccination. These adverse effects can be alleviated if vaccinated individuals do not revert to pre-pandemic contact rates, and if non-vaccinated individuals remain compliant with physical distancing measures. Convincing, clear and appropriately targeted communication strategies by public health authorities are required.

Introduction

More than one year after the outbreak of COVID-19 was declared a pandemic by the World Health Organisation [1], hospitalisations and death tolls continue to mount in many countries around the world. While physical distancing measures have been effective in significantly reducing SARS-CoV-2 transmission during the first wave [2, 3, 4, 5, 6], the virus continued to spread. Amid the second wave that started in fall 2020 [7, 8, 9], more transmissible [10, 11, 12] and potentially more deadly SARS-CoV-2 variants emerged (e.g., B.1.1.7) [13, 14], causing many countries to reinforce physical distancing measures in order to maintain healthcare capacities and to prevent deaths caused by COVID-19.

The approval of COVID-19 vaccines developed by BioNTech/Pfizer, Moderna and AstraZeneca, and more recently a vaccine by Johnson & Johnson [15], is fuelling hopes for the end of lockdown periods and relaxation of physical distancing measures. Phase 3 randomised clinical trials reported vaccine efficacies for preventing laboratory-confirmed symptomatic SARS-CoV-2 infection of 92% for BioNTech/Pfizer [16], 94.1% for Moderna [17], and at least 62% for AstraZeneca [18]. The data from the ongoing vaccination campaign in Israel supports the results of the randomised trials for BioNTech/Pfizer vaccine reporting that the effectiveness of this vaccine against symptomatic disease is 94% [19]. On 29 March 2021 CDC released a report that the Pfizer/BioNTech and Moderna vaccines have 80% effectiveness in preventing COVID-19 following 14 days or more after the first dose, but before the second dose, and 90% effectiveness following 14 or more days since the second dose [20]. These findings are consistent with earlier reports that the three vaccines (Pfizer/BioNTech, Moderna and AstraZeneca) have some effectiveness in blocking SARS-CoV-2 transmission [21, 22, 23].

The understanding of how the deployment of these vaccines will impact transmission is complicated by the emergence of new virus variants of concern such as B.1.1.7, B.1.351 and P.1 [24, 25]. Thus, to reduce the death toll and the burden on healthcare system, as well as to slow down the appearance rate of antigenically relevant mutations that may escape protection conferred by existing vaccines, a swift and rigorous vaccination campaign seems of utmost importance.

Vaccination rollout, however, faces multiple challenges. Public health services may be confronted with structural and logistical obstacles (e.g., sufficient supply size, capacity to administer shots [26, 27, 28]) illustrated by diverging vaccination rates among different countries [29]. Another factor that may affect vaccination rollout is vaccine acceptance [27] that varies greatly across countries from 23.6% in Kuwait to 97% in Ecuador [30, 31]. Mass vaccination may also have undesirable consequences such as reducing compliance with physical distancing measures. For example, vaccinated people may increase their contact rate as they perceive COVID-19 to pose a lower risk for them. Based on the notion that vaccination will end the COVID-19 pandemic, both vaccinated and non-vaccinated people may be less compliant with physical distancing measures due to factors such as lack of motivation or lack of knowledge about the necessity to maintain compliance post-vaccination. Hence, compliance may decline with increasing vaccination coverage. A number of modeling studies have shown that the feedback between the epidemic

dynamics and human behaviour has an important role in the disease transmission [32, 33, 34]. In an earlier modeling study [34] we showed that relaxation of compliance with physical distancing measures beyond a threshold may cause a significant increase in new infections and hospitalisations. This concern is especially relevant at the start of the vaccination campaign, when vaccination coverage is still low.

We developed a socio-epidemiological model (Figure 1) for SARS-CoV-2 transmission to investigate the effects of waning of compliance to physical distancing measures on the dynamics of COVID-19 as vaccine is rolled out in the population. The transmission dynamics is modelled through a susceptible-exposed-infectious-recovered (SEIR) framework. The vaccine works as all-or-nothing conferring perfect protection to a fraction of susceptible individuals who receive it. The vaccine delivered to individuals in other disease stages has no effect.

Compliance to physical distancing is captured by a reduction in the daily number of contacts relative to the pre-pandemic level of contacts. The population is divided into individuals who can be more compliant (henceforth referred to as "compliant") and less compliant ("non-compliant") to measures. The reduction in contacts is larger for compliant and smaller for non-compliant populations. Vaccinated individuals increase their contact rate above that of non-compliant individuals, thereby returning to nearly pre-pandemic level of contacts. Non-vaccinated individuals can move between compliant and non-compliant modes, and the rates of moving depend on the state of the epidemic and on vaccination coverage. Specifically, more individuals become compliant to physical distancing measures as the incidence of COVID-19 cases increases and lose compliance faster as the proportion of vaccinated individuals grows (see Methods).

We considered a baseline scenario without vaccination and several vaccination scenarios. As the speed of vaccination rollout differs between countries [29], we distinguished a slow and a fast vaccination rate, comparable to the rates in the Netherlands and in the UK between 7 January and 7 February 2021 [29]. We further considered scenarios for two types of SARS-CoV-2 variants. The first variant has the transmission potential of the original variant circulating in Europe prior to fall 2020. The second variant is a more transmissible, B.1.1.7-like variant, that is currently gaining dominance in European countries [35]. We investigated the impact of compliance on the numbers of infected, vaccinated and compliant individuals over the course of the vaccination rollout. We also compared the cumulative numbers of infected individuals after one and two years into the vaccination programme to the numbers without vaccination. Next, we considered the potential effects of two interventions aimed at improving compliance. The first intervention is targeted at people who have not been vaccinated yet and aims at keeping their compliance with physical distancing at the level of prior to vaccination rollout. The second intervention is targeted at people who have been vaccinated and aims at keeping their contact rates low. Finally, we considered a combined intervention where both interventions are implemented simultaneously.

Results

Compliance and vaccination rollout

The model was calibrated to the state of the epidemic and the level of compliance prior to the start of vaccination in the Netherlands. The size of the population that recovered from SARS-CoV-2 infection was set based on seroprevalence data from the serological study in an age-stratified and regionally weighted representative sample of the Dutch population [36, 37]. The estimated seroprevalence was 4% in June/July of 2020 [36] and increased to 5% in September/October 2020 [37]. To account for the effects of the second wave until the start of vaccination, we fixed the recovered population at 8%. The proportion of compliant population was set at 65% using the study on behavioral measures and well-being conducted in the Netherlands by the National Institute for Public Health and the Environment (RIVM) [38]. The vaccination rates were based on data from the first four weeks of vaccination rollout in the Netherlands and the UK [29]. Henceforth, these rates are referred to as "slow" and "fast", respectively. Figure 2a shows vaccination coverage during the first two years after the start of vaccination rollout for slow and fast vaccination. The inset shows the fit of the model to the data. We fixed the contact rate for compliant and non-compliant individuals such that the effective reproduction number for the original variant prior to vaccination is 1.1, as estimated by the RIVM in November 2020 [39]. The effective reproduction number for the B.1.1.7-like variant was 1.65, i.e. 50% higher than for the original variant [11]. The contact rate of vaccinated individuals was assumed to be close to the pre-pandemic rate and 1.5 times higher than the contact rate of non-compliant individuals [40].

In our model, individuals become compliant if there are infectious individuals in the population. The rate of switching to the compliant mode is proportional to the per capita rate of moving to compliant mode and the incidence of infectious cases (see Methods, Table 2). The per capita rate of moving to compliant mode was fixed in the main analyses. The sensitivity analyses for this parameter are shown in the Supplementary materials. Furthermore, even in the absence of vaccination compliance can wane but this occurs more rapidly as the vaccination coverage increases. The proportion of compliant population for a constant incidence of infection (5,387 cases per day, approximated using the number of cases detected in the Netherlands in the period used for the model calibration [41]) is shown in Figure 2b where we used slow and fast vaccination rates from Figure 2**a**. For slow vaccination, 56% of the population is compliant one year after the start of vaccination and 40% is compliant after two years. For fast vaccination, the compliant population decreases more rapidly, with only 7% and 1% of individuals being compliant after one and two years, respectively.

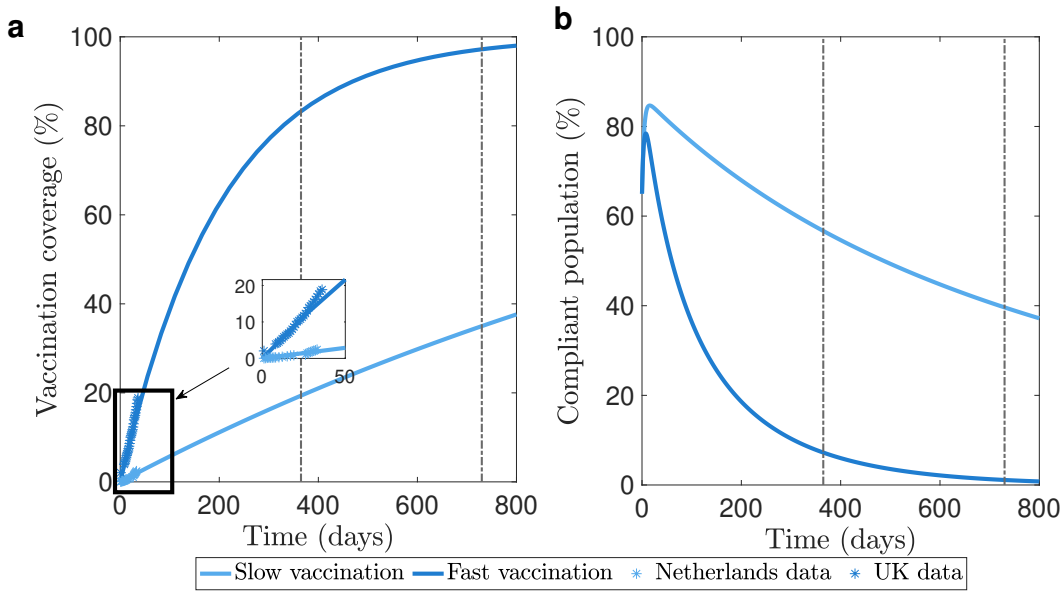

Figure 2: **Vaccination coverage and proportion of compliant population during the vaccination rollout.** **a** Increase in vaccination coverage for slow (light blue line) and fast (dark blue line) vaccination rates. Stars show data for the Netherlands (light blue) and the UK (dark blue) [29], respectively. **b** Decrease in the proportion of compliant population for slow and fast vaccination and a fixed incidence of infection (5,387 cases per day) observed in the Netherlands in the period used for the model calibration. Vertical brown lines mark one and two years since the start of vaccination.

The reason for the decline of compliance observed in Figure 2b is two-fold. First, as the vaccination coverage increases, the compliance in the non-vaccinated population decreases. Moreover, the speed of this decrease depends on how fast vaccination is rolled out. Second, since we assume that vaccinated people perceive themselves protected from COVID-19 they are no longer compelled to comply to physical distancing. These two processes translate into varying proportions of the compliant population depending on both the incidence of infection and vaccination coverage.

Epidemic dynamics with vaccination

The model predicts that depending on the speed of the vaccination rollout and transmissibility of the virus variant, as a result of decreasing compliance, the prevalence of infected individuals in the presence of vaccination can be higher than the prevalence in a situation without vaccination (Figure 3 and Table 1). This effect is much more pronounced for the B.1.1.7-like variant than for the original variant (Figures 3**a** and 3**b**). We quantify it as the excess of cumulative infections relative to the no-vaccination scenario level one and two years after the start of the vaccination rollout.

