# A venue-specific model for assessing the local impact of the Covid Safe Ticket

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# 1. Model description

We developed an individual-based simulation model to investigate the impact of the Covid Safe Ticket (CST) in limiting SARS-CoV-2 transmissions during events, extending a previously developed simulation framework based on the notion of the effective contact process (Torneri et al., 2020, Torneri et al. 2022). Venues are represented assuming a closed and homogenous population of size n, in which contacts between attendees occur according to the assumption of random mixing. The infection can be transmitted when an infectious case has a contact with a susceptible individual, assuming that such transmission probability depends on the viral load progression of the infectious individual and on the vaccination status of both the susceptible and infectious individuals. In such a model, we approximate the basic reproduction number ( $\Re_0$ ) with the average number of effective contacts, i.e. contacts

that lead to disease transmission when occurring between susceptible and infectious individuals. We simulate events lasting a limited amount of time, i.e. less than 1 day. Consequently, we assume that only index cases can spread the infection since their secondary cases are in their latent phase throughout the entire duration of the event. Before the epidemic starts, we sample a proportion of the population that we set to be vaccinated. For the vaccinated individuals, we assume that the vaccine-induced immunity gives both protection against infection and reduces the infectiousness, and we inform such values using vaccine effectiveness data. Index cases are drawn uniformly among the population members, and we assume that their time of infection is uniformly distributed in a time interval preceding the start of the event. A detailed description of the simulation model is reported in the Appendix.

#### 1.1 Covid-Safe-Ticket strategies

To investigate the impact of the CST in limiting infections during specific events we implemented three strategies:

- 1. No CST. No CST is required to join the event.
- 2. *CST.* Vaccinated or recently recovered individuals can join the event. Everyone else needs to take an antigen test within 48 hours from the event, and only test negative individuals can join the event.
- 3. *CST-X*. All the attendees need to take an antigen test within 48 hours from the event, and only test negative individuals can join the event.

When simulating the CST and CST-X strategies, we assume that the time of testing is uniformly distributed on the time interval from 2 days before the start of the event, in line with previously adopted guidelines. We do not account for a delay from testing to getting the test result since we consider using antigen tests, which give results in a short amount of time. Furthermore, we assume that individuals can test positive only during the infectious period (Figure S1), with a constant sensitivity of 83%. Individuals who test positive are not participating in the event.

# 1.2 Input parameters and simulation scenarios

In Table 1, we report the parameters that are given as input to the simulator. We report the values that we consider in the simulation study together with the respective reference when available. Parameter values reported in bold correspond to the values selected in the baseline scenario, while the other values indicate the choice made for the sensitivity analyses that we ran. Each sensitivity analysis is run by varying only one parameter value among the values assumed in the baseline scenario.

Parameter name	Parameter meaning	Parameter value	Reference
n	size of the event	100; <b>1k;</b> 10k	Assumed
pSeeds	proportion of index cases	0.05; <b>0.1</b> ; 0.2	Assumed
lambda.e	Average number of contacts each individual makes during the event	10	Assumed
VE <sub>S</sub>	vaccine effectiveness against susceptibility to infection	0.15; <b>0.3</b> ; 0.69	Lyngse et al. (2022)
VE <sub>I</sub>	vaccine effectiveness against infectiousness	0.15; <b>0.3</b> ; 45	Lyngse et al. (2022)
$\Re_0$	basic reproduction number	3.3 (Wuhan) <b>, 5</b> (Delta VoC); 8 (Omicron VoC)	Liu et al. (2022); Du et al. (2022)
test.sen	test sensitivity	0.5; 0.7; <b>0.83</b>	Butler-Laporte

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Table 1. Input parameters and	parameter values	assumed in the	simulation study

			et al. (2021)
test.time.int	maximum number of days before the event starts within individuals need to take the test	2	Assumed - in line with previous guidelines
min.inftime	maximum number of days before the event starts in which an index case can be infected	3; <b>5</b> ; 10	Assumed
vacc.coverage	proportion of vaccinated or recently recovered individuals	0.2; 0.4; <b>0.8</b>	https://covid-v accinatie.be/n I
ρ <sub>e</sub>	effect of environment on the transmission probability given a contact	1	Assumed

## 1.3 Summary measures

To investigate the effectiveness of the CST among the different scenarios, we compute the following quantities:

- Secondary Cases: total number of infections taking place during the event (index cases are not counted).
- *Detected:* total number of index cases who test positive and are not allowed to join the event.

