# The Stochastic Reproduction Rate of a Virus

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**Abstract**

We consider an SIR model where the probability of infections between infected and susceptible individuals are viewed as Poisson trials. The probabilities of infection between pairwise susceptible-infected matches are thus order statistics. This implies that the reproduction rate is a random variable. We derive the first two moments of the distribution of $\mathcal{R}_t$ conditional on the information available at time $t-1$ for Poisson trials drawn from an arbitrary parent distribution with finite mean. We show that the variance of $\mathcal{R}_t$ is increasing in the proportion of susceptible individuals in the population, and that *ex ante* identical populations can exhibit large differences in the path of the virus. This has a number of implications for policy during pandemics. We provide a rationale for why shelter-in-place orders may be a better containment measure than mandating the use of masks because of their impact on the variance of the reproduction rate.

## 1 Introduction

The COVID-19 pandemic has shown that the effective reproduction rate of the virus $\mathcal{R}_t$ is a crucial determinant not only if public health, but also of public policy. Social distancing, shelter-in-place and other containment measures aim to stop the spread of the virus, essentially by attempting to push $\mathcal{R}_t$ below 1.

It has been clear to epidemiologists, public health officials, and economists that with $\mathcal{R}_t > 1$ the virus spreads exponentially—overwhelming health systems and leading to substantial loss of life.

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Despite the critical importance of the reproduction rate it is treated in epidemiological models as a parameter, rather than as a random variable. Yet the primitives of so-called SIR models are, from a behavioral perspective, the proportion of the population that is infected and susceptible, the rate at which those sub-populations interact, and the probability that a given interaction leads an infected individual to infect a susceptible individual.

Those primitives *determine* the reproduction rate, yet $\mathcal{R}_t$ is typically modeled as an exogenous variable. The point-of-view we take in this paper is to derive $\mathcal{R}_t$ from primitives and explicitly model the stochastic nature of those primitives. This leads one to the seemingly obvious conclusion that $\mathcal{R}_t$ is a random variable—and as such it has an associated probability distribution. An immediate consequence of this is that the rate of susceptibility, infection, and recovery in a population are also random variables with their own probability distributions.

The first main result we derive is that the variance of $\mathcal{R}_t$ at any point in time, conditional on the information available that time, is increasing in the proportion of the population that is susceptible at that time. This implies that early on in an epidemic–when the susceptible proportion of the population is low–the variance of infection rates is the highest. This, in turn, implies that two *ex ante* identical jurisdictions could have very different infection rates simply due to chance, and this can create persistent difference in the path of the virus in those jurisdictions.

An optimal policy for controlling $\mathcal{R}_t$ must account for the complete distribution of $\mathcal{R}_t$ and the amount of mass of its probability distribution that lies on the part of the support above $\mathcal{R}_t = 1$. All else equal the amount of mass in this "danger zone" is larger when the proportion of the population that is susceptible is larger—i.e. at the onset of the virus. This implies that the optimal containment policy is stricter earlier in the evolution of the virus.

The remainder of the paper proceeds as follows. In section 2 we outline the statement of the problem. Section 3 characterizes the first two moments of the distribution of $\mathcal{R}_t$. Section 4 derives a number of implications for decision-makers and concludes. Proofs of results not contained in the text are relegated to an appendix.

# 2 The Model

## 2.1 Statement of the Problem

There are $N$ agents that are infinitely lived. Time is discrete and indexed by $t = 0, 1, 2, ....$ At each time $t$ an agent is in one of three states: *susceptible*, *infected*, or *recovered*. There are $S_t$ susceptible agents in the population, there are $I_t$ infected agents, and the number of recovered individuals is $R_t = N - S_t - I_t$, where $S_t, I_t, R_t \in \mathbb{N}$.

Following the standard convention, we shall refer to $I_t$ as the *prevalence* of the disease at time $t$.

At time $t$ each agent is pairwise random matched with $\phi = \phi(N)$ other agents *independently* with equal probability among agents irrespective of health status, where $\phi(N)$ captures the intensity of social interactions in the population. We assume that $\phi\colon \mathbb{N} \to \mathbb{N}$ is an integer valued function satisfying $0 \leq \phi(N) \leq N$ for all $N \in \mathbb{N}$. This allows us to consider both the simple case where $\phi(N) = d \in \mathbb{N}$ for all $N$, and more complex cases where for example $\phi(N) = \lfloor \log(N) \rfloor$ (here, $\lfloor \cdot \rfloor$ denotes the *floor* function, though we routinely omit floor and ceiling signs for readability). For notational convenience later on we also define here $m = \phi/N$ to be the proportion of the population that an individual matches up with in a given time period.

An interaction structure in the population can be formed by considering the *random regular graph* formed by the well known *configuration model* [6, 49] with $N$ individuals or vertices and degree sequence $(d_1, \dots d_N) = (\phi, \dots, \phi)$, conditioning on getting a simple graph. In the limit of a large population size, and for small $\phi(N)$, matches formed by the configuration model are approximately independent (this technique is used in [18] for example). Some notes on the reproductive rate with the independence condition removed are provided in the appendix.

If an infected agent $j$ is matched with a susceptible agent $k$ then with probability $p_j$ the susceptible agent becomes infected. We assume that $p_j$ is drawn from a distribution $\mathcal{D}$ on support $[0, 1]$ with finite mean $\mu$, and that the draw for each infected-susceptible match is stochastically independent.

Recovered agents are assumed to be immune to infection and are not contagious, and infected agents spontaneously recover with probability $\gamma \geq 0$.

## 2.2 Discussion

This paper contributes to the literature on heterogeneity (or 'heterogeneous infection rates' if you prefer) in epidemic models, much in the spirit of the now classic [21]. In

the existing literature, studies have focused on heterogeneity at the group level [28, 9, 3, 13, 8, 44, 43] (subdividing a heterogeneous population into homogeneous groups) as well as individual level [27, 41, 32, 22], whilst other authors have taken the approach of incorporating exogenous shocks into their models. Such shock are usually modeled by introducing discontinuities into the deterministic SIR (as well as SEIR, SIS, and SI) systems of differential equations via stochastic jumps or 'noise' [45, 47, 52, 53, 34]. Several studies built on variants of the SIR model have been applied directly to analysing the Covid-19 epidemic [51, 43, 39, 16], with some recent models taking into account the economic costs of various policy interventions (e.g. [1, 5, 11, 16, 12]). A significant amount of work has been done to describe the behavior of epidemics on networks with heterogeneous individuals [33, 29, 4, 30, 19, 46, 50, 40, 7, 15, 17], often making use of results from bond percolation or applying mean field approximations (e.g. see [35]).

Models which account for individual heterogeneity have become especially important in light of the accumulating theoretical [28, 26, 24, 32, 7] and empirical [10, 48, 14, 20, 4, 31] evidence that heterogeneity in a population strongly influences (and in particular, lowers) the probability of epidemic invasion.

Most of the models in the literature–with the exception of a few [8, 40, 22, 7] involve continuous time. In the way of discrete SIR models some work has been done (see [2] for example), though the disease-spreading mechanism that we employ has, as far as we are aware, not been considered in the epidemiological literature. Moreover, whilst many papers calculate the basic reproductive number $R_0$ under their models, this parameter –and consequently, the effective reproductive number $\mathcal{R}_t$ –often fail to accurately capture the threshold properties of epidemics, and can vary dramatically according to the method by which they are calculated [8, 23, 37]. For example, the observations of of super-spreading events (SSE's) which is well documented for the SARS outbreak of 2003 [38, 25, 42, 27] represent tail end draws from the (random) reproductive rate. Our main innovation is the explicit focus on the reproductive rate as a random variable, and our novel modeling approach of infected-susceptible matches as non-identical Bernoulli "trials"–or *Poisson trials* [36]. The paper perhaps closest to ours is [27].

