A Markovian Model for the Spread of the SARS-CoV-2 Virus ⋆

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Abstract

We propose a Markovian stochastic approach to model the spread of a SARS-CoV-2-like infection within a closed group of humans. The model takes the form of a Partially Observable Markov Decision Process (POMDP), whose states are given by the number of subjects in different health conditions. The model also exposes the different parameters that have an impact on the spread of the disease and the various decision variables that can be used to control it (e.g, social distancing, number of tests administered to single out infected subjects). The model describes the stochastic phenomena that underlie the spread of the epidemic and captures, in the form of deterministic parameters, some fundamental limitations in the availability of resources (hospital beds and test swabs). The model lends itself to different uses. For a given control policy, it is possible to *verify* if it satisfies an analytical property on the stochastic evolution of the state (e.g., to compute probability that the hospital beds will reach a fill level, or that a specified percentage of the population will die). If the control policy is not given, it is possible to apply POMDP techniques to identify an optimal control policy that fulfils some specified probabilistic goals. Whilst the paper primarily aims at the model description, we show with numeric examples some of its potential applications.

Key words: Modelling and Control of Biomedical Systems, Healthcare Management, Stochastic Systems, SARS-CoV-2, Virus

1 Introduction

The catastrophic outbreak of the COVID-19 pandemic caused by the SARS-CoV-2 virus has recently brought to the attention of academia, decisions makers, and common people the importance of reliable models to predict and control the evolution of the disease. Far from being an academic exercise, such models are key to define policies that reduce the risk of spreading the virus, minimising the impact on the economy and on the quality of social life. To be up to the task, a model has to meet a few requirements. First, it shall capture the multi-faceted nature of human relations. Most of human interactions take place within a limited circle of people (family members, colleagues, friends), and yet our lives are punctuated by random encounters and accidental events. A realistic model has to account both for the regularity and for the elements of randomness of human relations. Second, the level of details shall be sufficient to expose the impact of all the different parameters affecting the spread of the virus (e.g., use of masks, number of intensive care beds), and of the commands (e.g., social distance, number of tests administered) used to control its evolution. Finally, the model shall be analytically tractable for the synthesis of control policies and the evaluation of various scenarios.

Paper Contribution. In this paper, we propose a stochastic model for the evolution of a COVID-19-like epidemic. The model describes the evolution of a population of subjects, each one allowed to be in eight different states. Such parameters, as the probability of contracting the infection during a single meeting with asymptomatic subjects (which is directly connected to the use of protective masks) or the number of available beds in intensive care facilities, are first class citizens in the model. Besides, the model exposes two of the decision variables usable to control the evolution of the disease: social interaction limits and number of tests. Since part of the states are directly observable and part are not, the model takes the form of a partially observable Markov Decision Problem (POMDP). Our model is control–oriented, in that it exposes the impact of the command decisions on the transition probabilities, and hence on the evolution of the epidemic. Its main areas of applications are two-fold. First, given a control policy (e.g., a decision to apply lock-down depending on the estimated number of people affected by the disease), it is possible to exactly compute the probabilities and properties of interest, ranging from the probability that the deceased individuals will be beyond an acceptable

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threshold, to more general properties expressed in propositional temporal logic [8]. Second, it is possible to synthesise control policies where such properties are respected by construction [1]. Both goals can be fulfilled by using PRISM, STORM or similar tools.
Such methods are based on the study of the analytical evolution of the POMDP, therefore they are guaranteed to provide probabilistically correct results, insofar as the model is sufficiently close to the reality. Our primary effort in this paper has been toward modelling with a sufficient level of details such effects as the transition between the different states and the resource limitations (e.g., hospital beds or daily availability of tests). A potential price to pay for a detailed model is its level of mathematical tractability.
Indeed, our model remains tractable for groups having a population in the range of a few tens of people, which is the typical size of a circle of people that have daily interactions. The study of larger populations using the presented model is possible in different ways. A first possibility, discussed in the related work section, is by exploiting some recent results on scalable techniques for MDP analysis and design [25, 17]. Another possibility is by using a small size population model to develop a policy with guaranteed properties, and then recover the result on a heuristic basis for larger populations verifying its efficacy through Monte Carlo simulations, as shown in this paper.

Related work. The first pioneering work on mathematical epidemiology dates back to Bernoulli [5] with his study of smallpox models. However, most of the theory was developed only in the last century with the introduction of compartmental models [21]. The idea of a compartmental model is to break down a population into several compartments. The simplest is the Susceptible-Infected-Recovered (SIR) model, used under the assumption that a recovered subject is not infected again (in the short-term), while the Susceptible-Infected-Susceptible (SIS) model is used when a recovered subject can be infected again. The migration of subjects between the different compartments is ruled by a set of Differential Algebraic Equations (DAE), that is a set of Ordinary Differential Equations (ODEs) connected by an algebraic relation (the sum of the cardinality of the compartments yields the population). The outbreak of the SARS epidemic in 2002-2003 sparked a new interest in these models [2, 7], with the main goal of the researchers being a strategy to keep in check the evolution of the epidemic leveraging the control variables. The use of deterministic models paves the way for a number of interesting control theoretical results such as models with many compartments [16], flow control [15, 6] and stability [22], policies for COVID-19 based on Optimal Control (OC) [32]. These models have an important role in the identification of key epidemiological features that indicate the progress of the disease, such as the basic reproduction number. However, they are only a rough approximation of the reality because random factors naturally influence the evolution of an epidemic [3]. While deterministic models represent the mean disease dynamics, a much richer set of information can be derived from stochastic models. As an example, stochastic models may converge to a disease-free state even if the corresponding deterministic models converge to an endemic equilibrium [3]. In addition, stochastic models produce a variety of important information, such as the probability of an outbreak, the distribution of the final size of a population or the expected duration of an epidemic [7, 29]. Extensions of these models have been developed to account for stochastic fluctuations in some of the parameters [19, 24], but even so, these models do not entirely capture the natural randomness inherent in the very way humans interact.
The SARS-CoV-2/COVID-19 pandemic has given another boost to the research in the area, highlighting the importance of statistical techniques to elaborate the massive amount of data daily collected in most of the countries affected by the pandemics, in order to support the decision making process and the definition of long-term policies. A notable example is the work of Zardini et al. [33], where the authors apply statistical methods to quantify the probability of transition between different state of COVID-19-affected patients based on the age class. Another interesting example is a statistical study to evaluate the effects of lock-down policies in Italy after the outbreak of the pandemic [27]. Although these methods use compartmental models, they cannot directly be used for control design or to verify closed–loop properties.
An important class of models amenable to analytical analysis are the ones that respect the so-called Markov property, which we generally define Markov Processes [9, 2]. When we observe the system in discrete–time, Markov Models are called discrete-time Markov chains (DTMC). In continuous–time, we talk about continuous–time Markov chain (CTMC). Contrary to other stochastic models such as Stochastic Differential Equations (SDE), Markov models (DTMC and CTMC) adopt a numerable state space composed of discrete variables. When command variables become part of the model, Markov chains morph into Markov Decision Processes (MDP). Since we observe the system at periodic intervals, the modelling technique adopted in this paper is based on discrete–time MDPs and, since not all states are directly observable (e.g., asymptomatic persons), we have a partially observable MDP (POMDP).
There are, historically, two paradigms for modelling a disease spread as a DTMC, the Reed-Frost model and the Greenwood model. They are called chain-binomial models because the transition probabilities are governed by binomial random variables. In these models, infectious (I) are indexed in generations capable of infecting susceptibles (S) for one generation after which they are not involved in the process. If the population has constant size N, then the initial condition is $S_0 + I_0 = N$ and $S_{t+1} + I_{t+1} = S_t$, for times $t = 0, 1, \ldots$. Hence $S_t + \sum_{\tau=0}^{t} I_\tau = N$. The number of infectious at time $t+1$ is given by a binomial random variable of parameters S_t and $p(I_t)$ (the probability that a susceptible becomes infected if the infectious are I_t). The corresponding transition probability is given by the k-th component of the Binomial probability mass function (pmf):

$$\begin{aligned} &\Pr\{I_{t+1} = k \,|\, S_t = x, I_t = y\} = \\ &\quad \mathcal{B}(x, p(y))_k = \tbinom{x}{k} p(y)^k (1 - p(y))^{x-k}, \end{aligned} \tag{1}$$

which is trivially zero if $k > x$. A difference between the Greenwood and the Reed-Frost models is that in the former $p(y) \equiv p$ is constant, while in the latter we have that $p(y) = 1 - (1 - p(1))^y$, where $p(1)$ is the probability that a susceptible is infected by one infective. Extensions of this model were presented by [14, 30]. As discussed in Section 3, the model adopted in this paper can be seen as a generalisation of the Reed-Frost model.