The summary measures above are either computed at a population level or upon differentiating between the vaccination status, i.e. vaccinated or not vaccinated. Results are presented by reporting the boxplot of such summary measures, the average point estimate with the 95% quantile interval and the relative differences of the average number of secondary cases for the different CST scenarios. For each scenario, we run 1000 simulations and we compute the summary measures in each simulation.

# 2. Simulation Results

## 2.1 Baseline

Parameters in the baseline scenarios are set to represent the spreading of the Delta VoC and the vaccine effectiveness against such strain for individuals vaccinated 7-8 months before the event starts. Concerning vaccination coverage, we set the value of the current vaccination coverage in Belgium (80%).

As shown in Figure 1, the number of secondary cases decreases when either the CST is required or when all the attendees take an antigen test (CST-X) compared to

a strategy in which no CST is needed (NoCST). A high decrease in the number of secondary cases (64%) occurs when comparing the CST-X strategy with the NoCST strategy (Table 2). In fact, index cases are more often vaccinated, due to the high vaccination coverage, and testing also such individuals lead to substantially decrease the number of infections taking place during events (Figure 2). Nevertheless, the use of CST still leads to a 13-15% reduction of secondary cases.



Fig. 1. Boxplots of the number of secondary cases generated by the index cases for events of size n=100,1k,10k, when no Cst is required, CST is required and an antigen test is required for all the attendees (CST-X). Boxplots are reported for the total number of cases (AR\_Tot), for unvaccinated individuals (AR\_Unv) and for vaccinated individuals (AR\_Vac).



Fig. 2. Boxplots of the proportion of index cases detected by the antigen test for events of size n=100,1k,10k, when no Cst is required, CST is required and an antigen test is required for all the attendees (CST-X). Boxplots are reported for the aggregated set of index cases (detected\_Tot), considering only unvaccinated index cases (detected\_Unv) and considering only vaccinated index cases (detected\_Vac).

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Size	Strategy	Mean AR_Tot and 95% quantile interval	Relative Difference (With NoCST)
	NoCST	2 (0;5)	-
N=100	CST	1.7 (0;5)	13 %
	CST-X	0.7 (0;3)	66%
	NoCST	19.3 (11;29)	-
N=1k	CST	16.6 (8;26)	14%
	CST-X	6.9 (2; 13)	64%
	NoCST	192.5 (164;223)	-
N=10k	CST	164.1 (137;191)	15%
	CST-X	69.8 (52; 88)	64%

Table 2. Mean number of secondary cases with 95% quantile interval and relative difference with the NoCST strategy for events of size n=100,1k,10k

#### 2.2 Proportion of Index Cases

We varied the proportion of index cases to represent different prevalence values at the population level. While the number of secondary cases increases when more index cases are present, the relative effectiveness of the CST and CST-X does not substantially vary (Figure 3 and Table 3).



Fig. 3. Boxplots of the number of secondary cases generated by the index cases for a proportion of index cases equals to pSeeds=0.05,0.1,0.2, when no Cst is required, CST is required and an antigen test is required for all the attendees (CST-X). Boxplots are reported for the total number of cases (AR\_Tot), for unvaccinated individuals (AR\_Unv) and for vaccinated individuals (AR\_Vac).

pSeeds	Strategy	Mean AR_Tot and 95% quantile interval	Relative Difference (With NoCST)
	NoCST	10.2 (4;17)	-
0.05	CST	8.8 (3;15)	14 %
	CST-X	3.5 (0;8)	64%
	NoCST	19.3 (11;29)	-
0.1	CST	16.6 (8;26)	14%
	CST-X	6.9 (2; 13)	64 %
	NoCST	33.6 (22;47)	-
0.2	CST	29 (19;41)	14%
	CST-X	12.8 (6; 21)	62%

Table 3. Mean number of secondary cases with 95% quantile interval and relative difference with the NoCST strategy when pSeeds=0.05,0.1,0.2

#### 2.3 Basic Reproduction Number

We tested the effect of an increase in the basic reproduction number, which can represent an increase in infectiousness of a more contagious VoC, e.g. Omicron, or an increase of the individual contact rate during the event, or an increase of transmissibility due to environmental characteristics. Figure 4 shows that an increase in the basic reproduction number corresponds to an increase in the number of secondary cases. The relative differences between CST strategies are similar for the tested values of the basic reproduction number (Table 4).