Before we proceed with the analysis we pause briefly to remark that our main result about the variance of the reproduction rate at a particular point in time is stated as being conditional on the information available to an observer at that time. There are two reasons for this. The first is conceptual. Any policy maker should care about the variance at a given time accounting for the information that she has. Calculating the unconditional variance is to discard useful information. The second reason we focus on the conditional variance is practical. One can analytically derive the unconditional variance, but it has

a somewhat unwieldy recursive structure. In any case, we view it as being of limited interest for policy makers concerned with the evolution of a virus.

# 3 Analysis

## 3.1 The Distribution of the Reproduction Rate

We seek to characterize the distribution of the reproduction rate of the virus at time $t$, which we denote $\mathcal{R}_t$.

Notice that a given infected agent has $\phi$ pairwise random matches in the population and that infections can only occur when they are matched with a susceptible agent (which have proportion $S_t/N$ in the population). Assuming there is independence between matches (more on this assumption later), the expected number of susceptible matches of a given infected individual is therefore $\phi S_t/N$. Recalling that we defined $m = \phi/N$ we can write this as $mS_t$

Denote the probability that given such a match infection occurs as $p_j$. We define $\mathcal{R}_t$ as the random variable counting the number of people that are infected by a given infected person at time $t$. That is, we have

$$\mathcal{R}_t = \sum_{j=1}^{mS_t} \mathbb{I}_j, \tag{1}$$

where $\mathbb{I}_j$ is an indicator variable taking the value 1 if match $j$ leads to an infection and $0$ otherwise. That is, we define $\mathbb{I}_j$ by the conditional distribution

$$\mathbb{P}(\mathbb{I}_j = x \mid p_j = p) = xp + (1-x)(1-p).$$

Recalling that the mean of $p_j$ is finite and denoted by $\mu$, we may calculate the expectation of the indicator variable $\mathbb{I}_j$ by the *law of iterated expectations* as

$$E(\mathbb{I}_j) = E\big(E(\mathbb{I}_j \mid p_j)\big) = E\big(\mathbb{P}(\mathbb{I}_j = 1 \mid p_j)\big) = \mathbb{E}(p_j) = \mu. \tag{2}$$

We also ignore potential integer problems in the upper limit of the summation in the definition of $\mathcal{R}_t$, one could avoid this by explicitly taking $\lfloor mS_t \rfloor$ as the upper limit.

Defining $\mathcal{R}_t$ in this way gives us something analogous to a discrete-time SIR model which we will discuss how to specify shortly. By way of reference, we describe the classic discrete-time SIR model here. Let $\Delta S_{t+1} = S_{t+1} - S_t$ and define $\Delta I_{t+1}$ and $\Delta R_{t+1}$ similarly.

The classic discrete-time SIR model can be described as

$$\Delta S_{t+1} = -\beta I_t S_t \tag{3}$$

$$\Delta I_{t+1} = \beta I_t S_t - \gamma I_t \tag{4}$$

$$\Delta R_{t+1} = I_t \gamma, \tag{5}$$

where $\beta$ is the average rate of infection. Our model is substantially more complex than this since we account for the distribution of $\mathcal{R}_t$ and not just the point estimate $\beta$. As such, our rates of susceptibility, infection, and recover will also be random variables determined by some initial state vector $(S_0, I_0, R_0)$.

Let $\mathcal{R}_t^{pop}$ denote the population-level reproductive rate, that is, a random variable whose distribution is equal to the number of new infections in the population from time $t$ to time $t+1$. We saw that in the calculation of $\mathcal{R}_t$, the expected number of susceptible matches of a given infected individual is $mS_t$. Since there are $I_t$ infected individuals in the population at time $t$, and each infected-susceptible match is assumed to be independent, we have

$$\mathcal{R}_t^{pop} = \sum_{j=1}^{I_t m S_t} \mathbb{I}_j. \tag{6}$$

Taking $\phi(N) = \phi \in \mathbb{N}$ is sufficient for the independence properties we require, henceforth we assume $\phi \in \mathbb{N}$ is a constant. As a result, our SIR equations become

$$\Delta S_{t+1} = -\sum_{j=1}^{m I_t S_t} \mathbb{I}_j. \tag{7}$$

$$\Delta I_{t+1} = \sum_{j=1}^{m I_t S_t} \mathbb{I}_j - \gamma I_t \tag{8}$$

$$\Delta R_{t+1} = \gamma I_t. \tag{9}$$

We suppress the dependence of the indicator variables on the time period $t$ for notational convenience, in reality each new time period yields a new set of indicator variables $\mathbb{I}_j$ which are independent of those in any other time period. Notice by Wald's identity we have

$$\mathbb{E}\left(\mathcal{R}_t^{pop}\right) = \mathbb{E}\left(\sum_{j=1}^{m I_t S_t} \mathbb{I}_j\right) = \mathbb{E}(m S_t I_t)\mathbb{E}(\mathbb{I}_j) = m\mu\mathbb{E}(S_t I_t),$$

and hence

$$\begin{aligned}\mathbb{E}(\Delta S_{t+1}) &= -m\mu\mathbb{E}(S_t I_t).\\ \mathbb{E}(\Delta I_{t+1}) &= m\mu\mathbb{E}(S_t I_t) + \gamma\mathbb{E}(I_t)\\ \mathbb{E}(\Delta R_{t+1}) &= \gamma\mathbb{E}(I_t).\end{aligned}$$

That is, our model coincides with the standard model in expectation, with average infection rate $\beta = m\mu$.

Consider a policy maker who, at time $t$, knows the current state $\theta_{t-1} = \{S_{t-1}, I_{t-1}\}$ of susceptibility and infection in the population at all time periods before $t$. They will use this information to inform their policy. The key parameters of interest therefore are the mean and variance of $\mathcal{R}_t$, conditioned on knowing the history $\theta_{t-1}$. That is, we want to find expressions for $\mathbb{E}(\mathcal{R}_t \mid \theta_{t-1})$ and $\mathrm{Var}(\mathcal{R}_t \mid \theta_{t-1})$.

We begin by calculating $\mathbb{E}(\mathcal{R}_t \mid \theta_{t-1})$. By noting that $S_t$ is independent of the infection event captured by $\mathbb{I}_j$ for all $t, j \in \mathbb{N}$, we can calculate the conditional expectation of $\mathcal{R}_t$ by *Wald's identity* and (2) as

$$\begin{aligned}\mathbb{E}(\mathcal{R}_t \mid \theta_{t-1}) &= \mathbb{E}(mS_t \mid \theta_{t-1})E(\mathbb{I}_j \mid \theta_{t-1})\\ &= m\mathbb{E}(S_t \mid \theta_{t-1})E(\mathbb{I}_j)\\ &= m\mu\mathbb{E}(S_t \mid \theta_{t-1}).\end{aligned} \tag{10}$$

Therefore, the mean of $\mathcal{R}_t$ depends crucially on the mean of $S_t$, the number of susceptible individuals in the population at time $t$. We now derive the mean and variance of $S_t$ conditional on knowing the history of susceptibility and infection. This will allow us to derive the conditional mean and variance of $\mathcal{R}_t$. We work towards the theorem below in what follows.