The use of stochastic models opens for the possibility to use Stochastic Model Checking in order to study probabilistic temporal properties to evaluate the effects of a strategy on a population during the evolution of a disease. As an example, Razzaq and Ahmad [26] adapted a Susceptible-Exposed-Infectious-Recovered-Delayed-Quarantined (Susceptible/Recovered) CTMC model used to analyse the spread of internet worms using the PRISM model checker [23]. The same tool was used to validate a model and to compute the minimum number of influenza hemagglutinin trimmers required for fusion to be between one and eight [11]. Finally, Chauhan [10] uses a stochastic simulation to compute timing parameters for a timed automaton, which is then verified against some temporal properties.
All the stochastic models mentioned are abstract and simplified. For instance, the use of three states (Susceptible, Infectious, Recovered) certainly makes the model tractable, but does not provide enough details on the parameters and the command variables influencing the evolution of the disease. Another simplification frequently adopted to make the model tractable by model checkers is to enforce that only one subject can change her/his state across one transition (which is unrealistic), or to avoid the possibility of defining command variables. Only recently have more sophisticated models emerged. An interesting example is the work of Viet et al. [31], in which the authors use an MDP to model the spread of the Porcine Reproductive and Respiratory Syndrome (PPRS) and use it to synthesise a regional policy for its containment. A similar attempt has been made also for the SARS-CoV-2 by Nasir et al. [25]. The authors consider a segmented model for the population and model its evolution via a MDP with the purpose of synthesising an optimal policy for vaccination, hospitalisation and quarantine. The model is largely simplified and the paper omits any discussion on the computation of the transition probability as a function of the policy, which is essential for the definition of the MDP. Our work lies in this line of research. As in [25], we show how to construct a MDP describing the SARS-CoV-2 epidemics using a segmented model for the population, but our model is very detailed and contains an exhaustive discussion on how to compute the transition probabilities and on how to use the model for control design and analysis. Very interestingly, Nasir et al. [25] discuss in alarming terms the problem of scalability of epidemic models based on MDPs. The technique that they suggest to deal with large populations is by abstraction, i.e., by considering a unit of population in the model as the representative of a number of people. This is essentially equivalent to a process of quantisation in which the accumulated error can be controlled and shown to be tolerable, for practical purposes. Clearly, the same line of reasoning potentially applies to the model presented in this paper.
Another interesting technique to address the problem of scalability has been recently suggested by Haksar and Schwager [17] and is based on Graph Based MDPs. The authors consider a network of similar MDPs (i.e., MDP governed by the same update rules) that interact with their neighbors following a graph topology. By exploiting symmetry and the graph topologies, the authors show that the design of a sub-optimal policy for the MDPs can be achieved by approximate linear programming. This technique could be applied in our context considering the model proposed in this paper as the nodes of the graph.

Paper Organisation The paper is organised as follows. After introducing notation and definitions in Section 2, we describe the proposed stochastic model in Section 3, with the transition probabilities and computational remarks on the complexity of the model. The model checking techniques for the problem at hand and some numeric results to illustrate the potential application of the proposed model are reported in Section 4. Finally, Section 5 presents the discussion about the model and describes the lines of future work. In Appendix A, related to the computational complexity, we propose also a simplified model that may be used for computational efficiency.

2 Definitions and Notations

We model a population as a stochastic discrete–time system with a finite state. The evolution is observed at discrete time k and each subject can belong to one of eight possible states. We take into account the usual classes of susceptible, infected, recovered (SIR), the asymptomatic subjects A (i.e., a group of infected people that do not exhibit symptoms but are infective), hospitalised O and dead D people and we further add the group of people that recover from an asymptomatic state Ra and the case of swab-tested people, that are quarantined (Q) if they result positive. The subjects who are in a state at step k will be denoted by a calligraphic letter (e.g., $\mathcal{S}_k$ is the set of susceptible subjects). The table in Figure 1 reports the symbols used to denote the different sets, their cardinality (e.g., S_k is the cardinality of $\mathcal{S}_k$) and the different probabilities governing the transition of a subject between the different sets. Notice that, in order for the systems to be consistently defined, the following constraints shall hold on the probabilities: $C_{\beta,\delta} = (1 - \beta - \delta) \geq 0$, $C_{\mu,\psi,\alpha} = (1 - \mu - \psi - \alpha) \geq 0$, $C_{\sigma,\xi} = (1 - \sigma - \xi) \geq 0$, and $C_{\iota,\upsilon} = (1 - \iota - \upsilon) \geq 0$. These constraints will be used in the rest of the paper to prove the theorems. In our model two deterministic variables M and t are used as control variables, which represent respectively the number of meetings allowed in each period and the number of tests administered. In addition, two deterministic parameters (N, C) are demographic parameters related to the size of the population and to the availability of hospital beds. The other parameters are probabilities. For instance ω represents the probability for a subject to contract the infection in one meeting. Such prob-

Sets

$S_k = \lvert\mathcal{S}_k\rvert$	N. of susceptible sub. $\mathcal{S}_k$ at step k,
$A_k = \lvert\mathcal{A}_k\rvert$	N. of asymptomatic sub. $\mathcal{A}_k$ at step k,
$I_k = \lvert\mathcal{I}_k\rvert$	N. of symptomatic sub. $\mathcal{I}_k$ at step k,
$R_k = \lvert\mathcal{R}_k\rvert$	N. of recovered sub. $\mathcal{R}_k$ at step k,
$Ra_k = \lvert\mathcal{R}a_k\rvert$	N. of asympt. recovered sub. $\mathcal{R}a_k$ at step k,
$O_k = \lvert\mathcal{O}_k\rvert$	N. of hospitalised sub. $\mathcal{O}_k$ at step k,
$D_k = \lvert\mathcal{D}_k\rvert$	N. of deceased sub. $\mathcal{D}_k$ at step k,
$Q_k = \lvert\mathcal{Q}_k\rvert$	N. of quarantined sub. $\mathcal{Q}_k$ at step k,
$Q_k^{(R)} = \left\lvert\mathcal{Q}_k^{(R)}\right\rvert$	N. of quarantined sub. recovered $\mathcal{Q}_k^{(R)}$ at step k.

Deterministic Parameters

N	Total number of subjects,
C	Available beds in hospital facilities.

Probabilistic Parameters

ω	Prob. to contract the infection in one meeting,
β	Prob. for an infectious asympt. sub. to recover,
δ	Prob. for an asympt. sub. to develop symptoms,
μ	Prob. for a symptomatic sub. to recover,
α	Prob. for a symptomatic sub. to die,
σ	Prob. for an hospitalised sub. to die,
ξ	Prob. for an hospitalised sub. to recover,
γ	Prob. for a tested infectious sub. to be positive,
ψ	Prob. for a symptomatic sub. to be hospitalised,
ι	Prob. that a quarantined sub. develop symptoms,
υ	Prob. that a quarantined sub. recovers.

Command Variables

M	Num. of people met by any subject*,
t	Num. of people tested*.

Figure 1. Summary of symbols. *Can be variable with the state.

abilities depend on many factors (e.g., use of masks, vaccination) and can be estimated by means of statistics, such as the one presented by Zardini et al. [33]. In the rest of the paper, we consider that the binomial coefficient $\binom{a}{b}$ for generic $a \geq 0$ and $b \geq 0$ is: equal to 0 when $b > a$; equal to 1 when $a = b = 0$. Moreover, we recall the definition of the following quantities: a) the dispositions counting the different ways of arranging k items from a set of n elements are denoted with $\mathbb{D}_{n,k} = n(n-1)\dots(n-k+1) = \frac{n!}{(n-k)!}$; b) the permutations of n elements $\mathbb{P}_n = n!$. Therefore, the binomial coefficient is defined as $\binom{n}{k} = \frac{\mathbb{D}_{n,k}}{\mathbb{P}_k}$ and the multinomial coefficient (i.e. the permutations with repetitions obtained computing all the permutations of n elements taken from k sets with $n_1, n_2, \dots, n_k$ elements with $n_k = n - \sum_{i=1}^{k-1} n_i$) is defined as $\mathbb{M}_{n,n_1,n_2,\dots,n_{k-1}} = \binom{n}{n_1,n_2,\dots,n_k} = \frac{\mathbb{P}_n}{\prod_{i=1}^{k} \mathbb{P}_{n_i}}$.

3 Extended SAIROD Markov Model

In this section we discuss our Markov model representing the spread of a COVID-19-like infection. We will make the following assumptions that reflect the best knowledge currently available on COVID-19 and on its management: (1) if a tested subject is found infectious, she/he becomes quarantined and is isolated from other people until recovery (or death), (2) quarantined persons are constantly monitored; (3) persons that are recovered cannot be re-infected by the virus in the short period. The first two assumptions are pretty natural and are typically enshrined within the regulation of most of the COVID-19-affected countries. The third one restricts our analysis to an interval of time in which virus variant forms do not materialise. Furthermore, we do not consider other external death causes, and that there are no false positives for the test outcomes. Should this assumption be invalidated, the results of our analysis would be conservative in terms of the probability of deaths and therefore acceptable. The possible states of a subject are the ones depicted in Figure 2. Since we are observing the system at discrete–time and, under the assumptions made, the number of people in each state determines the evolution of the population regardless of how this configuration has been reached, our system can be conveniently modelled as a discrete–time Markov chain. The state of the Markov chain will be associated with the 8-tuple collected in the following vector

$$\mathbf{V}_k = [S_k, A_k, I_k, R_k, O_k, D_k, Q_k, Ra_k]\,,$$

where the values of all the different quantities are non-negative integers representing the cardinality of their respective sets (see Figure 1). The elements of the set are bound to respect the following constraints:

$$\begin{aligned} &S_k + A_k + I_k + R_k + Ra_k + Q_k + O_k + D_k = N, \\ &O_k \leq C, \end{aligned} \tag{2}$$

that is, the population cardinality is constant, and the number of hospitalised people is limited by a (positive) capacity C. We assume that the presence of a virus can be detected either if the subject starts to develop symptoms of the disease or when the subject is tested positive. The subject can be traced in all the states in which the presence of the virus has been diagnosed: $\mathcal{Q}, \mathcal{I}, \mathcal{O}, \mathcal{D}, \mathcal{R}$. Such states are *observable*. On the contrary, since it is not possible to distinguish a subject who is susceptible, asymptomatic or recovered without having developed symptoms, the states $\mathcal{S}$, $\mathcal{A}$, $\mathcal{R}_a$ are not observable. In the rest of this section, we show how to compute the transition probability:

$$\Pr\left\{\mathbf{V}_{k+1} = \mathbf{v}' \,|\, \mathbf{V}_k = \mathbf{v}\right\}, \tag{3}$$

where $\mathbf{v}$ and $\mathbf{v}'$ are (respectively) the state vector values before and after the transition. Let

$$\boldsymbol{\Delta} v = \mathbf{v}' - \mathbf{v} = [\Delta_S, \Delta_A, \Delta_I, \Delta_R, \Delta_O, \Delta_D, \Delta_Q, \Delta_{Ra}]^T$$

and consider Figure 2, where, by the notation Δ_i, we denote the flows of subjects between the different states (e.g., Δ_1 is the number of susceptible subjects who become infected). Each Δ_i is a number defined in the set $D = [0,\, N] \cap \mathbb{N}$. Thereby, we define a new vector $\boldsymbol{\Delta} = [\Delta_1,\, \dots,\, \Delta_{11}]$ whose components respect the balance equations (4), labelled by B_i for $i = 1, \dots, 8$ and collectively denoted as B:

$$\begin{aligned}
B_1: &\quad -\Delta_1 &&= \Delta_S, \\
B_2: &\quad \Delta_1 - \Delta_2 - \Delta_3 - \Delta_9 &&= \Delta_A, \\
B_3: &\quad \Delta_{10} + \Delta_2 - \Delta_4 - \Delta_5 - \Delta_6 &&= \Delta_I, \\
B_4: &\quad \Delta_4 + \Delta_8 + \Delta_{11} &&= \Delta_R, \\
B_5: &\quad \Delta_5 - \Delta_7 - \Delta_8 &&= \Delta_O, \\
B_6: &\quad \Delta_6 + \Delta_7 &&= \Delta_D, \\
B_7: &\quad \Delta_9 - \Delta_{10} - \Delta_{11} &&= \Delta_Q, \\
B_8: &\quad \Delta_3 &&= \Delta_{R_a}.
\end{aligned} \tag{4}$$

