Integrating stochasticity into epi-econ models and its implications for welfare evaluation

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Abstract

This paper investigates the implications of incorporating stochasticity into epidemiological-economic (epi-econ) models for evaluating welfare. While deterministic models assume completely predictable epidemic trajectories, infectious disease dynamics are inherently stochastic in nature, which is most pronounced and important to account for when case numbers are low. We adapt a specific epi-econ framework, namely the one proposed by Farboodi et al. [1], to include discrete-time stochastic transitions based on chain binomial processes. We find that inclusion of stochastic effects systematically reduces both health-related and activity-related costs as compared to deterministic model results. This discrepancy is primarily driven by the possibility of epidemic fade-out during the tail end of the outbreak, when case numbers are low and stochastic fluctuations in new cases or the absence thereof can eliminate the disease entirely. In such cases, subsequent infections and associated costs are avoided, and individuals return to normal activity sooner. As a result, deterministic models tend to overestimate expected welfare losses, potentially leading to overly conservative policy recommendations. Our results highlight the importance of accounting for stochasticity in disease transmission when relying on models for economic evaluation and policy design.

Keywords: epidemic modeling, stochastic SIR, welfare analysis, epi-econ, binomial processes

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1. Introduction

In 2020, the SARS-CoV-2 pandemic emerged and caused millions of deaths around the world. Similar to other infectious diseases, the focus was initially on disease incidence, severe disease and mortality [2]. However, other areas of society were also heavily affected. More specifically, it has been widely reported that the pandemic had an impact on education [3], loneliness [4], economic activity [5], crime [6], among other domains. As a consequence, it is clear that multidisciplinary expertise is required to guide policy decisions in such a context.

Particularly, mathematical models are often used as pragmatic tools to inform policy makers on public health interventions and their impact at the population-level in such a context. Moreover, these models are well suited to describe disease spread under varying conditions, through scenario analyses and short- and long-term forecasting. In addition to health-related outcomes, economists are especially interested in welfare [7] as a broader concept, mostly including economic variables, health and education. Relying solely on health-related variables leads to suboptimal societal decisions as other areas of interest are neglected, and therefore, a multidimensional, welfarist approach improves informed decisions.

In addition to this broader focus on welfare, economic models account for endogenous behavior by the inclusion of intelligent, rational agents, through game theory. From an epidemiological point of view, this implies that people choose the intensity of their protective behavior based on the current state of an epidemic. An example of this endogenized behavior are so-called 'free-riders', i.e., agents who do not get vaccinated in an almost fully vaccinated population, because they do not have an incentive anymore as their risk of getting infected is small.

There is a large tradition of joint epidemiologic-economic (epi-econ) models that focus on including this welfare and behavior to optimize pandemic policy decisions. Most published epi-econ models are deterministic in the sense that the implied unfolding of the epidemic is the same given a particular model parameter configuration. Consequently, transmission is assumed to follow a completely predictable pattern during an epidemic.

However, disease transmission is inherently influenced by risks of contracting the disease, probabilities of developing severe illness, and random events such as varying contact intensity rates, weather conditions and superspreading. While deterministic epi-econ models capture the average behavior in large populations, including stochastic effects could result in different epidemiological trajectories, ranging from minimal disease spread and extinction early on after introduction of the pathogen in a population, to large-scale outbreaks [8]. These variations should be accommodated when informing policymaking. The objective of this work is to contrast economic evaluations under results derived from deterministic and stochastic epi-econ models. We particularly focus on the evaluation in terms of social welfare in comparison to deterministic models.

Accounting for stochasticity is especially relevant as it can substantially affect the expected welfare outcomes of policy interventions. Decision theory provides a relevant framework here: expected value is a concept

based on the average benefit or cost of decisions in the presence of uncertainty, and which policy is based upon. When using a deterministic model, analysts implicitly assume that the fixed welfare outcomes align with expected values. However, due to the stochastic nature of outbreaks, this assumption may lead to policy recommendations that are either overly cautious or not protective enough.

Specifically, we hypothesize that incorporating stochasticity, and especially the possibility of epidemic fade-out during the tail end of an outbreak, systematically reduces welfare losses during an epidemic. This epidemic fade-out occurs when case numbers become sufficiently low, such that subsequent stochastic realizations of the number of new events per time step become zero, causing the disease to die out entirely. When this happens, further transmission is halted and society can go back to a pre-epidemic situation sooner. We hypothesize that this leads to lower cumulative welfare losses than predicted by deterministic models.

In this paper, we first integrate stochastic effects in an established epi-econ model. Our starting point is the work by Farboodi and colleagues [1] in which agents, maximizing their utility by choosing their activity level to balance social activity and infection risk, are embedded in an epidemic Susceptible-Infected-Recovered-Death (SIRD) compartmental model. Subsequently, a social welfare function accumulating the individual welfare of agents is computed to economically evaluate the model, allowing to quantify a trade-off between health and activity costs. Through simulation, we assess the impact of stochastic disease transmission on economic evaluation. The framework of Farboodi et al. [1] operates in continuous time, which complicates the inclusion of stochastic effects. Hence, we adapt the proposed epi-econ model to include a simpler activity function, making it possible to include discrete-time stochastic transitions following chain binomial processes.

This paper is structured as follows. Section 2 gives a brief outline of the relevant literature. In Section 3 an outline of the model is provided, while in Section 4 stochasticity is introduced in the model. Section 5 presents specific parameter choices and the general simulation setup. Results of the simulation approach are part of Section 6, before a discussion and conclusion is included in Section 7.

2. Literature review

We first perform a scoping literature review on deterministic epidemiological models, before we describe how epi-econ models enhance these models. Then, we discuss the stochastic extension of both types of models. At last, we discuss how our work contributes to the current literature.

Epidemiological models

Transmission models for infectious diseases can generally be divided in two subcategories; individual-based and compartmental models. Individual-based models simulate each individual in a population as an agent with unique characteristics, and explicitly model each interaction. This way, they are able to capture rich heterogeneity, however, at the cost of being more complex and more computationally intensive. A summary of the use of individual-based models for infectious disease transmission is provided in Willem et al. [9].

Compartmental models, on the other hand, subdivide the population in a limited number of disease-related states and transitions between states are described mathematically in terms of (ordinary) differential equa-

tions. Moreover, these models are computationally less demanding than individual-based models, however, at the cost of a population-level rather than individual-level perspective on disease transmission. Consequently, these models do not explicitly account for potential sources of heterogeneity inherent to disease transmission. Compartmental models are most commonly used in economic literature to illustrate disease dynamics and are well-suited for including behavioral aspects. Because we aim to illustrate how stochasticity affects economic evaluation in a behavioral epi-econ model, and do not require a complex, individual-level, and heterogeneity-explicit one, we choose to rather focus on this type of model [10].

In particular, a SIRD compartmental model implies that individuals are divided into compartments based on their health status. These states traditionally include (1) individuals who are susceptible to the infection under study, but likely become infected later on, and hereafter referred to as susceptibles (S), (2) individuals who are currently infected and infectious (I), (3) individuals who were infected previously, but have recovered and have now gained lifelong immunity (R), and (4) individuals who have deceased (D). Other relevant states can include individuals who are vaccinated, exposed, hospitalized, etc. Individuals then 'flow' from one state to another with transitions over time being described mathematically using a set of ordinary differential equations.
An illustration of the compartmental structure of the SIRD model is given in Figure 1.