**Theorem 1** *For all $t \in \mathbb{N}$*

1. $$\mathbb{E}(\mathcal{R}_t \mid \theta_{t-1}) = m\mu S_{t-1} + m^2\mu^2 I_{t-1}S_{t-1}.$$

2. $$\mathrm{Var}(\mathcal{R}_t \mid \theta_{t-1}) = m\mu(1-\mu)S_{t-1} + m^2\mu^2(1-\mu)(1+m\mu)I_{t-1}S_{t-1}.$$

We use the next result several times in the proofs that follow, so we state it here as a lemma.

**Lemma 1** *For all $t \in \mathbb{N}$, we have*

$$\mathbb{E}(\Delta S_t \mid \theta_{t-1}) = -m\mu I_{t-1} S_{t-1}.$$

**Proof.** The result can be obtained by a single application of Wald's identity. We thus have

$$\begin{aligned}
\mathbb{E}(\Delta S_t \mid \theta_{t-1}) &= \mathbb{E}\left( - \sum_{j=1}^{mI_{t-1}S_{t-1}} \mathbb{I}_j \;\middle|\; \theta_{t-1} \right) \\
&= -\mathbb{E}(mI_{t-1}S_{t-1} \mid \theta_{t-1})\mathbb{E}(\mathbb{I}_j \mid \theta_{t-1}) \\
&= -m\mu I_{t-1} S_{t-1},
\end{aligned}$$

as desired. ■

We now prove Part 1. of Theorem 1.

**Proof of Part 1.** We can compute $\mathbb{E}(S_t \mid \theta_{t-1})$ directly by noting that $S_t = S_{t-1} + \Delta S_t$. This gives us that

$$\begin{aligned}
\mathbb{E}(S_t \mid \theta_{t-1}) &= \mathbb{E}\left(S_{t-1} + \Delta S_t \mid \theta_{t-1}\right) \\
&= S_{t-1} + \mathbb{E}\left(\Delta S_t \mid \theta_{t-1}\right).
\end{aligned}$$

Now applying Lemma 1, we have

$$\mathbb{E}(S_t \mid \theta_{t-1}) = S_{t-1} - m\mu I_{t-1} S_{t-1}. \tag{11}$$

Applying (10) yields the desired result. ■

We will need to establish a few more lemmas in order to prove Part 2. of Theorem 1. We being by proving an equation for the conditional variance of $\mathcal{R}_t$ in terms of the conditional expectation and variance of $S_t$.

**Lemma 2** *For all $t \in \mathbb{N}$, we have*

$$\operatorname{Var}(\mathcal{R}_t \mid \theta_{t-1}) = m\mu(1-\mu)\mathbb{E}(S_t \mid \theta_{t-1}) + m^2\mu^2 \operatorname{Var}(S_t \mid \theta_{t-1}). \tag{12}$$

**Proof.** First, we have

$$\begin{aligned}\mathcal{R}_t^2 &= \left(\sum_{j=1}^{mS_t} \mathbb{I}_j\right)\left(\sum_{l=1}^{mS_t} \mathbb{I}_l\right) \\ &= \sum_{j=l} \mathbb{I}_j\mathbb{I}_l + \sum_{j\neq l} \mathbb{I}_j\mathbb{I}_l. \\ &= \sum_{j=l} \mathbb{I}_j + \sum_{j\neq l} \mathbb{I}_j\mathbb{I}_l.\end{aligned}$$

There are $(mS_t)^2$ terms in the expansion of $\mathcal{R}_t^2$. Exactly $mS_t$ of these fall under the sum where $j = l$, and the remaining $(mS_t)^2 - mS_t$ of them fall under the sum where $j \neq l$. Hence by Wald's identity and (2), we have

$$\begin{aligned}\mathbb{E}\left(\mathcal{R}_t^2 \mid \theta_{t-1}\right) &= \mathbb{E}\left(\sum_{j=l} \mathbb{I}_j \mid \theta_{t-1}\right) + \mathbb{E}\left(\sum_{j\neq l} \mathbb{I}_j\mathbb{I}_l \mid \theta_{t-1}\right) \\ &= \mathbb{E}(mS_t \mid \theta_{t-1})\mathbb{E}(\mathbb{I}_j \mid \theta_{t-1}) + \mathbb{E}\left((mS_t)^2 - mS_t \mid \theta_{t-1}\right)\mathbb{E}(\mathbb{I}_j\mathbb{I}_l \mid \theta_{t-1}) \\ &= \mu\mathbb{E}(mS_t \mid \theta_{t-1}) + \mu^2\mathbb{E}\left((mS_t)^2 - mS_t \mid \theta_{t-1}\right) \\ &= m\mu(1-\mu)\mathbb{E}(S_t \mid \theta_{t-1}) + m^2\mu^2\mathbb{E}(S_t^2 \mid \theta_{t-1}). \qquad (13)\end{aligned}$$

Where we have used independence of indicator variables on the second sum. Hence utilizing (10) and (13), the variance of $\mathcal{R}_t$ is given by

$$\begin{aligned}\mathrm{Var}(\mathcal{R}_t \mid \theta_{t-1}) &= \mathbb{E}(\mathcal{R}_t^2 \mid \theta_{t-1}) - \left(\mathbb{E}(\mathcal{R}_t \mid \theta_{t-1})\right)^2 \\ &= m\mu(1-\mu)\mathbb{E}(S_t \mid \theta_{t-1}) + m^2\mu^2\mathbb{E}(S_t^2 \mid \theta_{t-1}) - \left(m\mu\mathbb{E}\left(S_t \mid \theta_{t-1}\right)\right)^2 \\ &= m\mu(1-\mu)\mathbb{E}(S_t \mid \theta_{t-1}) + m^2\mu^2\,\mathrm{Var}(S_t \mid \theta_{t-1}),\end{aligned}$$

as required. ■

We require one more lemma before we complete the proof of Theorem 1.

**Lemma 3** *For all $t \in \mathbb{N}$ we have*

$$\mathbb{E}\left((\Delta S_t)^2 \mid \theta_{t-1}\right) = m\mu(1-\mu)I_{t-1}S_{t-1} + (m\mu I_{t-1}S_{t-1})^2.$$

**Proof.** The proof is similar to the proof of Lemma 2. We first note that

$$\begin{aligned}(\Delta S_t)^2 &= \left(-\sum_{j=1}^{mI_{t-1}S_{t-1}} \mathbb{I}_j\right)\left(-\sum_{l=1}^{mI_{t-1}S_{t-1}} \mathbb{I}_l\right)\\ &= \sum_{j=l} \mathbb{I}_j\mathbb{I}_l + \sum_{j\neq l} \mathbb{I}_j\mathbb{I}_l.\\ &= \sum_{j=l} \mathbb{I}_j + \sum_{j\neq l} \mathbb{I}_j\mathbb{I}_l.\end{aligned}$$

There are $(mI_{t-1}S_{t-1})^2$ terms in the expansion of $(\Delta S_t)^2$. Exactly $mI_{t-1}S_{t-1}$ of these fall under the sum where $j = l$, and the remaining $(mI_{t-1}S_{t-1})^2 - mI_{t-1}S_{t-1}$ of them fall under the sum where $j \neq l$. Hence by (2), we have

$$\begin{aligned}\mathbb{E}\left((\Delta S_k)^2 \mid \theta_{t-1}\right) &= \mathbb{E}\left(\sum_{j=l} \mathbb{I}_j \mid \theta_{t-1}\right) + \mathbb{E}\left(\sum_{j\neq l} \mathbb{I}_j\mathbb{I}_l \mid \theta_{t-1}\right).\\ &= \mu\mathbb{E}(mI_{t-1}S_{t-1} \mid \theta_{t-1}) + \mu^2\mathbb{E}\left((mI_{t-1}S_{t-1})^2 - mI_{t-1}S_{t-1} \mid \theta_{t-1}\right)\\ &= m\mu(1-\mu)I_{t-1}S_{t-1} + (m\mu I_{t-1}S_{t-1})^2, \qquad (14)\end{aligned}$$

as required. ■

We now prove Part 2. of Theorem 1.