B_1 captures that the flow from set $\mathcal{S}_k$ (susceptible) is only towards $\mathcal{A}_k$ (asymptomatic). B_2 captures that the population in $\mathcal{A}_k$ is increased by the inflow from $\mathcal{S}_k$ and decreased by

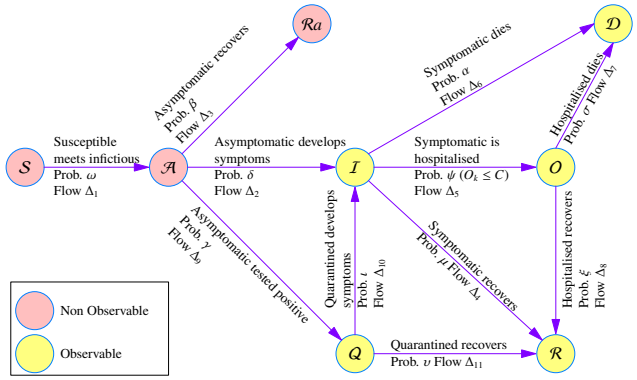

Figure 2. State transitions and flows (denoted with Δ_i) between the different states of the Markov model.

the outflow to $\mathcal{Q}_k$, $\mathcal{I}_k$ and $\mathcal{R}a_k$, meaning that an asymptomatic subject can either be tested positive and be quarantined (increasing Q_k), or exhibit symptoms and be classified as infected (increasing I_k) or recover (increasing R_a). B_3 captures that the subjects developing symptoms ($\mathcal{I}_k$) receives an inflow from set $\mathcal{A}_k$ and from set $\mathcal{Q}_k$, and has an outflow toward people who develop severe symptoms and are hospitalised (set $\mathcal{O}_k$), who recover (set $\mathcal{R}_k$) and who die (set $\mathcal{D}_k$). B_4 shows that, in our model, the set $\mathcal{R}_k$ has no outflow and receives inflows from $\mathcal{Q}_k$, $\mathcal{I}_k$ and $\mathcal{O}_k$. B_5 shows that the set of hospitalised people has an inflow from the infected state and produces two possible outcomes, a recovery or death. B_6 captures that the set $\mathcal{D}_k$ has no outflows, and only two inflows, from $\mathcal{I}_k$ and from $\mathcal{O}_k$. The set $\mathcal{Q}_k$ accounts for the quarantined subjects (i.e., tested positive). These subjects are constantly monitored and are not allowed to meet until they test negative. No transitions to quarantined state other than the ones that are asymptomatic subjects are modelled since no false positive test outcomes are considered. Therefore, the balance B_7 for $\mathcal{Q}_k$ is given by an inflow from $\mathcal{A}_k$ and two out flows towards $\mathcal{I}_k$ and $\mathcal{R}_k$. Finally, B_8 concerns the set $\mathcal{R}a_k$ including subjects that recover directly from the asymptomatic state. Therefore, they are assumed to be immune in the short-time, but there is no possibility of knowing that this is the case. As a consequence, contrary to the subjects who recovered from quarantine or known illness ($\mathcal{R}_k$), these subjects may be tested again and again with negative results. To summarise, the set $\mathcal{R}a_k$ has no outflows and has inflows only from $\mathcal{A}_k$. A direct consequence of this model is that the flows from the different states are subjected to the following constraints:

$$\begin{gathered}\Delta_1 \leq S_k, \quad \Delta_2+\Delta_3+\Delta_9 \leq A_k, \\ \Delta_{10}+\Delta_{11} \leq Q_k, \quad \Delta_4+\Delta_5+\Delta_6 \leq I_k, \\ \Delta_7+\Delta_8 \leq O_k.\end{gathered} \tag{5}$$

They ensure that the number of subjects remaining in the sets $\mathcal{S}_k$, $\mathcal{A}_k$, $\mathcal{I}_k$, $\mathcal{O}_k$ and $\mathcal{Q}_k$ is always non-negative. These constraints are not necessary for the absorbing sets $\mathcal{R}_k$, $\mathcal{D}_k$ and $\mathcal{R}a_k$.

We denote by $l(\cdot)$ an assignment of variables: $\Delta_i = \delta_i$, for $i = 1, \ldots, 11$. For an assignment of variable $l(\cdot)$ we use $l(\cdot) \models \varphi$ to mean that the assignment $l(\cdot)$ satisfies formula φ.

We say that an assignment is feasible if it satisfies the equations (4). For instance, $l\,(\Delta_1 = \delta_1,\ \Delta_2 = \delta_2,\ \Delta_3 = \delta_3) \models B_2$ means that the assignment δ_1, δ_2, δ_3 to the variables Δ_1, Δ_2 and Δ_3 satisfies balance equation B_2. Likewise, by $\boldsymbol{\Delta} \models B$ we mean that the vector $\boldsymbol{\Delta} = [\Delta_1, \ldots, \Delta_{11}]^T$ respects all the balance equations.

It is easy to re-write the definition of the transition probability (3) as follows:

$$\Pr\{\mathbf{V}_{k+1}=\mathbf{v}'\,|\mathbf{V}_k=\mathbf{v}\}=\Pr\{l(\boldsymbol{\Delta}) \models B\,|\mathbf{V}_k=\mathbf{v}\}\,. \tag{6}$$

For notational simplicity, we assume that $\mathbf{V}_k$ has been assigned to a particular value $\mathbf{v}$ and use the following notation: $\Pr\{l(\boldsymbol{\Delta}) \models B\,|\mathbf{V}_k\}$ as a shorthand for (3). Before stating and proving the theorem that allows us to compute the transition probability (6), let us introduce the following notations:

- l_1 is an assignment linking the variable Δ_1 defined via B_1 as: l_1: $(\delta_1 = -\Delta_S)$;
- l_2 is a function linking the variables Δ_2, Δ_3 and Δ_9 (with the variable Δ_9 obtained via equation B_2, and the variable Δ_3 obtained via equation B_8), defined as: l_2: $(\Delta_2 = \delta_2, \Delta_3 = \Delta_{R_a},\ \Delta_9 = -\Delta_S - \Delta_A - \Delta_{R_a} - \delta_2)$;
- l_3 is a function linking $\Delta_4, \Delta_5, \Delta_6$ and given by: l_3: $(\Delta_4 = \delta_4, \Delta_5 = \delta_5, \Delta_6 = \delta_6)$;
- l_4 is an assignment linking the remaining variables defined as: l_4: $(\Delta_7 = \Delta_D - \delta_6,\ \Delta_8 = \delta_5 + \delta_6 - \Delta_D - \Delta_O,\ \Delta_{10} = \Delta_I - \delta_2 + \delta_4 + \delta_5 + \delta_6, \Delta_{11} = \Delta_R + \Delta_D + \Delta_O - \delta_4 - \delta_5 - \delta_6)$;
- l_5, finally, assigns $(\Delta_2 = \delta_2, \Delta_3 = \delta_3, \Delta_9 = \delta_9)$.

We are now able to express (6) in a computable form.

Theorem 1 *The transition probability* (6) *is rewritten as*

$$\begin{aligned}&\Pr\{l(\boldsymbol{\Delta}) \models B\,|\mathbf{V}_k = \mathbf{v}\} = \\ &= \Pr\{l_1\,|\mathbf{V}_k\} \cdot \sum_{\delta_2=0}^{-\Delta_S-\Delta_A-\Delta_{R_a}} \Pr\{l_2\,|\mathbf{V}_k \wedge l_1\} \\ &\cdot \sum_{\delta_4=0}^{\delta_2-\Delta_I} \sum_{\delta_5=0}^{\delta_2-\Delta_I-\delta_4} \sum_{\delta_6=0}^{\delta_2-\Delta_I-\delta_4-\delta_5} \Pr\{l_3\,|\mathbf{V}_k \wedge l_1 \wedge l_2\} \\ &\cdot \Pr\{l_4\,|\mathbf{V}_k \wedge l_1 \wedge l_2 \wedge l_3\}\end{aligned} \tag{7}$$

Proof: By applying the theorem of total probability in the conditioned case to (6), we have:

$$\begin{aligned}&\Pr\{\mathbf{V}_{k+1} = \mathbf{v}'\,|\mathbf{V}_k\} = \Pr\{l(\boldsymbol{\Delta}) \models B\,|\mathbf{V}_k\} = \\ &\sum_{\delta_1 \in D} \Pr\{l(\boldsymbol{\Delta}) \models B\,|\mathbf{V}_k \wedge (\Delta_1 = \delta_1)\} \Pr\{\Delta_1 = \delta_1\,|\mathbf{V}_k\}.\end{aligned}$$