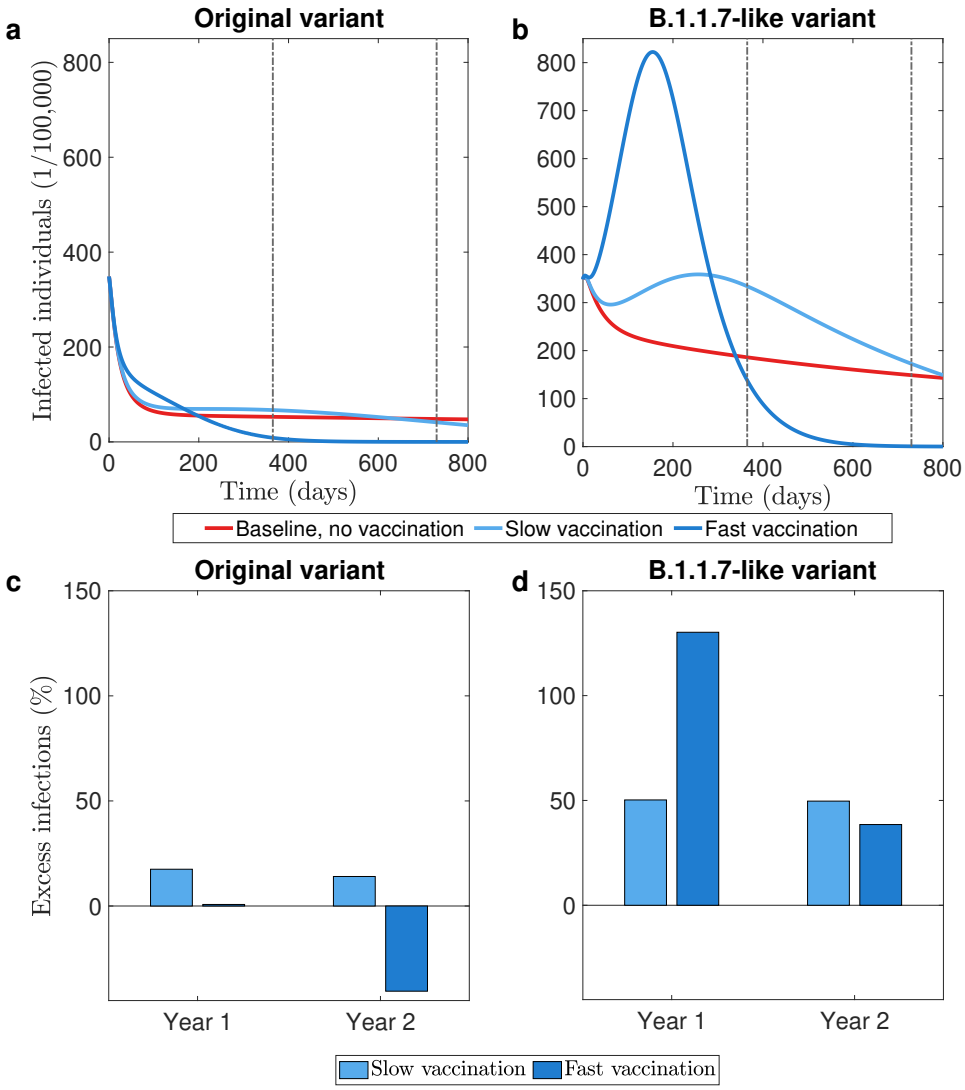

Figure 3: **Epidemic dynamics with and without vaccination**. **a** Prevalence of infected individuals versus time when the original variant circulates. **b** The same output when a B.1.1.7-like variant circulates. **c** Excess of cumulative infections relative to the no-vaccination scenario level for the original variant. **d** The same output when a B.1.1.7-like variant circulates. **c** and **d** show excess of infections relative to the no-vaccination levels when respective variants circulate. In **a** and **b**, vertical brown lines mark one and two years since the start of vaccination.

If the original variant is circulating (Figures 3**a** and 3**c**), vaccination can eliminate the virus after approximately one year (Figure 3**a**), provided the vaccination rollout is fast. Consequently, there are fewer cumulative infections two years into the rollout as compared to the no-vaccination scenario. Slow vaccination, in the presence of compliance waning associated with vaccine rollout, leads to an excess of cumulative infections at these time points (Figure 3**c**). The period of higher prevalence is shorter for fast vaccination (198 days) than for slow vaccination (633 days) (Figure 3**a**).

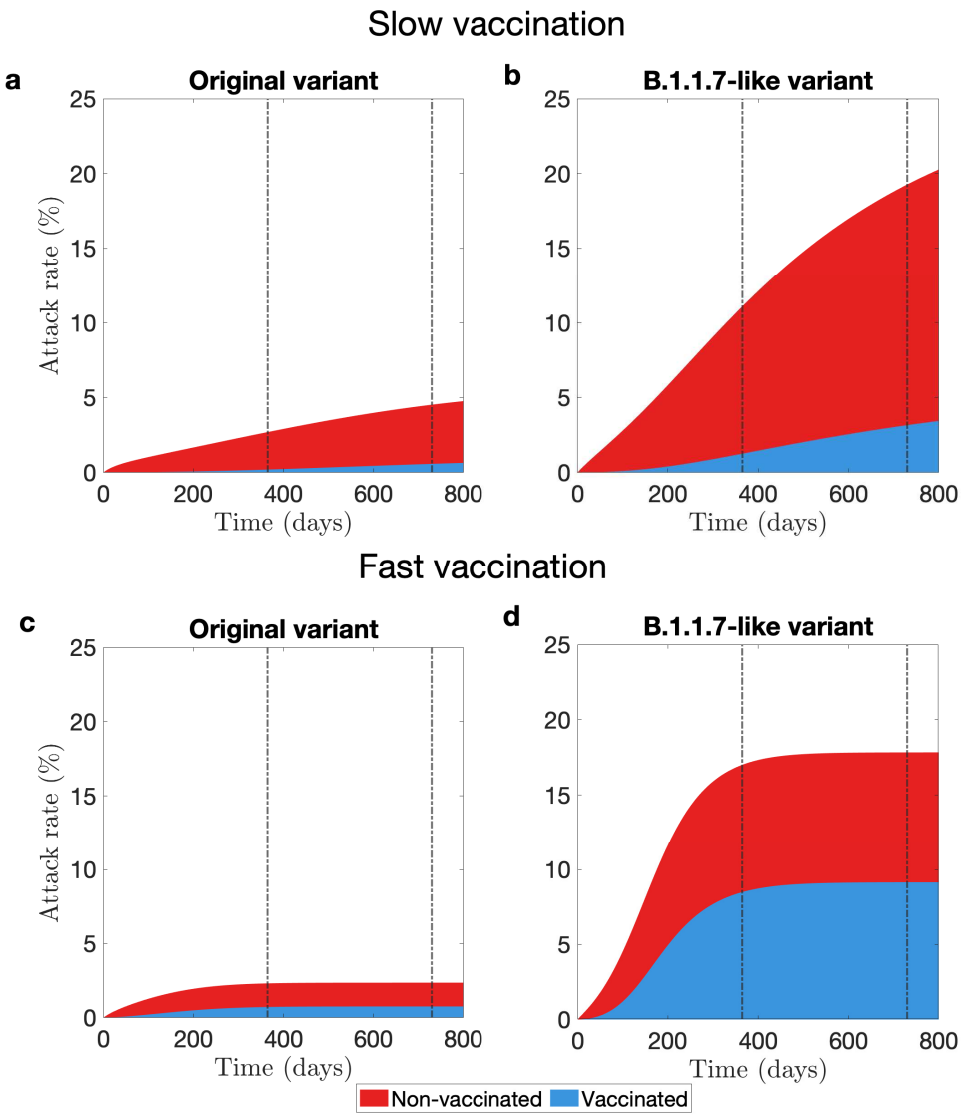

Figure 4: **Contribution of vaccinated and non-vaccinated individuals to attack rate during the vaccination rollout.** **a** and **b** show attack rates versus time given the slow vaccine uptake rate. **c** and **d** show attack rates versus time given the fast vaccine uptake rate. **a** and **c** show these quantities for the original variant, **b** and **d** for a B.1.1.7-like variant. Vertical brown lines mark one and two years time points since the start of the vaccination campaign. Attack rate is the proportion of the population that has been infected until a given time. We adjusted the attack rate so that it describes only new infections that appeared during the time interval that we considered.

If a more transmissible B.1.1.7-like variant is circulating (Figures 3**b** and 3**d**), decreased compliance can lead to an additional peak in prevalence (Figure 3**b**). In this case, vaccination leads to an excess of cumulative infections as compared to a situation without vaccination (Figure 3**d**). This occurs because waning of compliance coincides with an increased transmissibility of the virus. The period when the prevalence is higher as compared to the no-vaccination scenario lasts even longer than for the original variant, i.e., 340 days for fast vaccination and more than 800 days for slow vaccination (Figure 3**b**). As shown in Figure 3**d**, the cumulative number of infections at the first year mark is higher for a fast vaccination rate than for a slow vaccination rate (130% versus 50%) but the reverse relationship is observed at the second year mark (39% versus 50%).

To understand the role of vaccinated individuals in the transmission dynamics observed in Figure 3, we calculated the proportion of infections occurring in the vaccinated population over time (Figure 4). The analyses show that in the case of slow vaccine uptake (Figures 4**a** and 4**b**) vaccinated individuals comprise a small proportion of the infected population even at the end of the second year of the vaccination campaign (less than 17%, Figures 4**a** and 4**b**). Therefore, the increased prevalence among non-vaccinated can be attributed to the decrease of their compliance. In the case of fast vaccine uptake, the model predicts that a significant proportion of infections is among vaccinated individuals (at least 32%, Figures 4**c** and 4**d**). Thus, the observed rise in the prevalence is in part due to the increased contact rate of susceptible vaccinated individuals. These findings suggest that for slow vaccination the risk of severe disease and death in the population is hardly lowered, while for fast vaccination a significant proportion of the infected individuals will be protected against severe disease.

We refer to the analyses described in this section as to the epidemic dynamics without interventions. In the following, we compare the impact of interventions targeted at maintaining compliance with physical distancing to the epidemic dynamics without interventions and the scenario without vaccination.

Interventions targeting compliance

To investigate how interventions may improve the impact of vaccination rollout, we considered an intervention that targets compliance of those who are not yet vaccinated (Intervention 1) and an intervention targeted at the vaccinated population (Intervention 2). We assume that the first intervention targets non-vaccinated individuals and is successful in keeping the duration of compliance at the pre-vaccination length (30 days) as vaccination coverage grows. The second intervention, targeted at vaccinated individuals, succeeds in convincing vaccinated individuals to abstain from increasing the contact rate above that of the contact rate of non-compliant individuals. Our model predicts that a successful implementation of either of these interventions reduces the cumulative number of infections after vaccination rollout and can get this number below the level of no-vaccination scenario. The effectiveness of these interventions depends on the circulating variant and the vaccine uptake rate. We summarize our findings in Figures 5-7 and Table 1.

Intervention 1: Targeting compliance of non-vaccinated individuals

For the original variant (Figures 5**a** and 5**c**), the cumulative number of infections after one and two years is smaller than without vaccination regardless of vaccination rates. This number is smaller for the fast vaccination rate than for the slow vaccination rate as shown in Figure 5**c**. For a more transmissible, B.1.1.7-like variant (Figures 5**b** and 5**d**), the cumulative number of infections can be significantly reduced relative to the epidemic dynamics without interventions (compare Figures 5**d** and 3**d**, see Table 1), but will slightly exceed the level of no-vaccination scenario even at a two year mark (Figure 5**d**).

For the original variant, Intervention 1 improvement on the no-vaccination scenario are seen as early as 62 days into

the campaign, with larger prevalence reduction gained for the faster vaccination rate (Figure 5**a**). Moreover, in this case the model predicts that the epidemic can be extinguished in less than one year. Ultimately, after two years of the vaccination campaign, it is possible to reduce the cumulative number of infections by 26% if the vaccination rollout is slow and by 65% if it is fast, as compared to the no-vaccination scenario (Table 1).

For the B.1.1.7-like variant, Intervention 1 reduces the prevalence and the excess number of infections as compared to epidemic dynamics without intervention (Figures 3**b**, 3**d** 5**b**, 5**d**, Table 1). However, the prevalence in the first year of vaccination rollout is still higher than in the no-vaccination scenario. For slow vaccination rate the prevalence decreases below the level of the no-vaccination scenario after 485 days. For fast vaccination rate this happens 354 days after the start of the vaccination rollout. Subsequently, for slow and fast vaccination rates, there is a considerable excess of infections after the first year (11% and 70%, respectively) as compared to the no-vaccination scenario. For both vaccination rates, excess infections are slightly positive after two years vaccination campaign (Figures 5**c** and 5**d**). For the fast vaccination the cumulative number of infected individuals then lies 8% higher than in the no-vaccination scenario, while for slow vaccination there is only 3% difference (Figure 5**d**).

The impact of the intervention is smaller for the more transmissible variant, as a large proportion of infections originate in the vaccinated population and subsequently, are spread at a high rate by individuals who consider themselves immunized (Figure 2, Supplementary materials).