**Proof of Part 2.** We want to find a closed-form expression for $\mathrm{Var}(\mathcal{R}_t \mid \theta_{t-1})$. We will require (11) which we proved in Part 1. of Theorem 1. We recall this equation here as

$$\mathbb{E}\left(S_t \mid \theta_{t-1}\right) = S_{t-1} - m\mu I_{t-1}S_{t-1}.$$

Hence by Lemma 2 it only remains to calculate $\mathbb{E}\left(S_t^2 \mid \theta_{t-1}\right)$. Noting that

$$S_t^2 = (S_{t-1} + \Delta S_t)^2 = S_{t-1}^2 + 2S_{t-1}\Delta S_t + (\Delta S_t)^2$$

we can use Lemma 1 and Lemma 3 to compute

$$\begin{aligned}\mathbb{E}(S_t^2 \mid \theta_{t-1}) &= S_{t-1}^2 + 2S_{t-1}\mathbb{E}(\Delta S_t \mid \theta_{t-1}) + \mathbb{E}\left((\Delta S_t)^2 \mid \theta_{t-1}\right)\\ &= S_{t-1}^2 + 2S_{t-1}\left(-m\mu I_{t-1}S_{t-1}\right) + \left(m\mu(1-\mu)I_{t-1}S_{t-1} + (m\mu I_{t-1}S_{t-1})^2\right)\\ &= m\mu(1-\mu)I_{t-1}S_{t-1} + S_{t-1}^2 - 2m\mu I_{t-1}S_{t-1}^2 + (m\mu I_{t-1}S_{t-1})^2. \qquad (15)\end{aligned}$$

Further, we have by (11) that

$$\begin{aligned}(\mathbb{E}(S_t \mid \theta_{t-1}))^2 &= (S_{t-1} - m\mu I_{t-1}S_{t-1})^2 \\ &= S_{t-1}^2 - 2m\mu I_{t-1}S_{t-1}^2 + (m\mu I_{t-1}S_{t-1})^2 \end{aligned} \tag{16}$$

Now using (15) and (16) we can calculate

$$\begin{aligned}\operatorname{Var}(S_t \mid \theta_{t-1}) &= \mathbb{E}(S_t^2 \mid \theta_{t-1}) - (\mathbb{E}(S_t \mid \theta_{t-1}))^2 \\ &= \left(m\mu(1-\mu)I_{t-1}S_{t-1} + S_{t-1}^2 - 2m\mu I_{t-1}S_{t-1}^2 + (m\mu I_{t-1}S_{t-1})^2\right) \\ &\quad - \left(S_{t-1}^2 - 2m\mu I_{t-1}S_{t-1}^2 + (m\mu I_{t-1}S_{t-1})^2\right) \\ &= m\mu(1-\mu)I_{t-1}S_{t-1}. \end{aligned} \tag{17}$$

Finally, combining (17) and (11) together with Lemma 2, we have

$$\begin{aligned}\operatorname{Var}(\mathcal{R}_t \mid \theta_{t-1}) &= m\mu(1-\mu)\mathbb{E}(S_t \mid \theta_{t-1}) + m^2\mu^2 \operatorname{Var}(S_t \mid \theta_{t-1}) \\ &= m\mu(1-\mu)\left(S_{t-1} - m\mu I_{t-1}S_{t-1}\right) + m^2\mu^2\left(m\mu(1-\mu)I_{t-1}S_{t-1}\right) \\ &= m\mu(1-\mu)S_{t-1} - m^2\mu^2(1-\mu)(1-m\mu)I_{t-1}S_{t-1}, \end{aligned}$$

completing the proof. ■

The first consequence of Theorem 1 is the following important Corollary.

**Corollary 1** *For sufficiently large $N$, the variance of $\mathcal{R}_t$ given $\theta_{t-1}$ is increasing in $S_{t-1}$.*

In particular the proof of Corollary 1 which we present below shows that the conditional variance of $\mathcal{R}_t$ is increasing in $S_{t-1}$ when $t$ is small, since at the beginning of the SIR process, infection rates are low and susceptibility is high. This implies that if variance is a concern to policy makers, then containment policies should be stricter earlier on in the spread of the virus.

**Proof.** Note firstly that an immediate decrease in $S_{t-1}$ translates into a commensurate increase in $I_{t-1}$, that is, an individual who is no longer susceptible must have gotten infected. As such, it makes sense to think about an *increase* in $S_{t-1}$ as resulting from a commensurate *decrease* in $I_{t-1}$. One can think of this as having an additional susceptible individual at time $t-2$ who did not get infected by any of the $I_{t-2}$ infected individuals at that time period.

Now, suppose that we fix $S_{t-1} \in \mathbb{N}$ and $I_{t-1} \in \mathbb{N}$ with $S_{t-1} + I_{t-1} \leq N$. Consider what happens when the number of susceptible individuals at time $t-1$ increases by $1$.

Formally, let $S'_{t-1} = S_{t-1} + 1$ and let $I'_{t-1} = I_{t-1} - 1$, now consider what happens when $S_{t-1} \to S'_{t-1}$ and $I_{t-1} \to I'_{t-1}$. Letting $\theta'_{t-1} = \{S'_{t-1}, I'_{t-1}\}$, we have

$$\begin{aligned}
\mathrm{Var}(\mathcal{R}_t \mid \theta'_{t-1}) &= m\mu(1-\mu)S'_{t-1} - m^2\mu^2(1-\mu)(1-m\mu)I'_{t-1}S'_{t-1} \\
&= m\mu(1-\mu)\left(S_{t-1}+1\right) - m^2\mu^2(1-\mu)(1-m\mu)\left(I_{t-1}-1\right)\left(S_{t-1}+1\right) \\
&= \mathrm{Var}(\mathcal{R}_t \mid \theta_{t-1}) + m\mu(1-\mu) - m^2\mu^2(1-\mu)(1-m\mu)\left(I_{t-1}-S_{t-1}-1\right).
\end{aligned} \tag{18}$$

Now (18) tells us that $\mathrm{Var}(\mathcal{R}_t \mid \theta_{t-1})$ is increasing in $S_{t-1}$ if and only if

$$m\mu(1-\mu) - m^2\mu^2(1-\mu)(1-m\mu)\left(I_{t-1}-S_{t-1}-1\right) > 0. \tag{19}$$

Supposing $m \neq 0$ such that meetings occur, and that $\mu \neq 0, 1$ such that the Poisson trials are not degenerate, (19) holds if and only if

$$1 - m\mu(1-m\mu)(I_{t-1}-S_{t-1}-1) > 0. \tag{20}$$

If $I_{t-1} = S_{t-1} + 1$ then this equation holds trivially. Else writing $m = \frac{\phi}{N}$ and multiplying throughout by $N$ we can write this as

$$N > \phi\mu\left(1 - \frac{\phi\mu}{N}\right)(I_{t-1}-S_{t-1}-1). \tag{21}$$

Noting that the right hand side of (21) is $O(\phi(N))$ this inequality will hold for $N$ sufficiently large whenever $\phi(N) = o(N)$ (and in fact we have assumed that $\phi(N)$ is a constant), completing the proof. ■

We also present a second corollary regarding the asymptotic conditional variance of $\mathcal{R}_t$.