From the first balance equation (4), we observe that

$$l(\boldsymbol{\Delta}) \models B \implies (\Delta_1 = \delta_1) \models B_1 \implies \delta_1 = -\Delta_S.$$

When $\delta_1 \neq -\Delta_S$, it implies that

$$\Pr\{l(\boldsymbol{\Delta}) \models B\,|\mathbf{V}_k \wedge (\Delta_1 = \delta_1)\} = 0,$$

which makes all the terms in the summation vanish but the term $\Delta_1 = -\Delta_S$. Applying the above introduced shorthand

l_1, the transition probability is equal to

$$\Pr\{l(\boldsymbol{\Delta}) \models B \,|\mathbf{V}_k \wedge l_1\} \cdot \Pr\{l_1 \,|\mathbf{V}_k\}. \tag{8}$$

We can then iterate the application of the total probability theorem on the first term of (8) as follows

$$\sum_{(\delta_2,\delta_3,\delta_9)\in D^3} \Pr\{l(\boldsymbol{\Delta}) \models B \,|\mathbf{V}_k \wedge l_1 \wedge l_5\} \cdot \Pr\{l_5 \,|\mathbf{V}_k \wedge l_1\}.$$

We can again observe that the triple summation is only apparent, since for the terms that do not satisfy B_2, that is, $(\Delta_1 = \delta_1, l_5) \not\models B_2$, it holds that

$$\Pr\{l(\boldsymbol{\Delta}) \models B \,|\mathbf{V}_k \wedge l_1 \wedge l_5\} = 0.$$

As in the previous case, considering the second balance equation (4), we have introduced the shorthand l_2 which simplifies the notation for the above term to:

$$\Pr\{l(\boldsymbol{\Delta}) \models B \,|\mathbf{V}_k \wedge l_1 \wedge l_5\} = \sum_{\delta_2=0}^{-\Delta_S-\Delta_A-\Delta_{R_a}} \Pr\{l(\boldsymbol{\Delta}) \models B \,|\mathbf{V}_k \wedge l_1 \wedge l_2\} \Pr\{l_2 \,|\mathbf{V}_k \wedge l_1\}.$$

We can apply once again the theorem of total probability and enforce the validity of B_3 in shorthand l_3:

$$\Pr\{l(\boldsymbol{\Delta}) \models B \,|\mathbf{V}_k \wedge l_1 \wedge l_2\} = \sum_{\delta_4=0}^{\delta_2-\Delta_I} \sum_{\delta_5=0}^{\delta_2-\Delta_I-\delta_4} \sum_{\delta_6=0}^{\delta_2-\Delta_I-\delta_4-\delta_5} \Pr\{l_3 \,|\mathbf{V}_k \wedge l_1 \wedge l_2\} \cdot \cdot \Pr\{l(\boldsymbol{\Delta}) \models B \,|\mathbf{V}_k \wedge l_1 \wedge l_2 \wedge l_3\}.$$

Our last iteration of the theorem of total probability that enforces the validity of B_4, B_5 and B_6 by means of the shorthand l_4, leads to:

$$\Pr\{l(\boldsymbol{\Delta}) \models B \,|\mathbf{V}_k \wedge l_1 \wedge l_2 \wedge l_3\} = = \sum_{(\delta_7,\delta_8,\delta_{10},\delta_{11})\in D^4} \Pr\{l_4 \,|\mathbf{V}_k \wedge l_1 \wedge l_2 \wedge l_3\} \cdot \cdot \Pr\{l(\boldsymbol{\Delta}) \models B \,|\mathbf{V}_k \wedge l_1 \wedge l_2 \wedge l_3 \wedge l_4\},$$

It is easy to observe that $\mathbf{V}_k \wedge l_1 \wedge l_2 \wedge l_3 \wedge l_4$ is a certain event, thus

$$\Pr\{l(\boldsymbol{\Delta}) \models B \,|\mathbf{V}_k = \mathbf{v} \wedge l_1 \wedge l_2 \wedge l_3 \wedge l_4\} = 1,$$

which leads us to the final simplification and yields the theorem. □

In the following sections we will report the computations of the different probabilities that appear in (7).

3.1 Probability that a susceptible becomes infectious

A first step towards the computation of the different components of (7) is to compute the probability that a susceptible subject becomes infectious. To this end, we first introduce the following useful auxiliary events (the negated events are in parentheses) taking place at time k

$$\begin{cases} c_k\,(\overline{c}_k) & \text{A subject meets one person and (does, not) contracts the infection} \\ m_k\,(\overline{m}_k) & \text{A subject meets one person and he/she, is (not) infectious} \\ g_k\,(\overline{g}_k) & \text{A person (does not) becomes infected. after meeting an infectious person} \end{cases} \tag{9}$$

Hence, the event g_k can be defined as $g_k = \{p \in \mathcal{S}_k \wedge p \in \mathcal{A}_{k+1}\}$, where p is a generic person. Since each subject has M different meetings during time k, we have

$$\Pr\{g_k \,|\mathbf{V}_k\}_M = 1 - \Pr\{\overline{c}_k \,|\mathbf{V}_k\}^M,$$

i.e., the probability of contracting the infection is the complementary of the probability of remaining healthy after M meetings. It can be observed that a person remains healthy after one meeting either if the person met is herself healthy or if she is infectious but the infection is not transmitted in the meeting, i.e. $\overline{c}_k = \overline{m}_k \cup (m_k \cap \overline{g}_k)$. Thereby,

$$\begin{aligned} \Pr\{\overline{c}_k \,|\mathbf{V}_k\} &= \Pr\{\overline{m}_k \cup (m_k \cap \overline{g}_k) \,|\mathbf{V}_k\} \\ &= \Pr\{\overline{m}_k \,|\mathbf{V}_k\} + \Pr\{m_k \cap \overline{g}_k \,|\mathbf{V}_k\} \\ &= \Pr\{\overline{m}_k \,|\mathbf{V}_k\} + \Pr\{\overline{g}_k \,|m_k \wedge \mathbf{V}_k\} \Pr\{m_k \,|\mathbf{V}_k\}. \end{aligned}$$

Assuming a uniform distribution in the meetings across the different classes of subjects and recalling that $\Pr\{\overline{g}_k \,|m_k \wedge \mathbf{V}_k\} = 1 - \omega$, we have:

$$\begin{aligned} \Pr\{\overline{c}_k \,|\mathbf{V}_k\} &= \frac{S_k + R_k + R_{a_k}}{N - D_k - I_k - O_k - Q_k} \\ &\quad + \frac{A_k(1-\omega)}{N - D_k - I_k - O_k - Q_k} \\ &= 1 - \omega \frac{A_k}{N - D_k - I_k - O_k - Q_k}, \end{aligned}$$

where we applied (2). Therefore

$$\Pr\{g_k \,|\mathbf{V}_k\}_M = 1 - \left(1 - \frac{\omega A_k}{N - D_k - I_k - O_k - Q_k}\right)^M. \tag{10}$$

3.2 Probability that a number of infected subjects are identified and quarantined

In our setting, t_k tests are administered to a set of subjects in the set $\mathcal{N}_k = \mathcal{A}_k \cup \mathcal{S}_k \cup \mathcal{R}_{a_k}$, i.e. on the population that is not in the known set $\mathcal{Q}_k \cup \mathcal{I}_k \cup \mathcal{R}_k \cup \mathcal{D}_k \cup \mathcal{O}_k$.
Let the event s_H be defined as: "H people are tested positive, given the state $\mathbf{V}_k$". Assuming that each subject can be tested only once at time k (which implies $t_k \leq N_k = |\mathcal{N}_k|$), our goal is to find the probability $\Pr\{s_H\}$. To this end, we first recall (1) and, by defining the event a_p "p infected subjects are tested", we immediately have that the probability that H subjects are found positive given that $p \in \{0, \ldots, A_k\}$ infected subjects are tested, is given by

$$\Pr\{H \,|a_p\} = \mathcal{B}(p, \gamma)_H. \tag{11}$$

We are now in a position to prove the following theorem.

Theorem 2 *Given $t_k \leq N_k = A_k + S_k + R_{a_k}$ and $H \leq t_k$,*

we have that

$$\Pr\{s_H\} = \binom{N_k}{t_k}^{-1} \sum_{p=0}^{t_k} \binom{S_k + R_{a_k}}{t_k - p} \binom{A_k}{p} \mathcal{B}(p,\gamma)_H.$$

Proof: The probability $\Pr\{s_H\}$ is given by the sum of disjoint events (Total Probability Law) that enumerates all the possibilities of testing at most t_k infected in the set $\mathcal{A}_k$, i.e. $\Pr\{s_H\} = \sum_{p=0}^{t_k} \Pr\{a_p\} \Pr\{H\,|a_p\}$, where $\Pr\{H\,|a_p\}$ is given in (11). To determine $\Pr\{a_p\}$, we first consider the number p as the "number of infected people tested at time k". Hence, we have that the probability of one possible sequence having exactly p infected people tested is equal to: the number of the different ways of arranging $t_k - p$ people from the negative set of people $\mathcal{S}_k \cup \mathcal{R}_{a_k}$ times the number of the different ways of arranging p people from the positive set of people $\mathcal{A}_k$ divided by the number of the different ways of arranging t_k people from the overall set of people N_k, i.e.

$$\frac{\mathbb{D}_{S_k+R_{a_k},t_k-p}\mathbb{D}_{A_k,p}}{\mathbb{D}_{N_k,t_k}}.$$

This number has to be multiplied by the number of all the possible different sequences, which is given by all the permutations of t_k elements taken from the two sets with $t_k - p$ and p elements, i.e., $\binom{t_k}{p}$. By substituting the definitions given previously, we have that

$$\begin{aligned}\Pr\{a_p\} &= \frac{\mathbb{D}_{S_k+R_{a_k},t_k-p}\mathbb{D}_{A_k,p}}{\mathbb{D}_{N_k,t_k}} \frac{\mathbb{P}_{t_k}}{\mathbb{P}_{t_k-p}\mathbb{P}_p} \\ &= \binom{N_k}{t_k}^{-1} \binom{S_k + R_{a_k}}{t_k - p} \binom{A_k}{p}.\end{aligned}$$

Hence the proof follows. □

3.3 Probability of a state change

Instrumental to the construction of the transition matrix is to compute the probability that a given number of subjects simultaneously change their state, i.e. the different factors in equation (7) of Theorem 1. Considering Figure 2, such probabilities are discussed next.