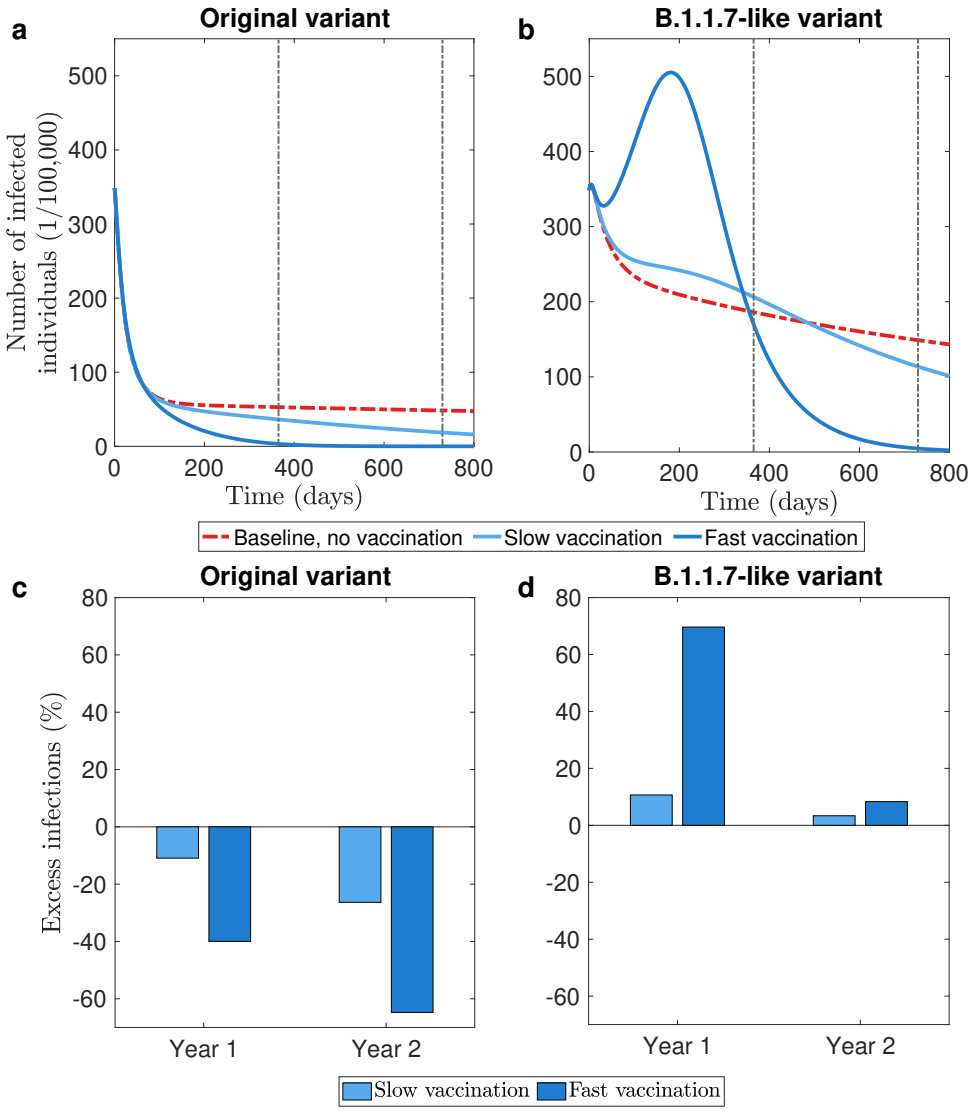

Figure 5: **Epidemic dynamics when intervention targeting compliance of non-vaccinated individuals is deployed.** **a** Prevalence of infected individuals versus time when the original variant circulates. **b** The same output when a B.1.1.7-like variant circulates. **c** Excess of cumulative infections relative to the no-vaccination scenario level for the original variant. **d** The same output when a B.1.1.7-like variant circulates. **c** and **d** show excess of infections relative to the no-vaccination levels when respective variants circulate. In **a** and **b**, vertical brown lines mark one and two years since the start of vaccination.

Intervention 2: Targeting compliance of vaccinated individuals

For both types of variants, regardless of vaccination rate, Intervention 2 yields improvement on epidemics dynamics without interventions both in the short and long terms (Figure 6). However improvements over the no-vaccination scenario can appear closer to two years since the start of the vaccination rollout (Figures 6**c** and 6**d**). Fast vaccination rates will produce larger reductions.

For the original variant the effects of Intervention 2 vary across the vaccination rates. If the vaccination rate is slow, the cumulative number of infections after 1 year is 7% higher than without vaccination, and only decreases

to negative 5% at the end of the second year (Figure 6**d**). If the vaccination rate is fast, the intervention produces significant improvements as compared to the no-vaccination scenario and as compared to the epidemic dynamics without interventions scenario (Table 1). After one year of vaccination the cumulative number of infections is reduced by 36% below the no-vaccination scenario level, and by 63% after two years. Respectively, the reduction of the cumulative number of infections compared to epidemic dynamics without interventions is equal to 36% after one year and 22% after two years of vaccination. Moreover, the vaccination campaign significantly and quickly reduces the prevalence of infections as compared to the no-vaccination scenario (Figure 6**a**). However, the outcomes of deploying Intervention 2 are worse than the outcomes of Intervention 1 as they produce smaller reduction of the cumulative number of infections (Table 1).

In the case of the B.1.1.7-like variant, Intervention 2 achieves improvements on both the epidemic dynamics with vaccination and on the no-vaccination scenario, the latter if the vaccination rate is fast. If the vaccination rate is slow, then we obtain improvement on epidemic dynamics without interventions scenario (with decrease of the cumulative number of infections equal to 27% and 27% after one and two years respectively) but still remain above the level of the no-vaccination scenario, with 23% and 23% excess infections in the first and second years, respectively (Table 1). If the vaccination rate is fast, Intervention 2 can produce improvements on the epidemic dynamics without interventions scenario and on the baseline, no vaccination scenario (Figures 6**b** and 6**d**, Table 1). In the first year, the outcomes of the vaccination campaign when intervention targeting compliance of the vaccinated individuals show an improvement on the epidemic dynamics without interventions scenario only (reduction by 124%). However, after two years, 38% reduction in the cumulative number of infected individuals as compared to the no-vaccination scenario is expected.

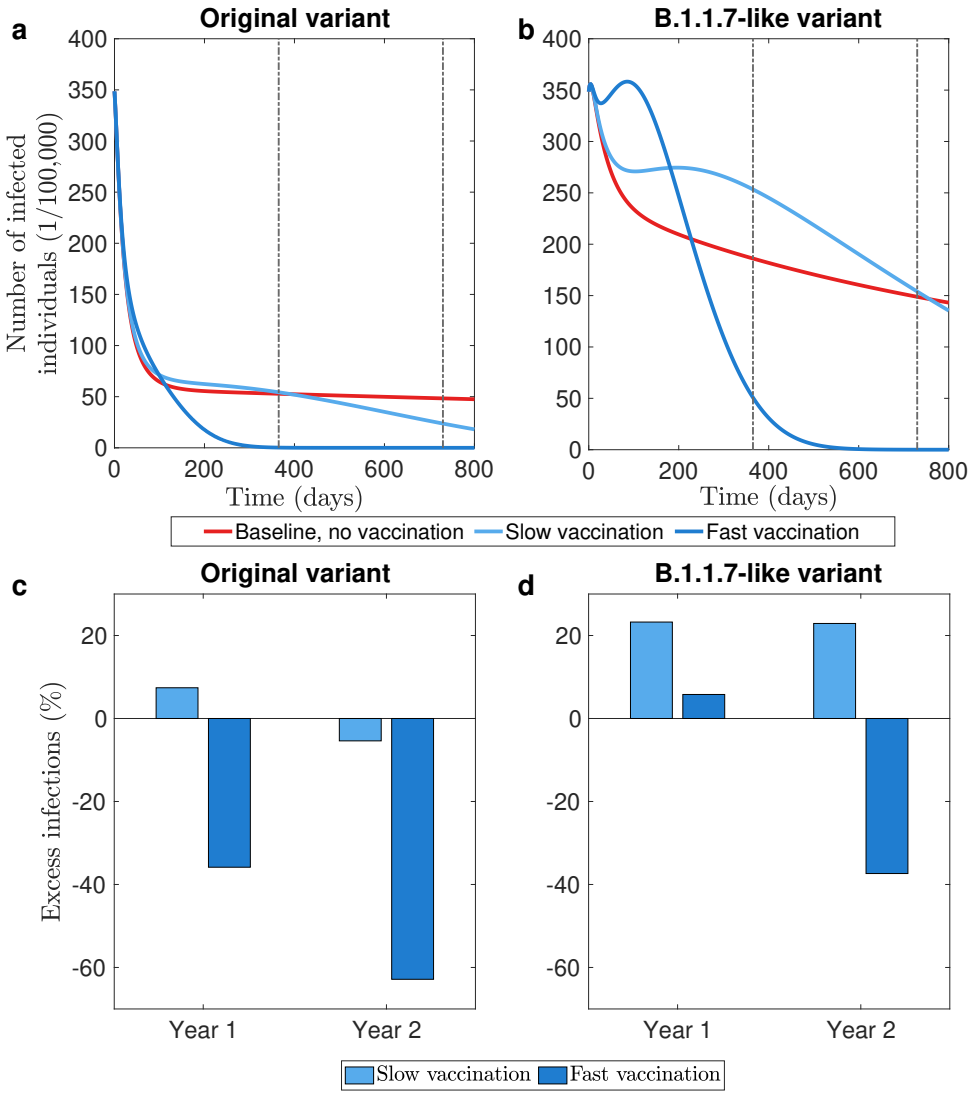

Figure 6: **Epidemic dynamics when intervention targeting compliance of vaccinated individuals is deployed.** **a** Prevalence of infected individuals versus time when the original variant circulates, **b** The same output when a B.1.1.7-like variant circulates, **c** Excess of cumulative infections relative to the no-vaccination scenario level for the original variant, **d** The same output when a B.1.1.7-like variant circulates. **c** and **d** show excess of infections relative to the no-vaccination levels when respective variants circulate. In **a** and **b**, vertical brown lines mark one and two years since the start of vaccination.

Combination of two interventions

Finally, combination of the two interventions leads to improvements that exceed the effects of individual interventions (Figure 7). For both variants, regardless of the vaccination uptake rates, the prevalence and the cumulative number of infections fall under the level of the no-vaccination scenario and epidemic dynamics without intervention scenarios as early as the first year of vaccination rollout. Universally, larger reductions are achieved for faster vaccination rates both in pure numbers and relative to the respective baseline scenario for the more transmissible variant, B.1.1.7 (Table 1).

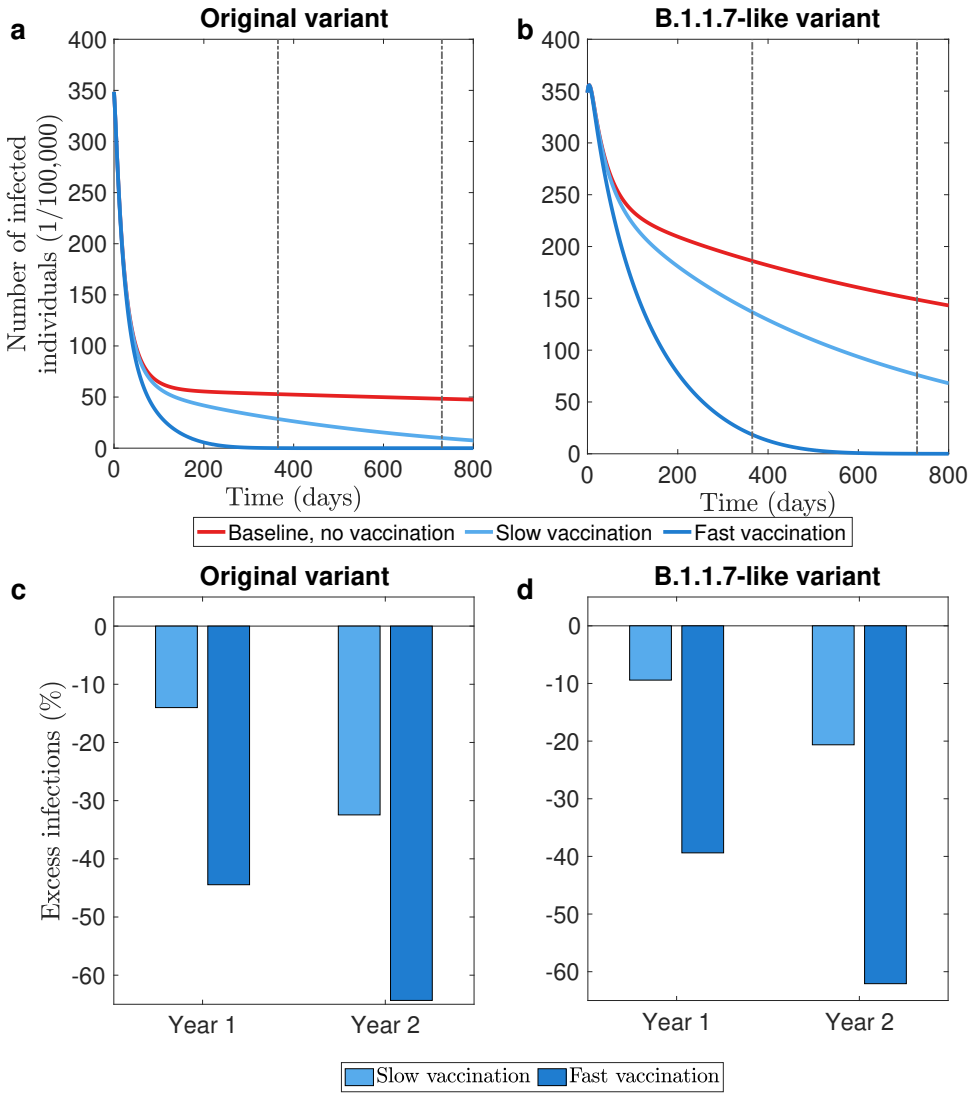

Figure 7: **Effects of the combination of interventions on the course of epidemic and excess infections.** **a** Prevalence of infected individuals versus time when the original variant circulates, **b** The same output when a B.1.1.7-like variant circulates, **c** Excess of cumulative infections relative to the no-vaccination scenario level for the original variant, **d** The same output when a B.1.1.7-like variant circulates. **c** and **d** show excess of infections relative to the no-vaccination levels when respective variants circulate. In **a** and **b**, vertical brown lines mark one and two years since the start of vaccination.