**Corollary 2** *As $N \to \infty$, we have* $\mathrm{Var}(\mathcal{R}_t \mid \theta_{t-1}) \to m\mu(1-\mu)S_{t-1} - m^2\mu^2(1-\mu)I_{t-1}S_{t-1}$.

**Proof.** The proof follows immediately from noting that

$$1 - m\mu = 1 - \frac{\phi\mu}{N}$$

which converges to 1 as $N \to \infty$. ■

## 3.2 Poisson trials and Superspreaders

The Poisson trials framework we have used allows us to observe that as the population size $N$ grows, the realized distribution of infection probabilities converges to the parent distribution.

**Theorem 2** *Let $X_1, \ldots, X_N$ be random variables drawn from an absolutely continuous distribution $F$, and let $\xi_k = \inf\{x\colon F(x) \geq k\}$ denote the $k$-th quantile of $F$, for $0 < k < 1$. Similarly, let $\hat{\xi}_{Nk} = \inf\{x\colon F_N(x) \geq k\}$ denote the $k$-th quantile of the sample distribution $X_1, \ldots, X_N$, where $F_N(x) = \frac{1}{N}\sum_{i=1}^{N} \mathbb{I}(X_i \leq x)$, and $\mathbb{I}(A)$ is an indicator variable for the event $A$. Then*

$$\mathbb{P}(|\hat{\xi}_{Nk} - \xi_k| > \epsilon) \leq 2\exp(-2N\delta_\epsilon^2),$$

*where $\delta_\epsilon = \min\{F(\xi_k + \epsilon) - k, k - F(\xi_k - \epsilon)\}$. That is, $\hat{\xi}_{Nk} \xrightarrow{P} \xi_k$ exponentially fast.*

**Proof.** This is a well known fact but we provide a proof of one of the bounds for completeness. Let $\epsilon > 0$, and note that

$$\mathbb{P}(|\hat{\xi}_{Nk} - \xi_k| > \epsilon) = \mathbb{P}(\hat{\xi}_{Nk} > \xi_k + \epsilon) + \mathbb{P}(\hat{\xi}_{Nk} < \xi_k + \epsilon).$$

By definition of the sample quantile $\hat{\xi}_{Nk}$, we have

$$\begin{aligned}
\mathbb{P}(\hat{\xi}_{Nk} > \xi_k + \epsilon) &= \mathbb{P}(k > F_N(\xi_p + \epsilon)) \\
&= \mathbb{P}\left(Nk > \sum_{i=1}^{N} \mathbb{I}(X_i \leq \xi_k + \epsilon)\right) \\
&= \mathbb{P}\left(\sum_{i=1}^{N} \mathbb{I}(X_i > \xi_k + \epsilon) > N(1-k)\right).
\end{aligned}$$

Now let $Y_i = \mathbb{I}(X_i > \xi_k + \epsilon)$. Then

$$\mathbb{E}(Y_i) = \mathbb{P}(X_i > \xi_p + \epsilon) = 1 - F(\xi_p + \epsilon).$$

Hence

$$\begin{aligned}
\mathbb{P}\left(\sum_{i=1}^{N} \mathbb{I}(X_i > \xi_k + \epsilon) > N(1-k)\right) &= \mathbb{P}\left(\sum_{i=1}^{N} Y_i - N(1 - F(\xi_k + \epsilon)) > N(F(\xi_p + \epsilon) - k)\right) \\
&= \mathbb{P}\left(\sum_{i=1}^{N} Y_i - \sum_{i=1}^{N} \mathbb{E}(Y_i) > N\delta_1\right),
\end{aligned}$$

where $\delta_1 = F(\xi_k + \epsilon) - k$. Hence by Hoeffding's inequality, we have

$$\mathbb{P}(\hat{\xi}_{Nk} > \xi_k + \epsilon) = \mathbb{P}\left(\sum_{i=1}^{N} Y_i - \sum_{i=1}^{N} \mathbb{E}(Y_i) > N\delta_1\right) \leq \exp(-2N\delta_1^2).$$

A similar method shows that

$$\mathbb{P}(\hat{\xi}_{Nk} < \xi_k + \epsilon) \leq \exp(-2N\delta_1^2),$$

and putting the two together yields the desired result. ■

Suppose for a moment that infection probabilities are individual-specific rather than match-specific. That is, each infected individual has the same probability of infecting any susceptible individual that they meet. Corollary 1 implies that, if we denote a person who has a very high probability of infecting other as being a *superspreader*, then however one defines that in terms of the threshold probability of infecting a susceptible person, the chance of a superspreader being present in a population grows exponentially in the size of the population. More generally when probabilities are match-specific as we have assumed, if we denote an event which causes a large number of new cases as being a *superspreading event*, then the probability of a superspreading event is increasing exponentially fast in the population size.

# 4 Implications and Conclusion

## 4.1 Path Dependence

An immediate implication that flows from our model is the possibility of *path dependence*, where two *ex ante* identical populations have persistent differences in observed infection rates over time. To see this, note the the infection rate evolves as follows.

**Lemma 4** *For all $t \in \mathbb{N}$, we have*

$$I_t = I_0(1-\gamma)^t - \sum_{k=1}^{t}(1-\gamma)^{t-k}\Delta S_k.$$

**Proof.** The proof is by induction. For $t = 0$, the result holds trivially. Now suppose the equation holds for some $t \geq 0$, and consider the $t+1$-th case. We have from Equations (7)

and (8) that

$$\begin{aligned} I_{t+1} &= I_t + \sum_{j=1}^{mI_tS_t} \mathbb{I}_j - \gamma I_t \\ &= (1-\gamma)I_t + \sum_{j=1}^{mI_tS_t} \mathbb{I}_j \\ &= (1-\gamma)I_t - \Delta S_{t+1}. \end{aligned}$$

Now applying the induction hypothesis yields

$$\begin{aligned} I_{t+1} &= (1-\gamma)\left(I_0(1-\gamma)^t - \sum_{k=1}^{t}(1-\gamma)^{t-k}\Delta S_k\right) - \Delta S_{t+1} \\ &= I_0(1-\gamma)^{t+1} - \sum_{k=1}^{t}(1-\gamma)^{t+1-k}\Delta S_k - \Delta S_{t+1} \\ &= I_0(1-\gamma)^{t+1} - \sum_{k=1}^{t+1}(1-\gamma)^{t+1-k}\Delta S_k. \end{aligned}$$

Hence the lemma is true for $t+1$ and is therefore true for all $t \in \mathbb{N}$ by induction. ■

We can see from Lemma 4 that the realized number of infections (the number of "successful" Poisson trials) at $t = 1$ has a persistent effect on the infection rate for all $t > 1$. That is, a bad round of early draws has persistent effect on the path of infection. Indeed, $\Delta S_t$ depends crucially on $\Delta S_k$ for every $k < t$, and so by Lemma 4 $I_t$ depends crucially on $I_k$ for $k < t$. This implies that a bad early realisation of trials from $\Delta s_1$ increases the number of infected people in the population, making transmission more likely since the probability of an infected-susceptible match in the population increases.

If the population is large enough, then the law of large numbers implies that the proportion of successful draws from the Poisson trials will converge to the mean of the trials. In particular, the number of new infected individuals in a given time period will be roughly equal to the mean, which we gave in Lemma 1 as $m\mu I_{t-1}S_{t-1}$.