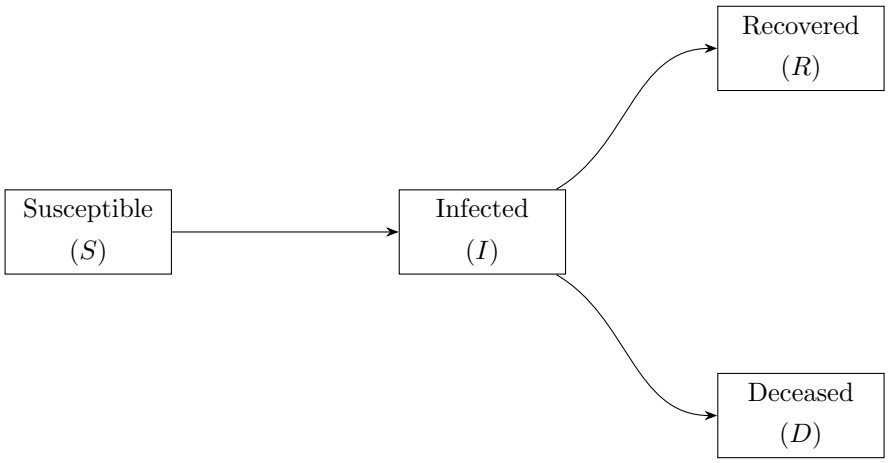


Figure 1: Flow diagram of the compartmental SIRD model. Individuals transition from being Susceptible (S) to Infected (I), and then either Recover (R) or become Deceased (D).

A central concept in epidemiology is the effective reproduction number, denoted as R_t. This metric represents the average number of secondary infections caused by an 'average' infectious individual infected at time t, during their entire infectious period in a population. In a deterministic modeling approach, an outbreak will always take place when R_0, the reproduction number at time 0 when the population is still completely susceptible to infection, exceeds the epidemic threshold value of one. For more details on compartmental models, the reader is referred to [11].

Epi-econ models

Integrated epi-econ models date back to the seminal contributions of Kremer [12] and Geoffard and Philipson [13]. Kremer [12] was the first to integrate rational, economic agents into epidemiological models. In his

model, which particularly focuses on AIDS, it is taken into account that people adapt their sexual behavior based on the prevalence of the disease. Independently from Kremer [12], Geoffard & Philipson [13] showed that epidemics give rise to a so-called 'infection externality'. These authors developed a theoretical model in which individuals choose their activity level to maximize utility in an epidemic. However, agents do not take into account in their decision that when they become infected, others' infection probability also increases. Because of this externality, agents always display an excessive level of activity that is suboptimal on a societal level, and there is scope for a policymaker to correct for this behavior by discouraging activity. Following these papers, a whole strand of literature on epi-econ models emerged. The interested reader is referred to Mcadams [14], Boucekinne et al. [15] and Auld et al. [16], while a short summary of relevant findings is presented hereunder.

Behavior

A key characteristic of epi-econ models is the presence of a feedback loop between the epidemic state and individual behavior. Empirical evidence supports this idea that individuals adjust their behavior in response to epidemic conditions. Research has shown that people tend to reduce risky activities as case numbers rise, and that they respond to changes in perceived infection risk or to policy interventions. These findings provide strong support for the behavioral foundations of epi-econ models [17, 18, 16, 19].

As a result, these epi-econ models are 'self-limiting', contrary to the traditional SIR-type transmission models. As infection awareness increases, people reduce risky behavior to avoid infection, lowering peak prevalence [16]. However, this feedback mechanism can also work in the opposite direction. Interventions that reduce prevalence, such as quarantine or masking, can induce a behavioral substitution effect: as perceived risk declines, individuals increase risky activity, partially offsetting the benefits of the intervention [20]. In extreme cases, such as when the number of infected individuals in the population approaches zero, incentives to protect oneself vanish completely, thereby complicating eradication. This implies that deterministic epi-econ models almost never reach zero infections, but rather feature a long, drawn-out tail phase of the epidemic.

Epi-econ models thus integrate behavior in epidemiological models. However, there is disagreement on how this behavior should be implemented. Concretely, this can be modeled in three main ways. First, behavior can follow a mechanistic approach, as is more traditional in the epidemiological literature. This implies that the behavior of agents follows a simple rule, and is defined exogenously [21]. An example is illustrated in the study by Bootsma and Ferguson [22], where individuals reduce their contacts in a given time period in relation to the number of deaths in the previous time period. An important drawback of such an approach is that it lacks microfoundations, meaning that agents do not optimize dynamically or weigh trade-offs. This not only makes it less suitable to evaluate welfare or design policies, but is also less accurate, as it often assumes overly simplistic responses to the time-varying epidemiological situation.

Second, agents can be modeled as optimally forward-looking, meaning that they choose activity to maximize their welfare function over time. This implies that they both perfectly discount their welfare over time and dispose of perfect information about their risk of infection. This is by far the most popular way to model behavior in epi-econ models [23, 24, 25, 1]. However, this assumption may be rather strict, as it is doubtful

whether agents are indeed perfectly informed about their welfare function at any time during the unfolding of the epidemic.

A third approach is to assume that agents maximize an instantaneous, simpler, 'rule-of-thumb' welfare function. For example, in Keppo et al. [26], agents maximize their welfare which solely depends on the current epidemiological state, their own distancing choice, and others' choices, at this present moment. This way agents are still strategic, yet 'myopic'. This methodology is used less in the literature [27, 26], however, it may be a more accurate way of reflecting reality [14]. Because this approach does not require intertemporal maximization, it is also easier to characterize the epidemic trajectory and thus to include stochastic effects. Hence, we rely on this methodology later.

Welfare

Most of the early epi-econ literature mainly focused on how agents adapt their behavior reacting to a pandemic and its implications for optimal policy. However, in the past decade, macroeconomic analyses have gained popularity to analyze due to which mechanisms welfare is lost as a result of pandemics[1]. These analyses have shown that welfare is effectively affected by epidemics, not only through direct health consequences, but also via indirect pathways. It is straightforward to see that epidemics negatively affect individual and public health. However, they also suppress both economic and social activity. For example, outbreaks often lead to a reduction in labor supply, which slows down the economy. In addition, non-pharmaceutical interventions like school closures can lower educational outcomes and reduce human capital accumulation [28]. To quantify by how much welfare is affected, epi-econ models are quintessential.

Specifically, we start in this paper from the framework introduced in Farboodi et al. [1] and adapt the model to include a rule-of-thumb welfare function to make it suitable for including stochastic effects. The reason for choosing this model is its clear and straightforward way to capture costs and benefits of widespread social distancing. It enhances a standard SIRD compartmental model by including agents maximizing a welfare function subject to differential equations, accounting for the fact that they choose their activity level based on their risk of getting infected. This way, their model includes behavior, but it also allows us to disentangle and quantify the activity-related and health-related costs associated with the pandemic. One then distinguishes between a 'laissez-faire' scenario where agents make their activity decisions to maximize their own welfare, but in the absence of policy, and an 'optimal policy' case in which the government maximizes the total welfare of the population by choosing aggregate activity. In this paper, we primarily focus on the former scenario to allow for smooth behavioral adjustments in contrast with large shocks that are induced by sudden and large-scale changes imposed by governmental decisions, and thus this approach suffices for our purpose. Note that the original approach by Farboodi and colleagues [1] includes a deterministic transmission process, hence, our approach extends the aforementioned work by including discrete-time stochastic transitions between compartments thereby capturing the stochastic nature of disease spread as pointed out earlier. In the next section, we will provide additional details thereon.