Table 1 summarize our findings across all epidemic scenarios that we considered, with and without vaccination and interventions.

In summary, we gain the following insights for different vaccination strategies and virus variants: (a) For the original variant slow vaccination leads to more excess infections than fast vaccination both in the short and long term, for the more transmissible variant fast vaccination is worse in the short term but better in the long run; (b) For the original variant an intervention targeting the non-vaccinated population is more effective regardless of vaccination rate; (c) For the more transmissible variant targeting non-vaccinated people is better if vaccination is slow, and targeting vaccinated people is better when vaccination is fast; fast vaccination and better compliance of vaccinated

Table 1: **Cumulative infections under different scenarios**

	Original variant		**B.1.1.7-like variant**	
	$n/100,000$ (% **of baseline**)		$n/100,000$ (% **of baseline**)	
	Year 1	**Year 2**	**Year 1**	**Year 2**
Baseline, no vaccination				
	2,301 (100)	3,975 (100)	7,390 (100)	12,878 (100)
Epidemic dynamics without interventions				
Slow vaccination	2,704 (117)	4,533 (114)	11,103 (150)	19,276 (150)
Fast vaccination	2,318 (101)	2,363 (59)	17,013 (230)	17,837 (139)
Intervention 1[£]				
Slow vaccination	2,050 (89)	2,928 (74)	8,178 (111)	13,310 (103)
Fast vaccination	1,381 (60)	1,400 (35)	12,537 (170)	13,950 (108)
Intervention 2[§]				
Slow vaccination	2,471 (107)	3,760 (95)	9,108 (123)	15,827 (123)
Fast vaccination	1,476 (64)	1,477 (37)	7,818 (106)	8,066 (62)
Interventions combined				
Slow vaccination	1,882 (81)	2,476 (62)	6,529 (88)	9,911 (77)
Fast vaccination	949 (41)	950 (24)	3,740 (51)	3,862 (30)

[£] targeting non-vaccinated individuals
[§] targeting vaccinated individuals

is the only way excess infections can be avoided in this case; (d) Slow vaccination with a combined intervention can reduce numbers of infections moderately for both virus variants; fast vaccination with a combined intervention is the only way to reach substantial vaccination impact within two years for both types of the virus variants.

Discussion

Using a compartmental model for the spread of SARS-CoV-2 in a population, where physical distancing measures are in place, we investigated the impact of vaccination rollout-related declining compliance to physical distancing on the numbers of infections. Our main finding is that, if compliance decays as the vaccination coverage grows, the speed of vaccination rollout has a strong impact on whether the cumulative number of infected can be decreased one or two years after the start of vaccination below the expected level of no-vaccination scenario. If vaccination rollout is slow, its positive effects on the incidence will be counteracted by fading compliance and increasing contact rates in the population. This might lead to an increase in the prevalence exceeding the number of infections in a situation without vaccination. If vaccination is rolled out faster, these detrimental effects can be avoided to some extent. However, in the short term, we may even see an additional epidemic peak. On a positive note, since among the excess infections a certain proportion of infected people will have been vaccinated, they will have a low probability of developing severe disease or death. An intervention that succeeds in maintaining the compliance to physical distancing in people not yet vaccinated on the same level as before the start of vaccination ensures significant decrease of the cumulative number of infections due to vaccination rollout throughout for the original virus variant, and substantial decrease in prevalence after a year of vaccination for the B.1.1.7 variant. An intervention that targets vaccinated individuals to prevent them from increasing their contact rates after being vaccinated, has a positive impact for fast rollout of vaccination, but cannot avoid detrimental effects of inevitable waning of compliance if vaccination rollout is slow. In the latter case, the cumulative number of infected is up to 23% higher as compared to a situation without vaccination. Only the combined effect of both interventions can consistently reduce the cumulative number of infections below the level of a no-vaccination scenario regardless of the rollout speed (in the vaccination rate range that we considered).

In their recent work Gozzi et al [42] also have considered the impact of the feedback between the epidemic dynamics, the vaccination rollout and compliance to physical distancing on infection transmission dynamics. The authors modeled compliant behavior modifications only as pertinent to the susceptible/vaccinated population, which may have resulted in more optimistic predictions for a disease like COVID-19 whose hallmark is asymptomatic spread. However, both ours and Gozzi et al [42] qualitative findings are in agreement between themselves and are consistent with the results of the earlier studies that have shown that factors that contribute to drastic increase of contact rates (such as vaccination-related behavioral change or premature reduction/removal of non-pharmaceutical interventions) may reduce the benefits of a vaccination programme [43, 44, 45, 46].

Motivated by the conclusions drawn by these studies, we considered the effect of supplementing the vaccination campaigns with communication strategies promoting maintenance of physical distancing behavior aimed at both vaccinated and non-vaccinated individuals and learned that 1) those interventions can significantly improve the outcome of vaccination campaign; 2) the choice of a specific information intervention should be informed by the epidemic circumstance of the situation (such as the dominant variant and speed of vaccination rollout).

Our results are based on some simplifying assumptions, one of them that physical distancing measures remain in place throughout the time period of analysis (two years). While this would be advantageous for preventing transmission of the virus, it might not be feasible out of societal and economic reasons. Therefore, compliance rates may even wane faster in real populations and contact rates may be up to higher, possibly pre-pandemic values during the rollout of vaccination. We do not expect that this would change our results much, as our results are obtained relative to a no-vaccination scenario, which would similarly be affected by a change in physical distancing measures. We expect therefore that the relative effects of vaccination would remain similar as in our simulations. We also assumed that the speed of vaccination rollout stays constant over the time period of 2 years, which is not the case in reality. In the Netherlands for example, vaccination rates have increased substantially after a slow start in January 2021 [29]. These rates will depend on many factors, nevertheless large differences will remain between countries. Finally, we have captured the dependence of rates of becoming compliant and non-compliant on the incidence of new infectious cases and vaccination coverage, respectively, using linear functions. As the vaccination in many countries continues and the population response data is collected, a more precise formulation of the response functions can be obtained. However, our results predominantly depend on the assumed monotonicity of these functions.

Furthermore, our model is relatively simple, not taking into account age structure and heterogeneity in contact patterns. Therefore, we do not attempt to make quantitative predictions on the impact of vaccination, but we provide qualitative insight into possible effects of waning compliance with physical distancing in the face of increasing vaccination coverage.

Recently, a number of studies/reports estimated the bounds for vaccine efficacy in terms of reducing the infection for some vaccines approved for use in Europe [19, 20, 22, 23]. Whether this reduction comes in the guise of reduction of susceptibility or transmissibility of vaccinated individuals is not known. Therefore, in this work we modeled the vaccination to be all-or-nothing and vaccine efficacy was given in terms of probability of conferring full protection from becoming infected. Our sensitivity analyses (Supplementary materials, Figures 15 and 16) show that the effect of a vaccination campaign and of individual interventions is highly sensitive with respect to this parameter. To implement the most efficient vaccination rollout it is important to know the boundaries of vaccine-conferred reduction of transmission.

Finally, in this work we have considered dynamics of circulation of two SARS-CoV-2 virus variants, the original variant and B.1.1.7 variants. With respect to the latter, we made a simplifying assumption that the vaccine has the same effectiveness with respect to the induction of immunity as it is against the original variant. Moreover, we

modeled as the immunity of the identical type (sterilising). However, already there is at least one variant, B.1.351, that was demonstrated to have a potential to diminish vaccine-induced immunity and increase risk of infection in vaccinated individuals [25, 47].

These results also show that speed of rollout of a vaccination campaign is important, because the speed of the rollout and subsequent changes in contact rates strongly impact cumulative case numbers. Although in the scenario where vaccination rollout is fast the epidemic may fair worse than it would have been without vaccination in the short term - especially for a more transmissible virus variant - on the longer term (> 1 year) it has vast advantages in terms of numbers of infections prevented.

Our results emphasize the importance of communication by public health professionals on continued adherence to self-imposed measures, to those who are awaiting vaccination as well as to those already vaccinated. Communication messages need to be different and targeted specifically to these two groups. We highlight the positive overall effects of vaccination campaigns in combination with continued adherence to non-pharmaceutical preventive measures.

Methods

Model

We developed a compartmental deterministic model that describes SARS-CoV-2 transmission and vaccination rollout in a population. Subsequently, we modified this model to include acquisition and loss of compliance by the population as individuals continuously get exposed to information about disease spread as well as the progress of vaccination rollout (Figure 1). We parametrized the model using parameter values from the literature as well as estimating a number of parameters by using publicly available data for the Netherlands. We used the model to investigate the effect of co-interaction between disease transmission, vaccination rollout, and changing compliance on the transmission dynamics.

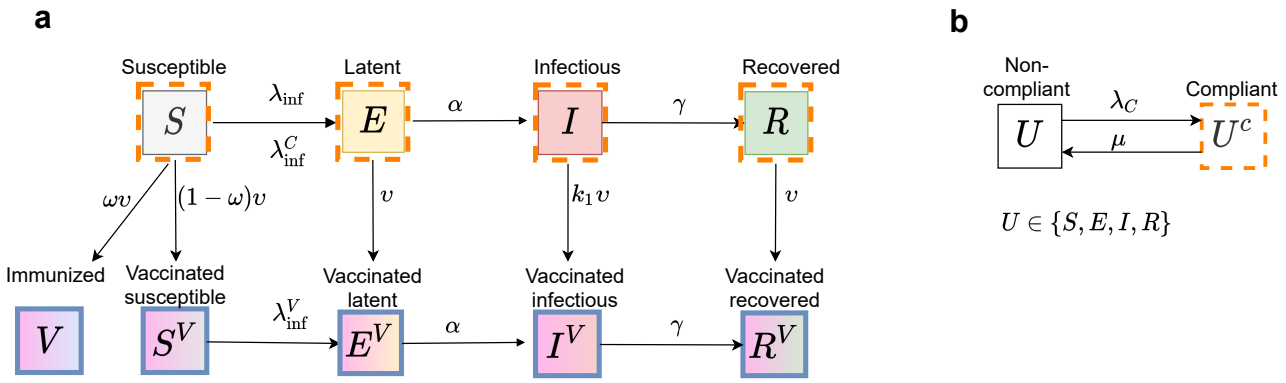

Figure 1: **Flow diagram of the infection transmission dynamics coupled with compliance and vaccination processes.** **a** shows dynamics of infection transmission and vaccination rollout, **b** shows the dynamics of acquisition and loss of compliance. Solid-colored rectangles denote non-vaccinated individuals, among them solid-bordered rectangles denote non-compliant individuals, orange dashed-bordered rectangles denote compliant individuals. Gradient-colored rectangles represent vaccinated individuals. Susceptible individuals (S, S^C, and S^V) become latently infected (E, E^C, and E^V, respectively) with rates λ_{inf}, λ^C_{inf}, and λ^V_{inf} through contact with infectious individuals (I, I^C, and I^V). Exposed individuals become infectious (I, I^C, and I^V, respectively) at rate α. Infectious individuals become recovered (R, R^C, and R^V) at rate γ. Compliance is gained with rate λ^C and lost with rate μ. Individuals in any state of infection or mode of compliance can get vaccinated. A proportion ω of susceptible individuals S, who were vaccinated will become fully protected, V. Individuals who were vaccinated but did not obtain protection are denoted with S^V, E^V, I^V and R^V and are epidemiologically indistinguishable from their non-vaccinated counterparts.