Fig. 4. Boxplots of the number of secondary cases generated by the index cases for a basic reproduction number of value R\_0=3.3,5,8, when no Cst is required, CST is required and an antigen test is required for all the attendees (CST-X). Boxplots are reported for the total number of cases (AR\_Tot), for unvaccinated individuals (AR\_Unv) and for vaccinated individuals (AR\_Vac).

R_0	Strategy	Mean AR_Tot and 95% quantile interval	Relative Difference (With NoCST)
	NoCST	12.7 (6;21)	-
3.3	CST	10.8 (4;18)	15%
	CST-X	4.5 (1; 9)	65 %
	NoCST	19.3 (11;29)	-
5	CST	16.6 (8;26)	14%
	CST-X	6.9 (2; 13)	64 %
	NoCST	30.7 (20;44)	-
8	CST	26.1 (16;38)	15%
	CST-X	11.2 (5; 19)	63%

Table 4. Mean number of secondary cases with 95% quantile interval and relative difference with the NoCST strategy when R\_0=3.3,5,8

#### 2.4 Vaccine effectiveness and vaccination coverage

Vaccinated individuals are shown to be less likely to acquire and to spread the infection (Lingsley et al. 2022). In the baseline scenario we selected vaccine effectiveness values for individuals who are vaccinated from 7-8 months. To challenge this assumption we selected a vaccine effectiveness with half value of the one chosen in the baseline, and the vaccine effectiveness reported in Lyngse et al. (2022) which estimates are not restricted to 7-8 months after vaccinations (VE\_s = 0.69; VE\_i = 0.45). Overall, the number of secondary cases decreases when the vaccine effectiveness is higher. Such a decrease is caused by an increased vaccine effectiveness which decreases the probability that individuals can acquire (Figure~5) or spread (Figure~6) the infection. The number of infection among unvaccinated is approximately the same when varying VE\_S since only the susceptibility of vaccinated cases varies, but we noticed a slight increase when varying VE\_I is low.



Fig. 5. Boxplots of the number of secondary cases generated by the index cases for a vaccine effectiveness against susceptibility to infection of value VE\_S=0.16,0.32,0.69, when no Cst is required, CST is required and an antigen test is required for all the attendees (CST-X). Boxplots are reported for the total number of cases (AR\_Tot), for unvaccinated individuals (AR\_Unv) and for vaccinated individuals (AR\_Vac).



Fig. 6. Boxplots of the number of secondary cases generated by the index cases for a vaccine effectiveness against susceptibility to infection of value VE\_I=0.16,0.32,0.45, when no Cst is required, CST is required and an antigen test is required for all the attendees (CST-X). Boxplots are reported for the total number of cases (AR\_Tot), for unvaccinated individuals (AR\_Unv) and for vaccinated individuals (AR\_Vac).

VE_S	Strategy	Mean AR_Tot and 95% quantile interval	Relative Difference (With NoCST)
	NoCST	22.3 (13;32)	-
0.15	CST	18.9 (10;29)	15%
	CST-X	8 (3; 15)	64 %
	NoCST	19.3 (11;29)	-
0.3	CST	16.6 (8;26)	14%
	CST-X	6.9 (2; 13)	64%
	NoCST	18.1 (10;27)	-
0.45	CST	15.5 (8;24)	15%
	CST-X	6.6 (2; 13)	64%

Table 5. Mean number of secondary cases with 95% quantile interval and relative difference with the NoCST strategy when VE\_S=0.16,0.32,0.69

Table 6. Mean number of secondary cases with 95% quantile interval and relative difference with the NoCST strategy when VE\_I=0.15,0.3,0.45

VE_I	Strategy	Mean AR_Tot and 95% quantile interval	Relative Difference (With NoCST)
	NoCST	22.3 (13;33)	-
0.15	CST	19.6 (11;30)	12%
	CST-X	7.9 (3; 15)	64 %
	NoCST	19.3 (11;29)	-
0.3	CST	16.6 (8;26)	14%
	CST-X	6.9 (2; 13)	64%
	NoCST	16 (8;25)	-
0.45	CST	13.2 (6;22)	18%
	CST-X	5.8 (1; 12)	64%