Transition from State S. The probability of the event "exactly Δ_1 susceptible subjects become infected at step k given the state $\mathbf{V}_k$ and a choice for M" is approximated by $\mathcal{B}(S_k, \Pr\{g_k\,|\mathbf{V}_k\}_M)_{\Delta_1}$. Hence, the probability of the assignment l_1 is approximated by

$$\Pr\{l_1\,|\mathbf{V}_k\} = \mathcal{B}(S_k, \Pr\{g_k\,|\mathbf{V}_k\}_M)_{-\Delta_S}. \quad (12)$$

Transition from State A. Consider the event $r^{(E)}_{\delta_2,\delta_3,\delta_9}$ defined as "exactly δ_3 asymptomatic infected subjects recover at step k without being quarantined, δ_2 subjects become symptomatic and δ_9 out of t_k are tested positive and become quarantined given the state $\mathbf{V}_k$". Let $\rho^{(E)}(\delta_2,\delta_3,\delta_9,\mathbf{V}_k)_{t_k}$ be the probability of this event given $\mathbf{V}_k$ and t_k. Instrumental to the computation of this probability, the following theorem proves useful.

Theorem 3 *Let $\rho(\delta_2,\delta_3,\mathbf{V}_k)$ be the probability of the event "exactly δ_3 asymptomatic infected subjects recover at step k and δ_2 subjects become symptomatic". Hence, $\rho(\delta_2,\delta_3,\mathbf{V}_k) = 0$ if $\delta_2 + \delta_3 > A_k$, otherwise*

$$\rho(\delta_2,\delta_3,\mathbf{V}_k) = \mathbb{M}_{A_k,\delta_2,\delta_3}\beta^{\delta_3}\,\delta^{\delta_2}\,C^{A_k-\delta_2-\delta_3}_{\beta,\delta}.$$

Proof: In order to define $\rho(\delta_2,\delta_3,\mathbf{V}_k)$ by using a combinatorial approach, we first need to compute all the possible combinations of $\delta_2 + \delta_3$ subjects out of the set A_k, which is given by the multinomial $\mathbb{M}_{A_k,\delta_2,\delta_3} = \binom{A_k}{\delta_2,\delta_3,A_k-\delta_2-\delta_3}$. Then, recalling that β is the probability for an infectious asymptomatic subject to recover, we have that the probability that exactly δ_3 subjects recover is given by β^{δ_3}. Similarly, since δ is the probability for an asymptomatic subject to develop symptoms, we have that the probability that exactly δ_2 subjects recover is given by δ^{δ_2}. Finally, using $C_{\beta,\delta} \geq 0$ defined in Section 2, the probability that exactly $A_k - \delta_2 - \delta_3$ subjects does not recover nor develop symptoms, is immediately given by $C^{A_k-\delta_2-\delta_3}_{\beta,\delta}$. Therefore

$$\rho(\delta_2,\delta_3,\mathbf{V}_k) = \mathbb{M}_{A_k,\delta_2,\delta_3}\beta^{\delta_3}\,\delta^{\delta_2}\,C^{A_k-\delta_2-\delta_3}_{\beta,\delta}.$$

Notice that, when $\delta_2 + \delta_3 > A_k$, we have by definition that $\mathbb{M}_{A_k,\delta_2,\delta_3} = 0$, which concludes the proof. □

We can now show the probability of the event $r^{(E)}_{\delta_2,\delta_3,\delta_9}$.

Theorem 4 *The probability $\rho^{(E)}(\delta_2,\delta_3,\delta_9,\mathbf{V}_k)_{t_k}$ is zero in the trivial conditions $\delta_2 + \delta_3 + \delta_9 > A_k \vee \delta_9 > t_k \vee \delta_2 < H - \delta_9$, and otherwise is given by*

$$\rho^{(E)}(\delta_2,\delta_3,\delta_9,\mathbf{V}_k)_{t_k} = \sum_{H=\delta_9}^{t_k} \Pr\{s_H\} \sum_{F=0}^{\delta_9} \rho(\delta_2,\delta_3+F,\mathbf{V}_k) \binom{A_k}{H}^{-1} K, \quad (13)$$

where

$$K = \binom{\delta_3 + F}{F} \binom{A_k - (\delta_2 + \delta_3 + F)}{\delta_9 - F} \binom{\delta_2}{H - \delta_9}. \quad (14)$$

Proof: Recalling the definition of the event s_H (i.e., H subjects are tested positive given the state $\mathbf{V}_k$), thus, conditioning over s_H, we have:

$$\begin{aligned}\rho^{(E)}(\delta_2,\delta_3,\delta_9,\mathbf{V}_k)_{t_k} &= \Pr\left\{r^{(E)}_{\delta_2,\delta_3,\delta_9}\right\} \\ &= \sum_{H=0}^{t_k} \Pr\{s_H\} \Pr\left\{r^{(E)}_{\delta_2,\delta_3,\delta_9}\,|s_H\right\},\end{aligned}$$

where the first equality follows by definition. Introduce the event $r_{\delta_2,\overline{\delta}_3}$ described as "exactly $\overline{\delta}_3$ asymptomatic infected subjects recover at step k and δ_2 subjects become symptomatic given the state $\mathbf{V}_k$", which, according to Theorem 3, has probability $\rho(\delta_2,\overline{\delta}_3,\mathbf{V}_k)$. It is important to observe that the δ_2 subjects will entirely move to state I, while among the $\overline{\delta}_3$ subjects, δ_3 that are not tested positive move to the state R_a, whilst $F = \overline{\delta}_3 - \delta_3$ are tested positive and move to the state Q. The number of the subjects F that recover

and are tested positive ranges from 0 to $\min(\overline{\delta}_3, \delta_9)$, with δ_9 the positive tested out of t_k. Thereby,

$$\Pr\left\{r^{(E)}_{\delta_2,\delta_3,\delta_9}\,|s_H\right\} = \sum_{F=0}^{\delta_9} \Pr\left\{r^{(E)}_{\delta_2,\delta_3,\delta_9}\,|s_H \wedge r_{\delta_2,\delta_3+F}\right\} \Pr\left\{r_{\delta_2,\delta_3+F}\,|s_H\right\}.$$

The event r_{δ_2,δ_3+F} is independent from the event s_H, since testing and healing are independent, hence

$$\Pr\left\{r_{\delta_2,\delta_3+F}\,|s_H\right\} = \Pr\left\{r_{\delta_2,\delta_3+F}\right\} = \rho(\delta_2, \delta_3+F, \mathbf{V}_k) = \rho(\delta_2, \overline{\delta}_3, \mathbf{V}_k).$$

We take into account now the conditional probability $\Pr\left\{r^{(E)}_{\delta_2,\delta_3,\delta_9}\,|s_H \wedge r_{\delta_2,\delta_3+F}\right\}$ that considers that H subjects are tested positive, we notice that: a) F of them are subjects that would recover (if they did not test positive) and are extracted from a group of $\overline{\delta}_3 = \delta_3 + F$ subjects, i.e. $\frac{\mathbb{D}_{\delta_3+F,F}}{\mathbb{P}_F}$; b) since a number of $A_k - (\delta_2+\delta_3+\delta_9)$ remains in the asymptomatic state $\mathcal{A}$, $\delta_9 - F$ are subjects that would remain asymptomatic and are extracted from a group of $A_k - (\delta_2 + \overline{\delta}_3)$ subjects, i.e. $\frac{\mathbb{D}_{A_k-(\delta_2+\delta_3+F),\delta_9-F}}{\mathbb{P}_{\delta_9-F}}$; c) $H - \delta_9$ are subjects that develop symptoms and are extracted from a group of δ_2 subjects, i.e. $\frac{\mathbb{D}_{\delta_2,H-\delta_9}}{\mathbb{P}_{H-\delta_9}}$. Therefore, by dividing by all the possible ways to arrange H elements tested positive in $\mathcal{A}$, we have

$$\Pr\left\{r^{(E)}_{\delta_2,\delta_3,\delta_9}\,|s_H \wedge r_{\delta_2,\delta_3+F}\right\} = \frac{\mathbb{D}_{\delta_3+F,F}}{\mathbb{P}_F}\frac{\mathbb{D}_{A_k-(\delta_2+\delta_3+F),\delta_9-F}}{\mathbb{P}_{\delta_9-F}}\frac{\mathbb{D}_{\delta_2,H-\delta_9}}{\mathbb{P}_{H-\delta_9}}\frac{\mathbb{P}_H}{\mathbb{D}_{A_k,H}},$$

and the proof follows. □

The probability $\Pr\{l_2\,|\mathbf{V}_k \wedge l_1\}$ is therefore given by:

$$\Pr\{l_2\,|\mathbf{V}_k \wedge l_1\} = \rho^{(E)}(\delta_2, \delta_3, \delta_9, \mathbf{V}_k), \qquad (15)$$

with $\rho^{(E)}(\cdot)$ given by equation (13).