[1]Macroeconomic models are not discussed in detail here, however, the interested reader is referred to the literature reviews of Bloom et al. [28] and Boucekinne et al. [15] for this purpose.

Stochasticity in epidemiological and epi-econ models

A commonly used way to describe disease transmission is based on the use of deterministic transition rates between susceptible, infected and recovered states. This deterministic modeling facilitates the optimization of policies and makes the solution of the problem easily tractable [29]. However, these rates do not account for stochastic effects inherent to disease spread. In epidemics, chance events are important as they can influence the course of the pandemic.

Several reasons are given in literature for why stochastic effects should be incorporated into epidemic models. First, they allow for the computation of the probability of a major outbreak. This is especially relevant in early phases of transmission, as early stochastic fade-out may lead to disease extinction before widespread transmission takes off, even with $R_t > 1$. Second, stochastic models more adequately quantify the duration of an epidemic and its final size distribution, as they capture the possibility of epidemic fade-out due to random chance, especially in the later phase when prevalence declines toward zero. This is a feature that deterministic models overlook, as there is no room for random chance to end the epidemic prematurely. Third, due to the stochastic nature of the transmission process, the population-level dynamics observed in practice often deviate from the population-averaged trajectories in deterministic models. This deviation is particularly pronounced at the start of an epidemic or in small populations where randomness can lead to extinction effects, as well as in periods of changing transmission conditions, such as the implementation of public health interventions or behavioral adaptations. Finally, statistical inference and parameter estimation with regard to the model parameters, although more complex due to stochasticity being present, provides a more comprehensive quantification of uncertainty, combining both structural and systematic measurement error [30]. These aspects are discussed in more detail in the works of King et al. [8] and Britton et al. [31].

Stochastic transmission rates can be included in epidemiological models in a variety of ways. For a summary, see Allen [32]. In this paper, we choose to model the transitions following a chain binomial model, as outlined in Abrams et al. [33] for a more complex model describing COVID-19 spread in Belgium, and based on the original work by Bailey [34]. The "chain" aspect refers to the Markovian structure of the model, in which the next state depends solely on the information from the previous time step. Modeling epidemic transitions following chain binomial processes is considered a contribution to the current (epi-)econ literature on this topic.

A key reason for favouring the chain binomial formulation is that it preserves the discrete, event-based nature of infection. Each susceptible individual faces a Bernoulli trial of infection in a given time step, so the number of new infections is an integer draw that directly reflects how many susceptibles remain; this "mimics the actual spread of infection" [35]. By contrast, a stochastic transmission rate treats the force of infection as a continuous diffusion and still relies on deterministic compartment sizes, which can misrepresent how infections occur. In addition to this, because the binomial process produces integer-valued counts, it admits epidemic fade-out. Diffusion-based models with a stochastic transmission rate cannot capture extinction in finite populations since they keep compartment sizes continuous. For analysing welfare losses and the implications of stochastic extinction at low prevalence, modelling transitions with a chain binomial process is therefore more appropriate than perturbing the transmission rate.

There are a few examples of stochastic epi-econ models. Federico and Ferrari [36] study optimal lockdown policy when the transmission rate follows a diffusive stochastic Wiener process whose drift can be influenced through costly containment. They show how stochastic fluctuations in the transmission rate accelerate and prolong interventions. Prieur et al. [37] incorporate random mutations of the SARS-CoV-2-virus into a piecewise deterministic control framework, combining discrete stochastic transitions between disease strains with deterministic epidemic dynamics. La Torre et al. [38] examine a setting where the transmission rate is subject to state-dependent stochastic shocks, capturing the idea that higher prevalence increases the probability of disruptive events (e.g., new strains). They show how complete eradication is never an optimal policy in such a framework. Hong et al. [39] are closest to our paper as they also directly compare deterministic and stochastic epidemic models, but they do so in an asset pricing context, showing how transmission volatility alters firm valuations. A recent contribution by Marsiglio and Tolotti [40] examines vaccination decisions in a stochastic SIR model with heterogeneous parents who compare the health and economic costs of vaccinating their children. By contrasting deterministic and stochastic frameworks, they show that focusing only on deterministic dynamics can be misleading because the stochastic system may exhibit metastability, multiple stable equilibria, chaotic dynamics and path

While these contributions highlight the importance of stochasticity, they do not systematically compare stochastic and deterministic versions of the same epi-econ model to evaluate and quantify the welfare implications of stochastic effects, especially epidemic fade-out. This is an important omission, as stochastic extinction is an important feature of real-world epidemics, particularly at low prevalence levels, and can strongly influence welfare evaluation. Moreover, these studies typically introduce stochasticity by modifying the transmission rate, rather than modeling it through binomial transition processes, which are suitable for capturing population-level dynamics. Our paper addresses these gaps by extending the framework of Farboodi et al. [1] to incorporate stochastic transmission dynamics. We explain how we do so in the next section.

3. Proposed stochastic SIRD epi-econ model

We include stochasticity in a deterministic epi-econ model published elsewhere. More specifically, we start from the framework introduced by Farboodi et al. [1] and describe the different changes made to their modeling approach below.

Setup and notation

We consider a homogeneous population of individuals who are unaware whether they are susceptible or infected, but they do know if they have recovered. This implies that individuals belonging to the infected and susceptible states display the same level of activity. Each of those individual's utility $u(t)$, depending on social activity level $a(t)$, is described by the function:

$$u[a(t)] = \log[a(t)] - a(t) + 1,$$

where $0 \leq t < T_{\max}$, with $T_{\max}$ the time horizon of interest, and $0 < a(t) \leq 1$. Consequently, utility $u(t)$ takes values from $-\infty$ to 0.

The utility function is specifically designed such that the implied utility level corresponding to the optimal social activity level in the absence of infection $a^*(t) = 1$ is equal to $u[a^*(t)] = 0$. Hence, as pointed out above, the utility level is always negative (or 0) and can be interpreted as the cost of being less active relative to a scenario without a pandemic (i.e., under maximal social activity). This definition of activity allows to remain agnostic about the specific type of activity, be it economic activity, leisure, or something else.

SIRD compartmental model and force of infection

To implement stochastic transitions, we convert the proportional population variables from Farboodi et al [1] to absolute counts, allowing us to model discrete transitions using binomial processes. Specifically, the disease transmission dynamics thus divide the population into four health states at time t (see Figure 1 for a schematic diagram): susceptible ($N_s(t)$), infected ($N_i(t)$), recovered ($N_r(t)$), and deceased ($N_d(t)$) individuals. These absolute counts represent the true number of individuals in each health state at time t, and are used to describe the aggregate evolution of the epidemic over time. Then, we define the normalized population shares, that is, the true proportions of the total population N in each state, as

$$n_s(t) = \frac{N_s(t)}{N}, \quad n_i(t) = \frac{N_i(t)}{N}, \quad n_r(t) = \frac{N_r(t)}{N}, \quad n_d(t) = \frac{N_d(t)}{N}.$$

These lowercase variables denote the actual share of the population in each compartment. Finally, we distinguish these true shares from subjective beliefs about an individual's own health status. Since individuals may not know their exact state, they form beliefs over it. These are captured by

$$\tilde{n}_s(t), \quad \tilde{n}_i(t), \quad \tilde{n}_r(t), \quad \tilde{n}_d(t),$$

which denote the perceived probabilities that an individual is susceptible, infected, recovered, or deceased, respectively.