Vaccination coverage has a high impact on the CST strategy, since a higher proportion of index cases are unvaccinated and possibly detected when the coverage is lower. This results in a higher relative difference between the CST and the NoCST strategies. Interestingly, the NoCST and CST-X strategies lead to a higher number of secondary cases for a lower vaccination coverage, while the opposite trend is observed in the case of the CST strategy. When the coverage is low, there is a high proportion of unvaccinated individuals, and their susceptibility and infectiousness are not affected by the vaccine-induced immunity. Therefore, a higher number of secondary cases is computed in such a scenario compared to the baseline. Stated



differently, the CST targets only the unvaccinated population, being consequently more effective when the coverage is low.

Fig. 7. Boxplots of the number of secondary cases generated by the index cases for a vaccination coverage of value Vacc\_Cov=0.2,0.4,0.8, when no Cst is required, CST is required and an antigen test is required for all the attendees (CST-X). Boxplots are reported for the total number of cases (AR Tot), for unvaccinated individuals (AR\_Unv) and for vaccinated individuals (AR\_Vac).

Table 7. Mean number of secondary cases with 95% quantile interval and relative difference with the NoCST strategy when the vaccination coverage is

Coverage	Strategy	Mean AR_Tot and 95% quantile interval	Relative Difference (With NoCST)
	NoCST	25.6 (16;37)	-
0.2	CST	12.0 (5;20)	53%
	CST-X	9.0 (3; 16)	65 %
0.4	NoCST	23.7 (15;34)	-
	CST	14.0 (7;23)	41%
	CST-X	8.3 (3; 15)	65 %
0.8	NoCST	19.3 (11;29)	-
	CST	16.6 (8;26)	14%
	CST-X	6.9 (2; 13)	64 %

Vacc Cov=0.2,0.4,0.8

#### 2.4.1 Highly effective vaccination campaign

When assuming a highly effective vaccination campaign, vaccinated individuals are less likely to get infected or spread the infection. Consequently, unvaccinated individuals are the driver of the epidemic. When this is the case, the effectiveness of a CST strategy increases substantially reducing the amount of infections that take place during events. In Figure 8, we simulate the effect of the testing strategies for events with different vaccination coverages when the vaccine effectiveness is of value 0.9 both for the effectiveness against susceptibility to infection and against infectiousness.



Fig. 8. Boxplots of the number of secondary cases generated by the index cases for a vaccination coverage of value Vacc\_Cov=0.2,0.4,0.8, when no CST is required, CST is required and an antigen test is required for all the attendees (CST-X) and the vaccine effectiveness against susceptibility to infection and against infectiousness are both of value 0.9. Boxplots are reported for the total number of cases (AR\_Tot), for unvaccinated individuals (AR\_Unv) and for vaccinated individuals (AR\_Vac).

Table 8. Mean number of secondary cases with 95% quantile interval and relative difference with the NoCST strategy when the vaccination coverage is Vacc\_Cov=0.2,0.4,0.8 and the vaccine effectiveness against susceptibility to infection and against infectiousness are both of value 0.9.

Coverage	Strategy	Mean AR_Tot and 95% quantile interval	Relative Difference (With NoCST)
	NoCST	19.3 (11;29)	-
0.2	CST	7.0 (2;13)	64%
	CST-X	6.7 (2; 13)	65 %
0.4	NoCST	11.9 (5;19)	-
	CST	4.6 (1;10)	61%
	CST-X	4.0 (1; 9)	66 %
0.8	NoCST	2.4 (11;29)	-
	CST	1.4 (0;4)	43%
	CST-X	1.4 (0; 4)	43 %

## 2.5 Test sensitivity

In Figures 9 and 10 we assume different values for the test sensitivity and we simulate epidemics during events characterized by a high vaccination coverage (i.e., 0.8 Figure 9) and low vaccination coverage (i.e., 0.2 Figure 10). We noticed that an increase in the test sensitivity corresponds to a decrease in the number of secondary cases. For the CST strategy this effect is more pronounced when the vaccination coverage is lower (Figure 10 and Table 10) since a higher proportion of index cases will be unvaccinated and potentially detected.