If $I_0 = \frac{1}{N}$, then standard branching process methods confirm that there are essentially 2 equilibria–either the infection persists forever in the population or $i_t \to 0$ as $N, t \to \infty$.

## 4.2 Containment Policy

An overarching message from our analysis is that it is incomplete to focus only on the mean of the reproduction rate. Because a virus grows exponentially when $\mathcal{R} > 1$ a policy

maker should consider the mass of the distribution of $\mathcal{R}$ above 1. It is this region that we have referred to as the *danger zone*, which is depicted in the following figure.

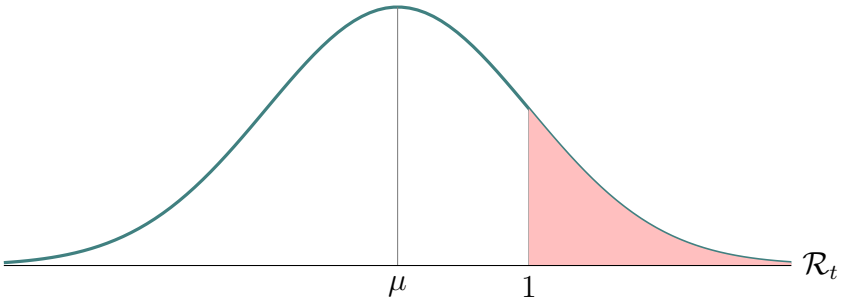


Figure 1: The danger zone.

We end this section with a suggestion for how one might measure the sensitivity the SIR system to perturbations. Fix the time at $t$ and the let $t_d(\phi) > t$ denote the first time period at which the expected number of total infections is greater than $2I_t$ when the number of matchings per individual in each time period is $\phi > 0$. That is, $t_d(\phi)$ denotes the number of time periods it takes for the expected number of total infections to double. We set $t_d(\phi) = \infty$ if infections are never expected to double.

We define the *stability index* of the virus at time $t$ as

$$\mathcal{S}_t = \frac{1}{t_d(\phi+1)} - \frac{1}{t_d(\phi)}. \tag{22}$$

The stability index measures how responsive the system is with respect to the number of matches per time period. It is one way to account for the mass of $\mathcal{R}_t$ which lies above 1.

A stability index of 0 indicates that the expected increase in the number of infections does not change substantially when the $\phi$ is increased by 1–in other words, the system is very stable to small perturbations of the number of matchings per time period. On the other hand, an index of 1 indicates that by increasing $\phi$ by 1, the number of infections is expected to double in the very next period whereas as $\phi$ currently stands, the number of infections is expected never to double. In other words the system is highly unstable to small perturbations of the number of matchings per time period.

We compute the stability index numerically when the underlying distribution $\mathcal{D}$ for the Poisson trials is $U[0,1]$ (so $\mu = \frac{1}{2}$). In particular we look at the cases when $N = 100,000$, $\gamma = 0.2$, $t = 1$ and $\phi = 1, 2, 3$. We find

$$(t_d(1),\, t_d(2),\, t_d(3)\, t_d(4)) = (\infty, \infty, 1, 1),$$

and hence for $\phi = 1$, $\mathcal{S}_t = 0$, for $\phi = 2$, $\mathcal{S}_t = 1$, and for $\phi = 3$, $\mathcal{S}_t = 0$. The reason the process takes off only when $\phi > 2$ is because this implies $\phi\mu > 1$ and so the infection grows exponentially.

We can also compute the conditional mean and variance (as in Theorem 1) of the reproductive rate numerically. Consider again the case when $\mathcal{D} = U[0, 1]$, $\phi = 2$, and $\gamma = 0.2$. We find

$$\begin{aligned}\mathbb{E}(\mathcal{R}_t \mid \theta_{t-1}) &= \frac{1}{N} S_{t-1} + \frac{1}{N^2} I_{t-1} S_{t-1} \\ \mathrm{Var}(\mathcal{R}_t \mid \theta_{t-1}) &= \frac{1}{4N} S_{t-1} + \frac{N+1}{8N^3} I_{t-1} S_{t-1},\end{aligned}$$

Taking $t = 20$ and $N = 100000$ (for the purpose of this example), we have $(S_{t-1}, I_{t-1}) = (44860.3, 40686.1)$, therefore

$$\begin{aligned}\mathbb{E}(\mathcal{R}_{20} \mid \theta_{19}) &= 0.631 \\ \mathrm{Var}(\mathcal{R}_{20} \mid \theta_{19}) &= 0.135,\end{aligned}$$

Finally, in general, given a critical value $c$, numerical mean and variance calculations such as the one above allow us to construct a "confidence interval" for $\mathcal{R}_t$ as

$$CI = \left[\mathbb{E}(\mathcal{R}_t \mid \theta_{t-1}) - c\sqrt{\mathrm{Var}(\mathcal{R}_t \mid \theta_{t-1})}, \mathbb{E}(\mathcal{R}_t \mid \theta_{t-1}) + c\sqrt{\mathrm{Var}(\mathcal{R}_t \mid \theta_{t-1})}\right],$$

giving us another measure for how much of the mass of $\mathcal{R}_t$ lies above 1. Taking $c = 1$ and using the example above we find $CI = [0.264, 0.998]$ and so in this case the system appears to be fairly stable.

### 4.3 Shelter-in-Place Orders versus Masks

Our model also speaks to the differential effectiveness of alternative containment policies. Given that the variance of the reproductive rate is increasing in $S_{t-1}$, consider a policy maker who seeks to implement a containment policy early in the spread of the virus. Such a planner has two lines of attack. The first is to try and lower the number of meetings an individual has per time period ($\phi$), for example, by a shelter-in-place order. The second is to try and lower the infectiousness of the virus ($\mu$), perhaps by mandating the widespread use of masks.

When $t$ is small, $I_{t-1}$ is approximately $1$, and $S_{t-1}$ is approximately $N$. Hence

$$(m\mu)^2 I_{t-1} S_{t-1} = \frac{\phi\mu}{N^2} I_{t-1} S_{t-1} \approx \frac{\phi^2\mu^2}{N^2} N,$$

which is close to $0$ for large enough $N$. It follows that early on in the spread of the virus,

$$E(\mathcal{R}_t \mid \theta_{t-1}) \approx m\mu S_{t-1} = \frac{\phi\mu}{N} S_{t-1},$$

and

$$Var(\mathcal{R}_t \mid \theta_{t-1}) \approx m\mu(1-\mu) S_{t-1} = \frac{\phi\mu}{N}(1-\mu) S_{t-1}.$$

While lowering $\phi$ or $\mu$ both result in a lower expected reproductive rate, the effect of such measures on the variance are less straightforward. The conditional variance of the reproductive rate is increasing in $\phi$ and thus a reduction in $\phi$ will indeed lower the variance of $\mathcal{R}_t$.

However, the variance is increasing in $\mu$ up until $\mu = 1/2$, at which point it is decreasing in $\mu$, leading to the perhaps counterintuitive result that the more contagious a virus is, the less effective it is to lower its infectiousness. The reason for this is that efforts to decrease $\mu$ may have the unintended result of also increasing the variance in the spread of the virus. By contrast, $\phi$ does not suffer from this problem, so a reduction in $\phi$ (e.g. shelter-in-place) will unambiguously result in a reduction in both the mean and variance of the reproductive rate of the virus.

### 4.4 Conclusion

By modeling the reproduction rate as something that emerges from primitives of how infections occur we have highlighted the importance of considering the whole distribution of the reproduction rate for understanding the spread of a virus, and the optimal policy response to it.