Transition from State I. Consider the event "exactly δ_4 symptomatic subjects recover, δ_5 become hospitalised and δ_6 die". Let $\phi(\delta_4, \delta_5, \delta_6, \mathbf{V}_k)$ be its probability given $\mathbf{V}_k$. The probability $\phi(\delta_4, \delta_5, \delta_6, \mathbf{V}_k)$ is zero if $\delta_4+\delta_5+\delta_6 > I_k$ or if $\delta_5 > C - O_k$ (the latter condition meaning that we are hospitalising more patients than allowed by the capacity of the hospital). We have two cases, when $\delta_5 < C - O_k = \overline{C}_k$, that is, we can hospitalise the patients, and when the maximum available capacity is saturated:

$$\phi(\delta_4, \delta_5, \delta_6, \mathbf{V}_k) = \begin{cases} 0 & \text{if } \delta_4+\delta_5+\delta_6 > I_k \vee \delta_5 > \overline{C}_k \\ M(\delta_4, \delta_5, \delta_6) & \text{if } \delta_5 < \overline{C}_k \wedge \delta_4+\delta_5+\delta_6 \leq I_k \\ M'(\delta_4, \delta_6) & \text{if } \delta_5 = \overline{C}_k \wedge \delta_4+\delta_5+\delta_6 \leq I_k \end{cases} \qquad (16)$$

where, similar to Theorem 3, we have

$$M(\delta_4, \delta_5, \delta_6) = \mathbb{M}_{I_k,\delta_4,\delta_5,\delta_6} \mu^{\delta_4}\, \psi^{\delta_5}\, \alpha^{\delta_6}\, C^{I_k-\delta_4-\delta_5-\delta_6}_{\mu,\psi,\alpha}.$$

The idea is that whenever the result for the hospitalised would exceed the maximum residual capacity $\overline{C}_k$, the extra patients are turned down and remain in state I. Thereby,

$$M'(\delta_4, \delta_6) = \sum_{h=0}^{I_k-\delta_4-\delta_6-\overline{C}_k} M(\delta_4, \overline{C}_k + h, \delta_6),$$

where we are accumulating into $M'(\delta_4, \delta_6)$ the probability of having more patients evaluated as in need of hospitalisation than it is possible to receive. We can now compute the probability

$$\Pr\{l_3\,|\mathbf{V}_k \wedge l_1 \wedge l_2\} = \phi(\delta_4, \delta_5, \delta_6, \mathbf{V}_k). \qquad (17)$$

Transition from State O: Consider the event "exactly δ_8 hospitalised subjects recover at step k and δ_7 hospitalised subjects die" and let $\zeta(\delta_7, \delta_8, \mathbf{V}_k)$ be its probability, given $\mathbf{V}_k$, which is obviously zero if $\delta_7 + \delta_8 > O_k$. The remaining cases show again a multinomial coefficient, i.e.,

$$\zeta(\delta_7, \delta_8, \mathbf{V}_k) = \mathbb{M}_{O_k,\delta_7,\delta_8} \sigma^{\delta_7}\, \xi^{\delta_8}\, C^{O_k-\delta_7-\delta_8}_{\sigma,\xi}. \qquad (18)$$

Transition from State Q. The transitions from state Q is similar to the previous case from state O, hence it is given by

$$\chi(\delta_{10}, \delta_{11}, \mathbf{V}_k) = \mathbb{M}_{Q_k,\delta_{10},\delta_{11}} \iota^{\delta_{10}}\, \upsilon^{\delta_{11}}\, C^{Q_k-\delta_{10}-\delta_{11}}_{\iota,\upsilon}. \qquad (19)$$

We can now compute the term $\Pr\{l_4\,|\mathbf{V}_k \wedge l_1 \wedge l_2 \wedge l_3\}$ that connects together the transitions from sates O and Q:

$$\begin{aligned} &\Pr\{l_4\,|\mathbf{V}_k \wedge l_1 \wedge l_2 \wedge l_3\} = \\ &= \zeta(\Delta_D - \delta_6, \delta_5 + \delta_6 - \Delta_D - \Delta_O, \mathbf{V}_k) \\ &\cdot\chi(\Delta_I - \delta_2 + \delta_4 + \delta_5 + \delta_6, \Delta_R + \Delta_D + \Delta_O - \delta_4 - \delta_5 - \delta_6, \mathbf{V}_k). \end{aligned} \qquad (20)$$

The computation of the transition probabilities defined in Theorem 1 is thus obtained by plugging (12), (15), (17) and (20) into (7).

3.4 Complexity analysis

For the analysis of the complexity, we will consider the overall number of states that can be generated by the model, assuming an arbitrary number of model configurations n, i.e., susceptible, asymptomatic, etc. Let N be the subjects in the population, we may assume that the overall number of states m that can be generated is given by $m = (N+1)^n$. However, this is a large upper bound to the states that can be actually reached: indeed, given α_i, $i = 1, \ldots, n$, the number of subjects in the i-th configuration, we have the constraint $\sum_{i=1}^{n} \alpha_i = N$. To account for this simplifying constraint, we first define $m = f(n, N)$, i.e., the overall number of possible states m given the n configurations and N subjects. The number m can be computed considering that the n-th configuration accounts for an arbitrary number $0 \leq i \leq N$ of subjects, while the remaining population of $N - i$ are allocated in the other $n - 1$ configurations. Therefore

$$m = f(n, N) = \sum_{i=0}^{N} f(n-1, N-i)$$

Hence, by simply considering that $f(1, N) = 1$, we have

$$f(2, N) = N + 1,$$

$$f(3,N) = \frac{(N+2)(N+1)}{2} = \binom{N+2}{2} = \binom{N+2}{N},$$

we end up with

$$m = f(n,N) = \binom{N+n-1}{n-1} = \binom{N+n-1}{N},$$

which is the Bose-Einstein statistics. Since the complexity is proportional to the number of reachable states, we have immediately the complexity figure for the presented algorithm.

4 Numeric Examples

To show the potential applications of the formalised model, we have developed in C++ a proof of concept open-source tool. [1] The tool operates both in the case t_k is set to zero (i.e., no people ever get quarantined) or not. For the rest of the section we consider the case where $t_k = 0$. In this simplified situation, the model takes a simpler form (see Section A). For this model, we could generate and analyse the Markov model for populations of size comparable to a typical circle of people who meet on a regular basis. What is more, the tool can be interfaced with the `PRISM` stochastic model checker [23]. The analysis of a system goes through the following steps. 1. reachability analysis: starting from an initial set of states and from a complete specification of the system's parameters (e.g., the different probabilities, the population size), compute all the states that are reachable and construct a transition table that associates each state-action pair `(cS, a)` with the next state `nS` and probability `p` to reach that state, where the `a` is associated with the number M of people that are allowed to be met for that state `cS`. 2. application of a `Policy`: apply a control law associating each state with one of the actions that are possible for that state generating a DTMC. After the completion of the second step, it is possible to either solve the DTMC (i.e., given a set of initial states and a known initial probability, compute the evolutions for different steps and/or at the steady-state), or apply the PRISM model checker to verify some Probabilistic Linear Temporal Logic (PLTL) properties [18].
Hereafter, we will use numeric examples to expose some interesting facts related to the properties of the formalised model. We used the following choice of probabilities (α,β,δ,μ,ω,ψ,σ,ξ)=(0.25,0.45,0.25,0.4,0.5,0.35,0.1,0.65).
We executed the experiments on a Linux PCs equipped with 256Gb of RAM, Intel® Xeon® 2.20GHz with 12 cores.

Equilibrium and Convergence. When we apply a policy, the system reduces to a DTMC. The presence of multiple absorbing states prevents the existence of a single steady-state equilibrium (trivially, any initial state such that all elements are zero but the absorbing states is an equilibrium point). The initial states worth to consider are those where most of the subjects are susceptible and a few of them are asymptomatic and they start spreading the infection. In this case, our question is to compute the probability that no more than a given percentage of the population (e.g., 20%) will eventually die owing to the virus. We considered a population of $N = 20$ subjects with the following three initial states: **IS 1** = (19,1,0,0,0,0), **IS 2** = (17,3,0,0,0,0), **IS 3** = (15,5,0,0,0,0). Each initial state is represented with a tuple $(S_0, A_0, I_0, R_0, O_0, D_0)$, where each element corresponds to the population in the respective state at step 0. In order to emphasise the impact of the choice of M on the equilibrium, we fixed parameter C (i.e., the capacity of the intensive care facility) to a relatively high value: $C = 5$. The result of the benchmark probability $\lim_{k\to\infty} \Pr\{D_k \geq 0.2N\}$ is reported in Figure 3. The figure reveals that the system converges to its equilibrium in a relatively small number of iterations (around 10). Another important fact is that the choice of a different initial state produces a different steady-state probability. Interestingly, these probabilities are closer for a permissive policy ($M = 5$), while they spread apart for a more restrictive policy ($M = 2$). The steady state Cumulative Distribution Functions (CDF) cast some light on this phenomenon. In Figure 4, we show the steady marginal distribution for the IS 3 and for two different values of M. The figure shows that aggressive restrictions gradually decrease the number of people in the asymptotic state without "fresh" replenishment from the susceptible state. As a consequence, at some point, with high probability, no more subjects will be infected, while the infected people will move to one of the

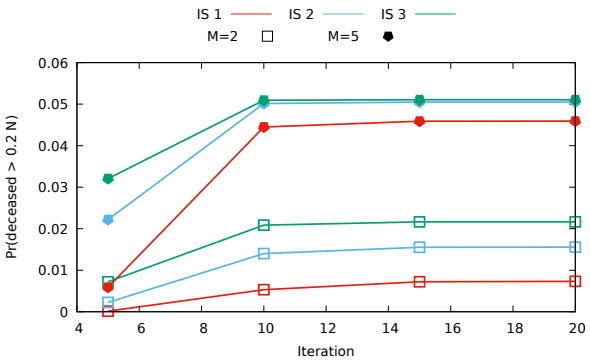

Figure 3. Probability that more than 20% of the population will die as a function of the iterations for two different values of M.

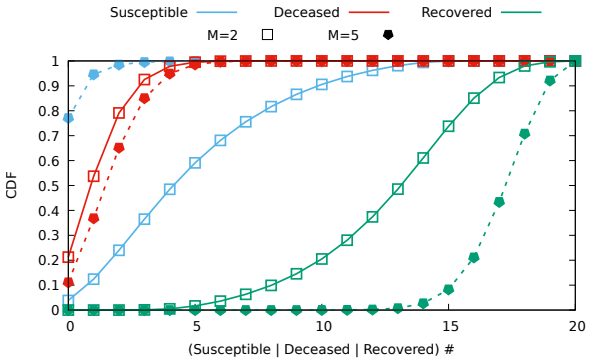

Figure 4. Steady-state CDFs of the deceased, the recovered and of the susceptible subjects for initial state 3 and variable M.