Fig. 9. Boxplots of the number of secondary cases generated by the index cases when the test sensitivity varies among 0.5,0.7,0.83, the vaccination coverage is 0.8 and when no Cst is required, CST is required and an antigen test is required for all the attendees (CST-X). Boxplots are reported for the total number of cases (AR\_Tot), for unvaccinated individuals (AR\_Unv) and for vaccinated individuals (AR\_Vac).



Fig. 10. Boxplots of the number of secondary cases generated by the index cases when the test sensitivity varies among 0.5,0.7,0.83, the vaccination coverage is 0.2 and when no Cst is required, CST is required and an antigen test is required for all the attendees (CST-X). Boxplots are reported for the total number of cases (AR\_Tot), for unvaccinated individuals (AR\_Unv) and for vaccinated individuals (AR\_Vac).

Table 9. Mean number of secondary cases with 95% quantile interval and relative difference with the NoCST strategy when the vaccination coverage is 0.8 and the test sensitivity varies among 0.5;0.7;0.83.

Test sensitivity	Strategy	Mean AR_Tot and 95% quantile interval	Relative Difference (With NoCST)
	NoCST	19.3 (11;29)	-
0.5	CST	17.7 (10;27)	8%
	CST-X	11.8 (5; 20)	39 %
0.7	NoCST	19.3 (11;29)	-
	CST	17.0 (8;27)	12%
	CST-X	8.9 (3;16)	54 %
	NoCST	19.3 (11;29)	-
0.83	CST	16.6 (8;26)	14%
	CST-X	6.9 (2; 13)	64 %

Table 10. Mean number of secondary cases with 95% quantile interval and relative difference with the NoCST strategy when the vaccination coverage is 0.2 and the test sensitivity varies among 0.5;0.7;0.83.

Test Sensitivity	Strategy	Mean AR_Tot and 95% quantile interval	Relative Difference (With NoCST)
	NoCST	25.6 (16;37)	-
0.5	CST	17.5 (9;28)	32%
	CST-X	15.8 (8; 25)	38 %
0.7	NoCST	25.6 (16;37)	-
	CST	14.0 (6;23)	45%
	CST-X	11.7 (5; 20)	54%
0.83	NoCST	25.6 (16;37)	-
	CST	12.0 (5;20)	53%
	CST-X	9.0 (3; 16)	65%

# 2.6 Time of infection index cases

We challenged the assumption on the time of infection for the index cases assuming that index cases could have contracted the infection in a time interval of length 3, 5 or 10 days before the event starts. The number of generated secondary cases and the effectiveness of the CST strategy depend on this assumption (Figure 8). More

precisely, the viral load value of the index cases at the starting of the event depends on when they got infected. If we assume that index cases can contract infection uniformly in a time interval from 0 to ten days from infection, the average infection time will be 5 days before the event. If this is the case, index cases attending are at the peak of their infectiousness, causing possibly a high number of secondary cases.



Fig. 11. Boxplots of the number of secondary cases generated by the index cases when the time of infection for the index cases can occur maximum 3, 5 and 10 days before the starts of the event and when no Cst is required, CST is required and an antigen test is required for all the attendees (CST-X). Boxplots are reported for the total number of cases (AR\_Tot), for unvaccinated individuals (AR\_Unv) and for vaccinated individuals (AR\_Vac).

Table 11. Mean number of secondary cases with 95% quantile interval and relative difference with the NoCST strategy when the time of infection for the index case can occur maximum 3, 5 and 10 days before the event starts.

MinIT	Strategy	Mean AR_Tot and 95% quantile interval	Relative Difference (With NoCST)
3	NoCST	6.5 (2;13)	-
	CST	6.1 (2;11)	6%
	CST-X	4.9 (1; 9)	25 %
5	NoCST	19.3 (11;29)	-
	CST	16.6 (8;26)	14%
	CST-X	6.9 (2; 13)	64 %
10	NoCST	22.5 (13;33)	-
	CST	18.4 (10;28)	18%
	CST-X	6 (1; 12)	73%

#### 2.7 Overdispersion in contact rate

We tested the impact of overdispersion on the contact rate of index cases, assuming that the contact rate follows a Negative Binomial distribution. Results indicate that the impact of different CST strategies do not vary when different overdispersion parameters are selected (Figure 12). Similar averages are computed among the baseline scenario, i.e. no overdispersion, and the scenarios where overdispersion is considered (Table 12).