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# 5 Appendix A

We make some remarks here about the matching technology when the independence assumption of matches is violated. The main effect of losing the independence of matches is that it invalidates $\mathcal{R}_t^{pop}$. Recall that $\mathcal{R}_t^{pop}$ is defined as the population-level reproductive rate, that is, a random variable whose distribution is equal to the number of new infections in the population from time $t$ to time $t+1$. In general, we can define the changes in

$S_t$, $I_t$, and $r_t$ by the discrete Markov process:

$$\Delta S_{t+1} = -\mathcal{R}_t^{pop} \tag{23}$$

$$\Delta I_{t+1} = \mathcal{R}_t^{pop} - I_t\gamma \tag{24}$$

$$\Delta R_{t+1} = I_t\gamma, \tag{25}$$

We want to work out a general form for $\mathcal{R}_t^{pop}$ without independence of matchings. Suppose we fix an interaction structure in the population at time $t$ by the configuration model or some other method. Let $m_k$ $(k = 1, \ldots, n_t)$ denote the number of susceptible individuals who interact with exactly $k$ *different* infected individuals. Then denoting by $[l]$ the set $\{1, 2, \ldots, l\}$, we can write the population reproduction rate as

$$\mathcal{R}_t^{pop} = S_t \left( \sum_{j_1=0}^{m_1} \mathbb{I}_{j_1} + \sum_{\substack{j_i=0 \\ i\in[2]}}^{m_2} \mathbb{I}_{j_1 \cup j_2} + \sum_{\substack{j_i=0 \\ i\in[3]}}^{m_3} \mathbb{I}_{j_1 \cup j_2 \cup} + \cdots + \sum_{\substack{j_i=0 \\ i\in[n_t]}}^{m_{n_t}} \mathbb{I}_{\bigcup_{k\le n_t} j_k} \right) \tag{26}$$

$$= S_t \sum_{l=1}^{n_t} \sum_{\substack{j_i=0 \\ i\in[l]}}^{m_l} I_{\bigcup_{k\le l} j_k}. \tag{27}$$

Note that for an arbitrary collection of events $\bigcup_k A_k$, the indicator variable of their union satisfies

$$\mathbb{I}_{\bigcup_k A_k} = 1 - \prod_k (1 - \mathbb{I}_{A_k}). \tag{28}$$

Which allows us to rewrite the above as

$$\mathcal{R}_t^{pop} = \sum_{l=1}^{n_t} \sum_{j_i=0, i\in[l]}^{m_l} \left( 1 - \prod_{k\le l} (1 - \mathbb{I}_{j_k}) \right) S_t. \tag{29}$$

It is clear that this makes the system of equations in Equations (23) to (25) particularly difficult to work with let alone to solve in some kind of closed form. At this point it is worth looking into the matching technology in the hope that $\mathcal{R}_t^{pop}$ can be simplified. Indeed, under the assumption of a large $N$ and a small $\phi(N)$ as outlined in Section 2.1, matches are approximately independent. This means that $m_k \to 0$ for all $k \geq 2$ It follows that the number of infected-susceptible matches in the population is $m_1 = I_t m S_t$, which gives us

$$\mathcal{R}_t^{pop} = \sum_{j=1}^{m_1 I_t S_t} \mathbb{I}_j,$$

as used in the paper.

# 6 Appendix B

A natural question is whether or not there exists a closed form expression for the unconditional mean and variance $\mathbb{E}(\mathcal{R}_t)$ and $\mathrm{Var}(\mathcal{R}_t)$. We present some results here which indicate that whilst in theory such an expression exists, it is rather difficult to write down. First, recall that the mean of $\mathcal{R}_t$ depends entirely on the mean of $S_t$. Our first lemma establishes an equation for the mean of $S_t$ in terms of lower order terms $I_k S_k$ for $k < t$.

**Lemma 5** *For all $t \in \mathbb{N}$,*

$$\mathbb{E}(S_t) = S_0 + \frac{m}{2} \sum_{k=0}^{t-1} \mathbb{E}(I_k S_k).$$

**Proof.** First, note that for all $t \in \mathbb{N}$, we have

$$S_t = \sum_{k=0}^{t} \Delta S_k, \tag{30}$$

where we let $\Delta S_0 = S_0$. Hence it follows that

$$\mathbb{E}(S_t) = S_0 + \sum_{k=1}^{t} \mathbb{E}(\Delta S_k). \tag{31}$$

Note then that since $\Delta S_{k+1} = \sum_{k=1}^{mI_k S_k} \mathbb{I}_j$, we have by *Wald's identity* that

$$\mathbb{E}(\Delta S_{k+1}) = \mathbb{E}(mI_k S_k)\mathbb{E}(\mathbb{I}_j) = \frac{m}{2}\mathbb{E}(I_k S_k). \tag{32}$$

Finally then substitution (32) into (31) and relabelling the index, we have

$$\mathbb{E}(S_t) = S_0 + \frac{m}{2} \sum_{k=0}^{t-1} \mathbb{E}(I_k S_k), \tag{33}$$

proving the lemma. ■

We can see from Lemma 5 that the expectation of $S_t$ depends entirely upon the expectation of $I_t S_t$.

We have from Lemma 4 an expression for $I_t$ in terms of $\Delta S_k$ for $k < t$. This allows us to find a closed form expression for $I_t S_t$ from which we can calculate its mean. However,

before we are ready to compute the expectation of $I_t S_t$, we need to know how to compute $\mathbb{E}(\Delta S_k \Delta S_l)$ for both $l = k$ and $l \neq k$. We establish this with the following lemma.

**Lemma 6** *Let $k, l \in \mathbb{N}$. Then*

$$\mathbb{E}(\Delta S_k \Delta S_l) = \frac{(Nd)^2}{4} \mathbb{E}\left(I_{k-1} S_{k-1} I_{l-1} S_{l-1}\right).$$

**Proof.** Notice firstly that $\Delta S_k$ and $\Delta S_l$ are not independent. Indeed, suppose $l < k$, then $S_{k-1}$ appears in the upper limit of the sum for $\Delta S_k$, and one has that $S_{k-1} = \sum_{j=0}^{k-1} \Delta s_j$. We have

$$\Delta S_k \Delta S_l = \left( \sum_{j=1}^{mI_{k-1}S_{k-1}} \mathbb{I}_j \right) \left( \sum_{q=1}^{mI_{l-1}S_{l-1}} \mathbb{I}_j \right).$$

Noting that this is a sum over $(mI_{k-1}S_{k-1})(mI_{l-1}S_{l-1})$ products of independent indicator variables, we apply Wald's identity to get

$$\begin{aligned}
\mathbb{E}(\Delta S_k \Delta S_l) &= \mathbb{E}\left((mI_{k-1}S_{k-1})(mI_{l-1}S_{l-1})\right) \mathbb{E}(\mathbb{I}_j \mathbb{I}_l) && (34)\\
&= (Nd)^2 \mathbb{E}\left(I_{k-1} S_{k-1} I_{l-1} S_{l-1}\right) \mathbb{E}(\mathbb{I}_j) \mathbb{E}(\mathbb{I}_l) && (35)\\
&= \frac{(Nd)^2}{4} \mathbb{E}\left(I_{k-1} S_{k-1} I_{l-1} S_{l-1}\right), && (36)
\end{aligned}$$

completing the proof. ■

We are now ready to provide a recursive formula for $I_t S_t$, which will allow us to compute its expectation.