[1] The tool and all the information to reproduce these experiments are available from `https://bit.ly/33jyiZU`.

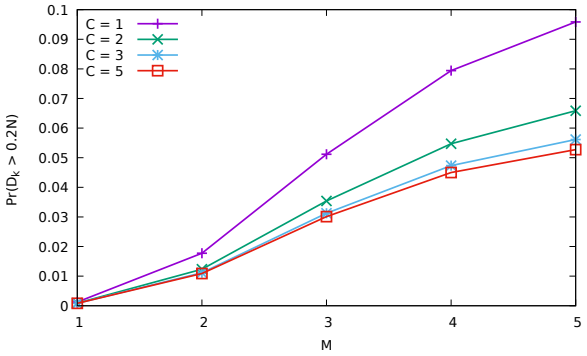

Figure 5. Probability that more than 20% of the population will die as a function of M for different capacities C.

absorbing state (deceased or recovered). On the contrary, a permissive policy causes all the subjects to be quickly infected. The consequences on the deceased are not substantial in this case because for this experiment, it is assumed to be a "generous" availability of intensive care beds, but the difference is visible on the number of subjects that get the disease and recover. When a high number of subjects remains confined to the susceptible state, the steady state becomes highly dependent on how many subjects are initially infected (which is the reason why for $M = 2$ the steady state probability is highly dependent on the initial state).

Impact of Social Distancing. In order to design adaptive policies, it is of paramount importance to look at the impact of the input variable M, i.e., the number of people that each one is allowed to meet at each step. For this experiment we assumed that at time 0 the system could be either in (15,3,0,0,0) or in (17,1,0,0,0) with equal probability 0.5, and the capacity of the intensive care C was chosen in the set $\{1, 2, 3, 5\}$. The plot of the probability of having more than 20% casualties is shown in Figure 5. The dependence is monotonic in M, but the amount of change clearly depends on the hospital capacity C. For $C = 1$ and $M = 5$, this probability is beyond 9%. Increasing the number of beds in the hospital has obvious benefits, but for the system at hand the performance gain flattens for $C \geq 3$, meaning that additional growths in the beds are not worth the effort.

Adaptive Policies. An adaptive policy is a feedback control law that decides the social distance M based on the current value $\mathbf{V}_k$. Adaptive policies for a system like the one described in this paper can be synthesised using the theory of POMDPs [12, 20]. The efficient synthesis of a POMDP tailored to the model is far from obvious and is reserved for future work. In this section, we opt for a simpler choice and present two heuristics, both based on the following equation:

$$\mathcal{M}(\mathbf{V}_k) = \begin{cases} M_\uparrow & \text{if } f(\mathbf{V}_k) \leq T_\downarrow \\ M_\downarrow & \text{if } f(\mathbf{V}_k) \geq T_\uparrow \\ M_\uparrow + (M_\downarrow - M_\uparrow)\,\frac{f(\mathbf{V}_k) - T_\downarrow}{T_\uparrow - T_\downarrow} & \text{otherwise.} \end{cases} \quad (21)$$

The two defined heuristics differ for the choice of the function $f(\mathbf{V}_k)$. The first one set $f(\mathbf{V}_k) = I_k + O_k/(N - D_k)$ and the rational is as follows. When the percentage of the number of symptomatic and hospitalised patients over the living population is below the threshold $T_\downarrow$, we are allowed to use the maximum value $M_\uparrow$ for M (meaning the least restrictions for social life). If this number is above a threshold $T_\uparrow$, we are forced to adopt the maximum restriction (the minimum value $M_\downarrow$ for M). If the number is in between the two thresholds we perform a linear interpolation between the minimum and the maximum values of M. The second heuristic sets $f(\mathbf{V}_k) = A_k/(N - D_k)$, i.e., we make the choice as for the first option but this time considering the ratio between asymptomatic infected subjects and the living population. Clearly, it is not possible to implement this policy (although A_k could be estimated by statistic means using tests), but still we believe it is useful to have it as a performance baseline. We performed a set of tests, considering the same scenario as in Section 1 assuming the worst-possible case scenario for hospital beds: $C = 1$. The parameters for the adaptive policies were chosen as $T_\downarrow = 0.05$, $T_\uparrow = 0.15$, $M_\downarrow = 1$ and $M_\uparrow = 5$. For the sake of completeness, we also compared the performance of the adaptive policies setting M to a constant value (the minimum and the maximum). The results are shown in Figure 6: on the left we report the benchmark probability $\Pr\{D_k \geq 0.2N\}$ for the different iterations; on the right we show the value of the average M (i.e., $\sum_{\mathbf{V}_k} \mathcal{M}(\mathbf{V}_k)\Pr\{\mathbf{V}_k\}$). The *Adaptive Asymptomatic (AA)* policy using asymptomatic subjects shows an excellent performance: the probability of the deceased subjects is very close to the one for $M = 1$, but the average value of M is higher (it starts from 2.5 and tends to 5 after a few iterations). The *Adaptive Symptomatic (AS)* policy using symptomatic subjects has a worse performance in terms of probability, and in the average applies more social restrictions (smaller M). This happens since the *AA* policy anticipates the restrictions at the first two steps, while the *AS* policy defers the decision after it can observe a growth of symptomatic subjects, too late to prevent many deaths.
We also run 1000 Monte Carlo simulations with a depth of 100 on a population of 100 individuals proportionally scaling M, the hospital capacity, and the individuals in the different states in the initial conditions. The $\Pr\{D_k > 0.2N\}$ is 0.0143 for AS, 0.0091 for AA, 0.0084 for the constant policy with $M = 5$, and 0.0143 for the constant policy with $M = 25$. Although the probabilities are lower than for the case $N = 25$, the results shows a similar trend.

We remark that, as shown in Section 3.4, an upperbound for the number of reachable states for a population of $N = 25$ can be computed as $f(N, n) = \binom{N+n-1}{N}$. Thus, it amounts to 142506 states for the simplified model ($n = 6$), and to 3365856 states for the complete model ($n = 8$). The transitions are of the order of $f(N, n)^2$, thus 2.0308e+10 transitions for $n = 6$, and 1.1329e+13 transitions for $n = 8$.

4.1 *Analysis using Probabilistic Model Checking*

Background on Probabilistic Model Checking (PMC). PMC [13] is an effective formal verification method [4] used for analysing stochastic transitions systems such as MDP,

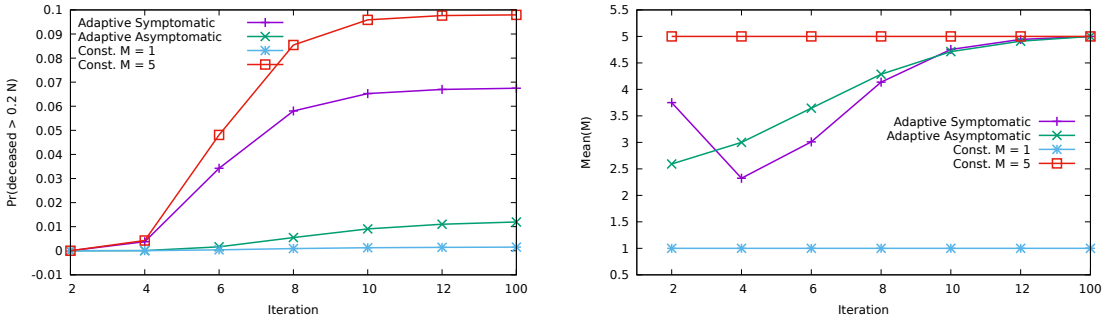

Figure 6. Probability that more the 20% of the population will die, average value of the action M used in the different iterations.

DTMC or CTMC. The analysis is performed by verifying whether the stochastic model of the system (in our case a policy) meets the requirements encoded in PLTL [18], which allows to express very rich properties. For instance, $\mathbf{P}_{<0.25}[\mathbf{F}\, O_k = C]$ means the probability of reaching a state where the intensive care facility is fully utilised is less than 0.25. $\mathbf{P}_{=?}[\mathbf{G}\,\mathbf{F}\, O_k < C]$ is an example of a quantitative property, PRISM computes the probability of visiting infinitely often states where the intensive care facility is not fully utilised. Intuitively, PMC amounts to automatically check whether all the computations starting from a given set of initial states of an MDP, DTMC or CTMC satisfy a given PLTL formula. As result of the check, PMC produces two kinds of outputs: a) qualitative answers to indicate whether the property holds or is violated (in this case generating a witness of the violation), b) quantitative answers to indicate the probability of satisfaction of the given PLTL formula. Moreover, PMC allows to compute the vector of steady-states with the corresponding probabilities, compute the transient probabilities after a given time, as well as compute the probability a certain condition holds in the steady-states. [2]

Applications of PMC to verify DTMC model. We performed a preliminary analysis, using the `PRISM` [23] model checker, to verify some PLTL properties on the DTMC model resulting from the application of the *AA* policy to our model. In this analysis, we considered a population of $N = 10$ persons, and all possible states (i.e. all possible consistent configurations for V) as initial. `PRISM` proved that i) $\mathbf{P}_{=?}[\mathbf{G}\,\mathbf{F}\, O_k < C]$ holds (the computed probability is 1) in about 5.0 seconds; ii) $\mathbf{P}_{=?}[\mathbf{G}\,\mathbf{F}\, O_k = C]$ is false (the computed probability is 0) in about 5.3 seconds; iii) $\mathbf{P}_{=?}[\mathbf{F}\, O_k = C]$ holds for about 42% of the initial states, does not hold for 4% of the initial states, and has a probability $p \in (0, 1)$ in the remaining 54% of the initial states. The last two properties aim at computing the probability to reach a state where the number of hospitalised is different/equal (resp.) to the number of available beds in the hospital. We also asked `PRISM` to compute the vector of steady-states with the corresponding probabilities (it took about 133.0 seconds) as well as the transient probabilities after 1000 time units (about 7.75 seconds).