We assume a closed population without demographic inflows or outflows: there are no births, migrations, or natural (non-disease) deaths. Consequently, the total population size N remains fixed over time, and all changes in health status are solely driven by disease-related transitions. This assumption is supported by the relatively short time horizon $T_{\max}$, which is chosen to be 1500 days.

The force of infection quantifies the rate at which susceptible individuals acquire infection, which accounts for the activity of both susceptible and infected individuals through a form of mass action [41]. More specifically, the force of infection $\tilde{\lambda}(t)$ is modeled using a quadratic matching function, in the spirit of Diamond and Maskin [42]:

$$\tilde{\lambda}(t) = \beta A(t)^2 n_i(t) \tag{1}$$

where β is the effective transmission rate and $A(t)$ denotes the activity level in the population.

Quadratic matching captures the idea that infectious contacts arise through the interaction between susceptible and infected individuals, with both contributing to transmission via their activity. Because individuals do not know whether they are susceptible or infected, they choose a common activity level $A(t)$, which leads to a force of infection proportional to $A(t)^2$. This quadratic form implies that infection risk rises more than proportionally if individual activity increases. It highlights how individuals, by not internalizing how their

behavior contributes to others' infection risk, may inadvertently increase transmission risk through their social interactions.

The aggregate dynamics of the population states over time are governed by the following system of ordinary differential equations, assuming that the exit rate from the I compartment γ (including both recovery or death) and the infection-fatality probability π are both time-invariant:

$$\begin{aligned}
\frac{dN_s(t)}{dt} &= -\tilde{\lambda}(t)N_s(t) \\
\frac{dN_i(t)}{dt} &= \tilde{\lambda}(t)N_s(t) - \gamma N_i(t), \\
\frac{dN_r(t)}{dt} &= (1-\pi)\gamma N_i(t), \\
\frac{dN_d(t)}{dt} &= \pi\gamma N_i(t).
\end{aligned}$$

The number of agents that transition from the susceptible to the infectious state depends on the rate governed by the force of infection. Subsequently, agents exit the infectious state, either recovering or dying at a rate γ, with probabilities determined by the infection fatality probability π.

Myopic welfare and activity derivation

We now discuss how an individual chooses their activity level. An individual chooses their activity $a(t)$(or $a_r(t)$ if recovered), such that $a(t), a_r(t) \in G = [0, 1]$, to maximize their instantaneous, myopic welfare: [2]

$$\underset{a(t),\, a_r(t) \in G}{\arg\max} \quad W[a(t), a_r(t)] = [\tilde{n}_s(t) + \tilde{n}_i(t)]\, u[a(t)] + \tilde{n}_r(t) u[a(t)] - [\beta\, A(t)\, a(t)\, n_i(t)\, \tilde{n}_s(t)\, \kappa]$$

where κ is the cost associated with infection and defined as the product of the infection-fatality probability π and the value of a statistical life (VSL) v:

$$\kappa = \pi \times v$$

The first term, $(\tilde{n}_s + \tilde{n}_i)u[a(t)] + \tilde{n}_r u[a_r(t)]$, captures the weighted instantaneous utility from social activity at time t and thus quantifies the activity cost. The weights $\tilde{n}_s$ and $\tilde{n}_i$ reflect the individual's subjective beliefs about being susceptible or infected, states in which individuals reduce their activity in response to infection risk. As recovered agents do not have an incentive anymore to shield themselves, they will impose an activity level of 1, corresponding to a utility level of 0. This term thus drops out of the equation.

The second term, $\beta\, a(t) A(t)\, n_i(t)\, \tilde{n}_s(t)\, \kappa$, represents the instantaneous expected health cost of infection at time t, by multiplying the expected probability of getting infected with the infection cost κ. We do not weight this term by all belief states, since only those who believe they are susceptible, are at risk of becoming infected and adapt their behavior. The product $a(t)A(t)$ reflects the fact that the probability of an individual becoming infected depends on both their own activity level and the average level of activity in the population.

In the original model, the health cost term reflects the probability of death conditional on being infected. In contrast, this formulation captures the unconditional risk to a susceptible agent of incurring the complete

[2]Note that this differs from the social welfare function we use later to compute costs, as described later.

health cost, linking it directly to current behavior. This shift in perspective is essential because it allows even a myopic individual, who does not solve an intertemporal optimization problem as in the original model, to influence their expected health cost through their current action $a(t)$. Since this new term appears directly in the individual's instantaneous welfare function, it creates an incentive to adjust behavior in response to infection risks.

In this framework, the agent only considers his individual welfare at the present moment, and is thus not forward-looking. We impose this 'rule-of-thumb' simplification in comparison to Farboodi et al. [1], as this does not require intertemporal maximization. This is necessary as to make it possible to include stochastic effects in a discrete-time framework later on, without destabilizing the model output. Note that this may also better reflect reality, as human beings are not necessarily perfectly rational. Ultimately, as described later, this specification does give a similar trajectory as the one obtained by Farboodi et al. [1]. This is in part because individuals in their model also heavily discount future outcomes, which makes their behavior partly resemble that of an instantaneous decision-maker.

Overall, this myopic welfare function implies that the disease inflicts health costs directly, through the chance of dying upon exposure, but activity costs only arise indirectly, through behavioral responses that reduce activity to mitigate infection risk.

Finally, we determine the activity level that maximizes the individual's myopic welfare, and balances the aforementioned costs. Taking the derivative of $W[a(t)]$ with respect to $a(t)$ and ultimately deriving the optimal activity level leads to:

$$A^*(t) = \frac{-[n_s(t) + n_i(t)] + \sqrt{[n_s(t) + n_i(t)]^2 + 4\,\beta\,\kappa\,n_s(t)\,n_i(t)\,[n_s(t) + n_i(t)]}}{2\,\beta\,\kappa\,n_s(t)\,n_i(t)}$$

The derivation of the optimal activity level is provided in detail in Appendix A.

Social welfare function

Individuals in our model are considered to be myopic and therefore base themselves on a rule-of-thumb welfare function. However, we still use the cost definitions, for both activity and health costs, based on the social welfare functions by Farboodi et al. [1]. The reason for this is that we consider agents to make their decisions based on the rule-of-thumb welfare function, and thus their perceived costs, however, the actual costs remain perfectly discounted and accurate. This social welfare function, based on [1], which accumulates costs across all individuals and is used for cost calculation is given by:

$$\text{Social welfare} = \int_0^\infty e^{-\rho t}\left[(N_s(t) + N_i(t))\,u[a(t)] + N_r(t)\,u[a_r(t)] - N_{d,t}^{\text{new}}\,v\right]dt$$

The function $u[a(t)]$ captures the utility from social activity level $a(t)$, $u[a_r(t)]$ denotes the utility of recovered individuals, and this term thus drops out of the equation. We divide this function by the population size to get the per capita costs as reported in our figures.

While individuals in our model are myopic and therefore do not discount future outcomes, the social welfare formulation does incorporate a combined discount rate of

$$\rho = r + \delta = \frac{0.05}{365} + \frac{0.67}{365},$$

where $r = \frac{0.05}{365}$ reflects a standard annual time preference of 5%, and $\delta = \frac{0.67}{365}$ represents the hazard rate of cure arrival, as in Farboodi et al. [1].