Population compartments The SARS-CoV-2 transmission dynamics follow a Susceptible-Exposed-Infectious-Recovered (SEIR) framework that divides the population into the following compartments: susceptible (S), latently infected (also referred to as "exposed", E), infectious (I), and recovered (R). Susceptible individuals (S) become latently infected (E) with rate λ_{inf} proportional to the fraction of infectious individuals (I/N, where N is the total population size). Individuals stay latently infected (E) for an average duration of $1/\alpha$ days after which they become infectious (I). Infectious individuals recover after $1/\gamma$ days and move to compartment R. Because of a relatively short time horizon of our analyses (not exceeding two years) and relatively small case fatality ratio, we disregarded demographic processes such as births and deaths, and therefore the population size N is constant. Additionally, we assumed that once individuals recover they acquire permanent immunity and cannot be re-infected. Since we are interested in understanding the qualitative dynamics that follow from co-interaction of infection transmission, changes in compliance, and vaccination rollout, we did not consider different outcomes that infection with SARS-CoV-2 may cause (e.g., asymptomatic or symptomatic infection, hospitalisation, death etc.). The infectious compartment (I), therefore, contains individuals who are asymptomatic, or have mild or severe symptoms.

The dynamics of the infection transmission are modelled for two different variants of the SARS-CoV-2 virus. The first variant represents the original variant that was predominant in Europe prior to fall 2020. The second, more transmissible variant represents the B.1.1.7 variant that was initially detected in the UK. We reflect the difference between these two variants by using different probabilities of infection per contact, ϵ. We assume that in all other

respects the variants have the same properties. We investigate model dynamics where only one of the two variants circulates in the population.

To model vaccination, the population is stratified into vaccinated and non-vaccinated classes. While for some vaccines authorised for use in Europe (BioNTech/Pfizer, Moderna and AstraZeneca, [15]), two vaccine doses are required to provide the full immunisation effect as well as a certain amount of time to pass from the moment of obtaining the second shot, we model the vaccination as a single event that confers protection instantaneously. We assume that individuals do not obtain either a diagnostic or an antibody test prior to vaccination, and therefore infected and recovered individuals also get vaccinated. Therefore, individuals in all epidemiological compartments can get vaccinated, but only those who were susceptible (S) at the time of vaccination may become immunised (V). The vaccination rate of susceptible, exposed, and recovered individuals is denoted with υ. We define a parameter k_1, $0 \leq k_1 \leq 1$, such that $k_1\upsilon$ denotes the vaccination rate for individuals in the infectious compartment, to reflect that a fraction of infectious individuals (who have symptoms) might not be eligible for or might decide against vaccination. In the main analysis we considered the case where infectious individuals get vaccinated at the same rate as individuals in other compartments ($k_1 = 1$). We explored sensitivity of the dynamics to variations of k_1 and observed that the outputs are not sensitive to perturbations in this parameter (the Supplementary materials). We assume that the vaccine works as all-or-nothing, i.e. upon vaccination a proportion, ω, of susceptible individuals (S) becomes fully protected from becoming infected (V), while in a proportion $1 - \omega$ of susceptible individuals the vaccine has no effect. We refer to ω as "vaccine efficacy" in the context of conferring sterilising immunity. Vaccination does not confer protection to individuals who were in other infection compartments (E, I and R) at the time of vaccination. The infection progression of vaccinated persons who do not become immunised is identical to the progression of non-vaccinated individuals. Individuals who were vaccinated but did not obtain the protection are denoted with S^V, E^V, I^V and R^V.

Recently, the Israel Ministry of Health, Pfizer Inc., and BioNTech SE in a joint press release announced that the BNT162b2 mRNA COVID-19 vaccine, developed by Pfizer/BioNTech can reduce the acquisition rate for asymptomatic SARS-CoV-2 infection by as much as 94% [21]. The extent of the decrease for other vaccines on the market is not yet known, although Lipsitch and Kahn estimated the lower bound for efficacy conferring reduction in susceptibility after a single dose of Moderna vaccine to be greater or equal to 61% [22]. Therefore, in our analyses, we varied ω on the interval of values ranging between 0.6 to 0.95. In this work we did not address the possibility of a variant escaping the immune response induced by vaccination and assumed the same vaccine efficacy for both variants.

Finally, in addition to infection status and vaccination status, individuals in the model are either compliant or non-compliant (compliant compartments denoted by superscript C: S^C, E^C, I^C, and R^C). Individuals who are compliant have on a lower contact rate than non-compliant individuals due to practicing physical distancing; both contact rates are assumed to be lower than pre-pandemic levels. We denote the contact rate of non-compliant

individuals with c and define the reduction factor in contact rate of compliant individuals compared to non-compliant is denoted by the parameter r_1, $0 \leq r_1 \leq 1$. Compliance can be gained and lost. Using a disease-behavior framework similar to the one developed by Perra et al [32] and used previously to assesses effects of self-imposed measures on SARS-CoV-2 dynamics in a population [34], we modeled the compliance acquisition rate, λ_C, as a function of the incidence of infection, assuming that individuals obtain information about numbers of cases through mass-media and health authorities. We assumed compliance wanes if case numbers drop or if the disease is no longer present, and individuals return to the non-compliant mode at rate μ. If there is no vaccination programme in place then this rate is constant. However, if vaccination rollout is in progress and as vaccination coverage increases, individuals may feel less motivated to remain compliant; we implemented this effect by an increased rate of losing compliance with increasing vaccination coverage, i.e. μ is taken as a linear function of vaccination coverage. We assumed that only non-vaccinated individuals can move into a more compliant mode, while vaccinated individuals move into a non-compliant mode permanently and even have higher contact rates than non-vaccinated non-compliant individuals. Compliant individuals get vaccinated at the same rate as non-compliant individuals.

We assumed that once vaccinated, individuals no longer comply with physical distancing. Vaccinated individuals who view themselves as well-protected from infection, are assumed to have a higher contact rate than the contact rate of non-compliant non-vaccinated individuals. We use $r_2 \geq 1$ to denote the increase in the contact rates of vaccinated individuals relative to the contact rate of non-compliant individuals, c. All individuals who were vaccinated will have the same (increased) contact rate regardless of whether vaccination was successful.

Rates In this section we discuss formulation of transition rates that depend on the epidemic state of the population and on vaccination coverage: rates of infection acquisition, and rates of acquisition and loss of compliance.

We assumed that individuals become infected at a rate that depends on the fractions of different types of infectious individuals, as well as on the mixing of compliant, non-compliant and vaccinated individuals. Therefore, infection acquisition rates as well as infection transmission rates depend on compliance and vaccination status of susceptible and infectious individuals. We define the following matrix to summarize transmission rates between different types of susceptible and infectious individuals.

$$M = \frac{c\epsilon}{N(t) + r_1 N^C(t) + r_2 N^V(t)} \begin{bmatrix} 1 & r_1 & r_2 \\ r_1 & r_1^2 & r_1 r_2 \\ r_2 & r_1 r_2 & r_2^2 \end{bmatrix} \tag{1}$$

with

$$
\begin{aligned}
N(t) &= S(t) + E(t) + I(t) + R(t) \\
N^C(t) &= S^C(t) + E^C(t) + I^C(t) + R^C(t) \\
N^V(t) &= V(t) + S^V + E^V(t) + I^V(t) + R^V(t),
\end{aligned}
$$

where $[M]_{11}$ captures the transmission of infection from non-compliant I to non-compliant S, $[M]_{12}$ from compliant I to non-compliant S, and $[M]_{13}$ from vaccinated I to non-compliant S. Similarly, the second row of the matrix captures the transmission of infection to susceptible individuals who are compliant, S^C. Finally, the third row of the matrix captures the transmission of infection to individuals who are susceptible despite vaccination, S^V.

We assumed that as the individuals learn about new infections they become compliant with physical distancing measures and therefore compliance is gained at the rate λ_C which is a positive increasing function of the incidence of the infectious cases:

$$\lambda_{\mathrm{C}}(t) = \delta \cdot \alpha \cdot \left[E(t) + E^C(t) + E^V(t)\right]. \tag{2}$$

On the other hand, we assumed that compliance is not permanent and eventually compliant individuals become non-compliant. Moreover, as the vaccination coverage increases, individuals stay compliant for a shorter period of time. Therefore, we model compliant mode to have an average duration $1/\mu$, such that μ is a positive increasing function of the vaccination coverage, $\bar{V}(t)/N$:

$$\mu(t) = \mu_0 + \mu_1 \bar{V}(t)/N. \tag{3}$$

Equations The system of ordinary differential equations (4) provides a full description of the model.

Dynamics of non-compliant individuals:

$$\frac{\mathrm{d}S(t)}{\mathrm{d}t} = -\lambda_{\mathrm{inf}}(t)S(t) - \lambda_{\mathrm{C}}(t)S(t) + \mu(t)S^C(t) - \upsilon S(t)$$
$$\frac{\mathrm{d}E(t)}{\mathrm{d}t} = \lambda_{\mathrm{inf}}(t)S(t) - \alpha E(t) - \lambda_{\mathrm{C}}(t)E(t) + \mu(t)E^C(t) - \upsilon E(t)$$
$$\frac{\mathrm{d}I(t)}{\mathrm{d}t} = \alpha E(t) - \gamma I(t) - \lambda_{\mathrm{C}}(t)I(t) + \mu(t)I^C(t) - k_1\upsilon I(t)$$
$$\frac{\mathrm{d}R(t)}{\mathrm{d}t} = \gamma I(t) - \lambda_{\mathrm{C}}(t) + R(t)\mu(t)R^C(t) - \upsilon R(t)$$

Dynamics of compliant individuals:

$$\frac{\mathrm{d}S^C(t)}{\mathrm{d}t} = -\lambda^C_{\mathrm{inf}}(t)S^C(t) + \lambda_{\mathrm{C}}(t)S(t) - \mu(t)S^C(t) - \upsilon S^C(t)$$
$$\frac{\mathrm{d}E^C(t)}{\mathrm{d}t} = \lambda^C_{\mathrm{inf}}(t)S^C(t) - \alpha E^C(t) + \lambda_{\mathrm{C}}(t)E(t) - \mu(t)E^C(t) - \upsilon E^C(t)$$
$$\frac{\mathrm{d}I^C(t)}{\mathrm{d}t} = \alpha E^C(t) - \gamma I^C(t) + \lambda_{\mathrm{C}}(t)I(t) - \mu(t)I^C(t) - k_1\upsilon I^C(t) \tag{4}$$
$$\frac{\mathrm{d}R^C(t)}{\mathrm{d}t} = \gamma I^C(t) + \lambda_{\mathrm{C}}(t)R(t) - \mu(t)R^C(t) - \upsilon R^C(t)$$

Dynamics of vaccinated individuals:

$$\frac{\mathrm{d}V(t)}{\mathrm{d}t} = \omega\upsilon\left(S(t) + S^C(t)\right)$$
$$\frac{\mathrm{d}S^V(t)}{\mathrm{d}t} = (1-\omega)\upsilon\left(S(t) + S^C(t)\right) - \lambda^V_{\mathrm{inf}}(t)S^V(t)$$
$$\frac{\mathrm{d}E^V(t)}{\mathrm{d}t} = \lambda^V_{\mathrm{inf}}(t)S^V(t) + \upsilon\left(E(t) + E^C(t)\right) - \alpha E^V(t)$$
$$\frac{\mathrm{d}I^V(t)}{\mathrm{d}t} = \alpha E^V(t) + k_1\upsilon\left(I(t) + I^C(t)\right) - \gamma I^V(t)$$
$$\frac{\mathrm{d}R^V(t)}{\mathrm{d}t} = \gamma I^V(t) + \upsilon\left(R(t) + R^C(t)\right)$$
$$\frac{\mathrm{d}\bar{V}(t)}{\mathrm{d}t} = \upsilon\left(S(t) + E(t) + R(t) + S^C(t) + E^C(t) + R^C(t) + k_1\left(I(t) + I^C(t)\right)\right),$$

where

$$\lambda_{\mathrm{inf}}(t) = [M(t)]_{11}I(t) + [M(t)]_{12}I^C(t) + [M(t)]_{13}I^V(t) \tag{5a}$$
$$\lambda^C_{\mathrm{inf}}(t) = [M(t)]_{21}I(t) + [M(t)]_{22}I^C(t) + [M(t)]_{23}I^V(t) \tag{5b}$$
$$\lambda^V_{\mathrm{inf}}(t) = [M(t)]_{31}I(t) + [M(t)]_{32}I^C(t) + [M(t)]_{33}I^V(t). \tag{5c}$$

Parameters and initial data

A full list of parameters and their values are given in Table 2. Here we elaborate on our calculations of the initial condition, as well as on the calculation of the behavioral parameters.