Scaling to large population size. These experiments have been carried out with a small population. However, we remark that the population can be scaled using quantisation, as in [25]. The price to pay in this case is an accuracy reduction due to the quantisation error. Indeed, approximating a population of $\hat{N}$ with N representatives, accounts for $\hat{N}/N$ subjects per representative. Since each representative can be either one or zero, we have a maximum approximation error of $\hat{N}/(2N)$ subjects. As an example, modelling with $N = 20$ representatives (as discussed above) a population of one million (a medium sized city), we can estimate a maximum error of $2.5 \cdot 10^4$ subjects, which corresponds to an error of less than 2.5% of the population, i.e., a relative error of $\hat{N}/(2N) \cdot 1/\hat{N} = 1/(2N)$.

5 Conclusions and future work

A new stochastic model for epidemiology has been proposed. Starting from the classic susceptible, infected, recovered (SIR) model, we have added additional states, i.e. asymptomatic A, hospitalised O, dead D, recover from asymptomatic Ra and quarantined Q, in order to model the peculiarities of a COVID-19-like infection. The key advantages of the model are: 1. the model is control oriented and is analytical, therefore it exposes all parameters and decision variables that make for design and evaluation of control laws; 2. The model captures in a detailed way the stochastic nature of the virus transmission and the impact of resource limitations (hospital beds, number of test swabs). The price to pay for this level of detail is the scalability of the analysis. For illustrative purposes, we have shown numeric examples related to the evolution of a small population and to the application of PMC to assess the validity of PLTL properties. This work poses the basis for several future works in different directions. At model level, the command variables M and t (assumed deterministic in this paper) could be generalised to stochastic variables with "controllable" mean. Second, the people in need of intensive care who are denied access to the hospital because of a saturation of the available sets currently remain in the $\mathcal{I}_k$ set (see (16)). A different possibility is to introduce a new state to express people in urgent need

[2] For the definitions of PLTL syntax and semantics see [13, 18], and visit `http://www.prismmodelchecker.org/`.

of care with high probability of death. This choice will have to be validated by considering the closest match with the existing data. Third, our model considers a restricted circle of people with no interactions with the remaining population. We will explore different possibilities to guarantee scalability in the analysis. A first possibility is to model a number of groups that evolve "almost" independently, except for a few possible interactions that could take place in mild lockdown situations (e.g., schools, sport activities, outdoor recreational activities, museums, cinemas, etc.). Such interactions could affect only the unobservable part of the different circles (i.e., the sets $\mathcal{S}$, $\mathcal{A}$, and $\mathcal{R}a$). In this case we can expect the probability transition matrix would have dominant diagonal blocks and a few off diagonal terms accounting for the interaction. The synthesis of the optimal control policy could be done applying the technique suggested by Haksar and Schwager [17]. A second possibility is to consider the abstraction technique suggested Nasir et al. [25]. In this case each individual would be considered as the representative of a few tens of people. A key issue would be in this case to produce a realistic analytical evaluation of the error made in probability. Third, carry out an accurate tuning of the probabilities, given the historical series that are now available. However, due to the heterogeneity of the aggregated data and the partial coverage with respect to the model state, this is a non trivial task. An additional possibility is to consider continuous–time finite state stochastic processes in which the state of the patient could switch at arbitrary times. In this class fall semi-Markov models and continuous–time Markov chains. While the former could be more general, the latter are based on exponentially distributed state–transitions and preserve the memory-less (Markov) property. We expect that this fact could significantly simplify the analysis and reduce the complexity of the approach. From a technological point of view, we aim at a tighter integration of our proof of concept tool with `PRISM` to avoid the generation and parsing of large files making the analysis of the full model tractable for meaningful population sizes. Preliminary results are in [28]. Moreover, we want to investigate the use of abstraction techniques to improve performance and, thus, including in the picture the vaccinated population, which may be studied to establish the interventions to reach, e.g., herd immunity. Finally, we want to investigate the use of POMDP and the PLTL automatic policy synthesis.

Acknowledgements. Work partially funded by project 40900028, Bando 2020 Università di Trento "Covid 19", lead by M. Roveri.

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A Simplified Markov Model: The Case of Untested Subjects

In this section, we focus on the case in which subjects are not tested (and hence are not quarantined), which is usually the situation at the beginning of a disease as COVID-19 where no clear medical test were known or developed. The possible states of a subject are the ones depicted in Figure 2 where $\Delta_3 = \Delta_9 = \Delta_{10} = \Delta_{11} = 0$ and where $|Q_k| = |R_{a,k}| = 0$, $\forall k$. Hence, the state of the Markov chain will be only associated to the 6-tuple given by $(S_k, A_k, I_k, R_k, O_k, D_k)$, while the constraints (2), the balance equations (4) and the flow constraints (5) are still valid under the previously reported assumption. As a consequence, the dynamic of the state vector

$$\mathbf{V}_k = \Big[S_k \;\; A_k \;\; I_k \;\; R_k \;\; O_k \;\; D_k\Big]^T,$$

will obey the transition probability rules in (3), where of course

$$\boldsymbol{\Delta} v = \mathbf{v}' - \mathbf{v} = \Big[\Delta_S \;\; \Delta_A \;\; \Delta_I \;\; \Delta_R \;\; \Delta_O \;\; \Delta_D\Big]^T.$$

With the same steps of the previous case and besides the variable assignment $l_1(\cdot)$, we introduce the shorthand notations for the variable assignments:

- l_2, defined as a function depending of δ_2 and δ_3, with the variable δ_3 obtained via equation B_2, that is l_2: $(\Delta_2 = \delta_2, \Delta_3 = -\Delta_S - \Delta_A - \delta_2)$;
- l_3, defined as a function depending of $\delta_4, \delta_5, \delta_6$, that is $l_3(\delta_4, \delta_5)$ and with δ_6 obtained via equation B_3, l_3: $(\Delta_4 = \delta_4, \Delta_5 = \delta_5, \Delta_6 = \delta_2 - \Delta_I - \delta_4 - \delta_5)$;
- l_4, defined as a function depending of $\delta_2, \delta_4, \delta_5$, with the relations obtained from B_4 and B_5, l_4: $(\Delta_7 = \Delta_D + \Delta_I - \delta_2 + \delta_4 + \delta_5, \Delta_8 = \delta_2 - \delta_4 - \Delta_D - \Delta_I - \Delta_O)$.

Proceeding as in Section 3, we can state the following:

Theorem 5 *The transition probability* (6) *is re-written as*

$$\begin{aligned}\Pr\{\boldsymbol{\Delta} \models B \,|\mathbf{V}_k = \mathbf{v}\} = \Pr\{l_1 \,|\mathbf{V}_k = \mathbf{v}\} \\ \sum_{\delta_2=0}^{-\Delta_S-\Delta_A} \Pr\{l_2 \,|\mathbf{V}_k \wedge l_1\} \cdot \\ \sum_{\delta_4=0}^{\delta_2-\Delta_I} \sum_{\delta_5=0}^{\delta_2-\Delta_I-\delta_4} (\Pr\{l_3 \,|\mathbf{V}_k \wedge l_1 \wedge l_2\} \cdot \\ \Pr\{l_4 \,|\mathbf{V}_k \wedge l_1 \wedge l_2 \wedge l_3\}).\end{aligned} \tag{A.1}$$

The proof is a simplified version of Theorem 1. We can now move to the computation of the four different probabilities that appear in (A.1).

A.1 Probability that a susceptible subject becomes infectious

The probability of $\Pr\{g_k \,|\mathbf{V}_k\}$ in this simplified case follows exactly the same lines of Section 3.1, where, obviously, (10) turns to

$$\Pr\{g_k \,|\mathbf{V}_k\}_M = 1 - \left(1 - \omega \frac{A_k}{N - D_k - I_k - O_k}\right)^M.$$

A.2 Probability of state change for a number of subjects

The probabilities of state change in (A.1) can be computed considering as reference Figure 2, where $\Delta_3 = \Delta_9 = \Delta_{10} = \Delta_{11} = 0$ and where $|Q_k| = |R_{a,k}| = 0$, $\forall k$.

Transition from State S. Nothing changes with respect to Section 3. Hence, the term $\Pr\{l_1 \,|\mathbf{V}_k\}$ is the same as in equation (12).

Transition from state A: Since the probability of the event "exactly δ_3 asymptomatic infected subjects recover at step k and δ_2 subjects become symptomatic" is given by Theorem 3, for the assignment l_2, we have

$$\Pr\{l_2 \,|\mathbf{V}_k \wedge l_1\} = \rho(\delta_2, -\Delta_S - \Delta_A - \delta_2, \mathbf{V}_k). \tag{A.2}$$

Transition from state I: With respect to the case considered in Section 3, the only difference is given by the constrained value of δ_6, i.e.

$$\begin{aligned}\Pr\{l_3 \,|\mathbf{V}_k \wedge l_1 \wedge l_2\} = \\ \phi(\delta_4, \delta_5, \delta_2 - \delta_4 - \delta_5 - \Delta_I, \mathbf{V}_k).\end{aligned} \tag{A.3}$$

Transition from State O: This case is identical to the previous case of Section 3, so we have

$$\begin{aligned}\Pr\{l_4 \,|\mathbf{V}_k \wedge l_1 \wedge l_2 \wedge l_3\} = \\ \zeta(\delta_D + \delta_I - \delta_2 + \delta_4 + \delta_5, \\ \delta_2 - \delta_4 - \Delta_D - \Delta_I - \Delta_O, \mathbf{V}_k).\end{aligned} \tag{A.4}$$

The computation of the transition probabilities is thus obtained by plugging (12), (A.2), (A.3) and (A.4) into (A.1).