This formulation accounts for the possibility that the pandemic may resolve early due to the arrival of a cure, thereby giving less weight to long-run costs. Even though the model simulates a fixed 1500-day horizon without explicitly modeling cure arrival, the exponential discounting mirrors the assumptions of the original model and ensures that later periods are appropriately downweighted. Note that this only affects costs, not the epidemic trajectory.

Importantly, the term $N_{d,t}^{\text{new}}$ appears explicitly and reflects the number of deaths at time t. In stochastic models, $N_{d,t}^{\text{new}}$ arises directly from realized binomial transitions, i.e., a stochastic fraction of infected individuals recover or die. This is not equivalent to the original, deterministic model [1], where deaths are computed as $\gamma\pi N_i(t)$. We do so as we assume myopic individuals to still calculate their probability of death based on the amount of infections as in the original model, and that they do not take this stochastic transition into account. As a result, $N_{d,t}^{\text{new}}$ in stochastic simulations does not necessarily equal $\gamma\pi N_i(t)$ at any given time as in the deterministic case, and must be tracked as a separate state variable over time.

4. Incorporating stochasticity in the SIRD model

To better capture the randomness inherent in disease transmission, we model transitions between compartments by using a chain binomial process [34]. We generate 500 stochastic realizations of the SIRD model with stochastic transitions to capture the full range of possible epidemiological outcomes given a specific parameter configuration (see Section 5).

In continuous-time models such as the one by Farboodi et al. [1], transition rates are typically defined as the expected number of events occurring per unit of time. To simulate these transitions stochastically in discrete time (e.g., per day), we convert rates into transition probabilities, i.e., the probability that an event occurs within a small time step. For daily updates, this corresponds to a step size of $\Delta t = 1$, and transition probabilities can be approximated accordingly as described later.

For illustrative purposes, we fully describe the chain binomial process for transitions from S to I. As pointed out earlier, the force of infection $\tilde{\lambda}(t)$ depends on the proportion of infected individuals $n_i(t)$ at time t as well as the population-level activity $A(t)$ (see Equation 1). Consequently, under a discrete-time perspective with time step Δt, one assumes that the time to infection is exponentially distributed with constant rate equal to the force of infection $\tilde{\lambda}(t)$ assumed constant between time t and $t + \Delta t$. This gives rise to the probability of infection per susceptible individual equal to $p_{i,t} = 1 - \exp\left(-\tilde{\lambda}(t)\,\Delta t\right)$. The number of new infections between time t and Δt is generated from a binomial distribution with $N_s(t)$ trials and probability of success equal to $p_{i,t}$, i.e., $N_{i,t+1}^{(\text{new})} \sim \text{Binomial}(N_s(t), p_{i,t})$. Similar expressions for the transition probabilities and binomial distributions can be derived for transitions from I to R and from I to D (see Appendix A.).
The dynamics of the population states in discrete time are given by:

$$
\begin{aligned}
N_s(t+1) &= N_s(t) - N_{i,t+1}^{\text{new}} \\
N_i(t+1) &= N_i(t) + N_{i,t+1}^{\text{new}} - N_{r,t+1}^{\text{new}} - N_{d,t+1}^{\text{new}} \\
N_r(t+1) &= N_r(t) + N_{r,t+1}^{\text{new}} \\
N_d(t+1) &= N_d(t) + N_{d,t+1}^{\text{new}},
\end{aligned}
$$

with $N_{r,t+1}^{\text{new}}$ and $N_{d,t+1}^{\text{new}}$ obtained from chain binomial processes for the number of new recoveries and deaths.

5. Simulation procedure and model parametrization

The model is calibrated to daily incidence data from the United States at the time of the COVID-19 epidemic, and largely based on Farboodi et al.[1]. We simulate the model for 1500 days, so we can capture the full fade-out effects. We consider a population of 10000 individuals. In our stochastic simulations, we impose an early fade-out threshold of 100 cumulative infections. If the total number of infections in a simulation remains below this threshold, we discard the run from further analysis. This is because such simulations represent cases where the epidemic fails to take off due to random chance, and we are specifically interested in outcomes conditional on the epidemic successfully invading into the population. Those cases are the ones in which the epidemic is actually noticed and in which policy interventions can have a meaningful impact.

The different parameter values for the model parameters in the epi-econ model are presented in Table 1, based on [1]. The baseline transmission rate is set to $\beta = 0.443$, which is the sum of an intrinsic transmission component (0.3) and the biological recovery rate $\gamma = 1/7 \approx 0.143$, consistent with early exponential growth of infections that was observed during the pandemic. The biological recovery rate γ reflects an average infectious period of one week. The infection fatality probability is calibrated at $\pi = 0.0062$, based on estimates from Hall et al. [43]. The value of a statistical life (VSL) is set at 31,755 dollars. This implies an expected cost of infection of $\kappa = \pi \cdot v = 197$ dollars. The initial state assumes a population of 10,000 individuals, with initial infections set at $N_i(0) = 5$. The combined number of initially recovered and dead individuals is $N_r(0) + N_d(0) = 0$. The remaining initially susceptible population is $N_s(0) = 9{,}995$, obtained as a residual.

Table 1: Model parametrisation based on Farboodi et al. (2021)

Description	**Symbol**	**Value**
Baseline transmission rate	β	0.443 (= 0.3 + γ)
Recovery rate (inverse of infectious period)	γ	$1/7 \approx 0.143$
Infection fatality probability	π	0.0062
Value of statistical life (VSL)	v	31,755 dollars
Utility-to-dollar conversion factor	fx	123
Expected cost of infection	$\kappa = \pi \cdot v$	197 dollars
Initially infected	$N_i(0)$	5
Initially recovered + dead	$N_r(0) + N_d(0)$	0
Initially susceptible	$N_s(0)$	9995

At last, the basic reproduction number is given by:

$$R_0 = \frac{\beta}{\gamma} = \frac{0.443}{1/7} \approx 3.1$$

The effective reproduction number is then defined as:

$$R_t \equiv R_0 A(t)^2 n_s(t).$$

In case of deterministic transmission, infections increase if and only if $R_t > 1$, meaning that a single infected individual, on average, causes more than one new infection. Therefore, keeping the effective reproduction number below one is essential for reducing disease prevalence and controlling the spread of the epidemic. Note, however, that this is not necessarily the case when using a stochastic transmission rate. Even if $R_t < 1$, an outbreak can still occur, and conversely, if $R_t > 1$, the outbreak can go extinct. The basic reproduction number in our model is set to 3.1, which is in line with observations for the Wuhan SARS-CoV-2 strain at the start of the epidemic. It makes the disease spread initially.

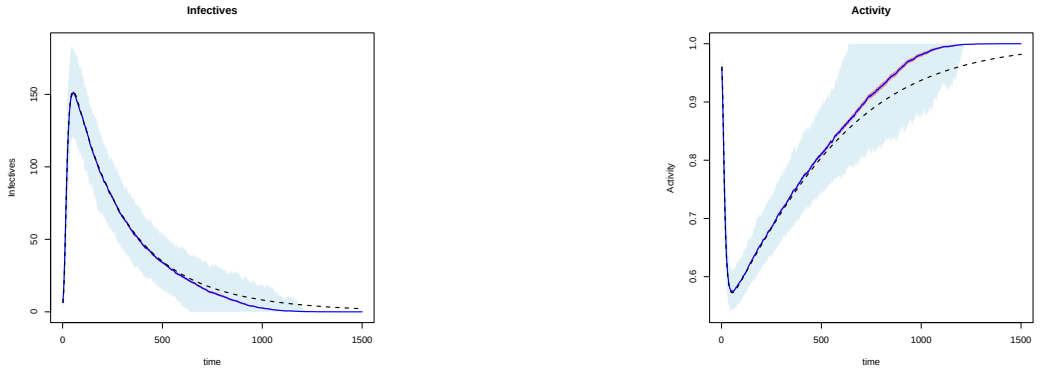
6. Simulation results

In this section, we discuss how the inclusion of stochastic effects impacts economic evaluation in an epidemic context. We focus on the laissez-faire equilibrium with no policy, as previously described. First, we briefly describe the baseline, deterministic model, before we move on to discuss how the implementation of stochasticity affects these results.