**Lemma 7** *The expectation $\mathbb{E}(I_t S_t)$ satisfies the recursive equation*

$$\begin{aligned}
\mathbb{E}(I_t S_t) = \left(1 - \gamma + \frac{m}{4}\right) \mathbb{E}(I_{t-1} S_{t-1}) - \frac{(Nd)^2}{4} \mathbb{E}\left((I_{t-1} S_{t-1})^2\right)\\
- \frac{(Nd)^2}{4} \sum_{k=1}^{t-1} \left(1 + (1-\gamma)^{t-k}\right) \mathbb{E}(S_{t-1} I_{t-1} S_{k-1} I_{k-1}).
\end{aligned}$$

**Proof.** Recall that in the proof of Lemma 4 we used the fact that $I_t = (1-\gamma) I_{t-1} + \Delta S_t$.

Hence we may write

$$\begin{aligned} I_t S_t &= ((1-\gamma) I_{t-1} + \Delta S_t) \left( S_0 + \sum_{k=1}^{t-1} \Delta S_k + \Delta S_t \right) \\ &= (1-\gamma) I_{t-1} S_{t-1} - S_0 \Delta S_t - \Delta S_t \sum_{k=1}^{t-1} \Delta S_k - (\Delta S_t)^2 + (1-\gamma) I_{t-1} \Delta S_t. \end{aligned}$$

Now using Lemma 4, we have

$$I_{t-1} = I_0 (1-\gamma)^t - \sum_{k=1}^{t-1} (1-\gamma)^{t-k} \Delta S_k,$$

and hence that

$$\begin{aligned} I_t S_t = (1-\gamma) I_{t-1} S_{t-1} - S_0 \Delta S_t - \Delta S_t \sum_{k=1}^{t-1} \Delta S_k - (\Delta S_t)^2 \\ + I_0 (1-\gamma)^t \Delta S_t - \Delta S_t \sum_{k=1}^{t-1} (1-\gamma)^{t-k} \Delta S_k. \end{aligned}$$

Which we can rewrite by collecting $\Delta S_t$ terms as

$$I_t S_t = (1-\gamma) I_{t-1} S_{t-1} - \left( S_0 + I_0 (1-\gamma)^t \right) \Delta S_t - \mathbb{E}\left( (\Delta S_t)^2 \right) - \sum_{k=1}^{t-1} (1 + (1-\gamma)^{t-k}) \mathbb{E}(\Delta S_t \Delta S_k).$$

Finally, applying Lemma 6 and (32), we have the desired result. ■

An alternative but less useful expression for $\mathbb{E}(I_t S_t)$ in terms of lower order terms can be derived by using the following lemma. This lemma is a little more involved but establishes $I_t S_t$ in terms of $I_k$ and $S_k$ for $k < t$.

**Lemma 8** *For all $t \in \mathbb{N}$ we have*

$$\begin{aligned} I_t S_t = S_0 I_0 (1-\gamma)^t + \sum_{k=1}^{t} \left( I_0 (1-\gamma)^t - S_0 (1-\gamma)^{t-k} \right) \Delta S_k \\ - \sum_{k=2}^{t} \sum_{l=1}^{k-1} \left[ (1-\gamma)^{t-k} + (1-\gamma)^{t-l} \right] \Delta S_k \Delta S_l - \sum_{k=1}^{t} (1-\gamma)^{t-k} (\Delta S_k)^2. \end{aligned}$$

**Proof.** Recall from (30) that $S_t = S_0 + \sum_{k=1}^{t} \Delta S_k$. Substituting this, and the equation we

established in Lemma 4 into the expression $I_t S_t$, we have for any $t \in \mathbb{N}$,

$$\begin{aligned} I_t S_t &= \left( I_0(1-\gamma)^t - \sum_{k=1}^{t} (1-\gamma)^{t-k} \Delta S_k \right) \left( S_0 + \sum_{l=1}^{t} \Delta S_l \right) \\ &= S_0 I_0 (1-\gamma)^t - S_0 \sum_{k=1}^{t} (1-\gamma)^{t-k} \Delta S_k + I_0 (1-\gamma)^t \sum_{l=1}^{t} \Delta S_l - \sum_{k=1}^{t} \sum_{l=1}^{t} (1-\gamma)^{t-k} \Delta S_k \Delta S_l \\ &= S_0 I_0 (1-\gamma)^t + \sum_{k=1}^{t} \left( I_0 (1-\gamma)^t - S_0 (1-\gamma)^{t-k} \right) \Delta S_k - \sum_{k=1}^{t} \sum_{l=1}^{t} (1-\gamma)^{t-k} \Delta S_k \Delta S_l. \end{aligned}$$

Now we work with the double sum to put it into the form given in the lemma. we have

$$\begin{aligned} \sum_{k=1}^{t} \sum_{l=1}^{t} (1-\gamma)^{t-k} \Delta S_k \Delta S_l &= \sum_{\substack{k,l \\ k=l}}^{t} (1-\gamma)^{t-k} \Delta S_k \Delta S_l + \sum_{\substack{k,l \\ k \neq l}}^{t} (1-\gamma)^{t-k} \Delta S_k \Delta S_l \\ &= \sum_{k=1}^{t} (1-\gamma)^{t-k} (\Delta S_k)^2 + \sum_{\substack{k,l \\ k \neq l}}^{t} (1-\gamma)^{t-k} \Delta S_k \Delta S_l. \end{aligned}$$

Focusing on the second summation, we note that by summing over the upper and lower triangles of an $l \times k$ matrix, we can write

$$\sum_{\substack{k,l \\ k \neq l}}^{t} (1-\gamma)^{t-k} \Delta S_k \Delta S_l = \sum_{k=2}^{t} \sum_{l=1}^{k-1} (1-\gamma)^{t-k} \Delta S_k \Delta S_l + \sum_{l=2}^{t} \sum_{k=1}^{l-1} (1-\gamma)^{t-k} \Delta S_k \Delta S_l.$$

Then by swapping the labels $l$ and $k$ on the second sum, this gives

$$\begin{aligned} \sum_{\substack{k,l \\ k \neq l}}^{t} (1-\gamma)^{t-k} \Delta S_k \Delta S_l &= \sum_{k=2}^{t} \sum_{l=1}^{k-1} (1-\gamma)^{t-k} \Delta S_k \Delta S_l + \sum_{k=2}^{t} \sum_{l=1}^{k-1} (1-\gamma)^{t-l} \Delta S_k \Delta S_l \\ &= \sum_{k=2}^{t} \sum_{l=1}^{k-1} \left[ (1-\gamma)^{t-k} + (1-\gamma)^{t-l} \right] \Delta S_k \Delta S_l, \end{aligned}$$

completing the proof. ■

Putting $I_t S_t$ in the above form allows us to apply the expectation operator in a clean way. In particular, applying Lemma 6 and (32), we can evaluate the expectation of $I_t S_t$

using Lemma 8 as

$$\begin{aligned}\mathbb{E}(I_t S_t) = S_0 I_0 (1-\gamma)^t + \sum_{k=1}^{t} \left( I_0 (1-\gamma)^t - S_0 (1-\gamma)^{t-k} \right) \frac{m}{2} \mathbb{E}(I_{k-1} S_{k-1}) \\ - \sum_{k=2}^{t} \sum_{l=1}^{k-1} \left[ (1-\gamma)^{t-k} + (1-\gamma)^{t-l} \right] \mathbb{E}(\Delta S_k \Delta S_l) \\ , - \sum_{k=1}^{t} (1-\gamma)^{t-k} \frac{1}{4} \left( (m I_{k-1} S_{k-1} + (m I_{k-1} S_{k-1})^2 \right) .\end{aligned}$$