Fig. 12. Boxplots of the number of secondary cases generated by the index cases when the contact rate is constant (Baseline) or distributed according to a Negative Binomial distribution (Shape=5, Shape=1 - shape indicates the shape of the Gamma mixture), when no Cst is required, CST is required and an antigen test is required for all the attendees (CST-X). A decrease of the shape parameter corresponds to a higher overdispersion. Boxplots are reported for the total number of cases (AR\_Tot), for unvaccinated individuals (AR\_Unv) and for vaccinated individuals (AR\_Vac).

Table 12. Mean number of secondary cases with 95% quantile interval and relative difference with the NoCST strategy when the time of infection for the index case can occur maximum 3, 5 and 10 days before the event starts.

Shape	Strategy	Mean AR_Tot and 95% quantile interval	Relative Difference (With NoCST)
	NoCST	19.3 (11;29)	-
Baseline	CST	16.6 (8;26)	14%
(No overdispersion)	CST-X	6.9 (2;13)	64 %
	NoCST	19.3 (10;31)	-
5	CST	16.7 (9;26)	14%
	CST-X	6.8 (2; 13)	65 %
	NoCST	19.3 (10;31)	-
1	CST	16.6 (8;27)	14%
	CST-X	7,1 (2; 15)	63%

# 3. Conclusion and Limitations

The simulation study that we performed shows the possible effect of a CST strategy in limiting the number of infections that can occur during an event. While some characteristics such as the event size, the basic reproduction number, and the proportion of index cases affect the total number of cases, other quantities such as the time of infection and the vaccine coverage affect the relative effectiveness of such strategies. In the baseline scenario characterized by high coverage and low vaccine effectiveness, the use of CST decreases with 13-15% the number of infections that might take place during an event. However, for events with a low vaccination coverage, i.e., 20%, the effectiveness of a CST strategy substantially increases (relative difference with No CST is 54%) since a higher proportion of index cases will be unvaccinated and therefore a target of the CST strategy. Thus, properly assessing the characteristics of participants would be valuable information to understand for which event the use of CST can be effective.

Furthermore, the effectiveness of the CST increases when the vaccine effectiveness is high since the infections that take place are most likely to happen between an unvaccinated-unvaccinated pair of individuals.

In this work we simulated infections that take place during isolated events at a specific time during the epidemic/pandemic. To compute the number of infections, and/or hospitalizations, at the population level it is necessary to embed this model in another framework that accounts also for waning of immunity and for human-to-human interactions taking place outside such events.

Several assumptions have been made in the present simulation study. Hereunder, we briefly discussed some of the limitations arising from the modeling choice made.

We assumed a homogeneous population, therefore no individual characteristics (e.g., age) are considered. This assumption is likely to affect the number of secondary cases and might affect the effectiveness of CST measures when different viral load profiles, vaccine effectiveness or coverage values are heterogeneously defined among the attendees.

We do not distinguish between asymptomatic and symptomatic carriers. This assumption affects the infection dynamic in two ways:

- a) asymptomatic individuals are often considered to be less infectious than symptomatic ones. Therefore, the overall reproduction number when asymptomatic carriers are considered is lower than in case all the infections are symptomatic.
- b) symptomatic and vaccinated individuals showing symptoms before the event starts could decide not to attend

Limitation a) can easily be overcome by varying the value of the basic reproduction number. To account for b), the code needs to be extended and a new parameter accounting for compliance given time since infection needs to be included. To date, there is a lack of information in the literature about such a quantity.

Nevertheless, we assume that the time of infection of the index cases is on average 2.5 days before the event starts. Such a value is lower than the average incubation period of Omicron and Delta. Therefore we are on average accounting only for index cases who will attend the event prior to symptom onset. In addition, infections post vaccinations are argued to be more likely asymptomatic (Tang et al. 2021).

We did not account for characteristics of the environment, e.g., ventilation. However, this can be represented by varying the value of the basic reproduction number.