Initial data According to the parameter values that we have selected the combined average duration of latent

Table 2: **Summary of model parameters.**

Name	Description (unit)	Value*	Source
Epidemiological parameters			
R_0	Basic reproduction number, original variant	2.5 (2–3)	[48, 49]
R_0^{new}	Basic reproduction number, B.1.1.7-like variant	3.75 (3.5–4.25)	[11, 12]
R_e	Effective reproduction number, original variant	1.1	Computed using the method in [50]
$\hat{c}$	Average contact rate prior to the epidemic (individuals/day)	14.9	[40]
ϵ	Probability of transmission per contact, original variant	2.4×10^{-2}	From $R_0 = \hat{c}\epsilon/\gamma = 2.5$
ϵ^{new}	Probability of transmission per contact, B.1.1.7-like variant	3.6×10^{-2}	From $R_0 = \hat{c}\epsilon^{new}/\gamma = 3.75$
c	Average contact rate of non-compliant individuals starting November 16, 2020 (individuals/day)	8.8	Obtained from solving $R_e(0) = 1.1$
r_1	Ratio between contact rates of compliant and non-compliant individuals	0.34 (0-1)	Assumed, control parameter
r_2	Ratio between contact rates of vaccinated and non-compliant individuals	1.5 (0-2)	Assumed, control parameter
$1/\alpha$	Duration of latent period (days)	4	[48, 49, 51]
$1/\gamma$	Duration of infectious period (days)	7	[52]
Compliance parameters			
δ	Per capita rate of moving to compliant mode (1/individual)	4×10^{-5} (1.5×10^{-6}–4×10^{-5})	Assumed, control parameter
$1/\mu_0$	Duration of compliant mode when there is no vaccination (days)	30 (7–30)	Sensitivity analyses
μ_1	Parameter describing how loss of compliance increases depending on vaccination coverage (1/day)	0, 0.3	Sensitivity analyses
Vaccination parameters			
υ	Vaccination uptake rate (1/day)	$(5.9, 49) \times 10^{-3}$	Based on vaccination data in [29]
ω	Vaccine efficacy in conferring protection against becoming infected	0.6 (0.4-1)	Based on estimates of efficacies in blocking SARS-CoV-2 infections for some of the existing vaccines [20, 22, 23], control parameter
k_1	Reduction factor in vaccination rate of infectious individuals	$1(0-1)$	Sensitivity analysis

* Interval was used in sensitivity analyses.
† National Institute for Public Health and the Environment in the Netherlands

and infectious stage is estimated to be 11 days (Table 2, [48, 49, 51, 53]). Since not all cases get detected, the weekly number of newly diagnosed individuals is a lower bound of the number of active cases that were detected. Using the data reported by RIVM for the week November 11-17, we set the total number of currently infectious individuals, $I + I^C$, at the start of vaccination rollout to 37,706. We have used this value in the main analysis and performed sensitivity analysis to investigate the sensitivity of our results to this choice. To estimate the fraction of recovered individuals, $R + R_C$, we used the seroprevalence data collected in a serological study in an age-stratified and regionally weighted representative sample of the Dutch population in the Netherlands in June and July of 2020 which estimated the seroprevalence to be 4% [36]. We set the number of recovered, $R + R^C$ such that at the start of vaccination rollout, the seroprevalence was equal to a higher value, 8% and performed sensitivity analysis with respect to the initial size of the recovered population. The size of the susceptible population $S + S^C$ follows. From the dynamics of the system we obtain

$$\frac{S}{S+S^C} = \frac{R}{R+R^C} = \frac{E+I}{E+I+E^C+I^C} \tag{6}$$

We have set the initial proportion of compliant individuals to be equal to 65% based on the compliance to maintaining the distance of 1.5m as reported by respondents of the study on behavioral measure and well-being conducted between November 11-15, 2020 in the Netherlands by RIVM [38]. Using the Eq. (6) and the percentage of compliant population , initial values for S, $E+I$, R, S^C, E^C+I^C, R^C follow. To obtain E and E^C, at the start of vaccination rollout we have estimated that the daily incidence of new cases is 16,149 people. Assuming that the system resides in (pseudo) equilibrium we have estimated $E + E^C$, and evaluated E and E^C using Eq. (6).

Setting the total population size to be equal to approximately that of the Netherlands, 1.7×10^7 we obtain:

$$S(0) = 5,453,262\ ,\ E(0) = 7,541\ ,\ I(0) = 13,197\ , R(0) = 476,000\ ,$$
$$S^C(0) = 10,127,486\ ,\ E^C(0) = 14,005\ ,\ I^C(0) = 24,509\ ,\ R^C(0) = 884,000\ .$$

The initial data for the rest of the compartments are set to 0.

Contact rates We defined a contact as an encounter with another individual that is sufficiently long to have a conversation or that involves physical interactions [40]. At the start of vaccination rollout a fraction of the population is more compliant with physical distancing measures and the remaining fraction is less compliant, such that contact rates in the compliant and non-compliant modes are constant and lower than pre-pandemic contact rates. However, as a consequence of vaccination and subsequent loss of compliance the average contact rate in the total population will change in time.

We fixed the contact rates for compliant and non-compliant individuals such that the effective reproduction number R_e at the start of the vaccination rollout was equal to 1.1, which is in agreement with the estimate of R_t reported by the RIVM for the population in the Netherlands in November 2020 [39]. We calculated $R_e(0)$ assuming that

$R_0 = \beta/\gamma = \hat{c}\epsilon/\gamma = 2.5$ [48, 49].

Recall that the contact rates of non-compliant and compliant individuals are denoted by c and $r_1 c$. We calculated the effective reproduction number using the method described in [50] as

$$R_e = \frac{\epsilon c S(0)}{\gamma(N(0) + N^c(0) r_1)} + \frac{\epsilon r_1 c S_c(0)\,(\mu_0(\alpha + \gamma + \mu_0) + \alpha\gamma r_1)}{\gamma(\alpha + \mu_0)(\gamma + \mu_0)(N(0) + N^c(0) r_1)}. \tag{7}$$

The value $R_e = 1.1$ is obtained for pairs of contact rates of non-compliant individuals, c, and contact rate of compliant individuals, $r_1 c$ (Figure 9).

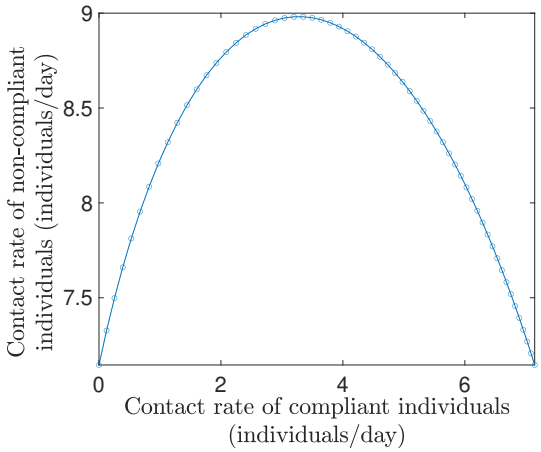

Figure 9: Pairs of contact rates of compliant and non-compliant individuals c and $r_1 c$ such that effective reproduction number is equal to 1.1.

Of all the pairs of contact rates that satisfy $R_e(0) = 1.1$, we have selected a combination such that the weighted average contact rate for the population at the start of the vaccination is 5 contacts per day. This value exceeds the reported number of contacts in the Netherlands during the government-initiated physical distancing measures in March 2020 by 1.5 contacts but is lower than the reported contact rate of 8.8 per day that was observed in June 2020, when some of the physical distancing measures were relaxed [40]. The chosen parameter pair is $c = 8.8$ and $r_1 c = 3$.

Contact rates of vaccinated individuals were taken to be 1.5 times the contact rate of non-compliant individuals, assuming that after vaccination individuals will nearly return to the pre-pandemic contact behaviour.

Compliance The proportion of compliant and non-compliant individuals in the population is determined by the compliance acquisition rate δ and compliance loss rate μ. For the main analysis we fixed the duration of compliance when there is no vaccination, $1/\mu_0$ to 30 days. We have selected the per capita rate of moving to compliant mode, $\delta = 4 \times 10^{-5}$ so that given a constant daily incidence of 5,387 cases, 86% of the population is expected to be compliant . In the regime where the epidemic is seeded in a population without any physical measures enforced (i.e. contact rate of non-compliant individuals is 14.9 individuals per day) as much as 84% of the population can be compliant provided there were no compliant individuals at the start of the epidemic. This value denotes the case

with high compliance acquisition rate. We investigated the sensitivity of the outputs to variation in per capita rate of moving to compliant mode and compliance loss rate using sensitivity analyses, Supplementary materials).

In the main analyses we considered a compliance decay scenario where as the vaccination coverage grows the duration of staying compliant decreases, such as when 1/3 of the population is vaccinated the compliant mode lasts 7 days. In other words, given a fixed daily incidence of 5,387 cases that we used to initialize the model, if the vaccination rate is slow then approximately 56% of the population is compliant one year after the start of vaccination rollout and if the vaccination is fast then approximately 7% is compliant (Figure 2**b**). This corresponds to $\mu_1 = 0.3$ per day.

Model code The model was implemented in MATLAB R2020b [54]. The code producing the analysis and figures for this study is available at https://github.com/aiteslya/VaccineCompliance [55].

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Figures

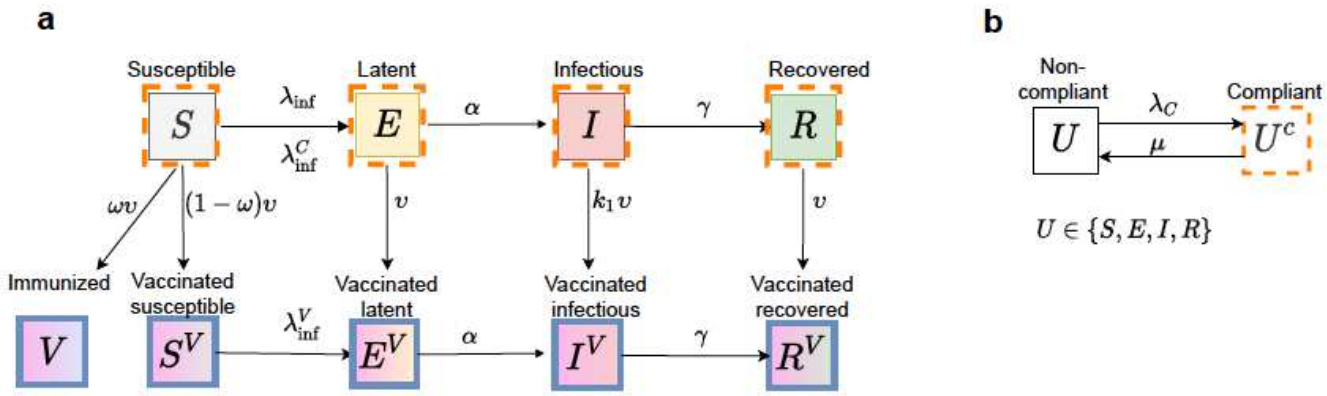

Figure 1

Flow diagram of the infection transmission dynamics coupled with compliance and vaccination processes. a shows dynamics of infection transmission and vaccination rollout, b shows the dynamics of acquisition and loss of compliance. Solid-colored rectangles denote non-vaccinated individuals, among them solid-bordered rectangles denote non-compliant individuals, orange dashed-bordered rectangles denote compliant individuals. Gradient-colored rectangles represent vaccinated individuals. Susceptible individuals (S, SC, and SV) become latently infected (E, EC, and EV , respectively) with rates λinf, λC inf, and λV inf through contact with infectious individuals (I, IC, and IV). Exposed individuals become infectious (I, IC, and IV , respectively) at rate □. Infectious individuals become recovered (R, RC, and RV) at rate γ. Compliance is gained with rate λC and lost with rate μ. Individuals in any state of infection or mode of compliance can get vaccinated. A proportion ω of susceptible individuals S, who were vaccinated will become fully protected, V . Individuals who were vaccinated but did not obtain protection are denoted with SV , EV , IV and RV and are epidemiologically indistinguishable from their non-vaccinated counterparts.