Deterministic model

At the onset of the pandemic, individuals maintain their baseline level of social activity (i.e., $a^*(t) = 1$), leading to an exponential increase in infections (see black dotted line in Figure 2a). The daily number of new cases increases rapidly and disease incidence peaks at 150 infections per day with a peak time of 61 days. In response to the heightened infection risk, individuals begin to adjust their behavior as depicted in Figure 2b by the black dotted line, reducing their activity to approximately 57% of their pre-pandemic level ($a(t) \approx 0.57$). This infection peak and subsequent behavioral response cause both daily health-related and daily activity-related costs to peak. However, health costs are higher than activity costs, because individuals do not fully take into account how their own behavior increases the risk of infection for others. As a result, they are excessively active and cause increased health costs for others.

Following this peak, activity levels gradually return to the optimal pre-pandemic level ($a^*(t) = 1$) as the number of infections declines due to the depletion of susceptibles. This behavioral pattern can be interpreted as individuals dynamically adjusting their activity to keep the effective reproduction number just below the critical threshold of $R_t = 1$, thereby preventing a resurgence of cases. As infections fall, activity rises; as infections rise, activity falls, thereby creating a feedback loop that stabilizes R_t near one. This mechanism results in a prolonged epidemic in which the disease neither explodes nor fades quickly. Note that these results are very similar to those presented in Farboodi et al. [1] and reflect the same trajectory dynamics.



(a) Infected individuals over time.

(b) Activity level over time.

Figure 2: Epidemic trajectories under deterministic and stochastic scenarios. **(a)** Infected individuals; **(b)** Activity level The black dotted line shows the deterministic trajectory. The dark blue line indicates the stochastic mean, the light blue area, the 95% credible interval (2.5th-97.5th percentiles) and the red interval the 95% confidence interval. Based on 500 stochastic simulations.

Including stochasticity in model output

Regarding stochastic effects, we notice that including these effects leads to substantial variation in activity level and infected people, thereby also impacting the distribution of both types of costs. First, we consider the stochastic evolution of N_i (in light blue), in Figure 2a. The timing, and also the height of the infection peak, differ between simulation runs. The peak infection level in some simulations deviates by $\pm 21\%$ from the deterministic case. For $a(t)$, the stochastic band around the deterministic scenario indicates a deviation of $\pm 6\%$ across simulations at this time, and this increases later on. A visual representation is shown in Figure 2b.

(a) Health cost distribution.

(b) Activity cost distribution.

Figure 3: Cost distributions across 500 stochastic simulations. **(a)** Health costs; **(b)** Social activity costs. Box plots show median (black line), mean (black star), 95% credible interval (blue dotted), 95% confidence interval (green line), and deterministic value (red square).

Another important conclusion can be drawn from these visualizations. In the deterministic model, the number of infected individuals $N_i(t)$ gradually decreases but never fully reaches zero, implying a prolonged tail of low-level (but non-zero) transmission. On the contrary, many stochastic simulations show epidemic extinction: once infections drop to sufficiently low levels, random chance often leads to a complete eradication of the disease. When this occurs, agents in those simulations immediately display full activity, setting $a(t) = 1$, since there is no longer any infection risk. This mechanism results in a (significant) divergence between the stochastic mean and the deterministic value at the end of the epidemic, as shown by the solid blue line and red interval.

This asymmetry in epidemic outcomes also creates an important difference in the cost distributions. In Figure 3a, the mean health costs of the stochastic simulations are 2.70% lower than in the deterministic case, which constitutes a statistically significant difference at the 95% level. The reason is straightforward: epidemics that end prematurely due to stochastic fade-out result in fewer cumulative infections, and therefore reduced health-related costs. Deterministic models on the other hand, assume an uninterrupted and predictable decline, which overstates the expected final epidemic size in a finite population and thereby health costs. Finally, the blue dotted line shows that health costs can vary by up to 34%, reflecting substantial variation across simulations.

Figure 3b reveals a similar pattern in activity costs. Here too, the stochastic mean falls 0.93% below the deterministic trajectory, which again consitutes a significant difference. Once the disease fades out in the stochastic setting, agents quickly revert to baseline behavior, hereby minimizing the activity cost. This contrasts with the deterministic model, in which even a small persistent infection level continues to suppress activity over an extended period. The blue dotted line indicates that activity costs can diverge by up to 4%.