Even though we tested different values of susceptibility to infection, we assumed that susceptibility is driven exclusively by vaccination. This choice was made because the population is assumed to be homogeneous and it is still not completely clear what drives susceptibility to infection. In our setting, higher or lower susceptibility values will merely lead to higher or lower basic reproduction number values. Furthermore, we set as a baseline scenario vaccine effectiveness values for the Delta VoC after two vaccine doses since both vaccine effectiveness against susceptibility and vaccine effectiveness against infectiousness are available in literature in such a case (Lyngse et al. 2022). However, we challenged this assumption by varying the vaccine effectiveness value and we noticed a remarkable difference only when vaccine effectiveness is very high.

We informed the intrinsic generation time of vaccinated and unvaccinated individuals using viral load data for young and healthy men infected with the Delta variant (Kissler et al. 2021), and we do not assume individual variation. This assumption might affect the effectiveness of the CST strategies, especially when attendees with different characteristics are attending the event. In addition, even though viral load profiles between Delta and other VOCs are comparable, precision would increase if viral data would be available for the current VoC (distinguishing between vaccinated and unvaccinated). When new viral load progression

data for other cohorts or other VOCs are available, the simulation model can easily be adapted to represent transmission dynamics in specific events.

We assumed that the sensitivity of the antigen test is constant over the infectious period and it is the same for both vaccinated and unvaccinated infected individuals. In addition, sensitivity might depend on the circulating VoC (Chu et al. 2022). A different sensitivity affects the effectiveness of the CST strategies, since less or more index cases can be detected. In addition, recent findings suggest that antigen test performance may differ in vaccinated vs unvaccinated individuals (Chu et al. 2022), possibly affecting the performance of the CST-X strategy.

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# S1. Appendix

### S1.1 Transmission probability and infection risk

We assume that the infection can be transmitted when an infected person has contact with a susceptible one. For each index case, we simulate the contacts this person has at the event, assuming the contact process of Poisson type. Each contact results in an infection event according to a time-varying probability value that, if assuming i to be an infectious individual and j a susceptible one, is given by:

$$p_{i,j}^{e}(t) = q_{i}^{e} \times v_{i}(t) \times \sigma_{j}$$

Here, *t* is the time since infection of individual *i*,  $q_i^e$  is the transmission potential of individual *i* in the specific environment *e*,  $v_i(t)$  is the intrinsic generation time density function of *i*, and  $\sigma_j$  is the susceptibility of individual j.

We describe susceptibility to infection using the vaccine effectiveness against infection, i.e.,

$$\sigma_i = (1 - VE_s)$$

By doing so, we assume that susceptibility to infection depends only on the vaccination status.

The transmission potential is set to depend on the vaccination status of individual *i* since this aspect has been argued to affect transmissions (Lyngse et al. 2022), and on characteristics of the environment (e.g., ventilation).

Precisely, we define

$$q_i^e = \frac{\Re_0}{\lambda} \times (1 - VE_I) \times \rho_e$$

indicating with  $\Re_0$  the basic reproduction number,  $\lambda$  the average daily contact rate,  $0 < VE_I < 1$  is the vaccine effectiveness against infectiousness, and  $\rho_e$  the effect of

the specific environment on the infectiousness. While  $\Re_0$ ,  $\lambda$  and  $VE_I$  can be set according to published scientific works (e.g., Liu et al. 2022, Mossong et al. 2008, Lyngse et al. 2022), it is currently not clear how the environment and vaccination affect droplet transmissions. To avoid complexity, we set this value to 1 and we represent the environmental effect by testing different values of the reproduction number. As soon as more precise indications on the effect of the environment on transmission are reported in literature, we can easily include such a component. The intrinsic generation time density function is set according to the viral load dynamic observed during the infectious period. We use data reported in Kissler et al. (2021), where they estimated the viral dynamics of SARS-CoV-2 in vaccinated and unvaccinated individuals. We normalized their mean estimates such that the area underneath the curves is equal to one, resulting in probability density functions. Furthermore, we assumed that for both vaccinated and unvaccinated cases the viral load peaks at 5.2 days after infection. The implemented curves are reported below.



Figure.S1 Intrinsic generation time density function for individuals who are vaccinated (orange continuous curve) and unvaccinated (green continuous curve).

Following this approach, the infectious period is modeled to start after 2 and 1.7 days from infection, and to end after 10.7 and 12.7 days from infection if the infected individuals are, respectively, vaccinated and unvaccinated. The start and end of the infectious period correspond to the time at which 40 or fewer Ct counts detected viral presence. In our implementation, we assume that all the individuals with the same vaccination status have the same viral progression.