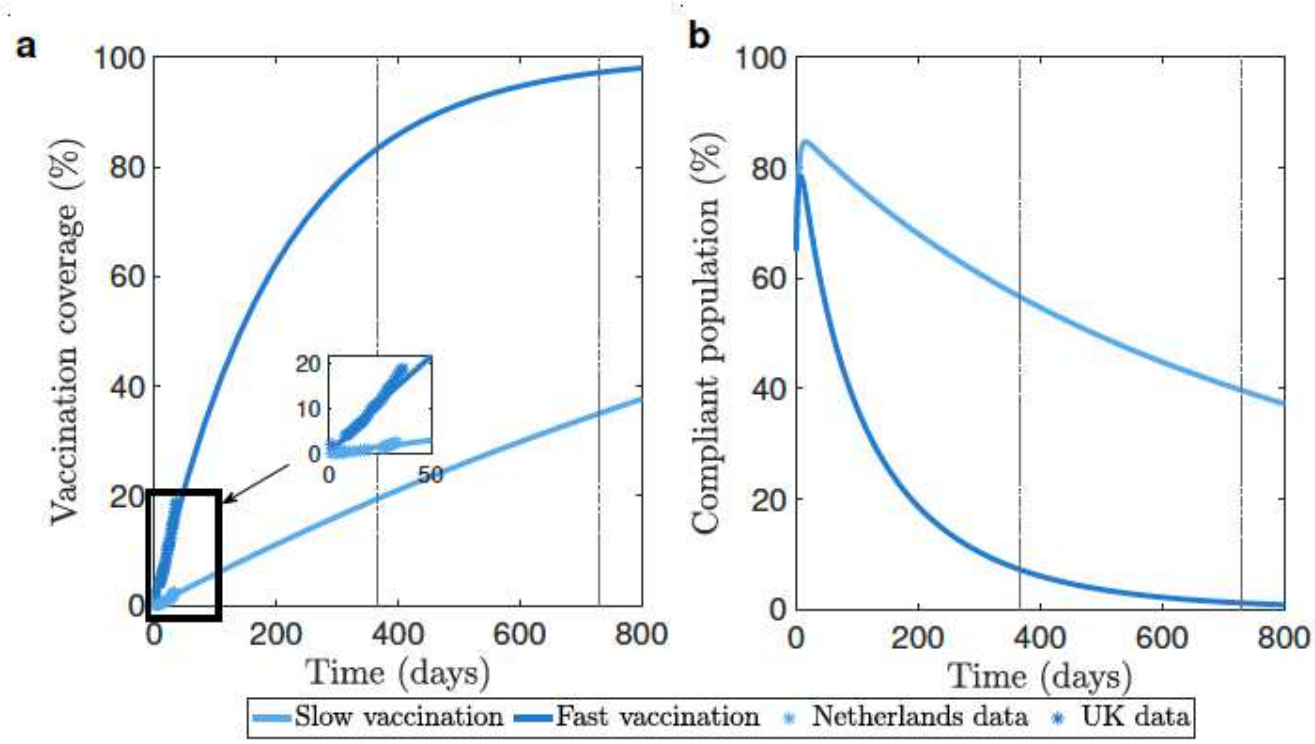

Figure 2

Vaccination coverage and proportion of compliant population during the vaccination rollout. a Increase in vaccination coverage for slow (light blue line) and fast (dark blue line) vaccination rates. Stars show data for the Netherlands (light blue) and the UK (dark blue) [29], respectively. b Decrease in the proportion of compliant population for slow and fast vaccination and a xed incidence of infection (5,387 cases per day) observed in the Netherlands in the period used for the model calibration. Vertical brown lines mark one and two years since the start of vaccination.

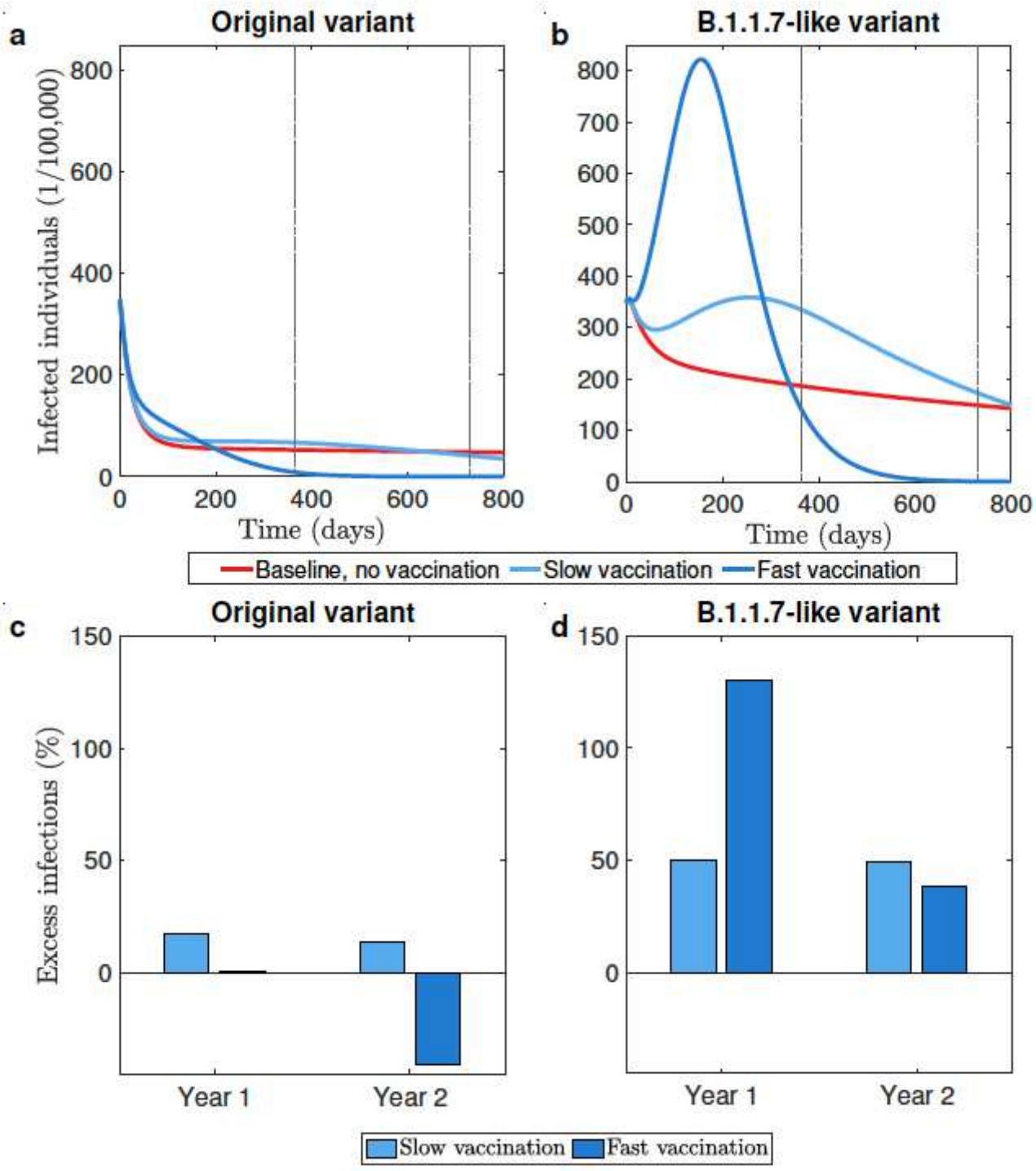

Figure 3

Epidemic dynamics with and without vaccination. a Prevalence of infected individuals versus time when the original variant circulates. b The same output when a B.1.1.7-like variant circulates. c Excess of cumulative infections relative to the no-vaccination scenario level for the original variant. d The same output when a B.1.1.7-like variant circulates. c and d show excess of infections relative to the no-vaccination levels when respective variants circulate. In a and b, vertical brown lines mark one and two years since the start of vaccination.

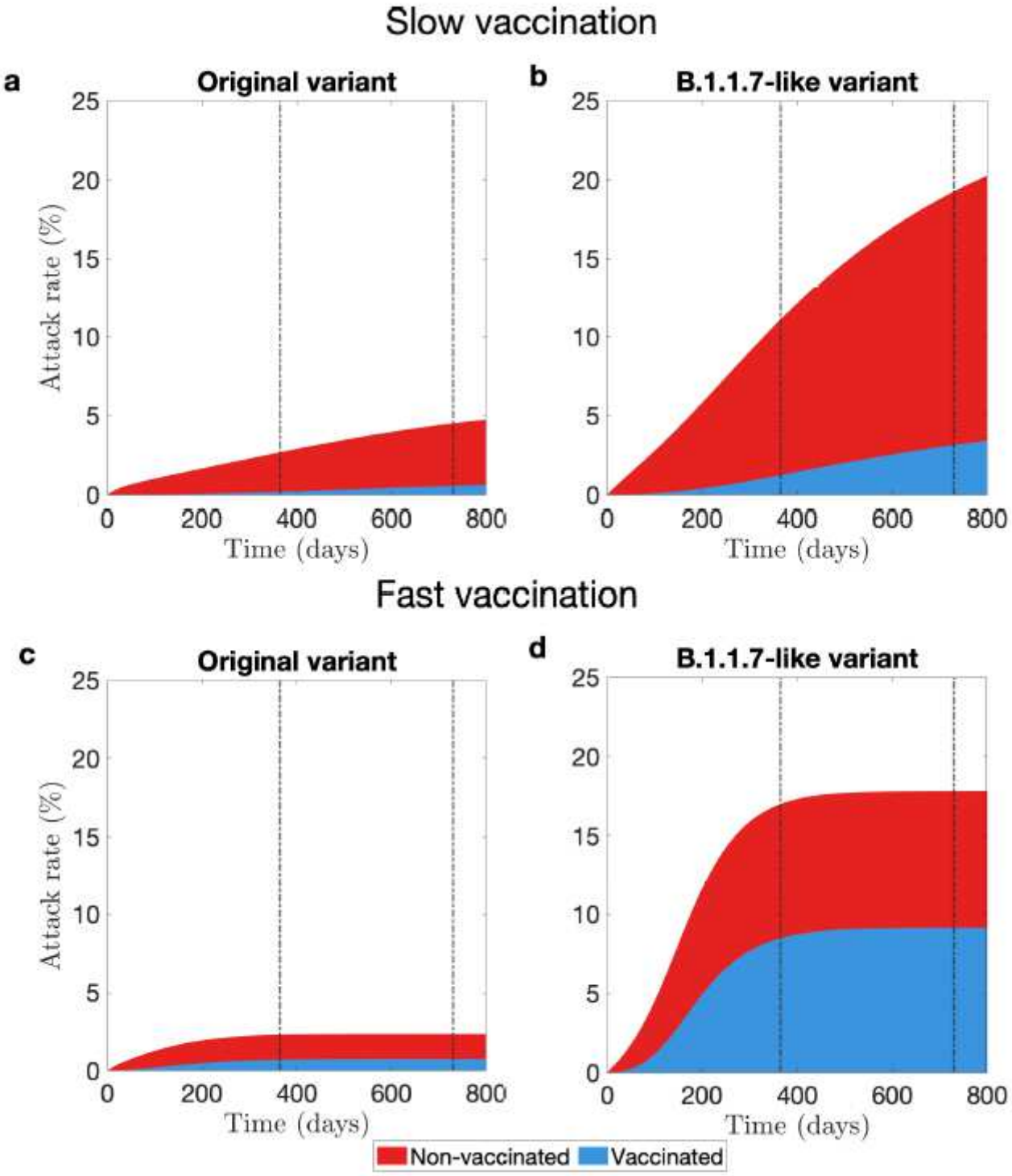

Figure 4

Contribution of vaccinated and non-vaccinated individuals to attack rate during the vacci- nation rollout. a and b show attack rates versus time given the slow vaccine uptake rate. c and d show attack rates versus time given the fast vaccine uptake rate. a and c show these quantities for the original variant, b and d for a B.1.1.7-like variant. Vertical brown lines mark one and two years time points since the start of the vaccination campaign. Attack rate is the proportion of the population that has been infected until a

given time. We adjusted the attack rate so that it describes only new infections that appeared during the time interval that we considered.