Sensitivity analysis

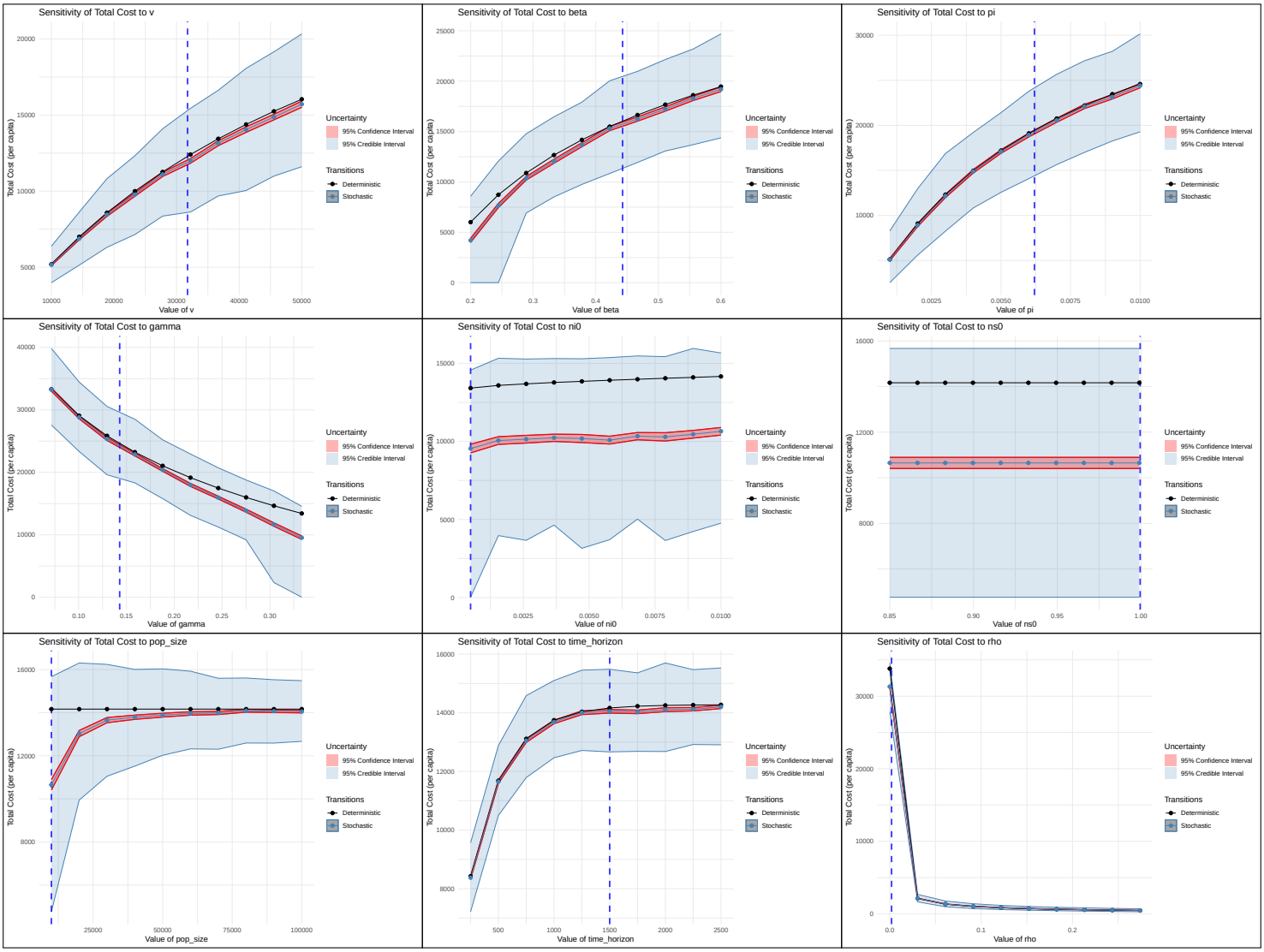

Sensitivity of Total Cost to v
Total Cost (per capita)
Value of v
Sensitivity of Total Cost to beta
Value of beta
Sensitivity of Total Cost to pi
Value of pi
Sensitivity of Total Cost to gamma
Value of gamma
Sensitivity of Total Cost to ni0
Value of ni0
Sensitivity of Total Cost to ns0
Value of ns0
Sensitivity of Total Cost to pop_size
Value of pop_size
Sensitivity of Total Cost to time_horizon
Value of time_horizon
Sensitivity of Total Cost to rho
Value of rho
Uncertainty
95% Confidence Interval
95% Credible Interval
Transitions
Deterministic
Stochastic

Figure 4: **Sensitivity analysis of total per capita cost with respect to key epidemiological and economic parameters.** Each subplot shows the relationship between the parameter (horizontal axis) and the total cost (vertical axis) under both deterministic (black) and stochastic (blue) SIRD simulations. The solid lines denote the deterministic and mean stochastic outcomes; shaded areas represent the 95% credible interval (blue) for the stochastic simulations and the 95% confidence interval (red) for the stochastic mean. The blue dashed vertical line indicates the baseline value used in the main simulation. Parameters explored include: (top row, left to right) recovery rate γ, transmission rate β, fatality rate π; (middle row) cost of infection κ, arrival rate of a cure δ, time preference rate ρ; (bottom row) population size N, initial infection level N_{i0}, and infection fadeout threshold.

Figure 4 presents a comprehensive sensitivity analysis of total per capita epidemic cost with respect to key model parameters, under both deterministic and stochastic transition assumptions. Each panel (a)–(i) corresponds to variation in a single parameter, holding all others constant. We highlight not just the divergence in expected costs, but also the mechanisms that drive these differences.

Time horizon.. As the simulation time horizon increases, both deterministic and stochastic models accumulate costs, so longer runs inevitably result in higher total costs. At the start of the epidemic, and until day 800, there is no significant difference between the stochastic mean and the deterministic scenario. This is because there is no fade-out yet and thus there is no mechanism that causes asymetric outcomes in the stochastic case. However, after this point, as epidemic fade-out starts to occur in the stochastic case, the difference between the deterministic and stochastic mean outcome does become significant. From day 1500, this difference remains the same for larger time horizons as the costs from this point on become negligible in both cases. Regarding the credible intervals, stochasticity increases variation in costs through time as this uncertainty becomes larger over large time horizons.

Value-of-life parameter v.. Increasing the value placed on each life (v) increases the cost associated with each death. Both deterministic and stochastic models show higher total costs as v rises, however there is no significant difference between the stochastic mean and the deterministic case. As (v) increases, variation in total costs logically increases as well.

Transmission rate β.. The transmission rate β determines how rapidly the disease spreads, shaping both the scale and duration of the epidemic. Higher β values result in more intense outbreaks, raising total costs in both deterministic and stochastic models. At lower β, the spread is more gradual and infections do not peak as high, and epidemic fade-out at the end of the epidemic becomes more likely. Consequently, the deterministic model tends to overestimate the total cost more at low β.

Fatality probability π.. The infection fatality probability π governs the fraction of infectious individuals who die rather than recover. It does not affect the spread of the disease; transmission dynamics remain unchanged as π increases, but it substantially alters the cost composition, as deaths carry higher economic cost. This is reflected in the sharp rise in total cost as π increases. The deterministic and stochastic curves largely coincide, indicating that the epidemic trajectory remains similar across π values. However, the credible interval widens with π, showing that uncertainty in cost outcomes grows as fatality becomes more consequential. This increased dispersion is driven by the greater variability in death tolls and thus economic impact.

Recovery rate γ.. Increasing γ reduces the infectious period, thereby lowering the basic reproduction number $R_0 = \beta/\gamma$. When γ is small, corresponding to longer infectious durations and a high R_0, the epidemic spreads

more rapidly, and the stochastic mean closely tracks the deterministic outcome. However, as γ increases, the force of infection weakens, and stochastic effects dominate: infections become smaller and more variable, and epidemic fade-out can occur at nearly any stage of the outbreak. This explains the striking pattern observed in the plot: the total cost declines with higher γ in both models, but the stochastic mean cost falls more steeply, diverging from the deterministic trajectory. Consequently, the deterministic case overestimates total costs.

Initial infected proportion ni_0.. A greater Ni0 leads to higher costs, however it does not lead to a significant difference between the deterministic and stochastic costs trajectory. Deterministic costs rise monotonically with ni_0, while the stochastic mean exhibits more irregularities and wider uncertainty bands, particularly when ni_0 is small.

Population size N.. Smaller populations exhibit greater stochastic variability because they have fewer total infections, making epidemic fade-out more likely and leading to lower average costs across simulation runs. As population size increases, the impact of fade-out events are reduced, causing the stochastic mean to converge toward the deterministic cost. As the population size increases, the credible interval also decreases.

Time Preference Rate ρ.. The time preference rate ρ influences cost through its role in discounting future outcomes in the social welfare function. A lower value of ρ places greater weight on long-term costs. As shown in the figure, total costs fall sharply with even small increases in ρ, reflecting the reduced importance of delayed costs under higher discounting. At lower values of ρ, there is a visible divergence between deterministic and stochastic costs. This is because fade-out effects at the end of the epidemic are given more weight. However, as ρ increases further, these differences rapidly diminish: future periods are increasingly discounted, making fade-out events less consequential in present-value terms.

Summary.. Among the parameters explored, the most substantial differences between deterministic and stochastic outcomes appear for the transmission rate β, recovery rate γ, and population size N. Each of these directly influences transmission dynamics and, as a result, affects the likelihood and timing of epidemic fade-out at the end of the outbreak. Because the deterministic model cannot capture this phenomenon, it tends to overestimate costs in settings where fade-out is frequent. This leads to notable divergence between the deterministic trajectory and the stochastic mean. For other parameters, such as the fatality probability π, value-of-life v, and initial infection share ni_0, the deterministic and stochastic results remain closer. These findings highlight how changes in epidemic dynamics themselves shape the divergence in economic outcomes across modeling approaches.

7. Discussion and conclusion

This paper highlights the importance of incorporating stochasticity into epi-econ models. Our results show that, relative to the deterministic benchmark, the inclusion of stochastic effects leads to a downward estimation of both health-related and social activity costs. This impact is even greater in the case of a higher γ, lower β, or lower ρ value. This underestimation is mainly driven by epidemic fade-out at the end of simulation paths. As the number of infected individuals decreases, random variation increasingly leads to early elimination of the virus without resurgence. Consequently, the average and median costs across stochastic

realizations lie below those estimated by the deterministic model. This effect is most pronounced in relatively small populations, where stochastic fade-out is more likely, and becomes negligible in larger populations.

Our findings complement and extend the emerging literature on stochastic epi-econ models. Previous studies have examined how stochasticicity in transmission parameters affects optimal policy choices. Unlike these earlier contributions, our model introduces randomness through binomial transition processes, which preserve the discrete nature of infection events and allow for epidemic extinction. By doing so, we quantify how stochastic fade-out systematically lowers both health and activity costs relative to deterministic models. Thus, our work fills a gap in the stochastic epi-econ literature by comparing welfare evaluation in deterministic and stochastic models and showing that policies based on deterministic projections may be overly pessimistic.

Deterministic models, by construction, underestimate the possibility of epidemic fade-out at the end of an epidemic and thus tend to overestimate the expected burden of disease and associated mitigation costs. This can lead to overly cautious policies, such as prolonged restrictions or excessive resource allocation with the purpose of eradication, which may not be justified when stochastic dynamics are taken into account. In particular, it may be more effective to implement policies that reduce infections to a low level, rather than to zero, since this can allow stochastic fade-out to occur naturally, avoiding the cost of stricter measures aimed at full eradication. This focus on policy implementation is deemed as a possible extension of this paper.

Stochastic models also uncover the full distribution of possible outcomes. This broader perspective is essential for effective policymaking. Rather than relying solely on expected outcomes, decision-makers must consider the range and likelihood of different scenarios. Depending on societal values, a policymaker may wish to give greater weight to worst-case outcomes to guard against excessive health system strain, or prioritize best-case scenarios under tight resource constraints [44, 45]. By evaluating policies across the entire stochastic distribution, rather than through a single deterministic lens, policy design should become more flexible and realistic.

Overall, our findings highlight the need for caution in deterministic cost projections. Epi-econ models should account for randomness in transmission and disease extinction when evaluating policy interventions.

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Appendix A.. Additional derivations

Myopic, individual welfare function

We consider an individual who myopically chooses their social activity $a(t)$, taking as given the population activity level $A(t)$ and the true infection prevalence $n_i(t)$.

The individual's instantaneous welfare is given by:

$$W(a(t)) = (\tilde{n}_s(t) + \tilde{n}_i(t))\, u[a(t)] + \tilde{n}_r(t) u[a_r(t)] - \beta\, a(t)\, A(t)\, n_i(t)\, \tilde{n}_s(t)\, \kappa$$

We assume the utility function is:

$$u[a(t)] = \log(a(t)) - a(t) + 1$$

Since $\tilde{n}_r(t) u[a_r(t)] = 0$, it drops out of the optimization problem. Taking the derivative of $W[a(t)]$ with respect to $a(t)$, we obtain the first-order condition:

$$\frac{dW[a(t)]}{da(t)} = (\tilde{n}_s(t) + \tilde{n}_i(t)) \left(\frac{1}{a(t)} - 1 \right) - \beta\, A(t)\, n_i(t)\, \tilde{n}_s(t)\, \kappa = 0$$

Equilibrium requires that individual and aggregate behaviors are consistent at every point in time:

$$a(t) = A(t), \quad \tilde{n}_s(t) = n_s(t), \quad \tilde{n}_i(t) = n_i(t)$$

Substituting these into the first-order condition:

$$(n_s(t) + n_i(t)) \left(\frac{1}{A(t)} - 1 \right) = \beta\, A(t)\, n_s(t)\, n_i(t)\, \kappa$$

Multiplying both sides by $A(t)$:

$$(n_s(t) + n_i(t))(1 - A(t)) = \beta\, \kappa\, n_s(t)\, n_i(t)\, A(t)^2$$

Rewriting:

$$\beta\, \kappa\, n_s(t)\, n_i(t)\, A(t)^2 + (n_s(t) + n_i(t)) A(t) - (n_s(t) + n_i(t)) = 0$$

Solving the quadratic equation gives the optimal activity:

$$A^*(t) = \frac{-(n_s(t) + n_i(t)) + \sqrt{(n_s(t) + n_i(t))^2 + 4\beta\, \kappa\, n_s(t)\, n_i(t)\, (n_s(t) + n_i(t))}}{2\beta\, \kappa\, n_s(t)\, n_i(t)}$$

Transition rates and probabilities

1. Transition Rates

The transition rate (Susceptible $\to$ Infected) is given by:

$$\text{Transition rate (Susceptible} \to \text{Infected)} = \beta A(t)^2 N_s(t) n_i(t)$$

The transition rate (Infected $\to$ Recovered) is given by:

$$\text{Transition rate (Infected} \to \text{Recovered)} = (1 - \pi)\gamma N_i(t)$$

The transition rate (Infected → Deceased) is given by:

$$\text{Transition rate (Infected} \rightarrow \text{Deceased)} = \pi\gamma N_i(t)$$

2. Transition Probabilities

Transition probabilities are derived based on the assumption that the time until each event (infection, recovery, or death) follows an exponential distribution with a corresponding rate. If an event occurs at a constant rate r, then the probability that it does not occur within a time interval of length Δt is given by the exponential distribution's survival function:

$$\exp(-r\Delta t)$$

Consequently, the probability that the event does occur within that interval is:

$$p = 1 - \exp(-r\Delta t)$$

Assuming a time step of one unit (i.e., $\Delta t = 1$), this simplifies to:

$$p = 1 - \exp(-r)$$

Plugging the previously described rates into this equation, gives the transition probabilities.

The infection probability per susceptible individual is given by:

$$p_i = 1 - \exp(-\beta A(t)^2 n_i(t))$$

The recovery probability per infected individual is given by:

$$p_r = 1 - \exp(-(1-\pi)\gamma)$$

The death probability per infected individual is given by:

$$p_d = 1 - \exp(-\pi\gamma)$$

3. Stochastic Transitions

We model the transitions following binomial processes. The binomial distribution describes the number of successful outcomes in a fixed number of independent trials, where each trial has the same probability of success. In our context, each trial represents an individual (e.g., a susceptible or infected person), and a 'success' corresponds to that individual undergoing a transition (e.g., becoming infected, recovering, or dying) during that specific day. The probability of success, denoted by p_i, p_r, and p_d, reflects the respective transition probabilities previously derived.

The number of new infections is then given as follows:

$$I_{\text{new},t+1} \sim \text{Binomial}(N_s(t), p_i)$$

The number of new recoveries is given by:

$$R_{\text{new},t+1} \sim \text{Binomial}(N_i(t), p_r)$$

The number of new deaths is given by:

$$D_{\text{new},t+1} \sim \text{Binomial}(N_i(t), p_d)$$

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