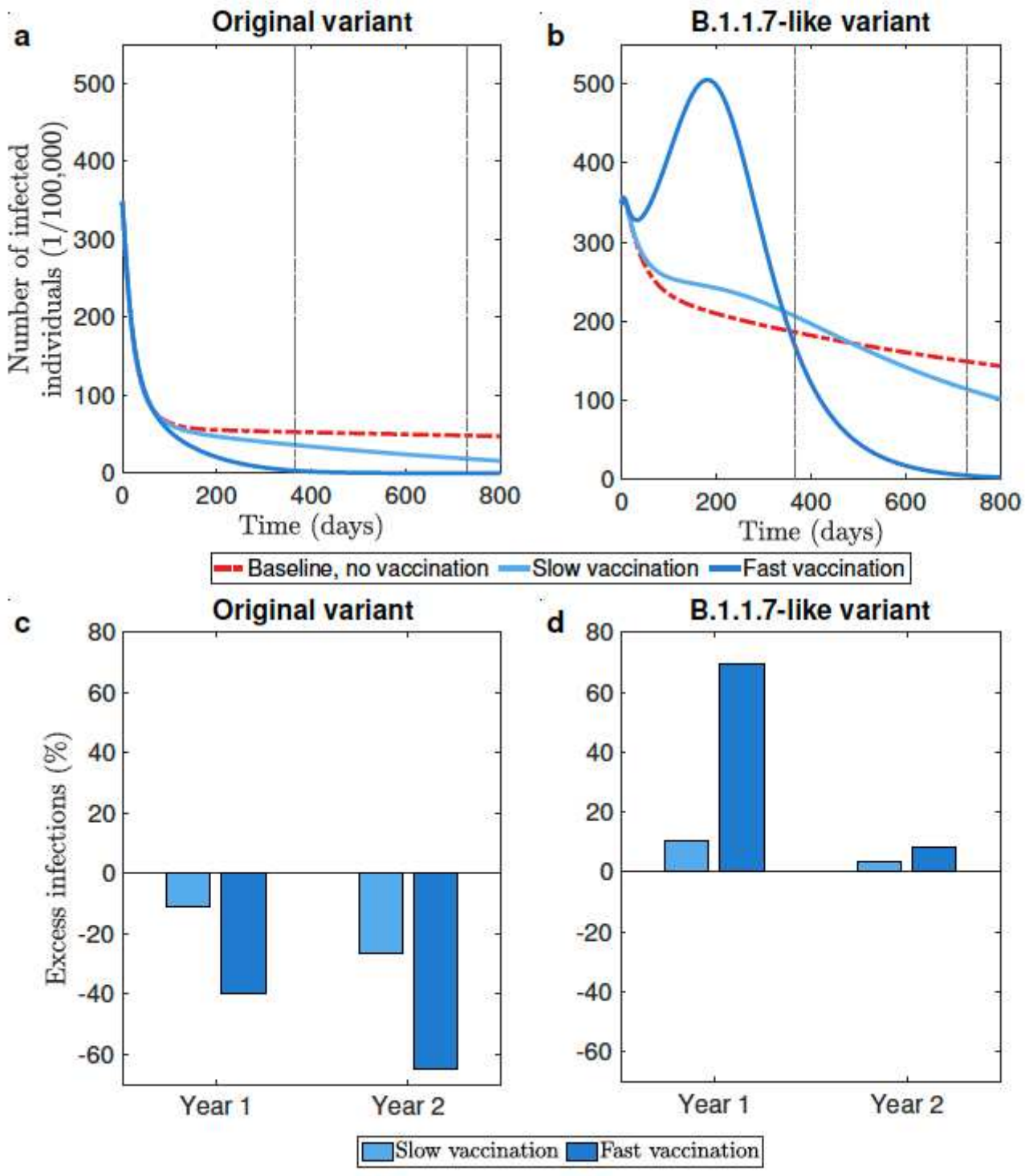

Figure 5

Epidemic dynamics when intervention targeting compliance of non-vaccinated individuals is deployed. a Prevalence of infected individuals versus time when the original variant circulates. b The same output when a B.1.1.7-like variant circulates. c Excess of cumulative infections relative to the no-vaccination

scenario level for the original variant. d The same output when a B.1.1.7-like variant circulates. c and d show excess of infections relative to the no-vaccination levels when respective variants circulate. In a and b, vertical brown lines mark one and two years since the start of vaccination.

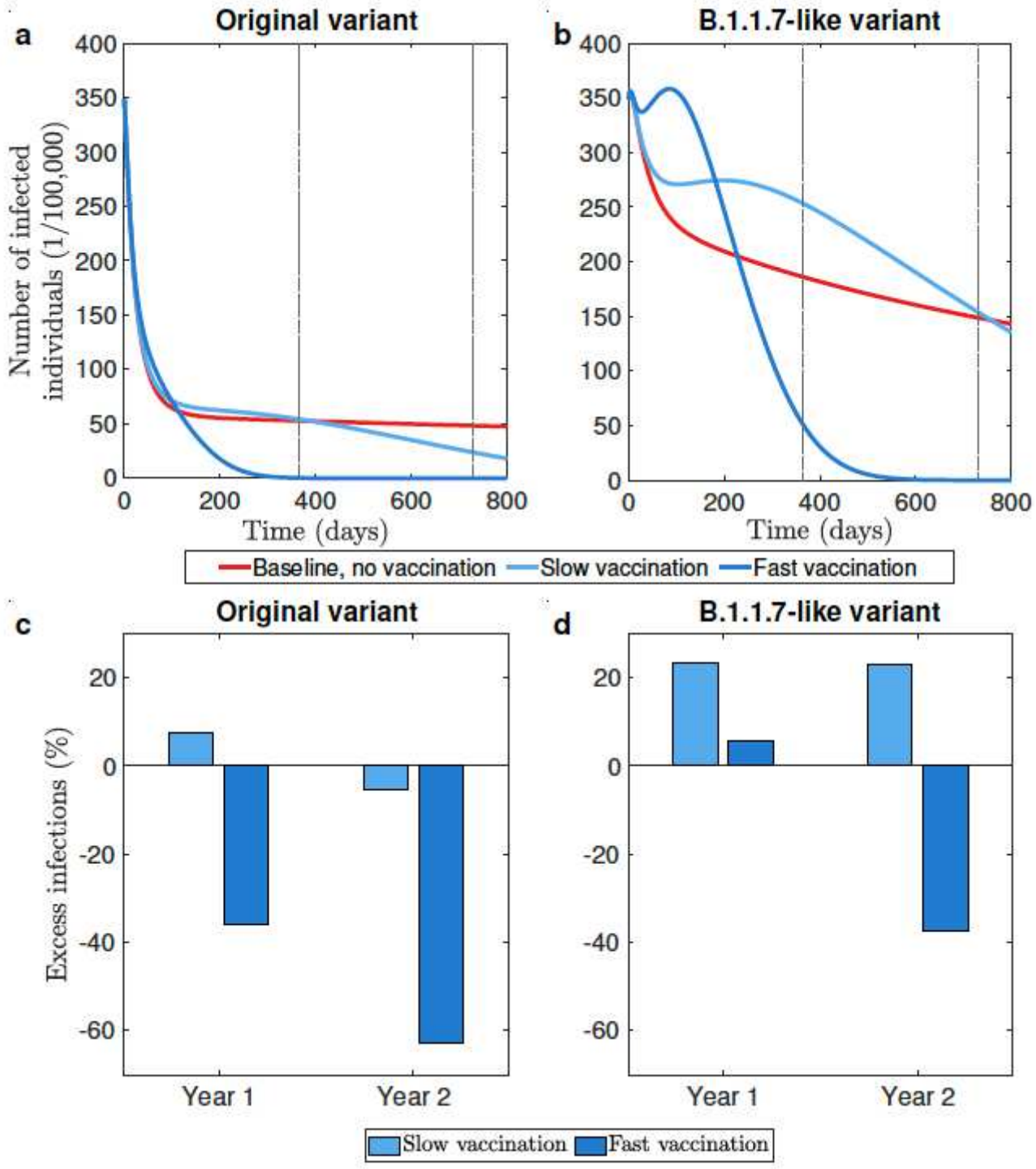

Figure 6

Epidemic dynamics when intervention targeting compliance of vaccinated individuals is deployed. a Prevalence of infected individuals versus time when the original variant circulates, b The same output when a B.1.1.7-like variant circulates, c Excess of cumulative infections relative to the no-vaccination

scenario level for the original variant, d The same output when a B.1.1.7-like variant circulates. c and d show excess of infections relative to the no-vaccination levels when respective variants circulate. In a and b, vertical brown lines mark one and two years since the start of vaccination.

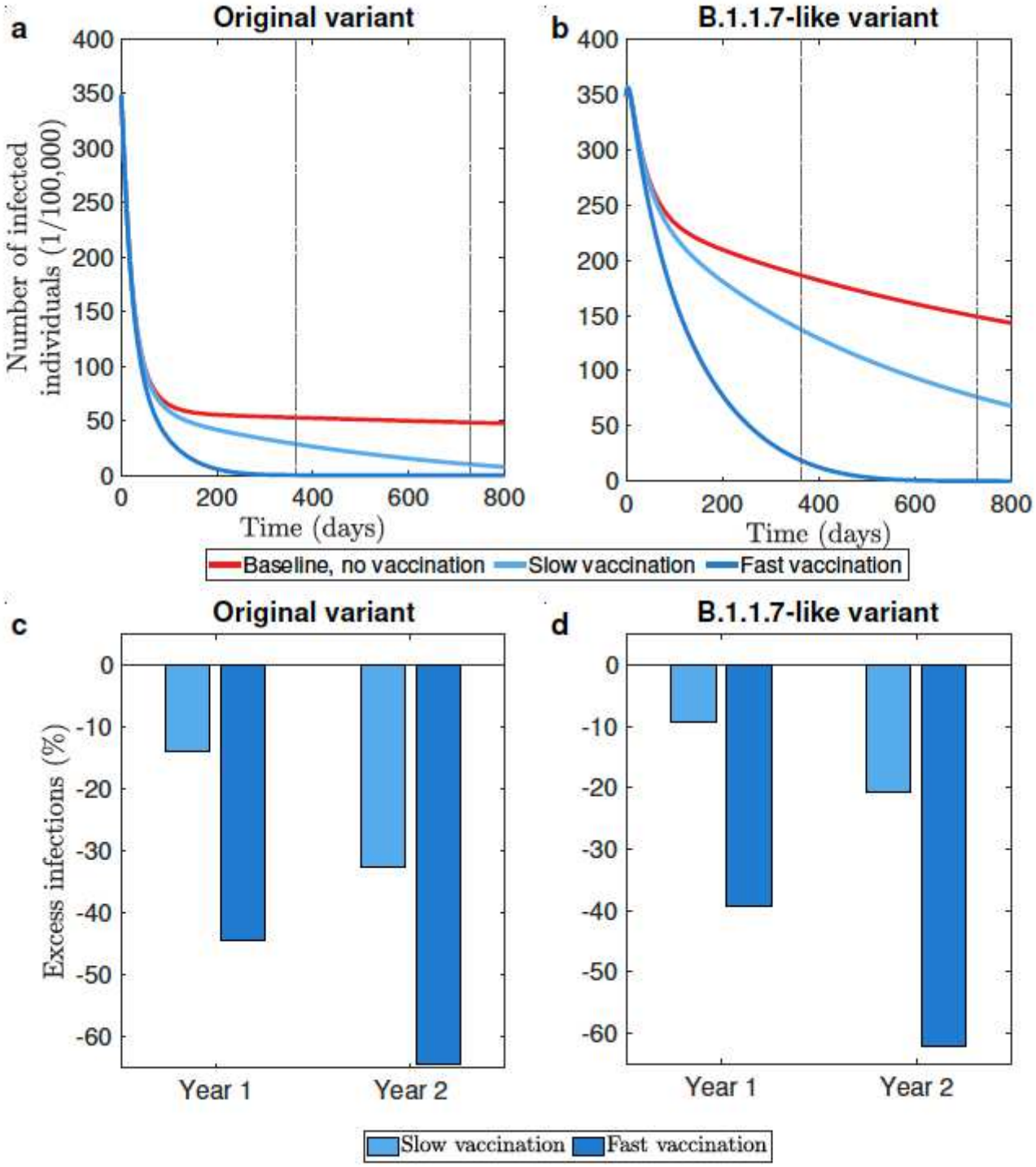

Figure 7

E□ects of the combination of interventions on the course of epidemic and excess infections. a Prevalence of infected individuals versus time when the original variant circulates, b The same output when a B.1.1.7-like variant circulates, c Excess of cumulative infections relative to the no-vaccination scenario

level for the original variant, d The same output when a B.1.1.7-like variant circulates. c and d show excess of infections relative to the no-vaccination levels when respective variants circulate. In a and b, vertical brown lines mark one and two years since the start of vaccination.

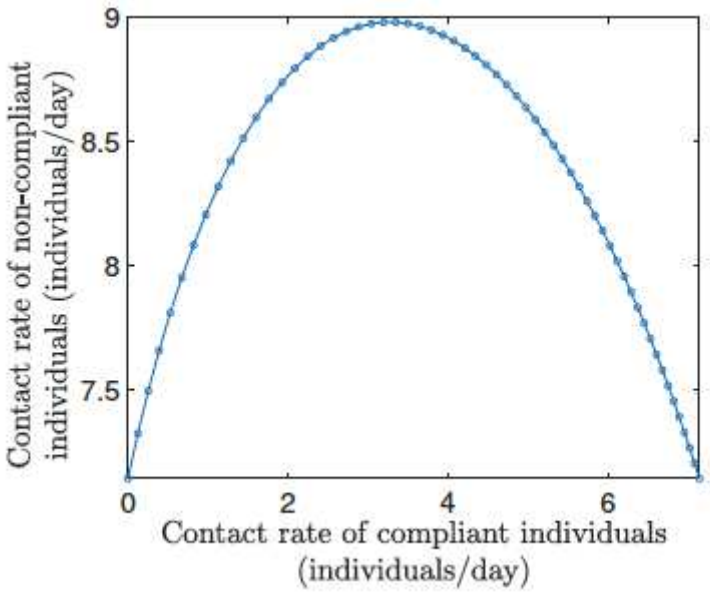

Figure 8

Figure 9: Pairs of contact rates of compliant and non-compliant individuals c and r1c such that effective reproduction number is equal to 1.1.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- SupplementaryMaterialSRAT2021.pdf